A word from the editor

The book series “DISEASES OF MARINE ANIMALS” (organized and edited by Otto Kinne and contributed to by numerous outstanding experts over years) is now freely available with online Open Access.

Published in the years 1980 to 1990, the copyright for “DISEASES OF MARINE ANIMALS” was graciously transferred by the original publishers, to the editor Otto Kinne.

The technical problems involved in the re-publication of the treatise were mastered by Konstantin Kambach (Inter-Research). Unavoidably, the print quality of the final product is somewhat inferior to the original.

Otto Kinne
Oldendorf/Luhe
21.01.2009

DISEASES OF MARINE ANIMALS

Volume I INTRODUCTION, GENERAL ASPECTS, PROTOZOA, MESOZOA, PORIFERA, CNIDARIA, CTENOPHORA, TENTACULATA, SIPUNCULIDA, PRIAPULIDA, ECHIURIDA, PLATYHELMINTHES, NEMERTEA, GASTROPODA

Volume II INTRODUCTION, BIVALVIA, AMPHINEURA, SCAPHOPODA

Volume III INTRODUCTION, CEPHALOPODA, ANNELIDA, CRUSTACEA, CHAETOGNATHA, ECHINODERMATA, UROCHORDATA

Volume IV

Part 1: INTRODUCTION, PISCES

Part 2: INTRODUCTION, REPTILIA, AVES, MAMMALIA: CARNIVORA, PINNIPEDIA, SIRENIA, CETACEA

DISEASES OF MARINE ANIMALS

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VOLUME III

Introduction, Cephalopoda, Annelida, Crustacea,
Chaetognatha, Echinodermata, Urochordata

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FOREWORD

Volume III of 'Diseases of Marine Animals' comes out late, 5 years after the appearance of Volume IV. As editor, I owe the readers a word of explanation and apology.

Comprehensive, integrated multi-author treatises, such as 'Diseases of Marine Animals', demand from all people involved a high degree of cooperation, dedication and reliability. The authors must accomplish their work in addition to other commitments, and the amount of time and effort required is often much larger than originally anticipated. My insistence on a rigid conceptual framework for the treatise certainly did not make their task any easier.

For the editor — once he has worked out his scientific concept and selected his crew — matters of steering cooperation and subject integration, motivating authors, and adhering to mutually agreed schedules become of prime concern. The means for making tardy authors to deliver are limited to the art of gentle, or not-so-gentle, prodding and, in the last resort, appeals to the authors' sense of fairness to other contributors who completed their work on schedule.

Sometimes all this fails. New authors must be found and the good boys in the crew be persuaded to wait and to update their chapters until all contributions are completed. No fun, either for the good boys or for the editor, that is what happened to Volume III of 'Diseases of Marine Animals'.

Well, all obstacles and problems now lie behind us. I thank the contributors to Volume III most cordially for their patience, diligence and hard work. Our combined efforts have produced the first integrated multi-author overview of what is known on disease phenomena in cephalopods, annelids, crustaceans, chaetognaths, echinoderms and urochordates, and — of equal importance — of what still has to be done in order to obtain a more complete picture. Volume III does not cover all of the animal groups that we would have liked to include. Intensive searches revealed that for several groups there simply was not enough information available to warrant reviews at the present time.

I would have liked to have had more time for editing and for streamlining, but for a few late submissions time pressure made that impossible.

Dr. Ellen Wahl (Biologische Anstalt Helgoland, Hamburg, Germany) assisted with proof reading and checked the Literature Cited sections. Frank Wohlgemuth (Hamburg) assembled the Taxonomic Index; Georg Petrausch (Hamburg), the Author Index. Helga Witt (Ecology Institute, Oldendorf/Luhe, Germany) helped in many ways throughout the years of organizing and completing this tome. I am most grateful for this support.

Oldendorf/Luhe, November 11, 1989 O. KINNE

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INTRODUCTION, CEPHALOPODA, ANNELIDA, CRUSTACEA, CHAETOGNATHA, ECHINODERMATA, UROCHORDATA

INTRODUCTION TO VOLUME III

O. KINNE

As in previous volumes of the treatise 'Diseases of Marine Animals', this introductory section provides a brief overview of the information presented for the benefit of the hurried reader and presents conclusions drawn by the editor. I have tried to keep the overview as short as possible and to concentrate on information immediately pertinent to disease phenomena.

Again, it is surprising how little definitive information is available on disease phenomena *sensu stricto*. The actual effects exerted by agents on their hosts, the hosts' responses to agent impact, ensuing disease phenomena, etiologies — and, where applicable, diagnoses and therapies — have been the focal points of attention only in a few cases, and then were largely restricted to host species of economic importance.

Headings such as 'Diseases of . . .' followed by a text statement saying 'No diseases are known in this group of animals' can be justified only by the aims and structural design adopted for 'Diseases of Marine Animals'. Our major aims were to: (i) review comprehensively and in detail all facts and interpretations thus far published in the scientific literature on disease phenomena in marine animals; (ii) consider all the circumstances that may contribute to, or cause, disease; and (iii) point out areas for which no or insufficient knowledge is available. While the latter requirement may be unusual in state-of-the-art overviews — which typically trace the routes along which sufficient information happened to have accumulated — I believe there is need to identify major gaps in the continuum of knowledge. Only by filling such gaps can we hope to achieve broad and reasonably balanced progress in science and thus to provide a solid foundation for future research. The structure of Volume III parallels that of previous volumes: This tome too has been organized around the 2 major entities involved, hosts and agents. For details consult Vol. I, pp. 1 to 2 and pp. 13 to 64.

SUMMARIES OF CHAPTER CONTENTS

Comments on Diseases of Mollusca: Cephalopoda

Our present knowledge on the diseases of cephalopod molluscs is limited, largely restricted to description, and characterized by an almost complete lack of information on etiologies, not to speak of potential measures of therapy under controlled conditions. Internal mechanisms of host defense and the means and ways of agent attack await thorough investigation. Nevertheless, the relatively few investigators who have devoted their research activities to the study of cephalopod diseases have brought to light many important facts and details.

An immunoglobulin-based defense against invading foreign particles is absent, as in other invertebrates. Thus far, virus-like particles have been detected only in 2 cases (pp. 23–25), and no definitive information on bacteria-caused diseases is available from

natural cephalopod populations. Of course, the absence of such information cannot be interpreted as a criterion for the absence of virus- or bacteria-caused diseases.

Records of bacterial species sampled from healthy and diseased squid have been summarized in Tables 1-1 and 1-2 (pp. 27, 28). Members of the genus *Vibrio* play an important role as disease-causing agents in cultured cephalopods. Under laboratory conditions, especially the delicate skin (particularly after injury) and the eyes may be sites of (secondary) bacterial invasion and subsequent disease phenomena, which in some cases proved lethal unless treated, e.g., with nifurpirinol or tetracycline. An apparent systemic infection was caused by *Vibrio carchariae* and led to sudden death in *Octopus bimaculoides*.

Under crowded conditions, laboratory-held *Octopus joubini* and *O. briareus* developed severe skin ulcerations due to secondary bacterial invasions of skin abrasions which resulted from octopus-octopus interactions. Cultures from these ulcerations revealed only Gram-negative bacteria: *Vibrio alginolyticus, V. damsela, Pseudomonas stutzeri* and *Aeromonas cavia* grew in cultures obtained from ulcers of *O. joubini; V. parahaemolyticus, V. damsela* and *P. stutzeri*, in cultures from ulcerated *O. briareus* skin. In reinfection experiments, only *V. alginolyticus* produced disease-like lesions. A highly virulent and lethal systemic bacterial infection in *Sepia officinalis*, apparently without external injury, was successfully treated with chloramphenicol and gentamycin.

The symbiotic interrelations between cephalopods and bacteria inhabiting light organs and nidamental glands require detailed attention. We may expect findings here that can shed new light on bacterial-caused cephalopod diseases.

Only 1 fungus *(Cladosporium sphaerospermum)* has thus far been clearly identified on the skin wounds of an octopus *(Eledone cirrhosa)*. Several pathologies of unknown etiology that may involve fungi remain to be analyzed.

Protistan and metazoan symbiotes abound on and in cephalopods, and the number of records has grown impressively over the last few decades (Table 1-5). To date about 150 species have been examined, of a total of 650 cephalopod species now recognized by science; the list of protistan and metazoan symbiotes of cephalopods may increase significantly in the decades to come. Mature individuals of large-sized host species harbor symbiotes almost without exception. Taxonomically, the symbiotes exhibit much diversity, with few exceptions paralleling that known from the fishes (Vol. IV). The symbiotes inhabit almost all host tissues and organs; however, skin, gills, digestive tract, excretory organs and musculature are preferred. Apparently, no definitive disease phenomena have thus far been attributed unequivocally to metazoan parasites.

In the transmission of parasitic agents, the Cephalopoda appear to play a role similar to that of the bony fishes (Vol. IV). They are primary hosts for protozoans, dicyemids and crustaceans, and reservoir, second or third intermediate hosts for larvae of digeneans, cestodes, acanthocephalans and nematodes.

Among the Protozoa, flagellates are rare as cephalopod symbiotes, but when present in large numbers they may cause severe damage, e.g., to skin and gills. Apicomplexa are represented only by species of *Aggregata* which infest the non-cuticularized portions of the digestive tract and appear to be very host specific (p. 61). Two Microspora species parasitize cephalopods. Of these, *Steinhausia spraguei* infects the renal appendages, completing its life cycle within a single host cell. Members of at least 5 families of ciliates abound in renal and digestive organs of cuttlefishes, squids and octopuses, and undeter-

mined, large-sized Protista *Incertae sedis* inhabit the gills of numerous pelagic cephalopod species.

Among the Metazoa, the dicyemids — a small, exclusively parasitic phylum — infest the excretory organs of benthic and epibenthic cephalopods, but do not seem to harm their hosts. The mode of host entry is not known. The life cycle of dicyemid parasites appears to progress as a function of host age and maturity. Chromodinid ciliates also inhabit the excretory organs. However, they attack preferably oceanic cephalopods which never contact the sea floor, while dicyemids coexist with hosts associating with the bottom. Such spatial separation renders concurrent infestations a rarity.

Monogenean Platyhelminthes have been found in mantle cavities and large blood vessels, as well as on gills and arms of cephalopods. Host invasion is facilitated by direct contact of adults (e.g., mating). The agents may prove to be common upon more extensive examination of cephalopods, especially loliginids. For larval or adult digenean Platyhelminthes, cephalopods serve as second intermediate, paratenic or final hosts (never as first intermediate hosts). Most abundant are larval didymozoids — acquired when the hosts consume invertebrates and fishes, and apparently causing little or no harm. For completing their life cycle, didymozoids appear to require 3 to 4 hosts. Most trematodes inhabit cephalopods as single individuals (hemiurids, accacoelids, hirudinellids); hence cephalopods seem to serve as paratenic hosts and are liable to infestation only when they consume intermediate hosts which are normally preyed upon by teleost fishes (the final hosts). While adult Cestoda have — with one possible exception — never been found in cephalopods, their larvae are common. Transferred from host to host through the food chain, the larvae may serve as indicators of trophic interrelations in marine ecosystems. Especially abundant are larvae of Tetraphyllidea and Trypanorhynchidea, presumably acquired by the cephalopods via feeding on crustaceans and fishes; the commonest larvae are those of the genus *Phyllobothrium*. Host-agent relations have been investigated only in the commercially important ommastrephid squids, and critical assessments of disease manifestations are not available.

An entirely parasitic phylum, the Acanthocephala mainly inhabit vertebrates; however, adults of 2 or 3 species have been regularly found in cephalopods, e.g., *Neorhadinorhynchus atlanticus* which lives in the stomach lumen (p. 164). No details on host-agent relations are available.

While larval Nematoda abound in cuttlefishes, squids and octopuses, the information at hand is almost exclusively restricted to recordings of presence or absence. Again, numerous unsolved taxonomic problems blur the picture. Larvae of *Contracaecum* species are common in muscles of *Todarodes pacificus*, and have been found in stomach and mantle of other cephalopods. Also common are *Anisakis* (life cycle on p. 182) larvae in secretory portions of visceral organs, the lining of the mantle cavity, and in mantle musculature; the larvae have also been found encysted in the ventricle of *T. sagittatus*. *A. simplex* exhibits pronounced differences in prevalence and intensity of parasitation. Where fishes and squids are consumed raw by humans (e.g., Japan, Korea, USA, Britain, Scandinavia), anisakiasis may result (ulcers or lesions, particularly in the stomach).

Among the Annelida, members of 3 hirudinean species have been reported from cephalopods *(Octopus dofleini)*. This relation appears to be temporary, however, and not to involve demonstrable harm to the host. Polychaetes are rare as cephalopod symbiotes; several species have been reported from egg masses of squids.

Crustacea do not seem to cause serious disease in cephalopods. They inhabit primarily the mantle cavity and gill surfaces, but may also move out over the skin of the head, mantle and anus. Thus far, members of 17 species of copepods, 1 brachyuran, 3 isopods and 1 amphipod have been reported. Details of host-agent relations remain to be studied.

Structural abnormalities and neoplasia have received anecdotal attention at best. Most reports on structural abnormalities stem from taxonomists studying preserved specimens. They have listed the abnormalities with an eye on keys useful for species descriptions, i.e., they have searched for reliable characteristics allowing them to describe and determine the different species present and to provide a solid platform for systematics. Attempts to link structural abnormalities to potential causes, to possible dysfunctions, or to disease phenomena are lacking.

There is no definitive information on neoplasia and tumors in cephalopods, although a few cases of 'tumors' have been reported. Squids, octopuses and other marine animals consumed as sea food by humans tend to contain high levels of amines that may play a role in the etiology of human stomach cancers and other gastrointestinal tumors.

Comments on Diseases of Annelida

Members of all 3 classes of Annelida — Oligochaeta, Polychaeta, Hirudinea — harbor parasites or commensals. Many more may be shown to entertain symbiotes, and hence be potential subjects of biotic diseases, as our knowledge grows. Most of the present documentations of parasitism focus on attempts to unriddle taxonomic identities of the symbiotes involved, and on the role of annelids as intermediate hosts for agents attacking animals of economic importance (especially molluscs, crustaceans and fishes). Little detailed information is available on agent virulence, agent effects, host defense and etiology, and few actual diseases have been identified.

There are a few reports on viral and bacterial infections, but their pathological consequences remain largely unknown. *Bacillus arenicolae* produces swelling and cell damage in the intestinal epithelium of *Arenicola ecaudata* and subsequently may cause death. Among the non-marine earthworms, *Lumbricus terrestris* is susceptible to fatal infection by *B. arenicolae,* and *Eisenia foetida* may suffer mortalities due to *B. thuringiensis.*

Flagellata, Gregarinida, Microspora, Myxospora and Ciliophora all include members that may infest annelids, and thus could potentially act as disease agents. However, unequivocal documentation of disease phenomena has not yet been published. The state of our knowledge is not much better with regard to metazoan agents.

Comments on Diseases of Crustacea

Diseases Caused by Microorganisms

We know more about the diseases of crustaceans than of any other host group treated in this volume. Especially the crustacean diseases caused by microorganisms have attracted considerable attention. Several crustaceans are a major, high-priced fishery commodity; some species are suitable for commercial cultivation (aquaculture). Economic interests, particularly for the cultured species, have kindled private and governmental support and thus catalyzed and intensified scientific inquiry. Nevertheless, much additional terrain will

have to be covered before we possess a body of knowledge comparable to that already available for insects.

Among aquacultured Crustacea, infectious diseases have repeatedly caused heavy losses. In many cases multiple infections due to combined attacks of viruses, bacteria, fungi and a variety of protozoal agents have rendered disease diagnosis and therapeutic measures problematic. As is true for other host groups in confinement rearing, crowding, decline in water quality, handling and inadequate nutrition tend to modify agent/host interrelations as they exist in nature, and often favor the agent's capacity for establishment while reducing the host's capacity for defense. Both in culture and in nature, pollutants may predispose hosts to agent attack and virulence, but unequivocal evidence for such pollution effect on population structure in wild populations has still to be presented.

Most infectious diseases reviewed in Chapter 3 with demonstrated morbidity and mortality effects have been described from cultured hosts. According to the documentation currently available for marine Crustacea, most of the microbial agents occur as subclinical infections in nature. The notable exception to this generalization is black mat disease of tanner crabs. Catastrophic consequences of diseases in nature seem to be rare, at least as far as can be judged from numerical population data. Possible qualitative effects mediated via selection (changes in genetic material) have not yet been considered, but deserve attention.

Since free-living marine crustaceans harbor a large variety of pathogens, their artificial translocation (e.g., clearly documented in aquaculture and suspected via ship movements) often involves concomitant pathogen transfer. In the new environments these agents may establish new host interrelations with unpredictable outcomes. In aquaculture facilities these pathogen transfers have resulted in serious losses to production.

Among the microbial agents known to affect marine crustaceans, the **viruses** are the most numerous and diverse group. More than 30 viruses have been identified — mostly in crabs and shrimps. They have been classified, almost exclusively, only tentatively. These agents resemble described viruses in the following families: Parvoviridae, Herpesviridae, Baculoviridae, Picornaviridae, Reoviridae, Birnaviridae, Rhabdoviridae and Bunyaviridae. Success or failure of shrimp farming depends in part on the knowledge and management of viral diseases. Prevention and control of viroses is an important aspect for world-wide progress in the farming of marine shrimp and possibly some species of marine crabs. A number of viruses reported from Crustacea apparently do not cause disease. The ecological role that viruses play in natural crustacean populations remains uncertain.

From the detailed review of viral diseases presented in Chapter 3, I have selected the following examples.

Baculovirus penaei (BP) commonly invades the hepatopancreas of *Penaeus vannamei, P. aztecus* as well as that of other penaeids. BP is widely distributed in the Americas within a host range limited to penaeid shrimp. Larval and early post larval stages are susceptible to BP epidemics characterized by high mortality in commercial shrimp hatcheries. Older life-stages are susceptible to infection which commonly remains subclinical. Disease manifestations, when they occur in these stages, include reduced rates of feeding and growth as well as increased fouling of body surfaces and gills due to epibionts. *B. penaei,* as with the other identified baculoviruses from marine shrimp, infect hepatopancreas and mid-gut epithelium. Patent BP infection is characterized microscopically by large pyramidal-shaped, intranuclear occlusion bodies that are easily identified in wet-mount impression smears of the hepatopan-

creas and fluoresce under ultraviolet light after staining; this permits quick diagnosis. Control or prevention of BP disease requires: use of virus-free nauplii obtainable from specific-pathogen-free broodstock or by disruption of transmission between infected female shrimp and offspring at the time of spawning/hatching; careful hygiene and disinfection of equipment and containers to minimize horizontal transmission between hatchery tank larval populations; and use of source water free of significant levels of chlorinated hydrocarbon and/or heavy metal contaminants. Chemical treatment and vaccination have not been seriously tested as modalities for control of BP disease in shrimp hatcheries.

Monodon baculovirus (MBV) is common in cultured *Penaeus monodon,* but studies on distribution of this virus in wild shrimp populations are lacking. However, based on data from cultured shrimp, the virus infects a wide range of penaeids over an extensive geographic distribution including Indo-Pacific coasts of Asia, Australia, Africa, Mediterranean coasts of southern Europe, Kuwait and Israel. Similar to BP the severity and signs of MBV disease vary with host age and species, with younger life-stages being prone to clinical manifestations. The intranuclear, subspherical MBV occlusion bodies are an important diagnostic characteristic for the disease. These are easily identified in histological preparations of hepatopancreas tissue from infected shrimp. Prevention and therapy of MBV disease requires critical attention. Disease control is restricted to agent exclusion or culture under low stress conditions.

Baculovirus mid-gut gland necrosis virus (BMNV) initially caused heavy annual losses in hatchery-cultured larval *Penaeus japonicus* in southern Japan. BMNV infections have been reported from natural shrimp populations. As far as is known the host range is limited to *P. japonicus*. Similar to BP and MBV, BMNV is a hatchery disease, and infected mysis and post larvae may suffer up to 89 % mortality. Horizontal virus transmission was successful by exposing larvae and postlarvae to the virus, and by feeding infected hepatopancreas tissues to postlarvae. Older life-stages are susceptible to BMNV infection but manifestations remain subclinical. Diseased larvae exhibit a white, turbid hepatopancreas and may float inactively near the water surface. At the cellular level, agent effects include hepatopancreas collapse, hypertrophy of infected cells, chromatin margination, diminished nuclear chromatin, nucleolar dissociation and karyorrhexis. Intranuclear occlusion bodies are not formed. Diagnostic tools include: fluorescent antibody staining, squash unstained preparations exposed to dark-field illumination, and phase-contrast microscopy. Control for BMNV hatchery disease has been routinely achieved by blocking virus transmission between female broodstock and offspring at the time of spawning/hatching.

Natural and experimental Tau infections are known for *Carcinus mediterraneus*. Virus transmission in the laboratory (tissue extract injection or feeding hepatopancreas pieces) resulted in a cumulative mortality of 100 % in 25 days compared to 20 % in the controls. Diseased crabs exhibit aggression, lethargy and inappetence. All epithelial cell types of the hepatopancreas are attacked. Infected nuclei are markedly hypertrophied and reveal loss of nuclear chromatin, margination, as well as karyolysis. The cytoplasm is highly vacuolated and disorganized. Cellular breakdown is followed by discharge of cell contents into the tubule lumen. Virus particles often occupy peripheral areas of the nucleus. Cytoplasmic changes include numerical reduction of organelles, size increase of mitochondria, as well as the presence of vacuoles and vesicles — many containing virions, and free virions in the cytoplasm. Nothing is known about natural Tau distribution and *in situ* effects of the disease.

Baculo-PP was recovered from blue king crabs collected in Alaskan waters. Virus-infected hepatopancreas cells are moderately hypertrophied, and nucleoli are generally absent from infected nuclei. Infections studied thus far have been subclinical and no diseases due to Baculo-PP have been reported from natural blue king crab populations.

Four rod-shaped nuclear viruses (B_1, B_2, Baculo-B, RV-CM) of uncertain taxonomic affinity have been reported to infect the crabs *Carcinus maenas, C. mediterraneus* or *Callinectes sapidus*. The viruses invade nuclei of hemocytes, as well as hematopoietic and other mesoderm cells. None of the 4 viruses appear to cause disease in their respective hosts.

Three hexagonal nuclear viruses have been reported from *Callinectes sapidus, Rhithropanopeus harrisii,* and *Paralithodes platypus*. The viruses from *C. callinectes* and *P. platypus* are highly pathogenic.

A cytoplasmic virus — Chesapeake Bay virus (CBV) — was recovered from *Callinectes sapidus*. It has been associated with a lethal disease in captive juvenile *C. sapidus*. CBV is a non-enveloped icosahedron replicating in cytoplasm and forming large inclusion bodies entirely made up of virus particles. Diseased crabs exhibit disoriented swimming, erratic movements, and head-down position at rest. CBV preferably invades neurons, retina, gill, epidermis, stomach, hindgut lining, antennal glands and bladder epithelium. Infected cells become hypertrophied and contain cytoplasm filled with a homogeneous, Feulgen-negative material (Fig. 3-9,f, p. 282). CBV disease can be diagnosed on the basis of clinical signs and light microscopical demonstration of hypertrophied cells with dense Feulgen-negative cytoplasm. The ecological significance of CBV disease awaits investigation.

Infectious hypodermal and hematopoietic necrosis (IHHN) is a highly contagious lethal disease of *Penaeus stylirostris*. IHHN virus infects all penaeid species tested thus far and is widely distributed in shrimp culture operations worldwide. Its natural reservoir host(s) and its distributional range in nature are not known. The virus is transmitted horizontally by host exposure to infected water and by *per os* ingestion of infected shrimp tissues. Vertical transmission is highly probable but remains unproven experimentally. Diseased shrimp display erratic, inverted surface swimming, then stop moving and sink with a slowed righting response. Microscopically, multifocal areas of cellular necrosis are visible, as are nuclear hypertrophy, pyknosis, karyorrhexis and intranuclear inclusions (Fig. 3-9,a-c, p. 282). Disease diagnosis is based on histopathological demonstration of the Cowdry Type A inclusions. For disease management consult p. 284.

A number of other viruses have been reported from marine crustaceans (pp. 284 to 291). Among these, putative bunya-like forms were recovered from *Carcinus maenas* (crab hemocytopenic virus, CHV); *Macropipus depurator* and *C. mediterraneus* (S virus); and from the Y-organ tissue of *C. mediterraneus* (unnamed virus). The first 2 viruses may cause disease.

Among the **Rickettsia and Chlamydia**, 4 representatives infect crabs or penaeid shrimps. A definitive classification of the agents involved has not yet been possible. The rickettsia-like forms attack predominantly hepatopancreas cells; the chlamydia-like agents invade cells of meso- and ectodermal origin. The cause of the Dungeness crab chlamydial disease is a highly pathogenic agent closely related to the Chlamydiales. The disease has a potential impact on natural crab populations.

Bacteria constitute an important disease-causing symbiote group in numerous marine crustaceans. Again, almost all of the information available stems from crustacean hosts

maintained under controlled conditions (aquaculture, experimentation). Even here, most agent/host interrelations are poorly understood. In general, stress, crowding, reduced culture-water quality, and insufficient hygiene, as well as circumstances enhancing entry, establishment and transmission of the agent (e.g., handling, injury, molting) are etiologic co-factors necessary for disease development; they hold the key for disease control.

Crustaceans are often subject to epibiotic bacterial infection. The most important filamentous epibiont is *Leucothrix mucor*. A variety of non-filamentous bacteria cause larval mortality in Dungeness crabs as well as fouling disease in shrimp larvae. While low-density epibiont symbioses are normal in natural crustacean populations, heavy settlements may cause disease and death. In cultures, excessive microbial epibiont development can pose serious problems. Epibionts damage the cuticle and may critically interfere with host respiration (gas exchange). Natural defense against heavy epibiont growth includes molting and preening. Disease management is based on culture water monitoring, filtration and chemical treatment (copper-based compounds; antibiotics: chloramphenicol*, penicillin, streptomycin and malachite green** for egg treatment).

Bacterial shell disease (brown or black spot disease) results in ulcerative lesions of the chitinous exoskeleton (Fig. 3-15,a,b, p. 305). It involves Gram-negative chitinoclastic bacteria and/or fungi. Shell disease is, according to some reports, contagious and has been documented from numerous decapods; it is promoted by integument damage, chronic exposure to high levels of heterotrophic bacteria (e.g., due to high temperature and high nutrient loading), as well as pollutants which degrade the cuticle and interfere with molting. Control of shell disease includes avoidance of injury, proper husbandry and culture-system hygiene, system sterilization, disinfection of incoming water, waste removal, selective culling of affected individuals, and proper nutrition. Also important are low temperature, prompt removal of exuviae and curbing of microbial biomass. Variable response is the rule for chemical treatment applied for shell disease.

Gram-negative bacterial septicemic disease (vibriosis) — predominantly caused by members of the Vibrionaceae — is frequently encountered in recently collected crustaceans, traumatized during capture and transfer (agent invasion via cuticular wounds, reduced capacity of internal host defense mechanisms). Apparently, bacterial toxins are involved in the pathogenesis. Disease signs include lethargy and weakness; disorientation; prostration in ventral, dorsal or lateral recumbency; dorsal flexion of the tail or head-down position; continuous slow movement of pleopods and pereiopods; focal-to-diffuse opacity of the striated musculature; expansion of cuticular melanophores on the dorsal surfaces; expansion of cuticular erythrophores of pereiopods and pleopods; irregular-sized white, cloudy areas in the gill lamellae and other body parts; antemortem, acellular jelly-like clots in hemal sinuses; and increased turbidity as well as reduced clotting of withdrawn hemolymph. Death occurs often quite rapidly, i.e., within 2 to 4 h after the onset of clinical signs. Disease prevention includes water-quality management, low bacterial biomass (disinfection, filtration), avoidance of extreme and rapidly changing temperature, balanced diets, and restraint in handling and crowding. Immunoprophylaxis or vaccination may provide some protection. Chemical treatments included application of malachite

* Chloramphenicol is known to be a cause of aplastic anemia in humans. Its use in aquaculture is not recommended.

** Malachite green has recently been listed as a potential cancer-causing substance. Its use in aquaculture is now forbidden in several countries.

green, chloramphenicol, furazolidone, as well as ethylenediaminetetraacetic acid (EDTA). In juvenile-to-adult penaeids oxytetracycline or furacin are administered in the feed.

Rod-shaped bacteria are assumed to cause feminization in male *Leptomithrax longipes*. Spirochete bacteria were found in the cytoplasm of antennal gland cells, hypodermal cells, muscle fibers, hemocytes and in hemocoelic spaces of *Artemia* sp. Nothing is known about transmission, virulence and *in situ* distribution of the spirochete. The unique importance of brine shrimp as live food for numerous cultivated marine animals renders pertinent research urgent and significant.

Caused by the Gram-positive bacterium *Aerococcus viridans* var. *homari*, gaffkemia of lobsters is the most thoroughly investigated microbial disease of marine Crustacea. Gaffkemia can be an epidemic, acute-to-chronic, and is almost invariably lethal in impounded *Homarus americanus* and *H. gammarus*. *A. viridans* var. *homari*, a non-motile, catalase-negative, beta-hemolytic, facultative anaerobe, entertains natural reservoirs in lobsters along the Atlantic coasts of North America and Europe, as well as in other decapods and, possibly, in marine sediments. In the early days it caused high economic losses to lobster holding facilities. The bacterium enters its host through breaks in the cuticle. The degree of virulence is temperature dependent, decreasing dramatically from 20° to 1°C. Both cuticle wounding and environmental temperature are keys to prevention and management of gaffkemia in lobsters. Advanced gaffkemic lobsters are extremely weak. Typical external disease signs include discolored (pink) ventral abdomen, pinkish hemolymph, black spots on gills, irregular lesions in the antennal glands, numerical reduction of hemocytes, and tetrad-forming cocci in the hemolymph. While vaccination may constitute a preventative tool, practical techniques are in the development stage. If applied prior to infection, vancomycin treatment provides protection. Important therapeutic measures included holding at low temperatures (5° to 10°C) and penicillin G injection. Protection from wounding through proper handling and husbandry coupled with use of low temperature holding and antibiotic medication as needed should largely prevent devastating gaffkemia outbreaks in the future.

Mediterranean crab streptococcosis is caused by *Streptococcus faecalis liquefaciens*. The disease can be transferred via inoculation and feeding of hepatopancreas tissue from infected crabs. Diseased *Carcinus maenas* are inappetent and weak.

Red disease of *Penaeus monodon* is assumed to have an abiotic etiology but may also involve a Gram-positive coccus.

A variety of marine **fungi** induce diseases in Crustacea. Several fungi are the cause of epidemics in shrimp hatcheries and farms, and *in situ* epidemics attributed to fungal pathogens have been reported as well. Most of the fungi capable of causing disease in marine crustaceans belong to the Phycomycetes.

Members of the yeast genus *Metschnikowia* infect *Artemia* sp., several marine copepods and limnetic cladocerans. Yeast attach to the integument surface and invade digestive tract and other tissues. Advanced yeast infection of copepods results in weakness and facilitates secondary invasion by bacteria and protozoans.

Mottling disease, attributed to a chytrid-like fungus, has been occasionally diagnosed in lobsters. Disease signs include irregular, large, yellowish patches on the cuticle. Such discoloration results from necrosis of underlying tissues.

Black gill disease, characterized by a black, mottled appearance of gill filaments, has

been reported from *Dichelopandalus leptocerus* and may be an important cause of natural mortality, possibly also in other crustaceans.

The phycomycete *Lagenidium callinectes* parasitizes eggs and/or larval stages of *Callinectes sapidus, Panopeus herbstii, Libinia dubia, Cancer magister, Scylla serrata,* several *Penaeus* species, *Homarus americanus, Pandalus platyceros, Chthamalus fragilis* and *Chelonibia patula. L. callinectes* commands a wide geographic distribution and a high potential of rapidly invading (via zoospores) new hosts. Once host eggs are infected, they invariably die; thus large losses result in culture and, possibly, in nature as well. Heavily infected eggs and larvae are opaque white. Diseased eggs are reduced in size (by ca. 20 %) and larvae increasingly lose their ability to move and orientate. In terminal situations, the host body is almost completely filled by fungus hyphae. While young hosts may suffer up to 90 % mortality, disease susceptibility tends to decrease with host age. Disease management is largely based on prophylactic measures; it includes application of chemicals (trifuralin = treflan, malachite green), strict sanitation, disinfection and ultraviolet treatment (p. 333).

Sirolpidium infection leads to serious mycoses in cultured penaeid shrimp and possibly lobster larval stages. While the agent is widely distributed, its taxonomic status has been insufficiently investigated. Pathogenesis and treatment of *Sirolpidium* sp. infection are similar to those described for *Lagenidium callinectes* (see preceding paragraph).

Leptolegniella marina infects eggs, embryos and body of *Pinnotheres pisum;* it invariably causes death of successfully invaded crabs. Time-to-death ranges from 8 to 57 days at 8° to 17 °C. Infected crabs exhibit irregular, variable-sized opaque patches under their cuticle or diffuse white discoloration of gill lamellae. *L. marina* is known from both sides of the Atlantic Ocean.

Leptolegnia baltica caused mass mortalities of *Eurytemora hirundoides* in the Baltic Sea. The disease did not affect other copepods, and over the last 4 decades no further outbreaks have been reported.

Haliphthoros milfordensis has been isolated from *Penaeus setiferus.* After experimental infection, the agent produced 100 % mortality in blue crab eggs, brine shrimp, and pink shrimp, and 46 % mortality in postlarval lobsters. The agent replaces host tissues by its mycelia. It first destroys host fat and striated musculature, later, the gut. The fungus rapidly spreads via large motile zoospores. In culture, *H. milfordensis* mycosis is controlled primarily through hygiene. Agent growth can be affected or inhibited by malachite green, Furanace, formalin, potassium permanganate and treflan (p. 337).

Atkinsiella hamanaensis infects crab eggs and brine shrimp; *Pythium* sp., *Palaemon serratus* and possibly also other crustaceans.

Burn spot disease and black gill disease are caused by *Fusarium* sp., and thus are also known as *Fusarium* disease. *F. solani* infects *Penaeus japonicus, P. duorarum, P. setiferus, P. aztecus, P. vannamei, P. stylirostris, P. californiensis, Homarus americanus* and *H. gammarus.* Certain penaeid species (e.g., *P. japonicus* and *P. californiensis*) are highly susceptible to *Fusarium* disease. Cuticular wounds provide a portal of entry and substrate for agent establishment. Mycotoxins may be a major factor in the pathogenesis of the disease. Diagnosis of *Fusarium* disease requires demonstration of the canoe-shaped macroconidia in wet-mount smears of exoskeleton or gill lesions. Attempts to control the disease in cultured shrimp, other than prevention, have not yet been successful on a practical basis.

Black mat syndrome or black mat disease is limited to tanner crabs and caused by *Trichomaris invadens*. In some of the offshore areas surveyed, black mat disease prevalence attained epidemic levels (e.g., 65 %; in barren tanner crab females up to 94.7 %). It is not known whether the barren state was due to senescence or the disease. First indications of the disease are small focal black spots on the carapace which later enlarge and coalesce; eventually the whole dorsal carapace is overlayed by a dark, tar-like mass of tangled hyphae and fruiting bodies (Fig. 3-24, p. 347). Tissue sections reveal massive hyphae proliferation, e.g., in connective tissues and blood vessels.

Diseases Caused by Protistans

The most important protistan pathogens known to cause severe diseases in marine crustaceans belong to the dinoflagellates, amoebas and microsporidians. There are, however, numerous other protistans which live as symbiotes on or in crustaceans and which may produce significant negative deviations from the host's normal state. Their role as potential disease agents has in most cases received insufficient attention. From the detailed documentation of pathogens and parasites in Chapter 3.2, I have selected the following examples.

The dinoflagellates *Blastodinium hyalinum, Syndinium* sp. and *Paradinium pouncheti* inhabit body cavities or guts of copepods. These endoparasites may inflict castration and/or death of their hosts.

The dinoflagellate *Hematodinium perezi* infects the hemolymph of *Carcinus maenas* and *Portunus depurator* in European waters; and that of *Callinectes sapidus, Ovalipes oscellatus, Cancer irroratus* and *C. borealis* in North American waters. Another dinoflagellate of unknown identity infects the hemolymph of *Chionoecetes bairdi* and *C. opilio*. This dinoflagellate, though similar to *Hematodinium* sp., causes Bitter Crab Disease, first described for the commercially exploited Southeast Alaskan tanner crab *C. bairdi*. Clinical signs of Bitter Crab Disease are pink coloration of the carapace, lethargy and a milky hemolymph. The agent replicates in the hemolymph and imparts a bitter aftertaste to cooked crab meat. Ultimately it kills all infected individuals. The bitter taste renders crab meat unmarketable and, thus, has caused high financial losses in the Alaskan tanner crab industry. The disease is chronic, occurring over an 11 month period. Single-cell and plasmodial stages (Figs. 3-25 to 3-27, pp. 352, 353) are carried by hemolymph to all host organs and tissues. Within 5 to 6 months post infection many crabs die; the remaining crabs are killed shortly after parasite sporulation. Bitter Crab Disease seems to follow an annual cycle which may, in part, be controlled by water temperature. Recently, Bitter Crab Disease has been detected in the Bering Sea in *C. opilio*. The disease seems to be spreading to previously uninfected host populations. In view of the severe decimation of natural populations, major economic losses seem almost certain.

Management of Bitter Crab Disease involves avoidance of harvesting from seriously diseased crab populations, and destruction (burial, incineration, cooking) of infected crabs. Should it turn out that the agent completes its life cycle within 1 year in a single host, earlier annual harvesting would result in the collection of infected crabs before parasite establishment could degrade the meat and kill the host (p. 355). In such a case the crabs caught should be marketable and disease dissemination reduced.

Thalassomyces californiensis invades the eye stalk of *Pasiphaea emarginata* and develops a root system that enters brain, optic nerve and ventral nerve cord of the host

shrimp. Root portions of another ellobiopsid parasite, *T. marsupii,* attach to ganglia of the amphipod *Parathemisto pacifica.* The parasite causes hypertrophy of the nerve cord and disorganization of nerve fibers. Details of pathology and therapy remain to be investigated.

Most Flagellata and Ciliophora do not appear to play important roles as primary disease-causing symbiotes of crustaceans. *Lagenophrys callinectes* and *Ephelota* sp. live as ectocommensals on blue crabs and shrimps, and may contribute to host mortalities in captivity. *Paranophrys maggii* and/or closely related forms are apparently secondary invaders which infect the circulatory system of a variety of crabs; the agent has also been reported from the isopod *Gnorimosphaeroma oregonensis* and *Homarus americanus.* Related mortalities in captive crustaceans probably are caused by destruction of hemocytes and major organ systems. Clinical signs of infection are lethargy and anorexia, and ataxia prior to death. Diagnostic characteristics of the disease include a cloudy hemolymph harboring myriads of motile ciliates with a long trailing cilium. Primarily observed in captive hosts, *Paranophrys* disease has also been reported from free-living Dungeness crabs and *Carcinus maenas.* Possibilities of disease management are still to be explored.

Only 1 member of the Amoebae is known as a major disease agent in crustaceans: *Paramoeba perniciosa.* Causing Gray Crab Disease in blue crabs, this parasite imparts a gray color to the ventral carapace and hemolymph in severely infected hosts. *P. perniciosa* has a large vesicular nucleus with a large central endosome and a Nebenkörper. Parasitized crabs eventually become lethargic and die; their hemolymph does not clot and is clouded with amoeba which tend to replace all hemocytes. *P. perniciosa* enters its host via ingestion and also via external wounds, particularly those obtained during molting; it predominantly inhabits hemal spaces and connective tissues, occurring in the hemolymph only in terminal disease stages. Epizootics of Gray Crab Disease usually prevail in spring; hibernating crabs reveal no hemolymph infections. It is not known whether infections persist below detectable levels in host tissues or whether annual reinfection keeps the disease going. Hosts presumably die due to disorders in respiratory and organ function, as well as nutrient deficiency. Gray Crab Disease management requires further investigation. *Cancer irroratus* and *Homarus americanus* are alternative hosts for *P. perniciosa.* However, in these crustaceans negative effects of the parasite have not been observed.

Sporozoa are obligate inter- and intracellular parasites of numerous animals including crustaceans. Especially the microsporidia may cause serious diseases. Other Sporozoa — gregarines, eugregarines and coccidians — are mostly not considered particularly harmful to their crustacean hosts. In the following paragraphs I mention only some of the more important pathogens.

Thelohania herediteria infects ovaries and muscles of female *Gammarus duebeni* in the Elbe River (Germany), shifting the sex ratio of offspring strongly in favor of females. It is assumed that *T. herediteria* inhibits the development of the androgenic gland which induces maleness. A similar sex-determining effect has been observed for *Octosporea effeminans* infecting ovaries and adipose tissues of *G. duebeni* in the Elbe estuary and the western Baltic Sea.

Cotton or milky disease of penaeid shrimps, characterized by opaque white coloration of the abdomen, involves damage to muscles and other tissues. The disease apparently is caused by several microsporidians and can be very severe, rendering infected hosts less resistant to environmental stressors.

Cottage Cheese Disease, caused by 2 undescribed *Thelohania* species, has been reported from *Paralithodes camtschatica* and *P. platypus*. Massive spore development — grossly visible as white curdy material — occurs in many tissues including hepatopancreas, ovaries, tegumental glands, and digestive walls. Similar disease manifestations, mostly confined to musculature including the heart, are caused by an unidentified microsporidian in the golden king crab. The infections appear to be lethal but infected crabs are low in prevalence.

Ameson michaelis of blue crabs sporulates in muscles throughout the body causing lysis of myofibriles and an opaque, chalky gross appearance. Infected crabs do not survive handling; however, the economic impact of the disease cannot yet be assessed.

Diseases Caused by Metazoans

Among the numerous metazoan parasites of marine Crustacea, some isopods and rhizocephalans can cause devastating harm at the population level. These and other, mostly less virulent, parasites belonging to a variety of metazoan taxa are reviewed in detail in Chapter 3.2. For this summary I have selected only a few important cases.

The turbellarian *Kronborgia amphipodicola* parasitizes the body cavity of several amphipods and often kills its host. Related turbellarians (e.g., *K. caridicola*) infest shrimp species. Trematode metacercariae have been found in numerous crustaceans and may cause disease, especially in severely parasitized hosts (e.g., damage to nervous tissues, ataxia).

Cestodes, nematodes, acanthocephalans, nematomorphs, copepods, hirudineans, and molluscs do not seem to play a significant role as disease agents in crustaceans at the population level. In contrast, several isopods, nemerteans and cirripedians may cause considerable damage and induce demonstrable negative deviations from the host's normal state, i.e., disease in the sense defined in Vol. I (p. 14). *Carcinonemertes errans* has played a major role in Dungeness crab and, possibly, king crab populations. In the course of agent/host co-evolution, such parasites usually develop considerable structural and functional modifications. General trends of such modifications are outlined in Vol. I (p. 56); details, in this volume.

Parasitic copepods obtain from their hosts body materials via sucking of fluids or by feeding on tissues. Apparently, many of them were preadapted to parasitic life and did not have to develop pronounced specializations *de novo*, but rather elaborate already existing structures and functions. One of the specialists, *Rhizorhina ampeliscae*, lives in the gill tissues of gammarids with only its egg sacs protruding.

Most members of the isopod suborder Epicaridea live as larvae (epicaridium) in copepods and as adults in decapod crustaceans. Morphologically these parasites resemble free-living isopods. Larvae of the Bopyridae (cryptonisci) leave their copepod host and enter the branchial cavity of shrimp where they molt into a bopyridium. Interestingly, the first larva to enter develops into a female; all later arriving larvae become neotenic males and attach themselves to the female between her abdominal pleopods. In case several larvae enter a given host at the same time, all develop into females but only 1 larva matures. Cryptonisci of other isopods (Cryptoniscidae, Entoniscidae) reside as endoparasites in anomuran, brachyuran and, rarely, macruran crabs. The host damage induced by isopod parasites includes atrophy of gill and muscle tissues, blockage of ovary maturation, nutrient depletion, tissue necrosis, (partial) parasitic castration in both sexes, and increased host mortality.

Rhizocephalans have evolved sophisticated structural and functional adaptations enabling them to exploit especially decapod crustaceans and their own free-living relatives. Noted for their invasiveness, but general non-lethality in feral hosts, rhizocephalans tend to suppress fecundity by inducing castration, thus causing endocrinological changes in host physiology, behavior and appearance. The rhizocephalan *Sylon hippolytes* parasitizes shrimp such as *Spirontocaris lilljeborgi* and *Pandalus platyceros. Peltogaster paguri* associates with *Pagurus bernhardus* and *P. pubescens,* causing gonadal degeneration in host females and apparently depletion of body fat. *Lernaeodiscus porcellanae* castrates *Petrolisthes cabrilloi* females and males. *Sacculina carcini* infests *Carcinus maenas.* This well known parasite inhibits spermatogenesis and arrests vitellogenesis, and it modifies secondary sexual characteristics of its host. The detrimental deviations are induced via modifications in neuroendocrine control, apparently due to secretory products released from rhizocephalan rootlets. Neurolemma penetration by rootlets leads to the degeneration of neuroglia and neuropile with pyknosis and karyorrhexis of perikarya. The evidence at hand suggests that a rhizocephalan parasite can affect the distributional range of its host. Further examples of rhizocephalan parasitism include the cosmopolitan *Briarosaccus callosus* which parasitizes a variety of lithodid crabs and causes growth suppression. Parasitized Alaskan king crabs cannot contribute to population recruitment due to castration of both sexes. This may reduce the fisheries harvest and modify population dynamics.

Diseases Caused by Proliferative Disorders and Neoplasia

Proliferative disorders and neoplasia are rare among marine crustaceans. Only 8 cases have become known. Of these, 6 involve shrimp of the genus *Penaeus;* the remaining 2, a caridean shrimp and a wild-caught red king crab. Determination of the reasons behind the apparent scarcity of neoplastic events in Crustacea deserves careful analysis.

The reported cases include a papilliform tumor-like growth in *Penaeus aztecus,* assumed to represent a benign neoplasm. Hamartomas were found in wild, postlarval *P. aztecus.* Three conditions of unusual idiopathic hyperplasia or hypertrophy involve proliferation of epineural layers surrounding the ventral nerve cord *(P. japonicus);* an acellular proliferation of the mid-gut (*P. japonicus, P. plebejus* and *P. merquiensis*); and multiple proliferative lesions of the lymphoid ('Oka') organ, including metastatic foci. Cases of reported neoplasia include a lymphosarcoma of hematopoietic tissue in *P. vannamei* infected with IHHN virus; a putative hind-gut carcinoma in *Paralithodes camtschatica;* and a carcinoma of developing *Palaemon orientis* embryos.

Hamartoma-affected shrimp have been suggested to be more available to predation (older individuals with such lesions were absent in wild populations).

Gut-and-nerve-syndrome (GNS) was seen in various captive shrimp populations with incidences ranging from 50 to 100 %. Gross signs of this disease are lethargy, low stamina, reduced escape reaction, poor growth, focal-to-general abdominal muscle necrosis, as well as increased susceptibility to epibiotic fouling, systemic bacterial and *Fusarium solani* infections. Lesions are located in the anterior mid-gut (basement membrane hypertrophy) and the epineurium (hyperplasia) of the ventral nerve cord. The cause of GNS is not known. A possibly related syndrome appears to be midgut serosal hypertrophy.

The clinical effects of 'Oka' (lymphoid) organ hyperplasia and metastasis (OHM) of penaeid shrimp, and the causes of these conditions, are unknown.

Comments on Diseases of Chaetognatha

A unique phylum in terms of morphology and evolution, the chaetognaths do not appear to harbor a flora and fauna of symbiotes essentially different from those found in other marine invertebrate hosts. The diseases of chaetognaths have thus far received very little attention. Since most chaetognaths die soon after being transferred to laboratories, even anecdotal reports on their symbiotes are rare. While a number of microorganisms, protozoans and metazoans may live on or in chaetognaths, in most cases they did not seem to inflict severe damage on their host.

Bacterial infections have been claimed to result in 2 types of 'X diseases' in *Sagitta crassa* (p. 427), characterized by degeneration of the epithelium and malfunction of the ciliary organs, respectively. In other cases, bacterial infection caused tumor-like swellings, head damage, impairment of feeding activities and reproductive potential, as well as diminution in body size.

Members of the Amoeba, Flagellata, Gregarinida and Ciliata have been found in or on Chaetognatha but details on agent-host relations have still to be studied. Trematodes are the most abundant parasites of chaetognaths (Table 4-1, p. 430). Usually living in the trunk coelom and sometimes in the gut, the trematodes may cause atrophy or complete removal of ovaries and hence partial or total sterility. For *Derogenes varicus* the first intermediate hosts are snails (*Natica* sp.); the second, copepods, ctenophores and chaetognaths. In Canadian Atlantic waters, *Hemiurus levinseni* infests *Sagitta elegans* in spring and autumn (up to 6 parasites host^{-1}).

Very few Cestoda inhabit chaetognaths, but Nematoda (mostly larval ascaroids) have been recorded frequently. Polychaeta and Copepoda were found attached to chaetognaths. They appear to act as commensals or ectoparasites. Considering the delicate body walls of their hosts, such attachment *per se* could cause serious damage.

Only in one case have abnormal growth and tumor-like abnormalities on appendages been reported for chaetognaths (in the Japan Sea).

Comments on Diseases of Echinodermata

Most phyla include members that coexist intimately with echinoderms. However, only few of these symbiotes have become known to inflict disease on their host. Echinoderms command effective defense mechanisms: whole-cell mechanisms such as phagocytosis, and cell-product mechanisms involving humoral factors, e.g., hemolysin and hemagglutinin (Vol. I, p. 50); some echinoderms produce antiviral and antifungal substances.

No definitive information is available on viral agents infecting echinoderms. Among the bacterial agents, *Vibrio* and *Aeromonas* species have been reported to act as causative agents of the communicable and potentially very detrimental bald-sea-urchin disease (p. 441) which leads to focal destruction of the body surface. Where the lesions cover more than 30% of the body surface, or where they penetrate deeply, death prevails. Infected urchins exhibit inflammatory-like responses with massive coelomocyte migration. *In situ* mass mortalities of *Strongylocentrotus franciscanus* and *Paracentrotus lividus* are believed to have been due to bald-sea-urchin disease. In contrast, mass mortalities of *S. droebachiensis* with body-wall lesions appear to have been caused by non-bacterial factors.

A disease affecting several Antarctic cidaroid echinoids (species of *Rhynchocidaris*

and *Ctenocidaris*) is caused by *Echinophyces mirabilis,* assumed to be a fungus. The pathogen inhabits the primary spines, dwarfs the host and may modify test growth.

A condition similar to that of bald-sea-urchin disease, observed in *Echinus acutus,* may have been caused by the blue-green alga *Dactylococcopsis echini.* Mass mortalities of *Strongylocentrotus droebachiensis* along the Atlantic coast of Nova Scotia (Canada) resulted from *Paramoeba invadens* infection. Paramoebiasis leads to loss of peripheral muscle function in tube feet, spines and mouth; quantitative reduction in red and white spherule cells; and incomplete clotting.

Apicomplexa parasitize holothuroids and echinoids but have never been reported from crinoids, asteroids or ophiuroids. There is little information available on agent life cycles and biology, and on agent effects exerted on hosts.

Among the numerous ciliates known to associate closely with echinoderms, only *Orchitophrya stellarum* has definitely been shown to be parasitic. This agent affects asteroid gonads, and in male hosts may cause complete castration.

Ophiura texturata, O. albida and *O. sarsi* may be targets of infection by the unicellular green alga *Coccomyxa ophiura.* Generally inhabiting the organic meshes of the ophiuroid's calcareous plates, the algal agent progressivly forms irregular holes and grows in subepidermal green cushions which tend to unite. Soon afterwards the host's epidermis disintegrates and death follows. The closely related alga *C. astericola* invades the asteroids *Hippasteria phrygiana* and *Solaster endeca* causing similar disease signs as described above.

A population of the echinoid *Diadema antillarum* — the principal herbivore and the most effective bioeroder of the Caribbean region — suffered a devastating mass-mortality that reduced its density to values of 0.6 to 7 % of the previous level depending on the area. The mass mortality was due to an as yet unidentified pathogen that is said to have caused the most widespread epidemic ever documented for marine invertebrates.

The mesozoan *Rhopalura ophiocomae* parasitizes ophiuroids, causing regression of host ovaries, decrease in the host's regenerative capacities and, possibly, a reduction in host growth. While a number of Turbellaria, Nematoda, Mollusca, Myzostomida, Copepoda and Ascothoracida associate closely with echinoderms, their potential roles as disease-causing agents have largely remained unexplored. Essentially the same holds for other metazoan echinoderm associates.

Most echinoderm agents inhabit either body wall, digestive cavity or coelomic cavities. Accordingly, the reviewer distinguishes 3 groups of symbiotes: external (A), intradigestive (B) and internal (C) agents.

External symbiotes (A-agents), such as gastropods, crabs and copepods, often feed on the host's body materials; cause hypertrophy of skeletal ossicles; induce the formation of supernumerary skeletal ossicles: inhibit the development of skeletal ossicles; or provoke dermal outgrowths.

Intradigestive symbiotes (B-agents), such as turbellarians, gastropods and copepods, inhabit the echinoderm's digestive system. The effects of these agents on their respective hosts are poorly investigated. B-agents feed on digestive epithelia or participate in the host's meals (e.g., bivalves, crabs); they may cause structural deformations.

Internal symbiotes (C-agents) inhabit coelom, hemal system or gonads; examples are sporozoans, turbellarians, aberrant gastropods, copepods, ascothoracids and fishes. Their nutritional resources are poorly known. Sporozoans spend most of their life cycle in the host's hemal lacunae. Gastropods and spatangoid ascothoracids attach to the host's

coelomic wall and dangle in its body cavity. Fishes are either phoronts seeking shelter, or parasites consuming tissues of host gonads or respiratory trees. Several internal agents castrate their host. C-agent Protozoa, Trematoda and Nematoda mostly feed directly on gonadal host tissues. Typical invasion routes for C-agents are the echinoderm's mouth, anus, gonopores, bursal slits, or thin areas of the body wall (tube feet, respiratory papulae). The host's principal reactions to C-agent invasion are: inflammation, formation of fibrous sheets enclosing the invader, and phagocytation.

Structural abnormalities in echinoderms have received limited attention from investigators. Test abnormalities in echinoids are caused by a variety of circumstances, including injury, genetic damage, critical environmental conditions, and biotic or nutritional disease factors. The causes of abnormalities in asteroid arm numbers include irregularities of regeneration following injury and, possibly, high salinities during early development.

The ultimate ecological consequences of the microbial-caused diseases of echinoderms are seen by the reviewer in a reduction or even elimination of the natural host populations concerned. In contrast, most of the diseases caused by metazoan agents do not seem to result in major detrimental effects at the population level. Quantitative assessments of the potential ecological consequences of host castration have not yet been made. The impact of disease-mediated fluctuations in echinoderm population strength on echinoderm predators or prey may be considerable. The role of echinoderms as a vector of fish diseases caused by trematodes or nematodes needs to be more thoroughly investigated.

Comments on Diseases of Urochordata

While a number of Urochordata, especially the Tunicata, are known to harbor a wide variety of symbiotes, diseases *sensu stricto* have not been reported. The taxa of urochordate symbiotes include Bacteria, Sarcomastigophora, Apicomplexa and Acetospora, Ciliata, Protista *Incertae sedis,* Protophyta, Cnidaria, Ctenaria, Turbellaria, Annelida, Bryozoa, Nemertina, Gastropoda, Bivalvia, Cephalopoda, Copepoda, Amphipoda, Isopoda, Cirrepedia, Decapoda, and Pisces. In many cases, the Urochordata are primarily used as shelter, rendering the symbiotes phoronts or commensals rather than parasites. Some cephalopods, amphipods and fishes feed on host tissues. In the ascidians, a variety of symbiotes utilize the host's body cavities (cloacal or pharyngeal cavities) where they shelter (phoronts), participate in their host's meals or feed on their host's body parts (parasites).

Viruses have thus far not been reported as disease agents from urochordates. However, in polluted waters, the filter-feeding tunicates may retain and concentrate viruses, including those causing human hepatitis. Thus edible ascidians may have to be considered a potential health hazard. Bacteria often attack aquarium-held ascidians, especially after injury, and may cause death. Bacteria-urochordate relations in nature have been reported but the actual role of the bacteria as primary or secondary invaders remains unclear. Species of *Pyrosoma* depend on the presence of luminous bacteria which aggregate in their luminous organs, and are acquired by the host via a complicated transfer mechanism from generation to generation.

Protistans inhabit, as commensals or parasites, all Protochordata. A number of flagellates, gregarines and ciliates live closely associated with urochordates, and the

coccidian *Graseela microcosmi* parasitizes the hepatic gland epithelium of *Microcosmus sabatieri*. Numerous ciliates, both commensals and parasites, associate closely with urochordates.

For protophytan symbiotes, transfer of metabolites from alga to ascidian has been documented. Ascidian cells can phagocytose the algae. Details of the mutualistic relation between algae and ascidians remain to be studied. Better insights into such associations may open up interesting new views on the mechanisms of bilateral short-cutting and complementing of interspecific flows of material and energy.

The relations between ascidians and numerous species of copepods are quite intimate. Following a free-living juvenile phase, the copepods (some males and all females) must find a host to complete their development. The symbiote copepods are taxonomically and morphologically very diversified. This fact and the often elaborate adaptations witness long-standing co-evolutions. Interestingly, copepod symbiotes are absent in pelagic tunicates. Significant details have been disclosed on the influence exerted by urochordate hosts on their symbiote copepods and on the copepod's dietary requirements.

Amphipods often find shelter in the branchial and cloacal cavities of large-sized ascidians; they may destroy tunicate tissues (hyperiids). While the role of isopod and cirriped symbiotes awaits analysis, shrimps have been observed inside the branchial sacs of ascidians in numerous cases since the 19th century. Belonging to the genera *Pontonia*, *Dasella* and *Periclimenaeus* (Table 6-8, p. 623), the shrimps do not seem to cause damage. Similar relations, principally involving phoresis, exist in decapods (e.g., *Planes minutus*) and fishes (e.g., *Tetragonurus* spp.). For the fishes, the host provides shelter and may also be utilized as a food source.

CONCLUSIONS

The diseases of cephalopods, annelids, crustaceans, chaetognaths, echinoderms and urochordates have been the topic of relatively few studies. The body of knowledge available to this day is limited and fragmentary. For several major host taxa pertinent information is lacking or is so anecdotal that reviews of the state-of-the-art turned out to be impractical or impossible. Hence we had to limit our efforts to the groups listed above.

Even in these groups we know very little about disease phenomena prevailing under natural conditions in marine environments. And the potential ecological consequences of diseases — in terms of modifying population dynamics or flow patterns of energy and matter in ecosystems — have been addressed by very few authors.

Investigations on diseases caused by microorganisms can rely to a considerable degree on information obtained from non-marine living systems. This is also true for proliferative disorders and neoplasia, and for the principal effects exerted by extreme intensities of environmental factors on host predisposition, disease development, host defenses and disease manifestation. However, the respective conditions in marine animals require investigation in their own right. Some of the new insights to be expected are likely to open up important new vistas beyond the horizons of present-day science.

Much of the information available in the scientific literature was published by parasitologists. They have focused their efforts on (i) presence or absence of symbiotes; (ii) the taxonomy of symbiotes; (iii) symbiote life cycles: and (iv) symbiote distributions (macro- and microdistribution, Vol. I, p. 37). Only in relatively few cases has the type of symbiote activity — phoresis, commensalism, parasitism, mutualism — been clearly

defined (see also Vol. I, pp. 19 to 20). We know very little about agent virulence, host defense, etiologies and disease diagnosis, let alone disease prevention and therapy under controlled conditions. Here is a wide open field for future research!

However, documentations of host/parasite interrelations have considerable value in themselves. They provide baselines for future researchers. These investigators can now build on the reviews published in 'Diseases of Marine Animals' and thus identify host/parasite systems available for their particular research programs. Students of cephalopod parasites, for example, have developed and refined methods for maintaining and rearing cuttlefishes, squids and octopuses in large numbers, and this has made it possible to analyze selected host/parasite systems under controlled conditions. As more species come under cultivation the investigators may be able to resolve problems of long standing by resorting to the inventories presented in this treatise.

The information presently at hand on the diseases of marine animals largely encompasses a few (considering the total number of species) economically important forms and a few animals used for experimentation. Many records of parasite presence result from more or less accidental findings while examining preserved specimens sampled during collections or expeditions not necessarily planned to investigate disease phenomena.

Aquaculture (cultivation of aquatic organisms for commercial ends) research has been the single most important source of knowledge on disease phenomena prevailing in confined bodies of water. The overwhelming amount of the limited information thus far produced on agent virulence, host defense, etiologies and disease diagnosis — in a few cases also on disease prevention, control and therapy — has been the result of research conducted in aquaculture farms or related research units.

On the other hand, most of the *in situ* work conducted to this day was performed by fisheries scientists. With an eye on economically important species — but also with the thoroughness of devoted scientists and hence often looking beyond narrow economic interests — fishery biologists have laid the foundations for research on the diseases of marine animals under field conditions (see Vol. IV).

Financial support of scientific inquiries into disease phenomena in aquaculture and fisheries by sponsors with primarily economic interests is of course welcome. Without it we would know much less about diseases in marine animals. However, research dependent on economic interests is of necessity limited in scope and depth. True breakthroughs in scientific inquiry rely first of all on adequate support of basic research. And here lies a major obstacle to solid progress: considering the importance of disease research in marine animals for science at large and for the long-term development of modern human societies, the present world-wide support for pertinent basic research is totally inadequate.

As pointed out before (Vol. I, p. v), I consider diseases as a basic denominator of organismic coexistence. I believe that more knowledge about the diseases of marine animals will provide new leads for solving a variety of ecological and evolutionary problems, and will contribute to the understanding of the origin and evolution of disease phenomena in general, i.e., also in terrestrial and limnetic organisms and in humans. Life on earth originated in marine environments. Present-day diseases can presumably be fully understood only if more light is shed on the millions of years of coevolution of the biological partners involved in biotic diseases (host and agent), and on the evolutionary aspects of other diseases, including those due to failures in cellular coordination and integration (cancer). We need more and deeper insights into disease phenomena in marine

animals, both for progress in disease research and for sound assessments of the impacts on marine biota exerted by modern industrial human societies.

Major difficulties in assessing the significance of diseases in oceans and coastal waters originate from the lack of sustained long-term, large-scale, synoptic *in situ* research. In view of the enormous dimensions of marine environments, such research requires great technological, organizational and financial efforts, as well as the development of new strategies and methodologies. In particular, there is need for: (i) sustained ecosystem research in carefully selected, diverse habitats; (ii) standardization of quantitative sampling procedures; (iii) a more thorough understanding of the role of environmental variables (including those due to human activities) on disease predisposition and manifestation; (iv) more details on food chains and life cycles (i.e., on the dynamics and routes of host/agent interrelations); and (v) bringing more light into the still prevailing darkness which obscures the taxonomic identities of the organisms concerned, and into the resulting nomenclatural problems. The difficulties of *in situ* research on the diseases of marine animals are aggravated by the fact that diseased host individuals tend to disappear quickly and completely from the scene. They are usually more available as prey to numerous carnivores and for attack by a vast variety of microbes. Their bodies readily disintegrate and vanish leaving no trace.

I hypothesize (see also Vol. I, p. 17, pp. 21 to 23, as well as other volumes of this treatise) that diseases may act as significant parameters affecting flow patterns of energy and matter in ecosystems, and as modifiers and motors of evolutionary trends. This hypothesis requires critical testing. However, there can be little doubt that millions of years of host/agent coevolution leave ineradicable marks in the gene pools (and ecological potentials) of both. No organism evolves in a 'vacuum'. All forms of life are historical entities, formed by age-old organism-organism and organism-environment interrelations. Hence the impact of disease should not be judged solely in terms of quantitative changes in present-day performance (reproductive rate, population strength, distributional range) but also take into account qualitative changes of structures and functions acquired as a consequence of evolutionary change.

Examples documenting the ecological role of diseases in marine habitats include: (i) Bitter Crab Disease, Gray Crab Disease, and the effects of several isopod and rhizocephalan parasites that have caused devastating damage in natural crustacean populations (changes in population strength, reproductive potential, distributional range, etc.); (ii) microbial-caused diseases that have severely reduced or even eliminated natural echinoderm populations. Such pronounced impacts of disease affect also the population dynamics of prey and predators of the hosts concerned, and thus may modify structures and functions of the ecosystem concerned, at least temporarily.

There is need for detailed analyses of agent-caused modifications in the host's ecological potentials (growth, reproduction, survival, adaptability, competition). And we need to know more about the strategies and dynamics of interchange between agent and host. Such information is likely to reveal important new insights into the rules of organismic coexistence, and to provide models for analytical research in ecology.

1. DISEASES OF MOLLUSCA: CEPHALOPODA

The cephalopods comprise a small class of the phylum Mollusca. With the exception of the genus *Nautilus,* all living species belong to the subclass Coleoidea. Nearly 1,000 species of living cephalopods have been described, although that number has been significantly reduced in systematic revisions during the last 10 years. Currently, 650 species are recognized, compared with nearly 11,000 fossil cephalopods and a total of well over 100,000 species in the phylum.

Cephalopods range in body size from the tiny loliginid genus *Pickfordiateuthis* with an adult mantle length of 15 mm or less to genera of giant squids such as *Architeuthis* and *Moroteuthis* that may reach total lengths of 10 to 20 m or more. As soft-bodied invertebrates they are without equal for mobility and complexity of behavior. The basic body form is relatively homogeneous throughout the group. Cephalopods are active predators and live in all of the world's oceans. They occupy a variety of habitats, from pelagic forms in surface waters to solitary deep-sea forms to neritic forms that occur in dense schools in shallow coastal waters.

Interest in cephalopods has increased considerably in the last 40 years, principally because of their sudden entrance into the world market as a major fishery resource. In addition, cephalopods have proved to be valuable as experimental animals for biomedical and behavioral research. Although important and impressive advances have been made, especially over the last 20 years, our knowledge of the biology and systematics of all but a few species is sorely lacking.

The world-wide cephalopod fishery produced about 500,000 tons in 1945 and presently contributes 1 to 2 million metric tons. Some scientists have indicated a potential annual production of as much as 100 to 300 million tons. Without basic biological and fishery information, the cephalopod fishery has an uncertain future unless stocks can be managed adequately.

The role of cephalopods in the ecology of pelagic and benthic communities is a complicated topic for which we have only fragmentary knowledge. Many areas require critical attention. Not only is basic information needed on systematics, morphologies, distributions and life cycles, but we need a better understanding of the trophic relationships of the key cephalopod predators in the marine environment.

Most of our knowledge of the biology of cephalopods is derived from a few example species that are commercially important. Common coastal benthic and epibenthic forms such as *Octopus vulgaris* and *Sepia officinalis* have been studied extensively, especially in Europe and the Mediterranean. Members of squid genera such as *Loligo, Illex* and *Todarodes* also have been studied in detail because they are easily available in nearshore waters over the continental shelf. More recently, studies have been initiated on oceanic

squids that are fished commercially, such as members of the genera *Gonatus, Ommastrephes* and *Sthenoteuthis*. For such commercially important families as the Sepiidae, Loliginidae, Ommastrephidae, Gonatidae and Octopodidae we are only now beginning to develop a skeleton framework on which to hang the fragmented pieces of our knowledge.

Cephalopods have evolved a body plan distinctly different from that of other molluscs, with concomitant differences in behavior, reproduction and general life history strategies. Regarding the diseases of cephalopods, the 2 developments of greatest influence were (1) loss of the external shell combined with neural and muscular development enabling high-speed locomotion; (2) evolution of a delicate yet sophisticated skin. A cephalopod's outermost contact with the world is a single-celled microvillous epithelium covered by mucus (Fig. 1-1). This anatomical feature is superbly designed for locomotion (and perhaps respiration) but poorly adapted for withstanding physical impact. Thus it is

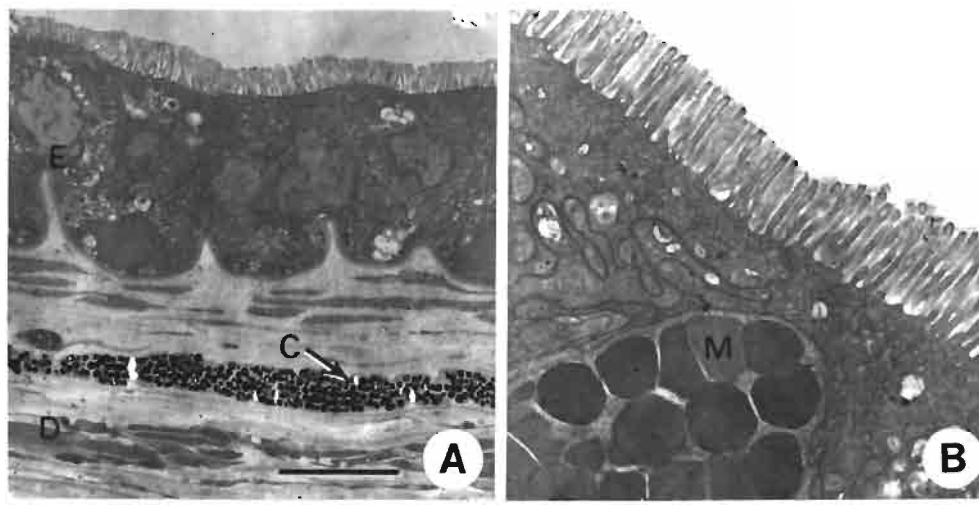


Fig. 1-1: Cephalopod skin. A: TEM section of normal skin from a young *Octopus joubini* (24 mm ML); epidermis (E) is one cell layer thick; dermis (D) contains a portion of an expanded chromatophore (C); bar = 10 μm. B: TEM section of microvillous border and a mucus-secreting cell (M) in the skin of the squid *Loligo plei*. (Original from R. T. Hanlon.)

no surprise that the most noticeable disease problems documented in cephalopods involve alterations of the skin followed by invasion of opportunistic pathogens.

There is a lack of information on diseases described from cephalopods collected in nature probably because injured or diseased individuals will be consumed quickly by their predators. In fact, the state of our knowledge concerning cephalopod diseases (as defined by Kinne, 1980*, for this treatise) is primitive, and very little is known about their internal defense mechanisms.

* Kinne, O. (1980). Diseases of marine animals: general aspects. In O. Kinne (Ed.), *Diseases of Marine Animals*, Vol. I. General Aspects, Protozoa to Gastropoda. Wiley, Chichester. pp. 13–73.

1.1 DISEASES CAUSED BY MICROORGANISMS

R. T. HANLON and J. W. FORSYTHE

Like other invertebrates, cephalopods do not have an immunoglobulin-based defense against foreign particles. They do have amoebocytes that play an important but as yet unspecified part in defense (Cowden and Curtis, 1981; Bayne, 1983). In cephalopods, wound healing sequentially involves muscular contraction, a dermal cellular reaction and epidermal migration (Lange, 1920; Polglase and co-authors, 1983; Feral, 1988). Furthermore, cephalopods are known to possess a haemoglutinin that may function as a lectin for immune responses (Russo and Tringali, 1983; Rögener and co-authors, 1985; Olafsen, 1988).

In attempting to be comprehensive, we include here the published literature as well as some unpublished pathology data from our laboratory, where 14 species of octopuses, squids and cuttlefishes have been maintained, reared or cultured for 13 years (Hanlon, 1987). Furthermore, since so few published reports were discovered, we solicited information (e.g., reports, obscure journal papers or chapters, etc.) from colleagues in France, Germany, Spain, England, the Soviet Union, Japan, Canada and the United States. We gratefully acknowledge the assistance of these colleagues as well as those who kindly provided illustrations (noted in captions).

Agents: Viruses

There are only 2 published reports on virus-like particles found in cephalopods. Rungger and co-authors (1971) found infected *Octopus vulgaris* in the Bay of Naples, Italy. The octopuses had tumors (ranging from 1 to 10 mm diameter) in the muscle tissue of the arms (Fig. 1-2, A), and in advanced stages tumors could be found on the funnel and the ventral surface of the mantle. Infected *O. vulgaris* brought to the laboratory eventually ceased feeding, became apathetic and often ate the affected parts of their arms; they usually died within 3 to 5 months of first appearance of visible tumors. Rungger and co-authors suggested that the infection is not uncommon in some years in market specimens and could be present in as many as 8 % of octopuses sold in Naples (Italy).

These tumors were embedded in arm musculature (Fig. 1-2, B), and in advanced stages the muscle tissue was completely degenerated. The tumors appeared to first invade the spaces between normal muscle fibers (Fig. 1-2, C, D); they then proliferated throughout the tissue to affect the interstitial septa and arteries, but not the nerves, cartilaginous tube or the epidermis; autophagy occurred at this time.

Electron microscopy revealed numerous virus-like particles that were hexagonal in section with electron-dense cores and were generally 120 to 140 nm long and 95 to 105 nm wide (Fig. 1-2, E–G). These particles occurred singly (Fig. 1-2, E) or in groups up to 300 in early stages of infection (Fig. 1-2, F); in the latter case the particles began to lose their coherence. Rungger and co-authors (1971) cautioned that experimental transmission of the disease and cultivation of the presumptive virus are required to firmly establish their

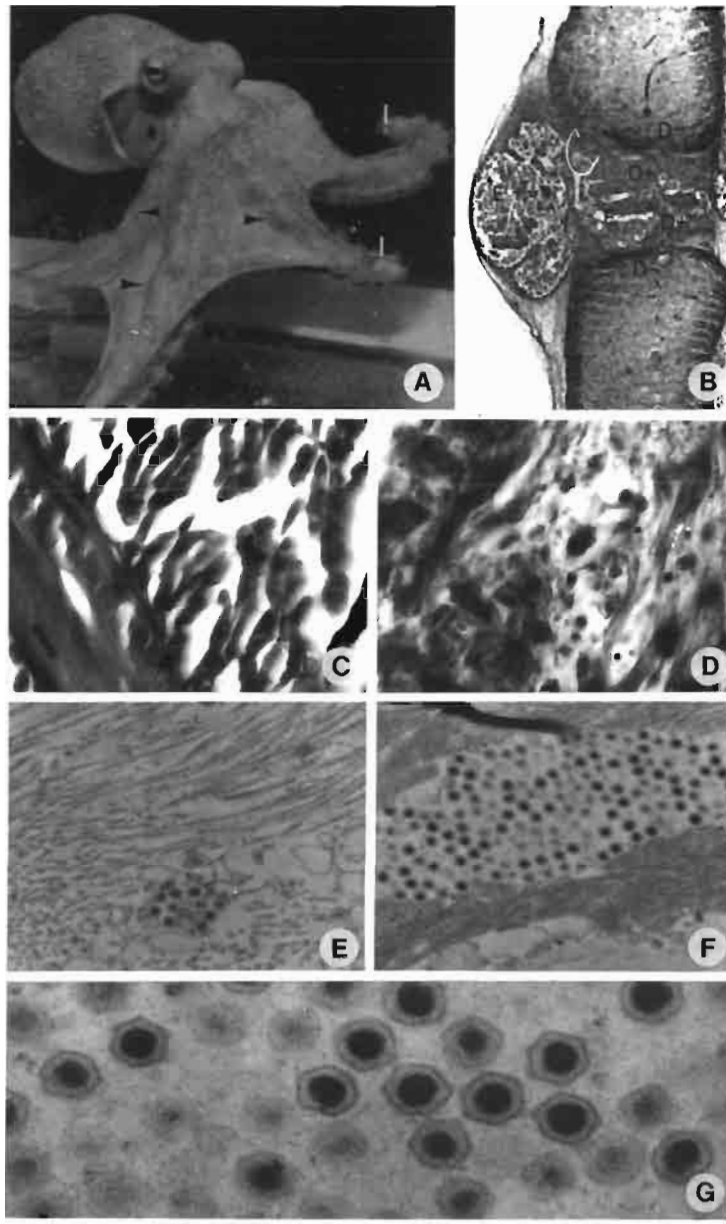


Fig. 1-2: *Octopus vulgaris*. A: Tumorous individual showing large knobs (arrows) on arms, the endings of which have been autotomized (white bars). B: Transverse section through mantle tumor; (E) edema, (D) area of decay, (D-) initial stage, (D+) advanced stage, (× 24). C: Section through normal arm muscle with elongate nuclei (× 1100). D: Necrotic area in arm showing rounded cells and nuclei containing dark granules; some muscle fibers and interstitial septa present; this corresponds to Area D- in Image B (× 1150). E: TEM of an area in early stage of degeneration (D- in Image B); muscle fibrils still present but already disaggregated; droplets and membranous structures present; single virus-like particles and a group of virus-like particles located between dissociating fibrils. F: Group of virus-like particles between decaying muscle fibrils (× 24,225). G: High magnification of virus-like particles with hexagonal structure of presumed capsid clearly recognizable; most capsids filled with electron-dense material (× 78,850). (After Rungger and co-authors, 1971.)

findings. Farley (1978), in reviewing the findings of Rungger and co-authors (1971), suggested that this might be an iridovirus because its size, morphology and location in the cytoplasm appear to be similar to that found in insect iridovirus.

The second report concerns virus-like particles in the stomach epithelium of the common European cuttlefish *Sepia officinalis* collected off the coast of France (Devauchelle and Vago, 1971). No details were given for the relative prevalence of this malady, nor of its deleterious effects on the cuttlefish. Most of the particles appeared in their micrographs as 5- or 6-sided icosahedrals ("elles sont spheriques ou paraspheriques" [sic]) approximately 75 nm in diameter with a nonenveloped capsid. Microtubular arrays were usually found in association with the particles, which appeared to develop within the cytoplasm. Devauchelle and Vago suggested that, although their observations were limited, these particles appear to be viral and that aspects of their development and structure indicate that they are similar to vertebrate 'Reovirus'.

There are 2 other incidental reports that indicate the possibility of virus-like particles in cephalopods. The first is in the loliginid squid *Loligo pealei,* in which F. G. Kern (Northeast Fisheries Center, NOAA, Oxford, MD, USA) found 'basophilic cytoplasmic inclusions in the epithelial cells of the tubules of the digestive gland, and that they are caused by a procaryotic organism' (pers. comm., 1988; also quoted in Otto and co-authors, 1979). The second report is by R. B. Short and F. G. Hochberg (1969 and pers. comm., 1988) that virus-like particles have been observed in sections of the renal appendages of several octopod species from New Zealand, Florida and California (USA), as well as in the nuclei of the somatic cells of the dicyemids that attach to the renal appendages.

From all of the aforementioned it is obvious that cephalopods have hardly been looked at with regard to virales. Hopefully these anecdotal reports will stimulate future workers to investigate and verify viral infections with the wider array of techniques now available.

Agents: Bacteria

There are no known bacterial diseases of cephalopods in nature, although there has never been a concentrated effort to look for such diseases. Fisheries biologists and ecologists who have studied extensively both exploited and unexploited cephalopod populations throughout the world have failed to observe any pathologic conditions indicative of bacterial disease (cf. Boyle, 1983, 1987). Compelling evidence also comes from long-standing traditional cephalopod fisheries of Asia (especially Japan), where the importance of aesthetic presentation of freshly-caught cephalopods has produced a keen eye for physical injury. The detection of chronic and acute bacterial diseases in juvenile age groups is problematic since it is difficult to locate young cephalopods in nature much less collect and sample them for disease. Nonetheless, the life history of cephalopods provides ample opportunity to encounter potentially pathogenic bacteria, particularly among the octopuses and cuttlefishes whose behavior dictates intimate contact with the benthic environment (Boyle, 1983).

Octopuses in Nature (Order Octopoda)

Forsythe and Hanlon (unpubl.) recently qualitatively sampled *in situ* the bacterial fauna of *Octopus bimaculatus* in the Channel Islands off the California coast, and *O. bimaculoides* in Long Beach harbor near Los Angeles, CA (USA). These data are still

being analyzed but these octopuses harbored 6 species of *Vibrio* alone and 6 to 8 non-fermenting bacteria species including *Pseudomonas*. Although far more sampling is needed, it seems that wild cephalopods host numerous, potentially pathogenic bacteria and thus the potential for disease clearly exists. We and other diving biologists have observed thousands of octopuses *in situ,* routinely seeing individuals missing varying portions of one or more arms, presumably lost to predators (e.g., Hartwick, 1983). In most cases, new arm tips were regenerated from the stumps with no signs of infection.

Squids in Nature (Order Teuthoidea)

Ford and co-authors (1986) sampled the external bacterial fauna of wild-caught *Lolliguncula brevis* in Galveston Bay, TX, USA (Table 1-1). Quantitatively, they found wild squids to have an average of 1,700 to 6,770 viable bacterial cells per cm^2 of dorsal mantle surface (depending on type of media used) while the sea-water samples from collection sites averaged 340 to 1,100 cells ml^{-1}. Qualitatively, there were at least 13 bacterial species isolated from the squids (Table 1-2); over half of them were potentially pathogenic species, while 2 of the 3 species isolated from seawater controls were potential pathogens. Externally, all of the squids sampled appeared to be in perfect condition.

Laboratory Cultured Octopuses

Most knowledge on bacterial diseases of cephalopods comes from husbandry, i.e., from members of this animal group kept in captivity. Octopuses have a long history of being held in captivity. Nevertheless, little is known of their bacterial diseases. Hanlon and co-authors (1984) described severe skin ulcerations of *Octopus joubini* and *O. briareus* cultured in large numbers in recirculating seawater systems (2,500 l capacity). The ulcerations were due to secondary bacterial invasions of skin abrasions caused by octopus-octopus interactions under crowded conditions. The first signs of disease were seen in *O. joubini* at 2 months post-hatching, and at 1 month in *O. briareus*. Gross anatomical manifestations of progressive infection were broken down into 4 arbitrary stages. The first stage involved extensive damage to the microvillous epidermis of the dorsal mantle and dysfunction of the underlying chromatophores due to destruction of the nerves and radial muscles involved with their expansion and retraction (Fig. 1-3, A, B). In the second stage, the epidermis and dermal chromatophores were totally destroyed, leaving a clear or white lesion (Fig. 1-4, A, E). The infection then penetrated through the dermis and into the underlying muscle layers of the mantle. In Stage 3 (Fig. 1-4, A, D), lesions spread to the lateral or ventral surfaces of the mantle and penetrated deep into or through the muscle layers, exposing the gills or other internal organs. Stage 4 (applying only to *O. briareus*) was the spread of lesions onto the head and arms. Octopuses died at Stages 3 and 4 with the entire progression requiring as little as 4 days. Without therapeutic intervention, the disease always ran the full course to death. No disease problems were encountered with individually reared siblings.

Only Gram-negative bacteria were cultured from ulcers (Fig. 1-4, B, C): *Vibrio alginolyticus, V. damsela, Pseudomonas stutzeri* and *Aeromonas cavia* were cultured from ulcerated skin of *O. joubini*; *V. parahaemolyticus, V. damsela* and *P. stutzeri* from that of *O. briareus*. These 5 species — as well as *Bacillus* sp., *Acinetobacter* sp., *Pleisiomonas* sp. and *Flavobacterium breve* — were cultured from water-culture-system samples. Reinfection experiments were carried out with all 5 bacterial species originally isolated from

Table 1-1
Total viable bacteria from *Lolliguncula brevis* mantle tissue and from sea-water (After Ford and Co-authors, 1986; modified)

Sample type	Number of samples	Culture medium[a]		
		TCBS	MA	GYP
Mantle tissue		Multiply these values by 100 for number of viable cells cm^{-2}		
Wild	30	17.0 ± 25.4	67.7 ± 58.8	31.8 ± 45.6
Laboratory—normal[b]	10	1114.9 ± 2229.2[d]	1804.5 ± 3413.3	48648.0 ± 140305.7[d]
Laboratory—ulcerated[c]	8	2655.7 ± 4298.4[d]	9985.8 ± 36558.4[d]	3818.7 ± 7411.2
Sea-water		Multiply these values by 100 for number of viable cells ml^{-1}		
Bay	3	3.4 ± 1.1[e]	41.8 ± 12.4[e]	110.5 ± 164.1[e]
Maintenance system	7	2.5 ± 3.5[e]	21.2 ± 26.0[e]	10.7 ± 14.2[e]

[a] TCBS: Thiosulfate Citrate Bile Salts (Difco); MA: Marine Agar 2216 (Difco); GYP: Zobell, 1946. Numbers: means ± 1 SD.
[b] Includes tissue from normal individuals (n = 3) and normal tissue from ulcerated individuals. There was no significant difference between these 2 sample types.
[c] Includes 3 individuals with open lesions and 5 individuals with damage to epidermis but not with open lesions.
[d] Significantly different from wild squids ($p < 0.05$).
[e] No significant differences were detected between bay and laboratory maintenance sea-water.

Table 1-2
Bacteria most frequently isolated from squid skin and sea-water (After Ford and co-authors, 1986)

Skin of wild squid	Normal skin of laboratory-maintained squid	Ulcerated skin of laboratory-maintained squid	Bay sea-water	Maintenance system sea-water
Gram-negative bacteria				
Aeromonas sp. (9)	*V. alginolyticus* (6)	*V. alginolyticus* (4)	*Vibrio* sp. (2)	*Vibrio* sp. (4)
Flavobacterium sp. (7)	*Vibrio* sp. (4)	*Vibrio* sp. (1)	*V. parahaemolyticus* (1)	*Proteus* sp. (1)
Pseudomonas sp. (5)	*Aeromonas* sp. (3)	*V. metschnikovii* (1)		*Flavobacterium* sp. (1)
Vibrio sp. (3)	*V. parahaemolyticus* (1)	*Aeromonas* sp. (1)		
Proteus sp. (3)	*V. metschnikovii* (1)	*Pseudomonas* sp. (1)		
V. parahaemolyticus (2)	*Flavobacterium* sp. (1)			
V. alginolyticus (2)	*Proteus* sp. (1)			
Flexibacter (1)				
Gram-positive bacteria				
Bacillus sp. (21)	*Bacillus* sp. (2)	*Bacillus* sp. (4)	*Bacillus* sp. (2)	*Bacillus* sp. (1)
Streptococcus sp. (2)	*Streptococcus* sp. (2)	*Staphylococcus* sp. (2)		
Staphylococcus sp. (2)				
Micrococcus sp. (2)				
Planococcus sp. (2)				

Bracketed numbers: number of isolates obtained from the sample.

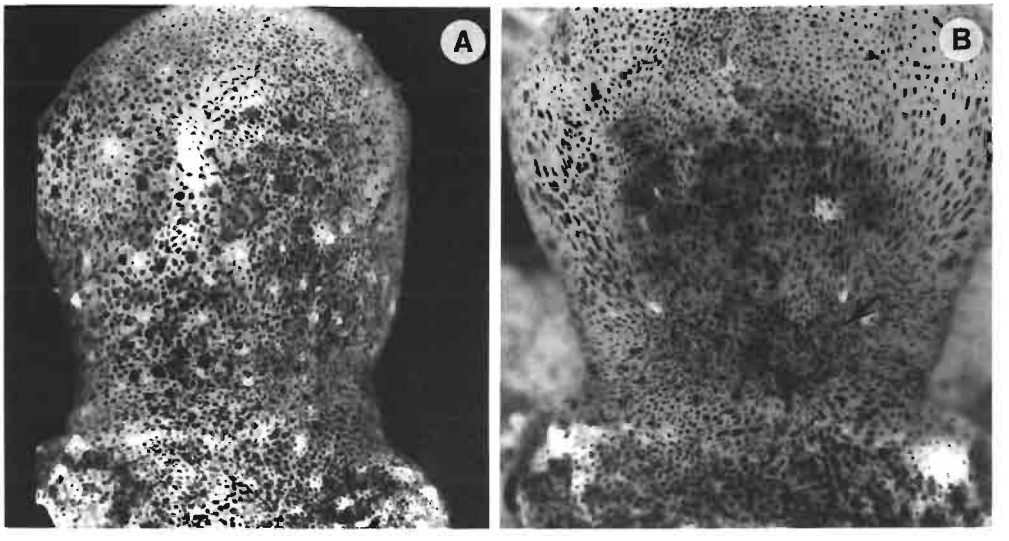


Fig. 1-3: *Octopus joubini*. A: Dorsal mantle of young individual (7 mm ML) with Stage 1 ulceration (altered chromatophores). B: Same-sized individual with Stage 1 ulceration indicated by darkened area of dispersed chromatophore pigment granules (arrow). (After Hanlon and co-authors, 1984.)

ulcers. Three-month-old *Octopus joubini* (1 g) were anesthetized and a 2-mm diameter lesion cut into the skin. The cut was deep enough to disrupt the dermal chromatophore layer, leaving a clear area that could be seen for 2 to 3 weeks. Immediately after the incision the octopuses were revived in sterile sea water then placed in a 1 l bath of sterile sea water innoculated with a pure culture of bacteria at 10^6 colony forming units (cfu)/ml for 60 min. Only *V. alginolyticus* produced disease-like lesions, which appeared in 3 days (Fig. 1-5, A, B). At concentrations of 10^5 and 10^4 cfu/ml, no disease symptoms were produced by *V. alginolyticus*.

The effect of several antibiotics were tested on halting disease progression and promoting healing (Table 1-3; Forsythe and co-authors, 1990). Only nifurpirinol significantly reduced mortality by stopping ulcer penetration and promoting healing, although best results were obtained with isolated octopuses. Group-reared octopuses still suffered high mortality (45 %) even with nifurpirinol treatments. Complete healing of lesions required 1 to 2 months. Ulcers typically progressed to the next stage of damage during the first week of treatment with nifurpirinol. Healing became evident thereafter with the formation of a smooth, continuous epidermis across the ulcerated area (Fig. 1-5, C, D) although chromatophores were still absent. Regeneration of chromatophores required 2 or more months.

Stoskopf and co-authors (1987) reported treating skin lesions of *Octopus dofleini* on display at the National Aquarium in Baltimore, Maryland (USA). A 7 kg individual had a chronic lesion 1.5 cm diameter for 2 months after arrival. After this initial period the lesion began to enlarge and deepen while additional small lesions (3 to 4 mm diameter) began to appear. Cultures from the large lesion revealed *Pseudomonas* sp. and *Acinetobacter anitratus*, both susceptible to tetracycline. The octopus received tetracycline injected into its live food organisms at a dosage of 10 mg kg^{-1} body weight once per day. The small lesions disappeared in 7 days, and the large lesion healed completely in 28 days.

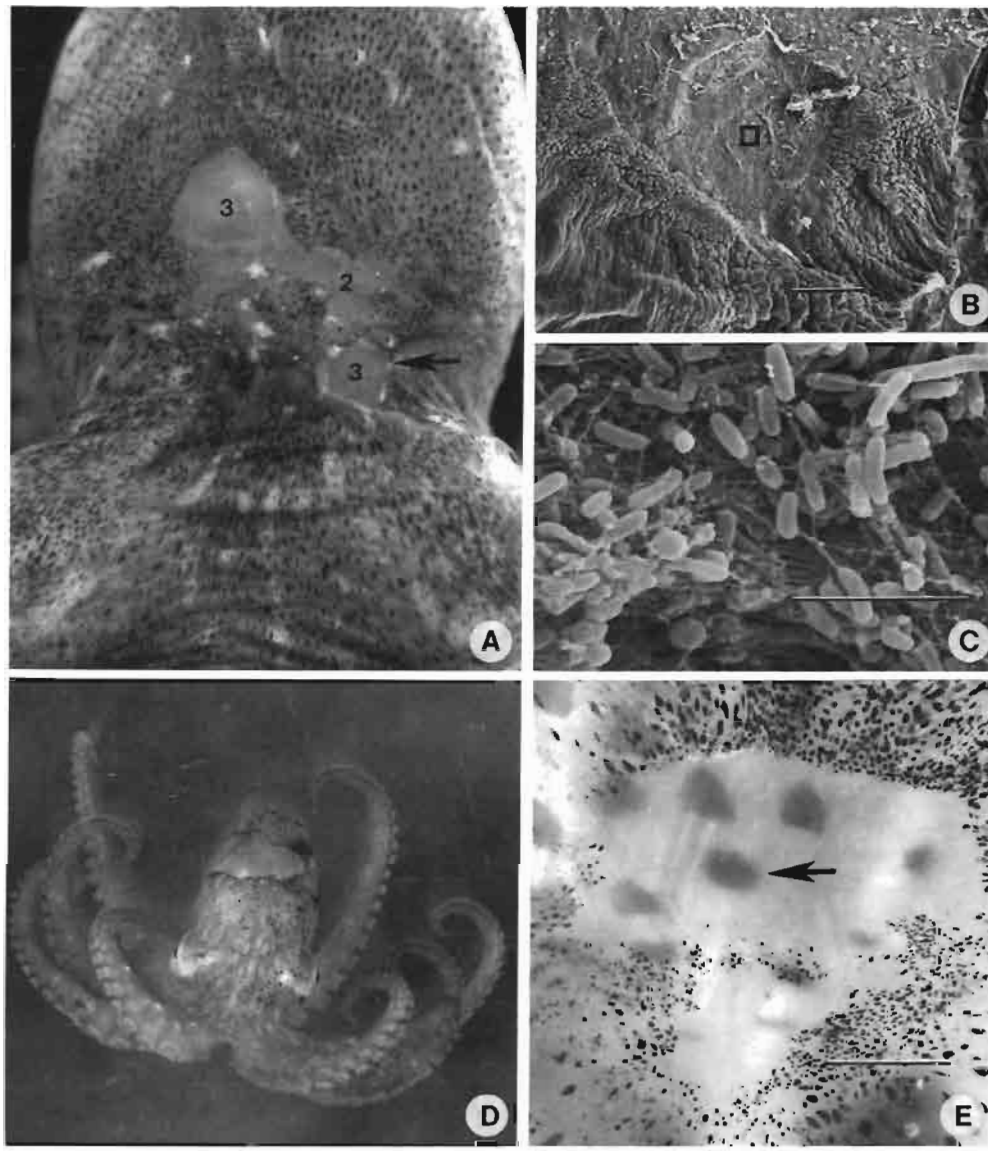


Fig. 1-4: *Octopus joubini*. A: Preserved individual (8 mm ML) with Stage 2 and 3 ulcers (marked with numbers). B: Detailed SEM view of smaller Stage 3 ulcer in Image A; wrinkled epidermis conspicuous in bottom of photograph; area in box enlarged in Image C; bar = 20 μm. C: Dense bacterial mat in center of lesion (see B above); bar = 5 μm. D: Diseased individual (7 mm ML) with spreading Stage 3 ulcer on mantle and darkened chromatophores around periphery of the ulcer. E: Close-up photograph of a Stage 2 ulcer on a young individual (7 mm ML); large, round, extrategumental chromatophores (arrow) are on the dorsal visceral surface well below the ulcer; dermis is gone and translucent muscle tissue is exposed; bar = 1 mm. (After Hanlon and co-authors, 1984.)

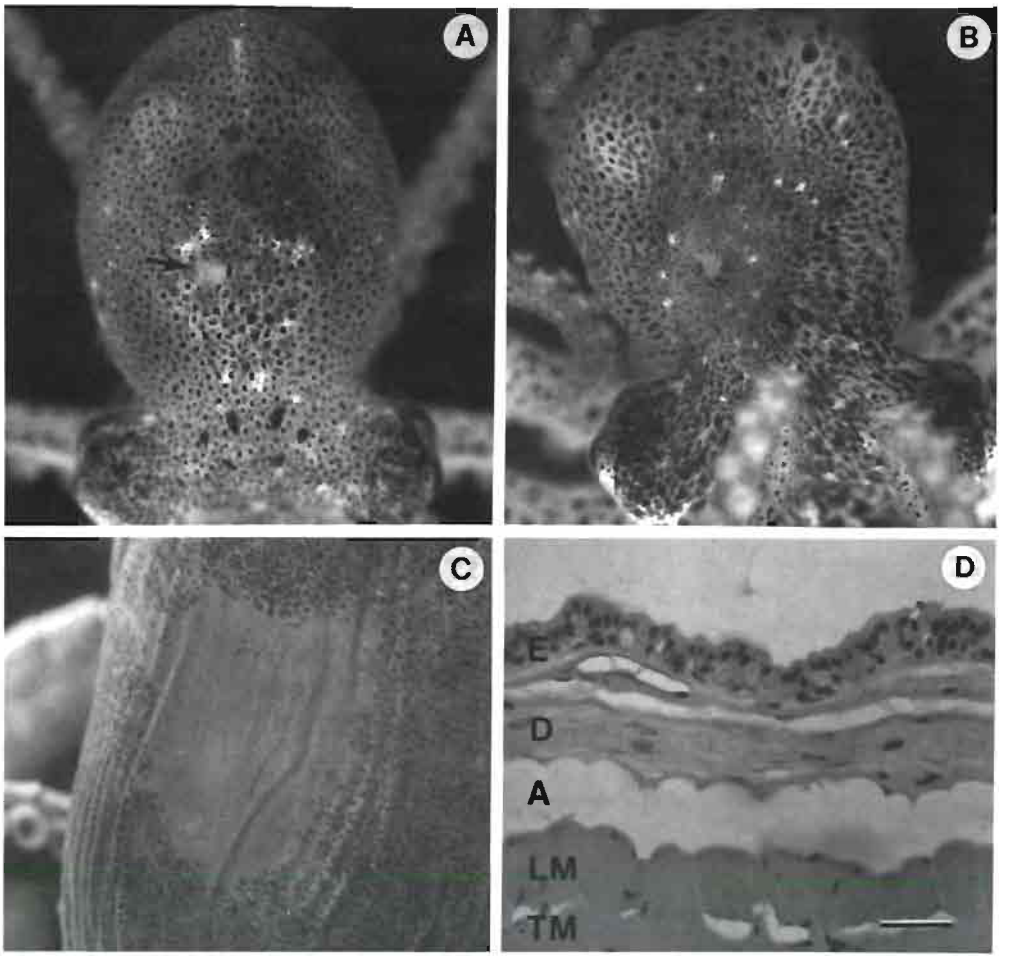


Fig. 1-5: *Octopus joubini*. A: Young octopus (6 mm ML) 5 days after small incision (arrow) was made on the mantle; compare normal distribution of chromatophores on this control individual with that on the individual in Image B. B: Stage 1 ulcer spreading from induced lesion 3 days after inoculation with the bacterium *Vibrio alginolyticus*. C: Healed ulcer with intact epidermis, chromatophores have not yet regenerated. D: LM section of partially healed ulcer in Image B; (E) epidermis, (D) dermis, (A) artifact, (LM) longitudinal muscle, (TM) transverse muscle. Haemotoxylin and Eosin stain; bar = 20 µm. (After Hanlon and co-authors, 1984.)

An apparent systemic infection caused by *Vibrio carchariae* has recently been observed by us (unpubl.) in *Octopus bimaculoides* and *O. maya* in laboratory culture. This disease was characterized by the sudden death of octopuses, with no visible external or behavioral symptoms in the 12 to 18 h preceding death. In some cases, octopuses fed normally within 8 h of death. Dead octopuses typically had no external damage or only a few (< 5) very small (< 3 mm diameter) skin lesions, judged to be too small to cause death. In about half of the mortalities, the octopus autotamized 1 or 2 arms at their midpoint in the last few hours prior to death. On moribund octopuses without such autophagy, palpation of the arms (normally firm due to muscle tone) invariably revealed 1 or 2 arms having a short soft section in the mid-region. Histological examination of tissue from near

Table 1-3
Octopus joubini. Treatment of skin ulcers (After Hanlon and co-authors, 1984)

Medication		Concentration (mg/liter)	Duration (days)	No. treated	Mortality (%)	Comments
Individual containers						
Control		—	—	—	100	Death within 4 days
Penicillin and chloramphenicol	1 hr/b.i.d.[a]	500/100	6	31	74	No improvement
Chloramphenicol	Continuous	13–16	19	20	45	Very slight improvement in 30 %
Minocycline hydrochloride	Continuous	0.53	20	17	29	Slight improvement in 71 %
Nifurpirinol	3 min/day	1.0[b]	4	28	18	Near-complete healing in 73 %
	3 min/b.i.d.	1.5[b]	33			
	7 min/day	2.0[b]	22			
	10 min/2 days	2.0[b]	23			
Group trays						
Nifurpirinol	5 min/day	1.5[b]	20	248	41	No individual evaluations
	5 min/day	2.0[b]	26			
	10 min/day	2.0[b]	28			

[a] b.i.d. twice a day.
[b] Actual concentrations of nifurpirinol, even though treatment baths were prepared with Furanace granules that contained 10 % nifurpirinol.

the stumps or soft areas of intact arms revealed heavy proliferation of rod-shaped bacteria (Fig. 1-6, A). Aseptically excised internal tissue swabbed on TCBS agar grew exclusively *V. carchariae*. Further histological and microbiological examinations revealed this same bacterium in branchial hearts and kidneys. All afflicted octopuses were at least 6 months old and from individually-cultured laboratory populations having no physical animal-to-animal contact. Furthermore, these octopuses were on diets consisting predominantly of fish and penaeid shrimps frozen after field collection.

Vibrio carchariae has previously been implicated as a food-borne pathogen of fishes being fed frozen shrimp (Nelson Herwig, Curator Houston Aquarium, pers. comm., 1988) and this seems to be the most likely route of infection in this case. This bacterial infection was highly sensitive to chloramphenicol injected into live or frozen penaeid shrimp at a dose of 150 mg kg^{-1} body weight of octopuses once a day for 7 to 10 days. No mortalities occurred while octopuses were on this therapeutic protocol; however, within 1 to 3 weeks of stopping this treatment, subsequent mortalities sometimes occurred, requiring reinstitution of drug therapy.

Wild-caught Squids Maintained in the Laboratory

Over the past 40 years, wild loliginid squids have increasingly been collected live for use in biomedical research. This group of squids possesses extremely delicate skin and often incurs severe skin damage during collection, transport to holding facilities and final

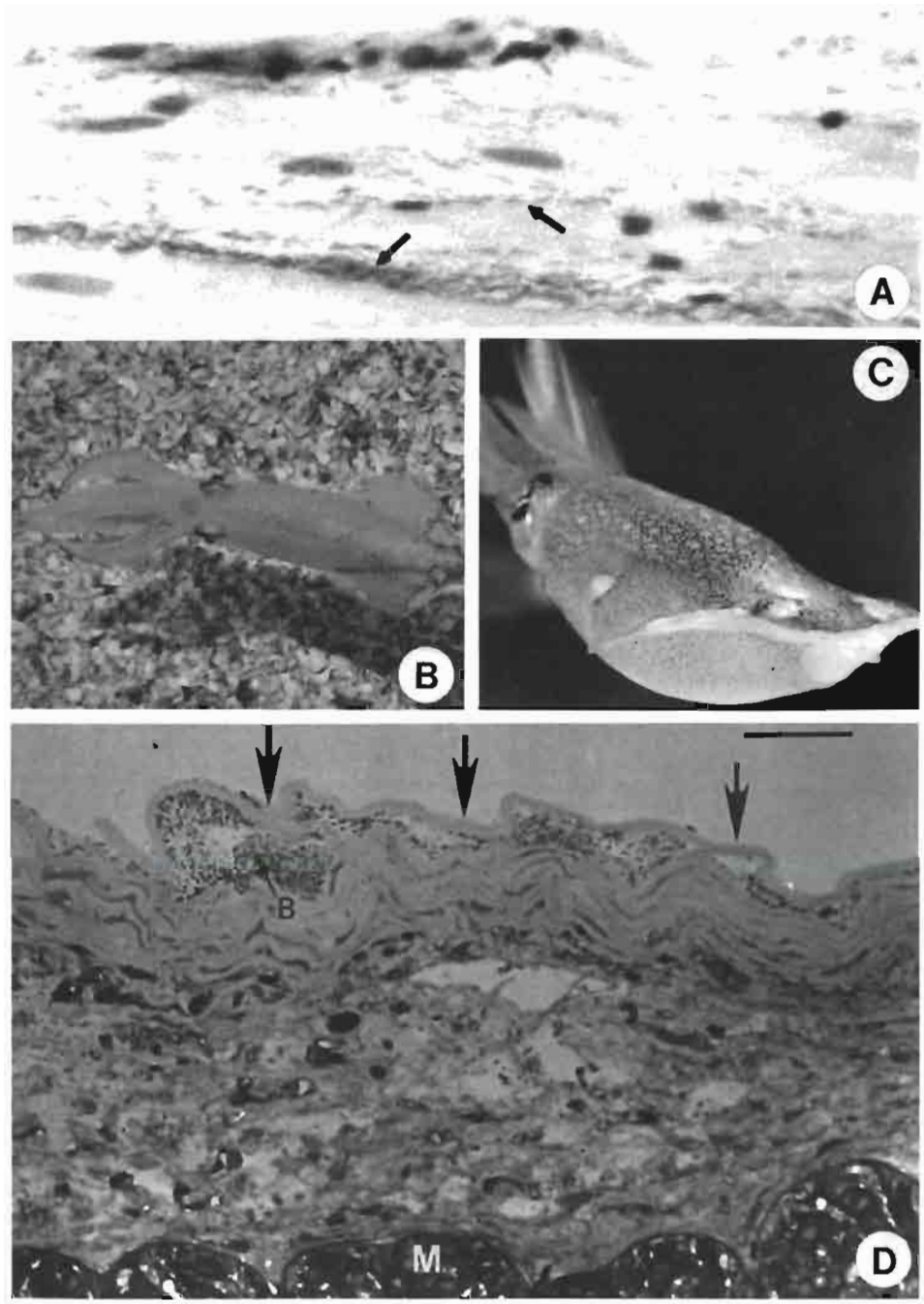


Fig. 1-6: *Vibrio carchariae*. A: Transverse rows of bacteria (arrows) in arm muscle tissue of *Octopus bimaculoides;* Geimsa stain; × 1000 (Original). *Loligo pealei*. B: Fairly severe fin damage incurred initially from transport: C: Same individual in Image B showing amount of damage on posterior fin and ventral mantle that resulted from hitting transport-tank walls (after Hanlon and co-authors, 1983.). *Ommastrephes pteropus*. D: Section through dorsal surface of abraded fin on an adult squid fixed 72 h after capture; epidermis is completely absent and arrows indicate exposed surface of the underlying dermis; bacteria (B) are multiplying in the dermis and in adjacent muscle tissue (M). Paragon polychrome stain. Line = 57 μm. (After Hulet and co-authors, 1979.)

transfer to holding tanks. Most squids die within 72 h of collection (Hanlon and co-authors, 1983) with mortalities attributed primarily to extensive skin damage to the fins (Fig. 1-6, B, C). Leibovitz and co-authors (1977), O'Dor (1978) and Hulet and co-authors (1979) showed that skin and fin damage are highly susceptible to secondary bacterial infections.

Leibovitz and co-authors (1977) described a progressive necrotic exfoliative dermatitis in *Loligo pealei.* The first stage was an acute necrotizing dermatitis characterized by epithelial necrosis and desquamation. Amoebocyte infiltration of inner and outer connective tissue layers was prominent in some areas and accompanied by edema and stretching of the surface epithelium. The second stage was a chronic necrotizing ulcerative dermatitis with deeper exfoliation of skin layers resulting in total loss of the dermis containing the iridophore and chromatophore structures. At the base of ulcers, collagen infiltrated with densely packed chains of uniform long bacterial rods was frequently encountered. The third stage of the lesions was a necrotizing bacterial myositis in the total absence of all skin layers. Chains of bacteria were found infiltrating deep into muscle tissue, particularly along the transverse septa of muscle bundles. Microbiological sampling of the skin of healthy and lesioned squids produced 4 colony types, 2 of which were Gram-negative rods that failed to transfer during isolation attempts and were not identified. The third bacterium was an enterobacterium that failed to conform to biochemical characteristics of any known enterobacteria species. *Vibrio anguillarum* was the fourth species. Numerous squids were autopsied, including healthy individuals, animals that died for no apparent cause and severely lesioned individuals. Aside from skin lesions, no other marked changes were observed in internal organs. The authors did not establish the etiologic agent of the disease but mentioned that Gram-negative filamentous bacterial rods in tissue sections resembled morphology and organization of *Myxobacteria* spp. They did not suggest any disease-causing role for *Vibrio anguillarum* cultured from all squids.

Hulet and co-authors (1979) and Hanlon and co-authors (1983) also found fin damage to be the major factor in the mortality of wild-caught squids *Loligo pealei, L. plei, Lolliguncula brevis* and *Ommastrephes pteropus,* and of *Loligo opalescens* hatchlings cultured from eggs in the laboratory. In all wild-caught species, histological examination of damaged fins showed the presence of large numbers of Gram-negative rods in the tissue (Fig. 1-6, D). The lesions resembled in almost all respects the pathological changes described by Leibovitz and co-authors (1977) in *L. pealei.* Ford and co-authors (1986), studying the bay squid *Lolliguncula brevis,* examined the external bacterial populations of normal skin on wild and laboratory-maintained squids versus ulcerated skin on laboratory-maintained individuals (Fig. 1-7, A). They found that laboratory-maintained squids had 100 times more viable cells cm^{-2} than wild squids (Table 1-1), although the wild squids had a slightly higher species diversity (Table 1-2). Due to large variations in bacterial counts (see standard deviations in Table 1-1), there was no statistically significant difference in numbers of viable cells from normal skin versus ulcerated skin of laboratory-maintained squids. However, on 2 of 3 media types, the mean number of viable bacteria was always higher on ulcerated skin. Microscopic observations using light, transmission and scanning electron microscopy consistently revealed large numbers of bacteria in ulcerated skin and muscle versus few on healthy skin and none in normal healthy tissue. Overall their results supported the contention that skin ulcers are not induced by bacteria but rather opportunistically invaded by bacteria subsequent to injury. This potential for bacterial invasion

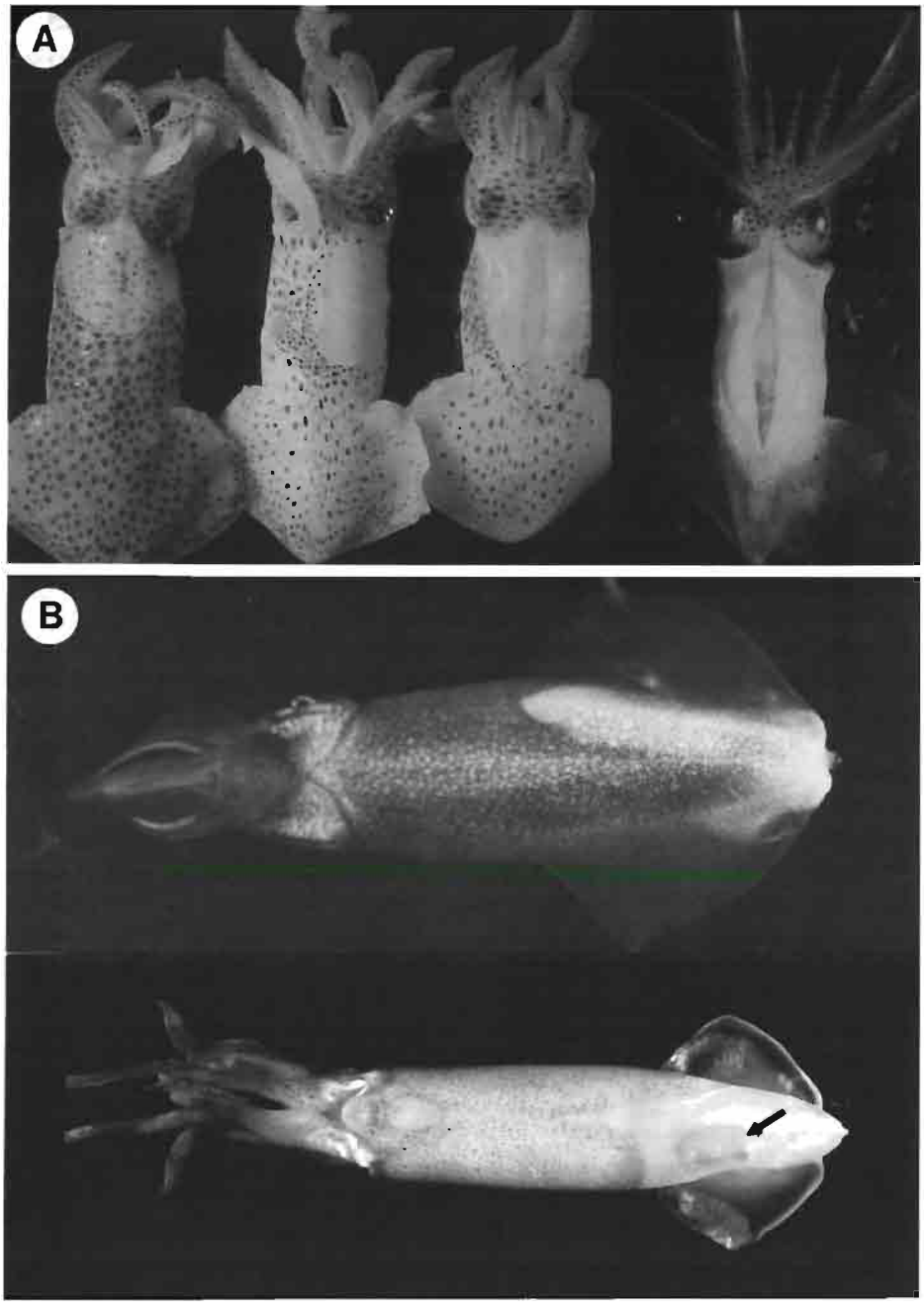


Fig. 1-7: Bacterial infections of loliginid squids. A: Preserved *Lolliguncula brevis* displaying a range of increasing skin ulcer severity (left to right); mantle muscle of the 2 squids on the right has eroded to the point where the mantle has split, revealing the pen (after Ford and co-authors, 1986.). B: Upper, cultured *Loligo forbesi* showing internal abscess (longitudinal white area in posterior mantle) resulting from infection by *Vibrio alginolyticus;* lower, ventral side of cultured *L. opalescens* showing how repeated bottom contact allowed secondary bacterial infections to initiate and spread; arrow: hole abraded through mantle. (Original.)

of a skin wound is exacerbated by the proliferation of potentially pathogenic species of *Pseudomonas, Aeromonas* and *Vibrio* in the laboratory environment.

Laboratory Cultured Squids

Squids reared successfully through their life cycle incur fin and mantle trauma from long confinement in the laboratory. In describing 4 major culture experiments with *Loligo forbesi* (1 experiment lasting 16 months), Hanlon and co-authors (1989) noted that many squids accrued fin and mantle skin damage from occasionally hitting tank walls over long periods of time. In squids over 3 months of age, 30 to 50 % of mortalities had chronic fin and distal mantle tip damage (Fig. 1-7, B). Bacterial sampling showed 5 *Vibrio* and 3 *Pseudomonas* species associated with necrotic tissue. Exposure to 0.2 mg/l nifurpirinol (active ingredient) was 100 % lethal within 24 h to *L. forbesi* suffering severe tail infections. A small number of squids had abcesses or infections of one or both eyes, but no further information was given.

Additional (although unpublished) information is now available related to these cases of eye damage reported by Hanlon and co-authors (1989). In one condition, *Loligo forbesi* were seen with one eye swollen much larger than the other (Fig. 1-8, C). Apparently the cause of swelling was a buildup of humor in the affected eye. Necropsy of one of these individuals revealed the fluid content of the posterior eye chamber to be slightly yellow as opposed to clear in the normal eye. The cornea of the swollen eye remained clear while the lens was white and nearly opaque (Fig. 1-8, A, B). *Micrococcus* sp. was cultured from vitreous humor, posterior lens surface and hemolymph of affected squids. It was not cultured from squids in the same tanks with typical mantle tip and fin damage but normal eyes. Other specimens of *L. forbesi* were observed with cloudy-to-opaque corneal tissue as well as opaque lenses. Typically there was only unilateral involvement, and the affected eye swelled to a larger size. It was believed this damage was initiated through mechanical abrasion of the external surface of the cornea with subsequent bacterial infection. In some cases prior to death, a large opening appeared in the cornea (Fig. 1-8, D), allowing direct contact of the sea water with the lens.

More recently, the Pacific oval squid *Sepioteuthis lessoniana* was observed to have bacterial infections of the eye, primarily involving damage to the cornea. In one individual bilateral, though unequal, involvement was seen. One eye had a slightly whitish clouding of the cornea. Aspiration cultures of the posterior eye chamber yielded a pure culture of *Vibrio harveyi.* The other eye was more severely damaged with a part of the cornea actually missing and the lens opaque. Aspiration cultures of the posterior chamber of this eye produced *V. anguillarum* and *V. carchariae.*

A case of ovarian infection was also observed in laboratory-cultured *Sepioteuthis lessoniana.* A large mature female was caught for neurophysiological experimentation. Upon dissection, the fully mature ovary was noted to have numerous opaque ova interspersed with the normal clear ova (Fig. 1-8, E). Clear and opaque eggs were aseptically removed from the ovary and cultured separately. Both egg samples cultured *Vibrio splendidus* (Biogroup 1) while the opaque eggs also harbored a species of *Pseudomonas.*

Laboratory Cultured Cuttlefishes (Order Sepioidea)

We are aware of no publications or reports dealing with bacterial diseases in wild or captured cuttlefishes. However, during the past 3 years we have cultured several hundred

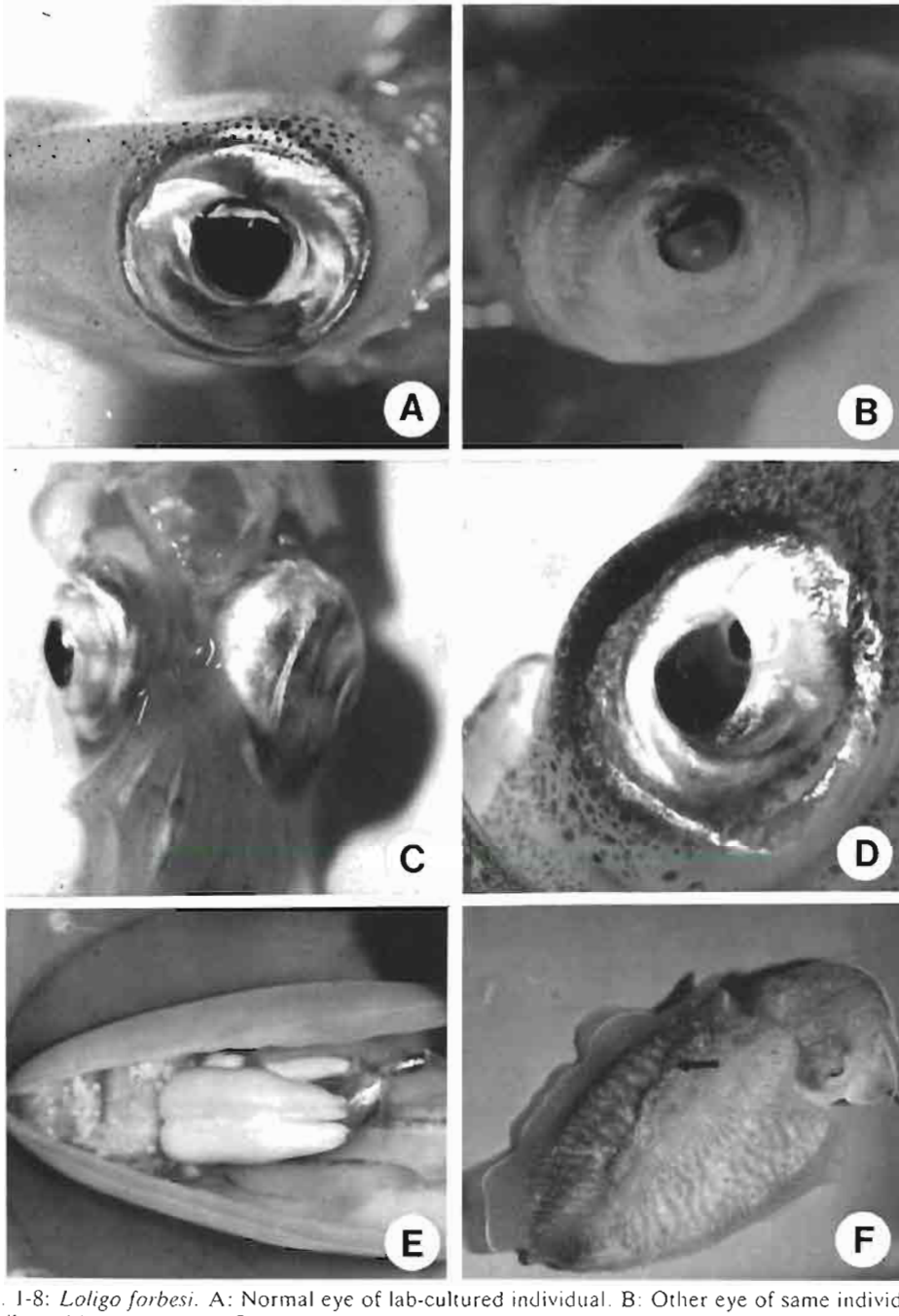


Fig. 1-8: *Loligo forbesi*. A: Normal eye of lab-cultured individual. B: Other eye of same individual, swollen with opaque lens. C: Ventral view of individual above showing normal versus swollen eye. D: Individual with hole through cornea. *Sepioteuthis lessoniana*. E: Ventral view of opened mantle cavity revealing 2 very large, normal white nidamental glands and, posterior to them (left), various dead embryos (white) amidst normal translucent embryos in the ovary. *Sepia officinalis*. F: Lab-cultured adult showing longitudinal ridge-like haematoma (arrow) along the mantle; systemic bacterial infection suspected. (Original.)

cuttlefish of 2 species through the life cycle to sexual maturation and spawning. We have encountered one minor and one major disease condition. The primary species cultured was the European cuttlefish *Sepia officinalis;* only 15 *S. latimanus* from the southwestern Pacific were reared to maturity. Both species incurred mantle-tip damage due to collisions with tank walls. However, *S. latimanus* was far more prone to this type of injury often sustaining major chronic mantle-tip lesions. These lesions persisted for up to 2 months. Secondary bacterial infections of this exposed tissue were highly suspected although no bacterial sampling was conducted. A group of 4 adult individuals with extremely severe damage was isolated and given daily intramuscular injections of chloramphenicol (75 mg kg^{-1} body weight) directly into the inflamed tissue. In 3 of the 4 cuttlefish, epidermis gradually migrated back over the injured area in about 3 weeks. The fourth individual died after 4 days of injections. *S. officinalis* rarely incurs such severe tail damage and it is not a significant problem.

Sepia officinalis has been susceptible to a highly virulent systemic infection which does not appear to be related to external injury. The first sign of this disease is a long narrow ridge, 5 to 10 cm long, running along the dorsal surface of the mantle. The dorsal epidermis, dermis and muscle layer over the cuttlebone is extremely thin (less than 2 mm thick) in an adult individual and the appearance of these ridges is quite prominent. In normal healthy cuttlefish, several large blood vessels traverse the length of this dorsal skin layer, and it is possible that the swelling ridges follow the path of these blood vessels. Within 12 to 18 h of the initial appearance of the ridge, it becomes a fairly continuous swollen haematoma, blue in color due to large amounts of oxygenated haemocyanin (Fig. 1-8, F). In the following 12 to 24 h there is massive destruction of the dorsal epidermis with death occurring at this point. Only rarely is the ventral mantle skin and musculature involved. This suggests an internal infection of the dorsal blood vessels which eventually swell and rupture allowing the causative agent to spread quickly through the dermis but beneath the epidermis. Smears of hemolymph from moribund cuttlefish produced 3 isolates, *Vibrio pelagius* (Biogroup II), *V. splendidus* and *Pseudomonas stutzeri.* The condition is epizootic and will result in over 90 % mortality of an infected population in less than 1 week without therapeutic intervention. Both chloramphenicol (40 mg kg^{-1}) and gentamycin (20 mg kg^{-1}) administered by intramuscular injection or via food have been highly effective in halting progression of the disease. If antibiotics are given immediately when the first sign of ridge swelling appears, symptoms will disappear completely within 18 h (overnight). Once signs of haematoma appear, intramuscular injections for 2 days preceding oral administration of antibiotics are necessary to assure survival. When injected, chloramphenicol and gentamycin concentrations should not exceed 100 mg ml^{-1} and 40 mg ml^{-1} respectively to avoid excessive tissue toxicity at injection sites. A single injection is usually divided among 4 sites, often 4 different arm bases, to avoid tissue trauma.

Our knowledge of bacterial diseases in cephalopods is primitive. Only one study (Hanlon and co-authors, 1983) has attempted to reinfect healthy individuals with a suspected pathogen isolated from sick cephalopods to prove causation. All other studies have provided only circumstantial evidence pointing to a probable disease agent. Elston (1984), in reviewing the status of disease prevention and management in intensive mollusc husbandry, states that vibriosis is the most significant disease encountered in bivalve and gastropod molluscs. Experience to date indicates the same is true for cephalopods. Of the

36 bacteria species the authors have found associated with diseased cephalopods, over half have been of the genus *Vibrio* (Table 1-4). Some combination of events regularly occurs when molluscs are intensively cultured in captivity that causes or heightens the virulence of certain commonly occurring *Vibrio* species while impairing or diminishing the resistance to infection of the mollusc species in question. The degree to which this sequence of events is eventually understood will bear heavily on the long-term success of cephalopod mariculture.

Table 1-4
Bacteria isolated by the authors from diseased octopusesm, squids and cuttlefish in culture at the cephalopod biology laboratory of the Marine Biomedical Institute. The bacteria were cultured from 1: skin; 2: muscle; 3: haemolymph; 4: eye; or 5: ovary (Original)

Bacteria	Octopuses	Squids	Cuttlefish
Acinetobacter lwoffi		1	
Aeromonas caviae	1		
A. hydrophila	1		
A. sobria		1	
Alcaligenes faecalis		1	
Cytophaga sp.	1	1,3	
Flavobacterium sp.	1		
Klebsiella pneumoniae	1		
Plesiomonas shigelloides		3	
Proteus sp.		1	
Pseudomonas alcaligenes		1	
P. diminuta		1,3	
P. maltophilia		1	
P. putrifaciens	1	1	
P. vesicularis		1,3	
P. stutzeri	1		3
Vibrio alginolyticus	1	1,3	
V. anguillarum	1	4	
V. carchariae	2,3	1,3,4	
V. cambelli		1,3	
V. costicola	1		
V. cholerae (non-01)	1		
V. damsela	1	1,3	
V. fluvialis	1	1	
V. harveyi	3	4	
V. hollisae		1,3	
V. mediterranei		1	
V. metschnikovii		1	
V. mimicus		1	
V. natriegenes		1	
V. ordalii		1	
V. parahaemolyticus	1	1,3	3
V. pelagius (biovar 2)	1	1	3
V. splendidus (biovar 2)		1,4,5	3
V. tubiashi		1,3	
V. vulnificus		1	

Agents: Fungi and Labyrinthomorpha ('Fungus-like Protistans')

The only mycotic occurrence reported in the literature is that by Polglase and co-authors (1984) on skin wounds in the octopus *Eledone cirrhosa*. They noted mycelia emanating from a skin wound made (by them, for other studies) 52 days earlier (Fig. 1-9, A). These mycelia were growing within the underlying dermis (Fig. 1-9, B). The fungus was isolated and cultured, and they were able to reinoculate 2 of 3 octopuses and to reisolate the fungus from a total of 4 reinoculation sites. The fungus was identified as *Cladosporium sphaerospermum* Penz. At the inoculation site, mycelia were growing into the surrounding dermal tissue (Fig. 1-9, C). These authors were careful to point out that they were unsure whether this organism was a pathogen or merely an opportunistic saprophyte. They also stress that the genus *Cladosporium* consists mainly of terrestrial species, although one species is authentically marine, and they found records from the Commonwealth Mycological Institute in the UK verifying that *C. sphaerospermum* has been isolated from seawater.

There are 2 reports of thraustochytrid-like organisms causing mortality in field-collected cephalopods. The taxonomic position of the labyrinthulids is uncertain and has been reviewed elsewhere in this series (Lauckner, 1983); currently it appears acceptable to consider them as 'fungus-like' protistans although they show similarities with fungi, protozoans and even algae.

Polglase (1980) described progressive ulceration of the skin of the octopus *Eledone cirrhosa* collected near St. Andrews (Scotland) and maintained in flow-through seawater systems. Octopuses were collected consistently from 1976 through 1980 and, despite stringent cleaning of the tanks, it was impossible to keep them alive more than a week or so. This must have been a fairly restricted phenomenon since Boyle (1983) reported that the same species collected regularly near Aberdeen (Scotland) never exhibited this malady. Polglase cited a personal communication from J. B. Messenger that a similar condition had been observed occasionally in *Octopus vulgaris* collected in the Bay of Naples (Italy) and maintained in the Stazione Zoologica. Lesions could be manifest on any part of the external skin, and they generally appeared first as grey patches of inactivated chromatophores followed by progressively larger and whiter patches in which the entire epidermis and dermis were missing (Fig. 1-10, A, D, E). The number and size of ulcers increased and led to death within 2 or 3 days. Sickened octopuses often ran their arms over their body continually and in some cases shook their arms violently in advanced stages of ulceration. Examination of fresh and fixed scrapes of the lesions revealed a variety of organisms, including ciliates and bacteria, but only one type of organism was present in all scrapes from diseased tissue and absent in normal tissue. Silver staining showed rounded cells, about 6 μm diameter, with a large nucleus and conspicuous nucleolus; these cells were commonly seen as tetrads but were also present as diads and multiples of each (Fig. 1-10, B). From light and electron microscopy, that organism was suspected to be a thraustochytrid but this was not confirmed. One other organism was found in a large number of scrapes — it had fusiform cells with lightly staining, non-cellular envelopes in silver preparations (Fig. 1-10, C). In fresh smears, these ". . . could be seen gliding one after the other across the field of the microscope. Clusters of the organisms were also seen" (Polglase, 1980; p. 703). Morphology and behavior indicated to Polglase (1980) that these were similar to *Labyrinthula* sp.

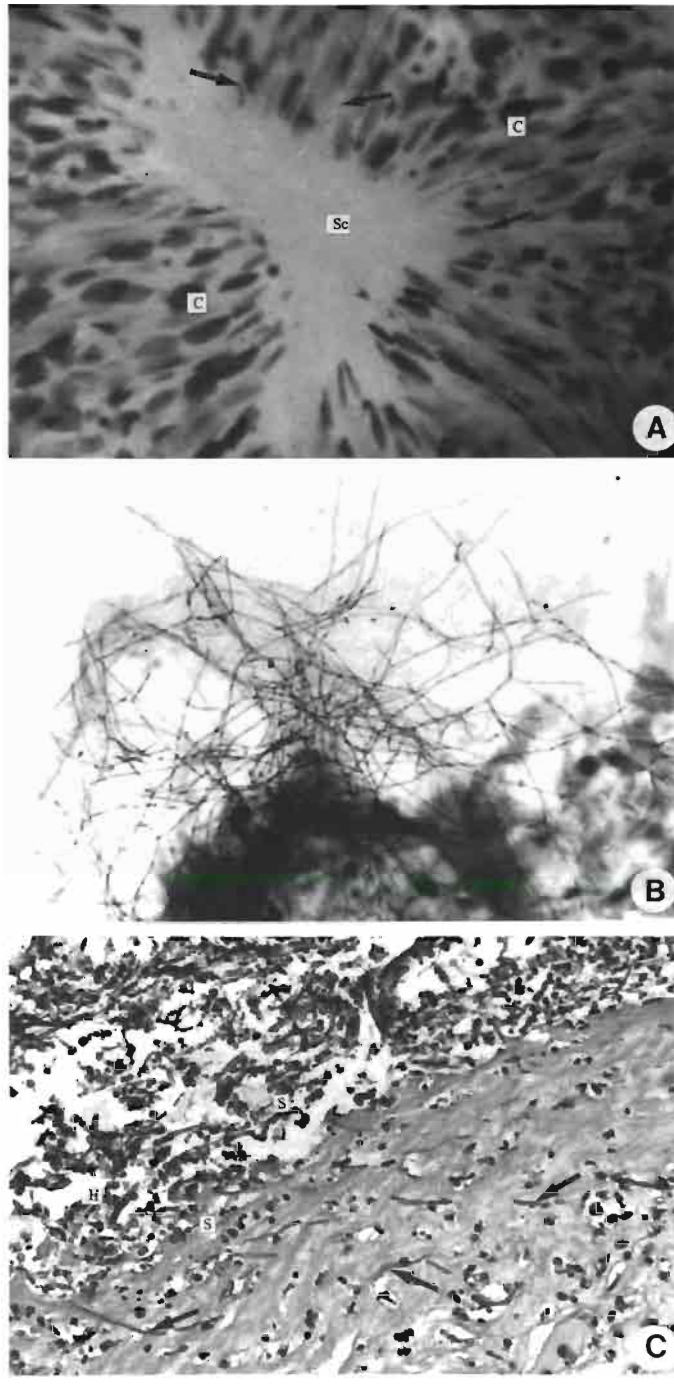


Fig. 1-9: *Eledone cirrhosa*. A: Infected skin wound with growth of mycelium (arrows) on external surface; (C) chromatophores. (Sc) scar tissue (× 75). B: Section of wound illustrated in A showing fungal growth above and in the upper layer of the dermis (× 125). C: Section of reinoculation site revealing original spores (S) and hyphae (H); new hyphae (arrows) growing with the surrounding tissue (× 320). (After Polglase and co-authors, 1984.)

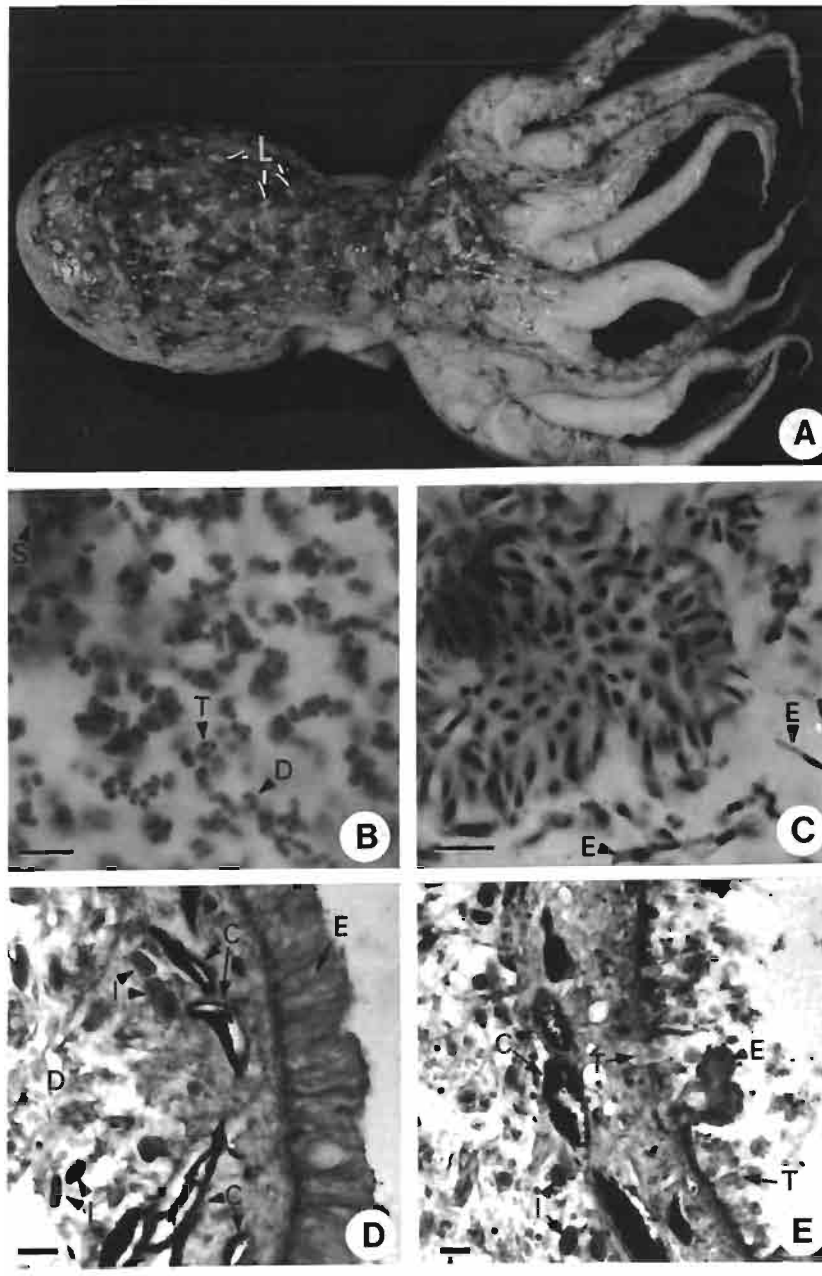


Fig. 1-10: *Eledone cirrhosa*. A: Adult with lesions (L) over entire body from thraustochytrids. B: Silver-stained smear preparation showing presumed thraustochytrids present as single-cells (S), diads (D) and tetrads (T), bar = 20 μm. C: Silver-stained smear preparation showing a cluster of *Labyrinthula*-like organisms; note tenuous, non-cellular envelope (E) surrounding the cells, bar = 20 μm. D: Normal undamaged epithelium (E) and underlying dermis (D) with chromatophores (C) and iridophores (I), Mallory's triple stain, bar = 20 μm. E: Section from center of lesion; note almost complete absence of epithelium (E) and damage to underlying structures; presumed thraustochytrids (T) associated with surface of epidermis and deeper in tissue, (C) chromatophores, (I) iridophores, Mallory's triple stain, bar = 20 μm. (After Polglase, 1980.)

Jones and O'Dor (1983) described gill lesions in squids *(Illex illecebrosus)* maintained in a very large seawater system after having been collected off the coast of Nova Scotia (Canada). In 1979, in particular, extensive gill lesions were considered to be the main cause of delayed mortality in the laboratory population, although lesions were not found in squids in the wild. Males and females were susceptible and the condition had a high degree of fatality. The lesions appeared as white patches (1.5 mm wide) scattered over the whole gill (Fig. 1-11, A, B). Light and electron microscopy indicated a single organism type within lesions. Numerous spherical cells up to 7 μm in diameter were prominent and appeared as diads, triads or tetrads (Fig. 1-11, C, D). Ultrastructural characteristics indicated that this organism was a thraustrochytrid but the authors warn that isolation and pure culture are necessary to verify its identity. Where large numbers were present, considerable epidermal and connective tissue damage was apparent (Fig. 1-11, C). In a typical pattern of host-tissue damage, it was apparent that the ectoplasmic net (and not the thallus) caused marked damage and even obliteration of cell structure. Lytic damage was confined to the area occupied by ectoplasmic net profiles (Fig. 1-12, A, B). Bacteria were common outside the gill but never inside gill tissue even in regions of maximum damage and disruption of the epithelium. Jones and O'Dor noted that the process of lysis and digestion was similar to that described by Perkins (1973). There was little evidence of a host response other than the occasional presence of amoebocytes, i.e., no encapsulation of the invading organism was observed. Jones and O'Dor suggest that these squids suffered from reduced resistance to infection due to general laboratory stress or to the onset of sexual maturation, during which time cephalopods are known to experience decreased ability for wound repair and regeneration of somatic tissue (O'Dor and Wells, 1978).

Pathologies of Unknown Etiology

Boyle (1981) described skin abrasions and 'bruising' in field-collected *Eledone cirrhosa* that led to death of many individuals from an unknown agent. Octopuses maintained for long periods in aquaria also developed skin lesions at the apex of the mantle that could increase rapidly and lead to death from unknown reasons.

Several eye conditions have been observed among cultured octopus that were not epidemic and because of logistical limitations were not closely studied. *Octopus joubini* has periodically suffered unilateral swelling and rupture of the posterior chamber of an eye (Fig. 1-13, A). This condition appeared suddenly and the octopuses died within 24 h of rupture; damage or opacity of cornea and lens were never noted. *O. maya* occasionally develops opaque lenses (Fig. 1-13, B) both unilaterally and bilaterally while the cornea remains normal and clear. Such afflicted octopuses live for months with this condition. When the condition is bilateral the functionally blind octopuses apparently feed by tactile means.

Individuals of *Octopus joubini* and *O. maya* have occasionally shown severe edema of mantle and arms where epidermis and dermis are separated from the underlying muscle layers. In several *O. joubini* the entire dermis of the mantle was separated from the muscle layers by a watery, almost gelatinous, layer of fluid. A similar condition has been seen on arms and arm bases of *O. maya*. In both species the condition is fatal within 48 h.

In most laboratory populations of *Octopus maya* a few individuals, at an age of 6 to 8 months, take on a unique static (or fixed) skin pattern (Fig. 1-13, C) markedly different from the normal dynamic color-pattern repertoire of this species. Octopuses showing this

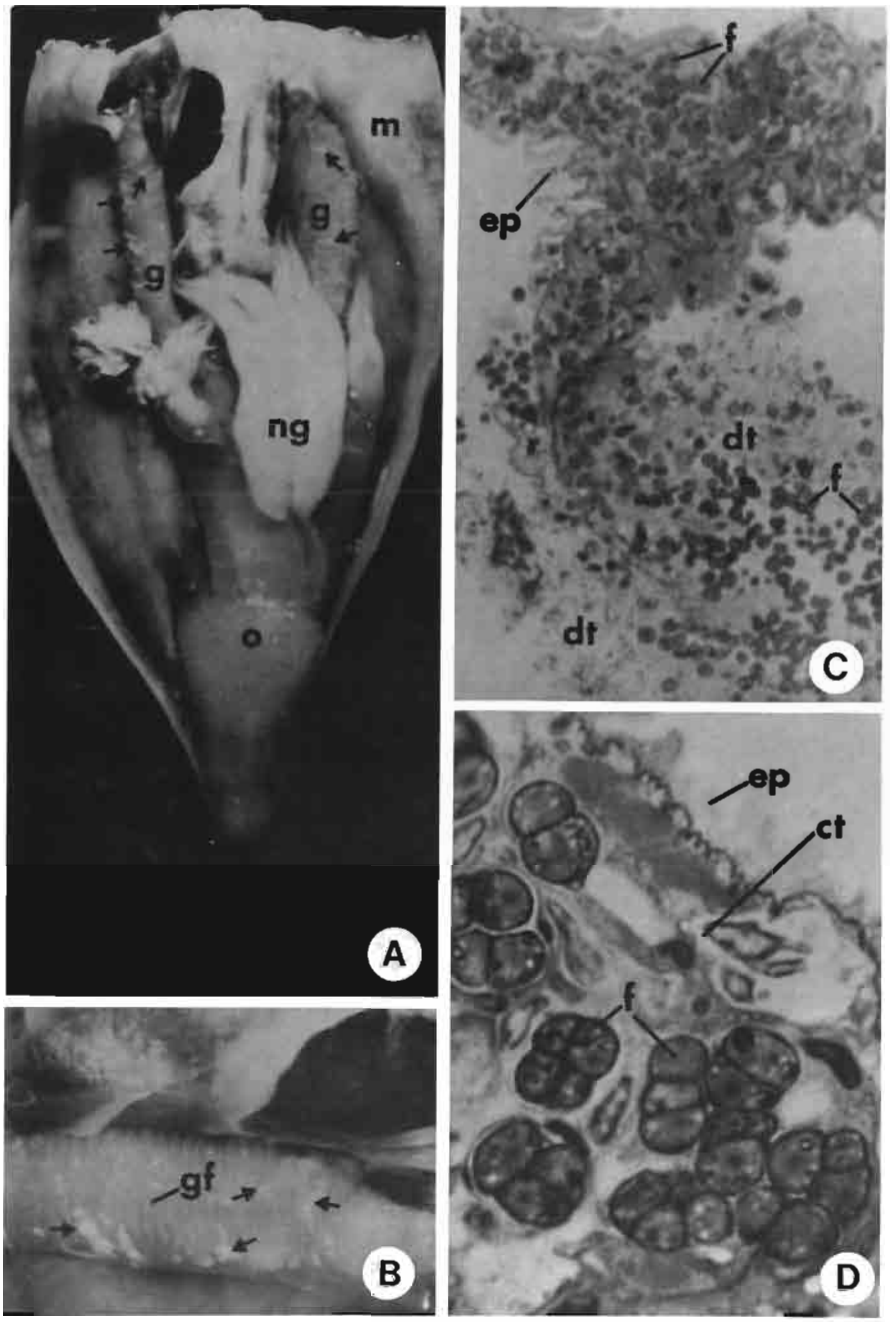


Fig. 1-11: *Illex illecebrosus*. A: Ventral view of cut-open mantle: note lesions (arrows) on gills (g) in a moderately infected female: (m) mantle, (ng) nidamental gland, (o) ovary (ML = 20 cm). B: Close-up of gill showing white thraustochytrid parasites in the gill filaments (gf). C: Section of gill filaments through lesioned area (1 μm section): numerous thraustochytrids (f) occur beneath the epithelium (ep); in places the gill tissue is disrupted and thraustochytrids and cell debris spill out into the mantle cavity: (dt) disrupted tissue, bar = 25 μm. D: Thalli (f) in gill connective tissue (ct): note diads, triads and tetrads of the thraustochytrid cells; (ep) epithelium, bar = 5 μm. (After Jones and O'Dor, 1983.)

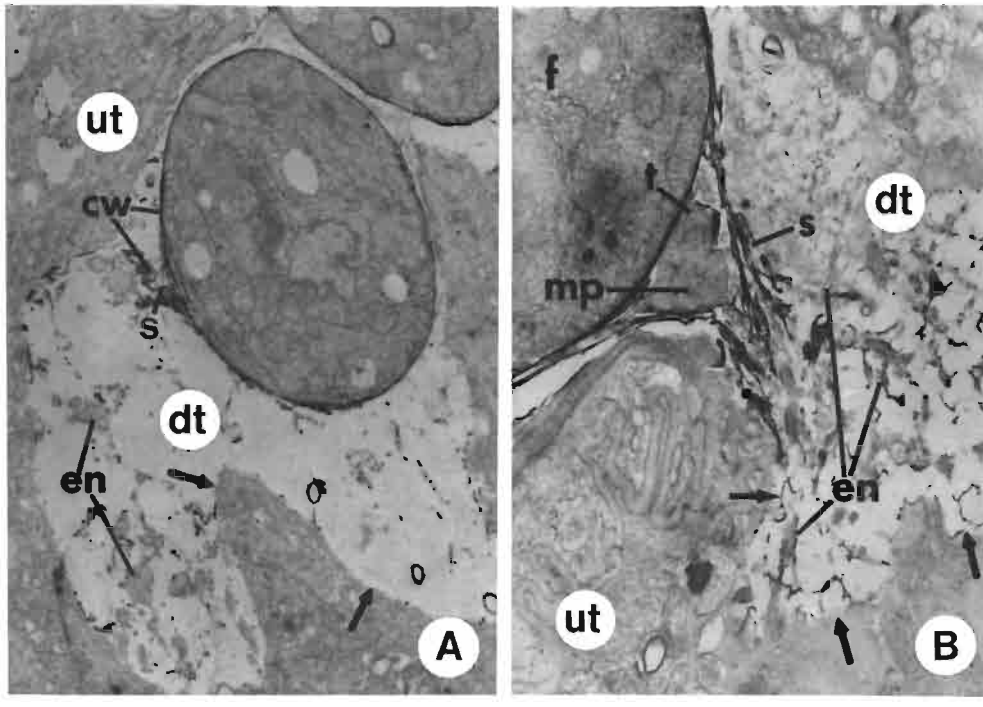


Fig. 1-12: *Illex illecebrosus*. A: Lytic damage to host cells due to thraustochytrids is confined to the area occupied by ectoplasmic net profiles (en); host cells apposed to the thallus do not show similar damage; Transition between damaged (dt) and relatively undamaged (ut) host tissue is abrupt (arrows); (cw) cell wall, (s) scale. B: Region of host-cell destruction adjacent to emerging trunk of ectoplasmic net; zone of cell damage (dt) distinct from adjacent, undamaged host tissue (ut); transition between them abrupt (arrows); (f) fungal thallus, (mp) tubular membrane profile, (s) cell wall scale, (t) trunk (× 14,000). (After Jones and O'Dor, 1983.)

pattern cease feeding and gradually become lethargic over a 3 to 4 day period and often just hang motionless on the tank walls. This condition is invariably fatal once it appears, but has never affected an entire laboratory population. There is no external skin pathology associated with this condition and because it is manifested via the nervously-controlled chromatophore system, it is probably a dysfunction at some level of the central nervous system.

Forsythe (unpubl.) observed a pathologic condition while collecting California mud-flat octopus *Octopus bimaculoides* from trap (or pot) lines in shallow nearshore areas of Los Angeles harbor near San Pedro Island (USA). The collection method was atraumatic because the octopus merely occupied one of the many clay pots or pieces of pipe attached to the line. Approximately 1 % of the octopuses collected had severe ulceration of their dorsal mantle skin and occassionally of their arms. The damage observed was not indicative of injury caused by predators but was seemingly identical to the bacterial lesioning often seen in laboratory-cultured octopuses. These observations led us to sample the bacterial fauna on the skin of this species at this location. The environment where these octopuses were collected was rather polluted, and featured a large resident cephalopod population. Most of the trap lines were in crowded marinas and anchorages with bottom

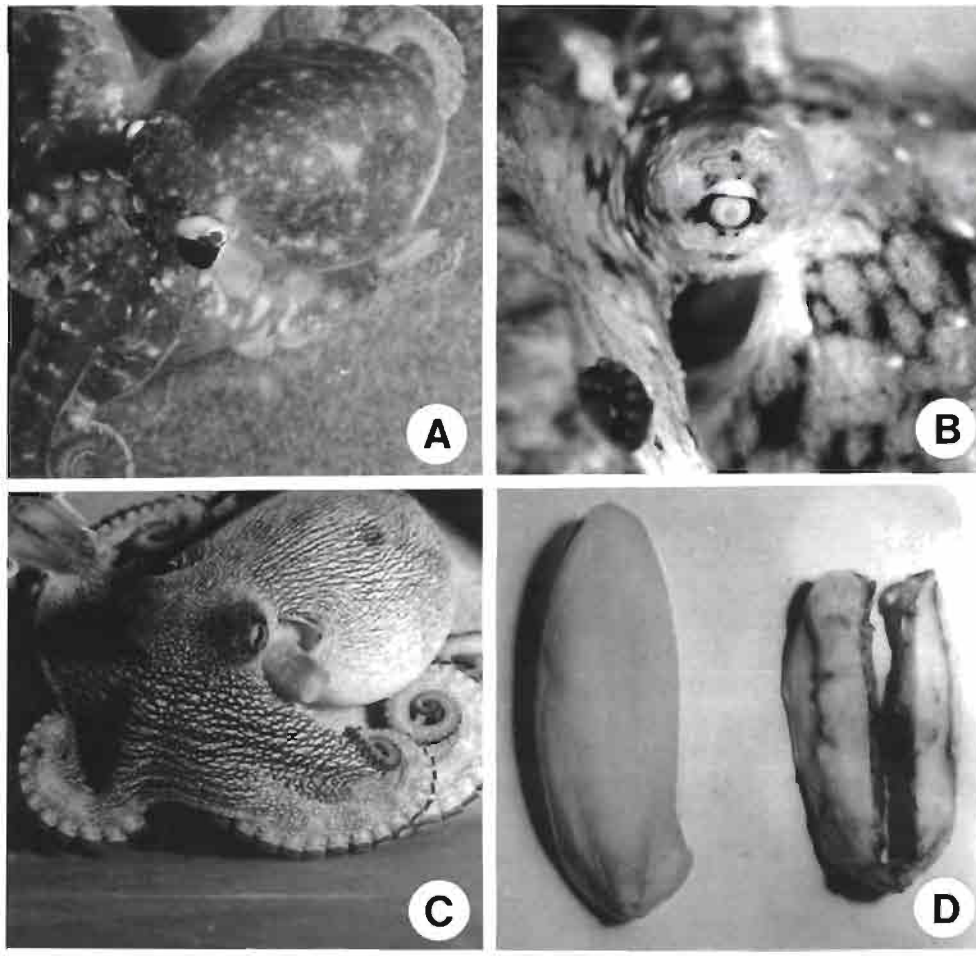


Fig. 1-13: Diseases of unknown etiology. A: Ruptured eye in an adult *Octopus joubini*. B: Opaque lens in juvenile *O. maya*. C: Static, abnormal body pattern in a diseased *O. maya*. D: Internal damage to the cuttlebone of the cuttlefish *Sepia officinalis;* normal cuttlebone (left) versus diseased one (right). (Original.)

sediment that was black, highly oxidized hydrogen sulfide mud. This population represents an ideal opportunity to evaluate how a degraded environment may affect the health of a resident cephalopod population.

Fig. 1-13, D illustrates an extraordinary case of internal infection of the cuttlebone in *Sepia officinalis*. The individual pictured was observed for 2 weeks to exhibit progressive severe swelling on its dorsal mantle, beginning at the anterior tip of the cuttlebone nearest the head and moving toward the distal mantle tip. The somewhat deformed individual became lethargic and was anesthetized and examined. Upon dissection the cuttlebone was found to have been nearly split in half along its length. No bacterial sampling was performed.

Acknowledgement. Much of the work presented in this Subchapter was conducted during grants RR 01024 and RR 01279 from the National Institute of Health.

1.2 DISEASES CAUSED BY PROTISTANS AND METAZOANS

F. G. HOCHBERG

Historically, the first known reference to a cephalopod parasite was in a book by Redi (1684). During the ensuing decades as cephalopods attracted more attention there has been a dramatic increase in the number of phyla and the number of species of parasites recorded from these molluscs. Table 1-5 summarizes the groups of parasites currently known to be associated with cephalopods. In this table are included all the published and unpublished records which could be located, in order to present as complete an overview of the diversity and distribution of parasites and hosts in which they occur as possible. The total spectrum of organisms living symbiotically on or in cephalopods is a great as that found associated with most other marine organisms. With the exception of the dicyemid mesozoans and the apostome ciliates, which are restricted to cephalopod hosts, the cephalopod parasite fauna most closely parallels the fauna present in marine fishes.

In the older literature considerable confusion exists. The identifications of the parasites and sometimes even the hosts are often in doubt. No attempt was made to resolve the many taxonomic problems that exist, especially among the larval cestodes and nematodes. In many cases the lack of adequate descriptions and figures makes it impossible to determine whether the parasite was a protozoan, mesozoan, metazoan or even part of the host. Only a very few groups of cephalopod parasites have been reviewed critically in the last 50 years, namely: chromidinid ciliates (Chatton and Lwoff, 1935; Hochberg, 1971, 1982a); dicyemid mesozoans (Nouvel, 1947, 1948; McConnaughey, 1949a, 1951; Hochberg, 1982a); and the digenetic trematodes (Overstreet and Hochberg, 1975; Gaevskaya, 1977b). Dollfus (1958) reviewed the crustaceans and helminths of cephalopods in Europe and the Mediterranean. Hochberg (1983) provided the first comprehensive review of the parasites of cephalopods.

Table 1-5 shows that with few exceptions the total picture for the parasites of cephalopods is inadequately known. To date only 63 genera and about 150 species of cephalopods have been examined for parasites. This represents fewer than half the known genera and fewer than a quarter of the approximately 650 species of cephalopods currently recognized. In only 2 cases have the total parasite loads been documented: *Sepia officinalis* and *Octopus vulgaris*. Members of several genera of squids have been studied in some detail; these include *Loligo, Illex, Ommastrephes, Sthenoteuthis*, and *Todarodes*.

Almost without exception all large, mature cephalopods are infected with parasites. Viruses, bacteria, fungi (see Subchapter 1.1), 3 phyla of protists and 6 phyla of metazoans have been recorded. Parasites have been recovered from almost all the tissues and organs of cephalopods. In general terms, however, they are most commonly located: (1) on or in the skin, (2) on the gills, (3) in the digestive tract, (4) in the 'kidneys' or excretory organs, and (5) in the musculature. The excretory organs are unusual in that they provide a uniquely suitable environment for the establishment and maintenance of parasites and as such have been exploited by a number of phylogenetically distinct groups (Hochberg, 1982a).

Table 1-5
Summary of published and unpublished records of parasites encountered in genera of cephalopod molluscs (Original)

Parasite group: / Host genera:	Virales	Bacteria	Fungi	Flagellates	Coccidians	Microsporidians	Ciliates	Dicyemids	Monogeneans	Digeneans	Cestodes	Acanthocephalans	Nematodes	Polychaetes	Hirudineans	Branchiurans	Copepods	Isopods	Decapods
SUBCLASS NAUTILOIDEA																			
Nautilus (6)																	●		
SUBCLASS COLEOIDEA																			
ORDER SEPIOIDEA																			
Spirula (1)		●					●												
**Metasepia* (3)																			
Sepia (100+)	●	●	●		●	●	●	●		●	●		●			●	●	●	
Sepiella (6)											●								
**Sepiadarium* (5)																			
**Sepioloidea* (2)																			
ORDER SEPIOLIOIDEA																			
**Austrorossia* (4)																			
Euprymna (7+)		●																	
Heteroteuthis (6)		●					○				○								
**Inioteuthis* (3)																			
**Iridoteuthis* (2)																			
**Nectoteuthis* (1)																			
**Neorossia* (1)																			
Rondeletiola (1)		●						●											
Rossia (10+)		●					○	●		●	●								
Semirossia (3)		●																	
Sepietta (3)		●					○	●											
Sepiola (12+)		●					●	●		●	●		●						
Sepiolina (1)		●																	
**Stoloteuthis* (1)																			
**Idiosepius* (6)																			
ORDER TEUTHOIDEA																			
**Pickfordiateuthis* (2)																			
Alloteuthis (3)							○		●	●	○		●				○		
Doryteuthis (6)		●											●						
Heterololigo (4)											●		●					●	
Loligo (20)		●					●	●	●	●	●		●	●			●	●	●
Loliopsis (1)										●			○						
Loliolus (3)																			
Lolliguncula (6)		●					○			●	○		●						
**Nipponololigo* (6)											○								
Photololigo (3)								●					●						
Sepioteuthis (3)		●						●											
Uroteuthis (1)		●																	
Lycoteuthis (1)											●		●						
**Nematolampas* (1)																			
**Selenoteuthis* (1)																			

Table 1-5 (continued)

Parasite group: / Host genera:	Virales	Bacteria	Fungi	Flagellates	Coccidians	Microsporidians	Ciliates	Dicyemids	Monogeneans	Digeneans	Cestodes	Acanthocephalans	Nematodes	Polychaetes	Hirudineans	Branchiurans	Copepods	Isopods	Decapods
ORDER TEUTHOIDEA																			
**Lampadioteuthis* (1)																			
Abralia (17)							○				○		○						
Abraliopsis (10)							○			●	○		○					○	
Ancistrochirus (1)																			
**Enigmoteuthis* (1)																			
Enoploteuthis (10)							○				○		○						
Pterygioteuthis (3)							●			●	○		○						
Pyroteuthis (2)							○												
Thelidioteuthis (1)							○			○									
**Watasenia* (1)																			
Octopoteuthis (6)							○				○								
**Taningia* (2)																			
**Ancistroteuthis* (1)																			
Kondakovia (1)											●								
Moroteuthis (7)							○				●		●						
Onychoteuthis (3)							○						●						
Onykia (11)																			
Berryteuthis (2)							○			●	●	●	●						
**Boreoteuthis* (1)																			
**Eogonatus* (1)																			
Gonatopsis (3)							○												
Gonatus (15+)							○				●								
**Cycloteuthis* (2)																			
**Discoteuthis* (2)																			
**Psychroteuthis* (2)																			
Lepidoteuthis (1)							○				●		●						
Pholidoteuthis (2)										●	●		●						
Tetronychoteuthis (2)										●	●								
Architeuthis (4+)											●								
Histioteuthis (15+)							○				○		●						
Ctenopteryx (2)							○			○									
Bathyteuthis (3)																			
**Alluroteuthis* (1)																			
**Neoteuthis* (1)																			
**Walvisteuthis* (1)																			
**Brachioteuthis* (5)																			
**Batoteuthis* (1)																			
Dosidicus (1)							○			●	●		●						
Eucleoteuthis (1)										●	●								
Hyaloteuthis (1)										●									
Illex (4)			●				●			●	●		●						
Martialia (1)					●						●								
Nototodarus (4)											●		●						

Table 1-5 (continued)

Parasite group: / Host genera:	Virales	Bacteria	Fungi	Flagellates	Coccidians	Microsporidians	Ciliates	Dicyemids	Monogeneans	Digeneans	Cestodes	Acanthocephalans	Nematodes	Polychaetes	Hirudineans	Branchiurans	Copepods	Isopods	Decapods
ORDER TEUTHOIDEA																			
Ommastrephes (3)						○	○			●	●	●	●				●		
Ornithoteuthis (2)										●	●		●						
Sthenoteuthis (2)							○			●	●	●	●						
Todarodes (4)							●			●	●		●				●		
Todaropsis (1)										●	●		●				●		
**Cirrobrachium* (1)																			
Thysanoteuthis (1)										●									
Chiroteuthis (10)							○			●	○		○						
**Grimalditeuthis* (1)																			
**Tankaia* (1)																			
**Valbyteuthis* (3)																			
**Echinoteuthis* (1)																			
Mastigoteuthis (15)							○												
**Promachoteuthis* (1)																			
**Joubiniteuthis* (1)																			
Bathothauma (1)																			
**Belonella* (3)																			
Cranchia (1)																			
**Egea* (1)																			
Galiteuthis (5)											○								
Heliocranchia (3)																			
Leachia (8)																			
**Liguriella* (3)																			
Liocranchia (3)											○								
Megalocranchia (3)											○								
**Mesonychoteuthis* (1)																			
Sandalops (1)																			
Taonius (1)											○		○						
**Teuthowenia* (3)																			
ORDER VAMPYROMORPHA																			
Vampyroteuthis (1)							○				○		○						
ORDER OCTOPODA																			
**Cirroteuthis* (1)																			
**Cirroteuthopsis* (1)																			
**Cirrothauma* (1)																			
Chunioteuthis (2)											●								
**Froekenia* (1)																			
**Stauroteuthis* (1)																			
Grimpoteuthis (13)								●											
**Opisthoteuthis* (10)								○											
Bolitaena (1)							○												
Eledonella (1)							○												

Table 1-5 (continued)

Parasite group: / Host genera:	Virales	Bacteria	Fungi	Flagellates	Coccidians	Microsporidians	Ciliates	Dicyemids	Monogeneans	Digeneans	Cestodes	Acanthocephalans	Nematodes	Polychaetes	Hirudineans	Branchiurans	Copepods	Isopods	Decapods
ORDER OCTOPODA																			
Japatella (2)							○			●			○						
**Amphitretus* (1)																			
**Vitreledonella* (1)																			
Bathypolypus (8)								●									●		
Bentheledone (2)								●									●		
Benthoctopus (15)								●									●		
**Berrya* (2)																			
**Cistopus* (1)																			
**Danoctopus* (3)																			
Eledone (6)			●				○	●		●	●		●				●		
**Euaxoctopus* (2)																			
Graneledone (6)								○									●		
**Grimpella* (1)																			
**Hapalochlaena* (5)																			
Octopus (100+)	●	●	●	●	●		●	●		●	●		●		●		●		
Pareledone (8)								●									●		
Pteroctopus (2)							●				○								
Robsonella (5)								●		●									
**Sasakinella* (1)																			
Scaeurgus (2)							○	○			○								
**Teretoctopus* (2)																			
**Tetracheledone* (1)																			
Thaumeledone (2)								○											
**Velodona* (1)																			
**Vosseledone* (1)																			
**Tremoctopus* (2)																			
Ocythoe (1)											○								
Argonauta (6)										●									
**Alloposus* (1)																			

(n) Number of species in the genus ● Published reports of identified parasites
* Genera not examined for parasites ○ Unpubl. reports (Hochberg) or unident. parasites

As the search for additional fisheries resources expands, cephalopods are more commonly being marketed for human consumption. In Japan and other countries where cephalopods, especially squids, are eaten raw there is the very real possibility that larval nematodes will be transmitted to humans. Anisakiasis is currently recognized as an important medical problem which warrants further investigation. This is briefly discussed in the nematode section (p. 182, 183).

The role of cephalopods in the food web is only now beginning to be understood. One way ecological relationships have been elucidated is through examination of parasites. All

the evidence at hand indicates that cephalopods play a role similar to fishes in the transmission of parasites in the marine environment. Cephalopods serve as primary hosts for protozoans, dicyemids, and many crustaceans. More commonly, cephalopods function as reservoir hosts or even obligatory second or third intermediate hosts for larval stages of digeneans, cestodes, acanthocephalans and nematodes, and thus play a vital role in the transfer of parasites through the food web to final hosts such as elasmobranchs, bony fishes, sea birds and marine mammals.

Research on the parasites of cephalopods has only just begun. Canadian and Russian workers have demonstrated the importance of quantitative studies. In order to establish patterns of biological importance large numbers of a wide diversity of cephalopods need to be sampled and examined from a wide diversity of habitats and geographic locations. Details of predator/prey interactions need to be documented in order to elucidate trophic pathways. One of the key requirements for consistency in future work is to standardize the identification of not only the cephalopod hosts but their parasites. In addition to unraveling taxonomic problems we must also turn our attention to the critical tasks of completing life cycles, evaluating the effects of parasites on growth, reproduction and survival of cephalopods, and to clarifying the interactions of cephalopods and parasites in the marine environment. This subchapter will hopefully provide a starting point by bringing together the literature in one place and reviewing the work of many previous investigators.

DISEASES CAUSED BY PROTISTANS

Agents: Sarcomastigophora (Flagellata)

Flagellates rarely have been reported to be associated with cephalopods. Brocco (pers. comm.) discovered a blasidinid flagellate, tentatively identified as *Protoodinium* (Fig. 1-14), imbedded in the skin of *Octopus dofleini* collected in Washington (USA). Micrographs of the flagellate *in situ* show a dissolution of the epidermal layers associated with lesions in the mantle. A stomopode-like tube penetrates deep into the dermis of the host. No further information is available on this parasite and it has not been reported subsequently.

Laboratory populations of young octopuses, especially *Octopus bimaculoides* (between 0.5 and 25 g), are susceptible to outbreaks of an ectoparasitic flagellate which Forsythe and co-workers suggest is closely related to the freshwater bodonid, *Ichtyobodo* (Forsythe and co-authors, 1987, 1988). The disease was most likely introduced into laboratory culture from brood stock adult octopuses collected in the wild. The parasite may be similar to the flagellate found on wild-caught *Octopus dofleini* (see above).

In laboratory infestations (Forsythe and co-authors, unpubl.) the gills seemed to be the initial site of attack. As the population of parasites increased, the flagellates spread throughout the mantle cavity and then over the external surfaces of the body, head and arms. The pyriform shaped trophozoites measured 4 to 8 μm in length (Fig. 1-15). The flagellates attach to the host epithelium and feed by inserting a cytostome into the cell and digesting the cytoplasmal contents. When the host cell is destroyed the flagellate moves and taps into a new cell.

When present in large numbers the flagellates caused severe damage to the gills. As the disease condition progressed, small white spots appeared on the skin where epithelial

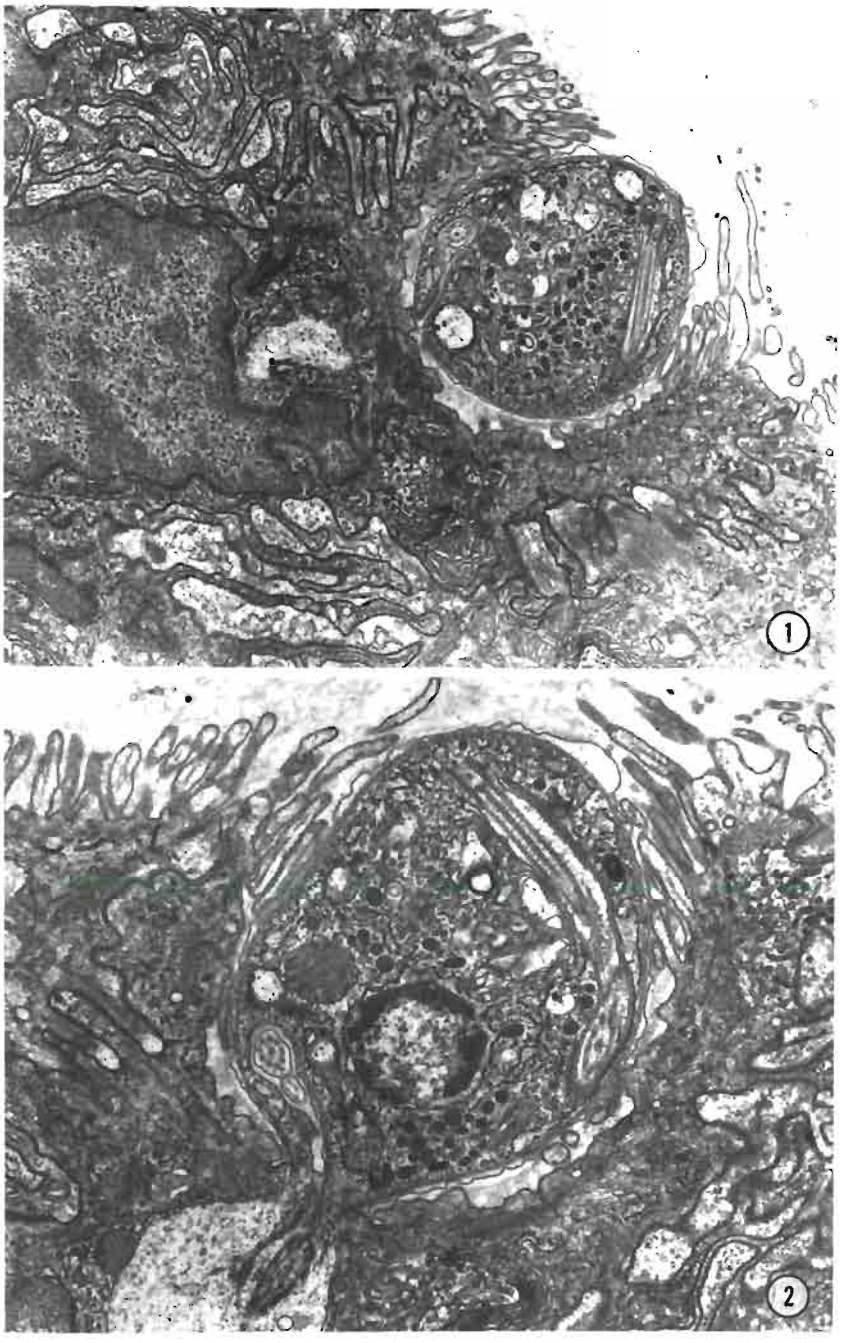


Fig. 1-14: 'Blasidinid' flagellate in skin of *Octopus dofleini*. 1: Section of skin of host with single parasite *in situ*. 2: Longitudinal section of parasite attached to skin; note suctorial tube embedded in epidermal cell. (Original, provided by S. Brocco.)

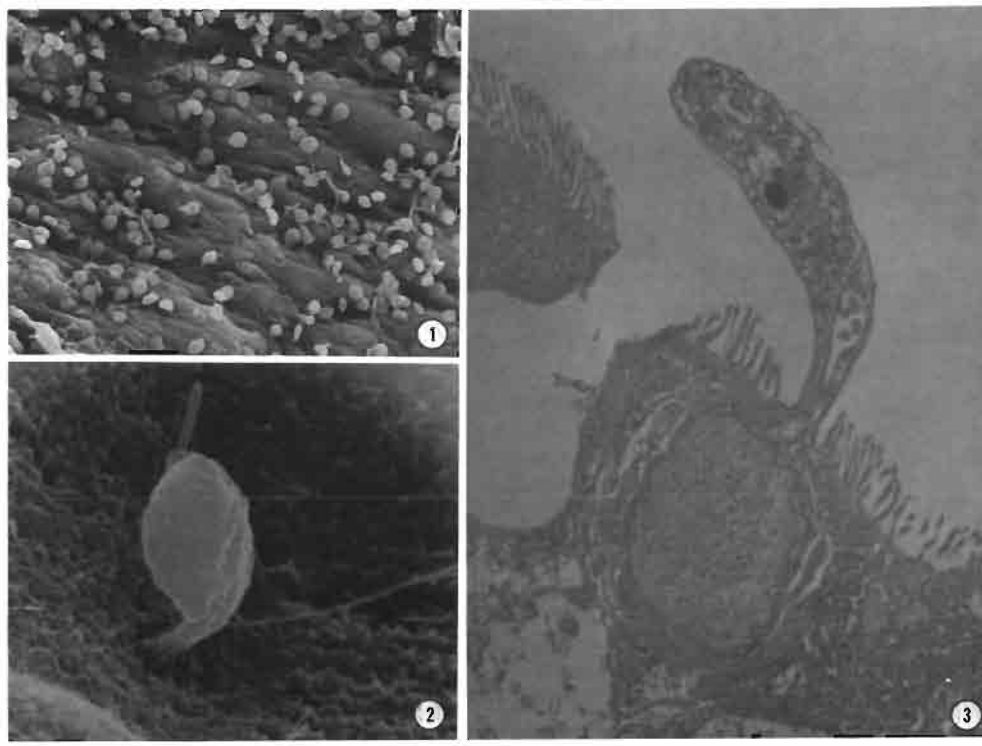


Fig. 1-15: 'Bodonid' flagellate from skin of *Octopus bimaculoides*. 1: Massive infestations of flagellates on skin of dorsal mantle of host octopus (SEM). 2: External morphology of flagellate attached to skin of host (SEM). 3: Longitudinal section of parasite attached to skin; note cytostome penetrating epithelial cell. (Original, provided by Forsythe and co-authors.)

cells had been killed. In the final stages of the disease large necrotic lesions, produced in association with secondary bacterial infections, covered the body (Fig. 1-16). It has not been determined if the flagellates inject lytic substances into the cells of the host as some authors have proposed for related parasites.

Severely infected octopuses appeared listless and emaciated. Within 2 to 4 weeks after the first appearance of white spots on the skin 100 % of cultured *Octopus bimaculoides* died. Although the disease was 100 % fatal for *O. bimaculoides,* it was only about 50 % fatal for *O. maya,* and did not appear to affect *O. digueti* cultured in the same system. No effective treatment was found to control the flagellate without killing the octopuses. Death appeared to result from flagellate damage to the gills which impaired respiratory function and to secondary bacterial infections associated with skin lesions. In mariculture situations the flagellate appears to be a serious pathogen.

Agents: Apicomplexa

The coccidian genus *Aggregata* has a 2-host life cycle (Fig. 1-17). Sexual stages occur in the digestive tracts of cephalopods, and asexual stages infect the digestive tracts of crustaceans. When first reported by Lieberkühn (1854) *Aggregata* was thought to be a

Fig. 1-16: *Octopus bimaculoides*. Lesions in skin of mantle, head and web, produced by combined flagellate and secondary bacterial infection. (Original, provided by Forsythe and co-authors, unpubl.)

gregarine and, in fact, was originally named *Monocystis sepiae* by Lankester (1863). It was correctly interpreted as a coccidian by Schneider (1883) and later placed in the family Aggregatidae by Labbé (see Pixell-Goodrich, 1914). Fine structure studies by Heller (1969, 1970a, b) and Heller and Scholtyseck (1969) indicated affinities with *Eimeria,* and placement of *Aggregata* in the Suborder Eimeriorina has been accepted by most protozoologists (see Grell, 1973; Levine, 1988; Levine and co-authors, 1980).

The best known cephalopod apicomplexan, *Aggregata eberthi,* infects the cuttlefish *Sepia officinalis* and the portunid crab *Macropipus* (= *Portunis*) *depurator* in the Mediterranean, English Channel and North Sea (Dobell, 1925; Fig. 1-17). The parasite probably occurs wherever the distributions of *S. officinalis* and species of *Macropipus* overlap. Two additional species of *Aggregata* have been reported from *Octopus vulgaris* in the Mediterranean and also in the English Channel. *A. octopiana* was described by Schneider (1875a, b), *A. spinosa* by Moroff (1906a). Moroff (1908) listed an additional 9 species he considered to be synonyms of the 3 species of *Aggregata* listed above (see Table 1-6). A number of species have been described from crustaceans in Europe but as yet these have not been identified in cephalopod hosts (Table 1-6).

Recently, Narasimhamurti (1979) described *Aggregata kudoi* (Fig. 1-18) from the cuttlefish *Sepia elliptica* collected off Visakhapatnam, India. The crustacean hosts for these latter 3 species are not known. Several undescribed species of *Aggregata* are known to occur in *Octopus* species off California (USA) and the west coast of Mexico (Hochberg, unpubl.), off Florida (USA) (McSweeney, pers. comm.), in the Caribbean off the Virgin

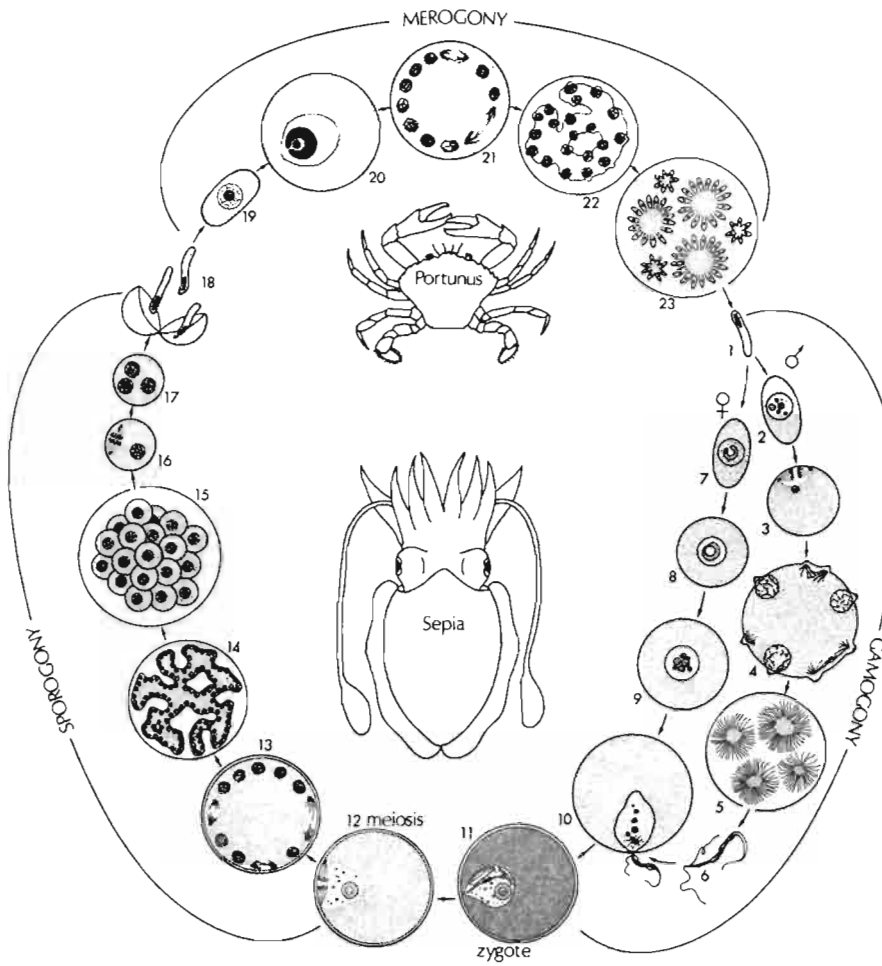


Fig. 1-17: *Aggregata eberthi*. Life cycle of this emeriid apicomplexan from the cuttlefish *Sepia officinalis* and the crab *Macropipus (= Portunus) depurator*. (1) Merozoite; (2–5) microgamont development; (6) microgamete; (7–9) macrogamont development; (10) macrogamete at time of fertilization; (11) zygote; (12–14) sporont development; (15) oocyst with developing sporocysts; (16–17) sporocyst development; (18) sporocyst (spore) with three sporozoites; (19–23) meront development. (After Hochberg, 1983.)

Islands (Hochberg and Couch, 1971) and off Argentina (Sardella and Ré, unpubl.). Species of *Aggregata* are distinguished on the basis of host, diameter of sporocysts, number and length of sporozoites (Table 1-7). The reports by DeHorne (1930a, b) of *Aggregata* in members of the polychaete genus *Nereis* most likely represent a misidentification.

The presence of a species of *Aggregata* in the neretic ommastrephid squid *Martialia hyadesi* off Argentina (Gaevskaya and co-authors, 1986a; Sardella, unpubl.) is not surprising considering the discovery that these coccidian parasites are common in mid-water decapod crustaceans (Théodoridès, 1965; Théodoridès and Desportes, 1975).

Species of *Aggregata* appear to be very host specific in cephalopods but not in crustaceans. Of the cephalopods examined by Dobell (1925) in the Mediterranean only

Table 1-6
Species of *Aggregata* from cephalopods and crustaceans (Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	Crustacean hosts	Locality	Source
ORDER SEPIOIDEA				
Sepia elliptica	*A. kudoi*	*[Ocypoda cordimana, O. platytarsis]*	Western Bay of Bengal (India)	Narasimhamurti (1979)
Sepia officinalis	*A. eberthi (= A. arcuata, A. frenzeli, A. mamillana, A. mingazzinii, A. minima, A. portunidarum)*	*Macropipus arcuatus, M. armatus, M. bolivari, M. corrugatus, M. depurator, M. holsatus, M. puber, M. tuberculata, M. vernalis, [M. arenatus]*	Mediterranean (Italy, Monaco, France, Spain, Tunisia)	Frenzel (1885), Eberth (1892), Labbé (1895, 1896, 1899), Léger and Duboscq (1906–1908), Dobell (1914, 1925), Pixell-Goodrich (1914), Bělař (1926), Wurmbach (1935), Vivares (1970, 1973a, b), Vivares and Rubio (1969)
S. officinalis	*A. eberthi*	*M. holsatus*	English Channel (France, England)	Moroff (1906–1908), Pixell-Goodrich (1914), Porchet-Hennere and Richard (1969–1971), Hochberg (unpubl.)
S. officinalis	*A. eberthi*	?	North Sea (Germany)	Heller (1969–1970)
ORDER SEPIOLIOIDEA				
+Rossia pacifica	*A.* sp. unident.	?	Eastern North Pacific Ocean (California, USA)	Hochberg (unpubl.)
ORDER TEUTHOIDEA				
Martialia hyadesi	*A.* sp. unident.	?	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)

Table 1-6 (continued)

Cephalopod hosts	Parasites	Crustacean hosts	Locality	Source
ORDER OCTOPODA				
+*Eledone massyae*	*A.* sp. unident.	?	Western South Atlantic Ocean (Argentina)	Ré and Taylor (1981)
+*Octopus bimaculatus*	*A.* sp. unident.	?	Eastern North Pacific Ocean (California, USA)	Lara (unpubl.)
+*Octopus bimaculoides*	*A.* sp. unident.	?	Eastern North Pacific Ocean (California, USA & Mexico)	Hochberg (unpubl.)
+*Octopus briareus*	*A.* sp. unident.	?	Caribbean (Virgin Islands); Straits of Florida (Florida, USA)	Hochberg and Couch (1971); McSweeney (unpubl.)
+*Octopus megalocyathus* (= *Enteroctopus m.*)	*A.* sp. unident.	?	Western South Atlantic Ocean (Argentina)	Ré (1980, 1984)
+*Octopus rubescens*	*A.* sp. unident.	?	Eastern North Pacific Ocean (California, USA)	Hochberg (unpubl.)
+*Octopus tehuelchus*	*A.* sp. unident.	?	Western South Atlantic Ocean (Argentina)	Sardella and Ré (in press)
+*Octopus veligero*	*A.* sp. unident.	?	Eastern North Pacific Ocean (Mexico)	Hochberg (unpubl.)
Octopus vulgaris	*A. octopiana* (= *A. duboscqi, A. reticulosa, Klossia, Benedenia*)	?	English Channel (France)	Frenzel (1885), Schneider (1875a, 1883), Jacquemet (1903), Moroff (1906–1908)
O. vulgaris	*A. octopiana* (= *A. duboscqi, A. reticulosa*)	?	Mediterranean (Italy, Monaco, France)	Mingazzini (1892a, b), Dobell (1925), Wurmbach (1935)

Table 1-6 (continued)

Cephalopod hosts	Parasites	Crustacean hosts	Locality	Source
ORDER OCTOPODA				
Octopus vulgaris	*A. spinosa* (= *A. jacquemeti, A. labbei, A. legeri, A. ovata, A. reticulosa, A. schneideri, A. siedleckii, A. stellata*)	Unknown	English Channel (France)	Moroff (1906–1908)
Unknown	*A. coelomica*	*Pinnotheres pisum, P. pinnotheres*	Mediterranean (France, Spain)	Léger (1901), Léger and Duboscq (1906a, 1908), Vivares and Rubio (1969), Vivares (1973a, b)
Unknown	*A. inachi*	*Inachus communissimus, I. dorsettensis, I. scorpio* (= *I. maure*)	Mediterranean (Italy, France, Spain, Tunisia)	Smith (1905), Léger and Duboscq (1906a, 1908), Dobell (1925), Vivares and Rubio (1969), Vivares (1970, 1973a, b)
Unknown	*A. leandri*	*Leander squilla, Acanthephyra eximia, Gennadas elegans, Solenocera membranacea*	Mediterranean (Italy, France)	Pixell-Goodrich (1950), Théodoridès (1965), Théodoridès and Desportes (1975)
Unknown	*A. maxima*	*Sergestes robustus*	Mediterranean (France)	Théodoridès and Desportes (1975)
Unknown	*A. vagans*	*Eupagurus cuanensis, E. prideauxi, E. sculptimanus*	Mediterranean (France)	Léger and Duboscq (1903, 1906a, 1908)

Table 1-6 (continued)

Cephalopod hosts	Parasites	Crustacean hosts	Locality	Source
ORDER OCTOPODA				
Unknown	*A.* sp. unident.	*Atelecyclus rotundatus, Carcinus maenas, C. mediterraneus, Corystes cassivelaunus, Dromia personata, Goneplax rhomboides, Homarus gammarus, Macropodia rostrata, Pagurus arrosor, Pachygrapsus marmoratus, Parathenope anguilifrons, Pilumnus hirtellus, P. spinifer, Pirimela denticulata, Portumnus latipes, Stenorhynchus phalangium, Xaiva biguttata, Xantho poressa*	Mediterranean (France, Spain, Tunisia)	Frenzel (1885), Léger and Duboscq (1908), Vivares and Rubio (1969), Vivares (1973a, b)
Unknown	*A.* sp. unident.	*Eucopia hanseni, Sergestes arcticus, S. corniculum, Pasiphaea multidentata, P. sivado*	Mediterranean (France)	Théodoridès and Desportes (1975)
Unknown	*A.* sp. unident.	*Parapeneopsis sculptilis*	Eastern Arabia Sea (India)	Setna and Bhatia (1934)

[ ] Experimentally infected hosts – not reported to be infected in nature; + New host records

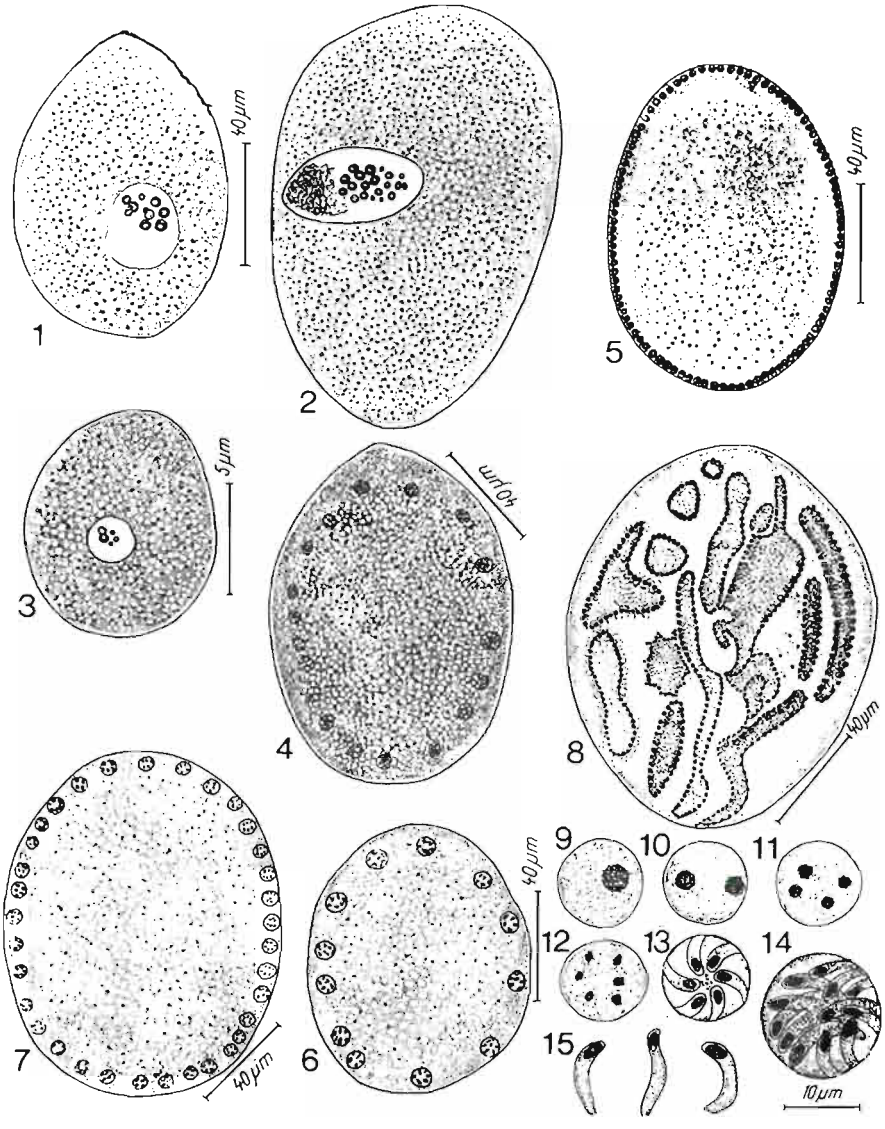


Fig. 1-18: *Aggregata kudoi*. Stages in the development of the parasite from cuttlefish *Sepia eliptica*. (1 to 2) Macrogamont development; (3 to 5) microgamont development; (6 to 8) sporont development; (9 to 12) sporocyst development; (13) normal sporocyst with 6 fully formed sporozoites; (14) abnormally large sporocyst with 12 sporozoites; (15) free sporozoites. (After Narasimhamurti, 1979.)

Sepia officinalis and *Octopus vulgaris* were infected (Table 1-8). In both cases *Aggregata* was present in 100 % of these hosts. Off California 100 % of *O. bimaculoides* and *O. rubescens* are infected with a new species of *Aggregata* (Hochberg, unpubl.) and in the Caribbean and off Florida 100 % of the *O. briareus* examined were infected (McSweeney, pers. comm.; Hochberg and Couch, 1971).

Aggregata selectively infects the non-cuticularized, nutrient uptake portions of the digestive tract of both cephalopod and crustacean hosts. In cephalopods the parasites are

Table 1-7
Species of *Aggregata* in cephalopods: diameter of sporocysts, number and length of sporozoites (Original)

Aggregata species	Sporocyst diameter (μm)	Sporozoite		Cephalopod host
		number	length (μm)	
A. eberthi	8–9	3	15–17	*Sepia officinalis*
A. kudoi	9–11	6	16–18	*S. elliptica*
A. octopiana	12–15	8–16	25–30	*Octopus vulgaris*
A. spinosa	?	12–28	20–27	*O. vulgaris*

Table 1-8
Cephalopods examined for the presence of *Aggregata* species in the Mediterranean at Naples (Italy) (After Dobell, 1925)

	Cephalopods	Number examined	Number infected
SEPIOIDEA	Family Sepiidae		
	Sepia officinalis	**73**	**73**
	S. orbignyana	8	0
	S. elegans	82	0
SEPIOLIOIDEA	Family Sepiolidae		
	Sepiola rondeletii	55	0
	Rossia macrosoma	7	0
TEUTHOIDEA	Family Loliginidae		
	Loligo vulgaris	13	0
	Alloteuthis media (= *Loligo marmorae*)	16	0
	Family Ommastrephidae		
	Illex coindetii	9	0
OCTOPODA	Family Octopodidae		
	Octopus vulgaris	**8**	**8**
	O. macropus	2	0
	O. defilippi	7	0
	Eledone moschata	25	0
	E. cirrhosa (= *E. aldrovandi*)	2	0
	Family Ocythoidae		
	Ocythoe tuberculata	3	0

located within epithelial cells of the mucous membrane and in the submucosal connective tissue of the caecum and intestine. As infective stages or merozoites (Fig. 1-19) migrate through the epithelium of the digestive tract, the invaded cells die and degenerate. Periodically, necrotic portions of the gut lining are sloughed off and eliminated. In heavy infections the submucosal tissue of the cephalopod may be almost completely replaced by parasite cells. When *Aggregata* parasites are present in large numbers the mechanical

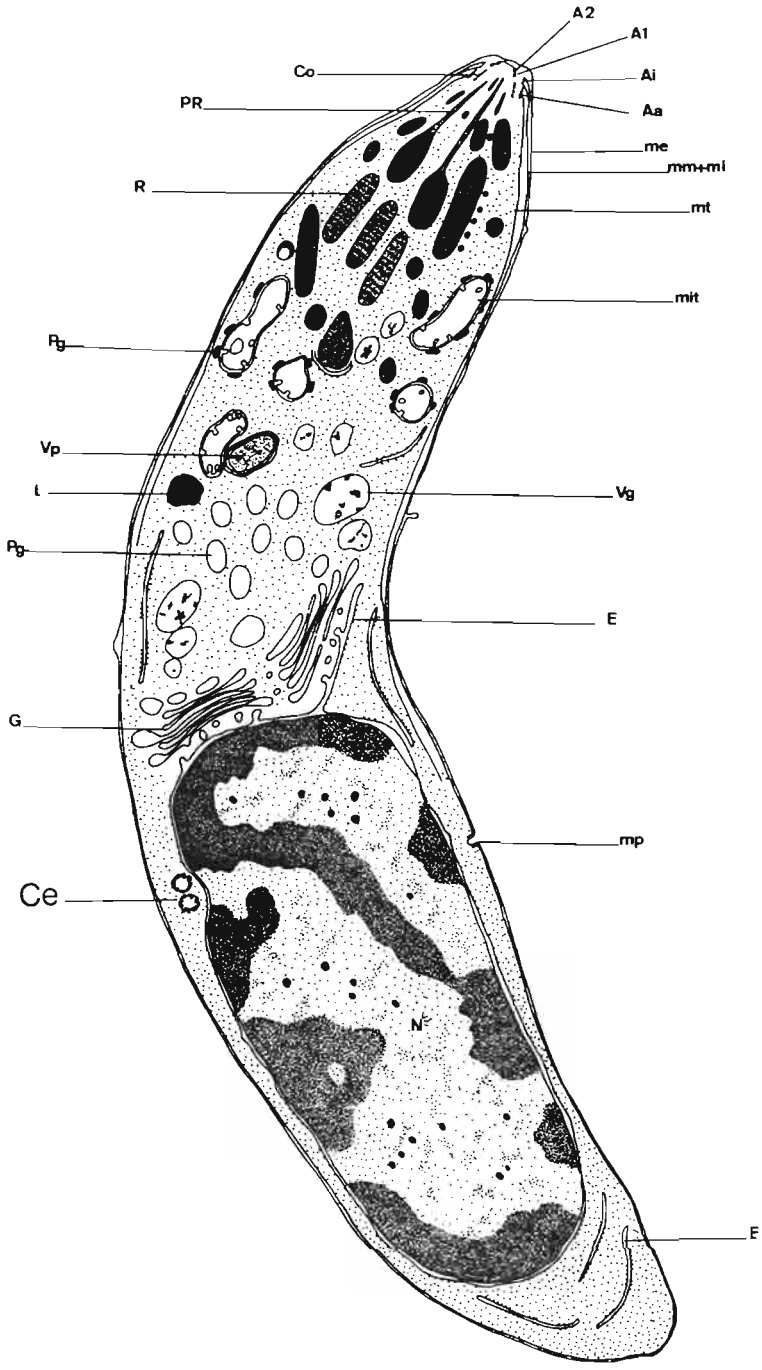


Fig. 1-19: *Aggregata eberthi* from *Macropipus* (= *Portunis*) *holsatus*. Schematic reconstruction of the merozoite or infective stage for the cephalopod host. (A1, A2) Anterior or polar rings; (Aa) ring where microtubules are inserted; (Ai) collar where cell wall is interrupted; (Ce) centriole; (Co) conoid; (E) ergastoplasm; (G) golgi apparatus; (L) lipid vacuole; (me) outer membrane; (mp) micropore; (mt) subpellicular microtubule; (mit) mitochondrion; (mm + mi) middle and internal membrane of pellicle; (N) nucleus; (Pg) paraglycogen; (PR) rhoptry peduncle; (r) rhoptry; (Vg) golgi derived vacuole; (Vp) multimembrane vacuole. (After Porchet-Henneré and Richard, 1971b.)

effects of compressing and deforming host tissue may prevent circulation and muscular activity in the gut wall. In *Sepia officinalis* the individual infected cells exhibit no apparent response to the presence of the parasite. However, in *Octopus vulgaris*, the invaded cells may undergo enormous nuclear and cytoplasmic hypertrophy (Figs 1-20 and 1-21; Brumpt, 1913; Dobell, 1925; Wurmbach, 1935).

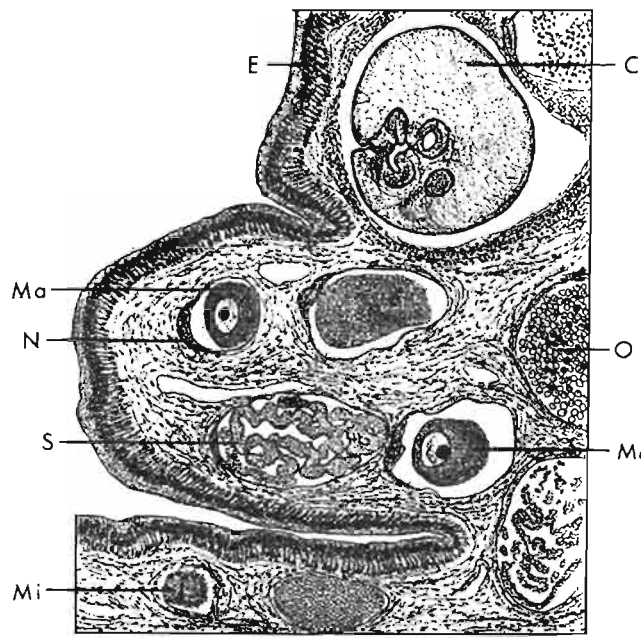


Fig. 1-20: *Aggregata octopiana* in *Octopus vulgaris*. Section of rectum showing various stages in the development of the coccidian and the larval cestode *Scolex* sp. (C) Larval cestode; (E) epithelium; (Ma) macrogamete; (Mi) microgamete; (N) nucleus of hypertrophied infected cell; (O) oocyst with fully developed sporocysts (spores); (S) developing sporont. (After Brumpt, 1913.)

The life cycle of *Aggregata eberthi* is one of the classics in parasitology (Fig. 1-17). Originally outlined by Léger and Dubosq (1906–1908), the cycle has been studied in detail by Siedlecki (1898a, b), Dobell (1914, 1925), Naville (1925) and by Bělař (1926). Fine structure studies of a number of the stages in the life cycle have confirmed the observations of earlier workers (see Porchet-Henneré and Richard, 1969–1971; Porchet-Henneré and Vivier, 1971; Vivier and co-authors, 1970).

The infection is initiated when *Sepia officinalis* feed on portunid crabs of the genus *Macropipus*. Ripe, infective stages (merozoites, Fig. 1-19), which reside in the coelom of the crab, are released into the digestive tract of the cuttlefish upon ingestion of the intermediate host. The merozoites actively bore through the epithelial lining of the caecum and intestine of *S. officinalis* and enter connective tissue cells in the submucosa. Growth occurs as nutrients are taken up from lymph spaces within the connective tissue of the cephalopod host. During gamogony the merozoites are transformed into gamonts of 2 types. Each macrogamont (Fig. 1-22) gives rise to a single macrogamete, and as these large cells develop the nucleus approaches the surface of the cell. Development of the microgamete (Fig. 1-23) proceeds until large numbers of biflagellated microgametes are produced.

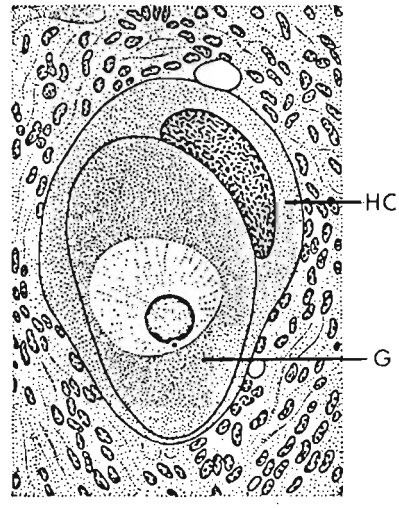


Fig. 1-21: *Aggregata octopiana* in *Octopus vulgaris*. Young gamont in the subepithelial connective tissue of the intestine; note enlargement of host cell and nucleus in response to presence of parasite. (G) Gamont; (HC) hypertrophied host cell. (After Wurmbach, 1935.)

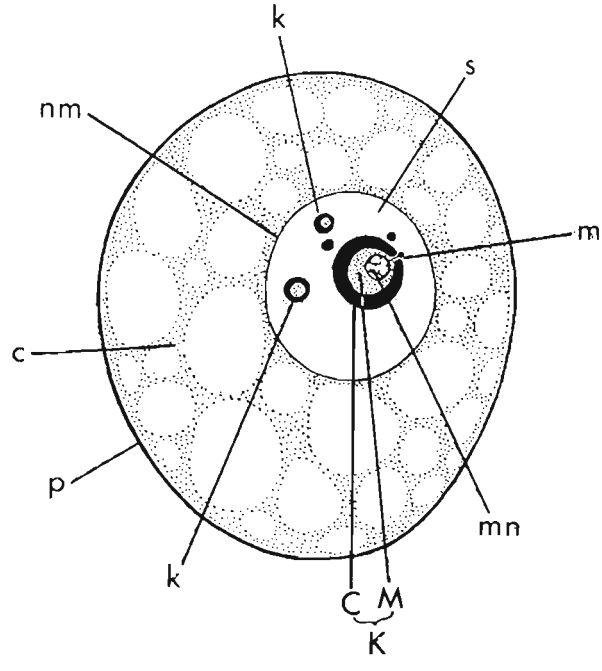


Fig. 1-22: *Aggregata eberthi* from *Sepia officinalis*. Diagrammatic representation of the young female parasite. (C) Cortex; (c) cytoplasm; (K) primary karyosome; (k) accessory karyosome; (M) medulla; (m) micropyle; (mn) micronucleus; (nm) nuclear membrane; (p) pellicle; (s) nuclear space. (After Dobell, 1925.)

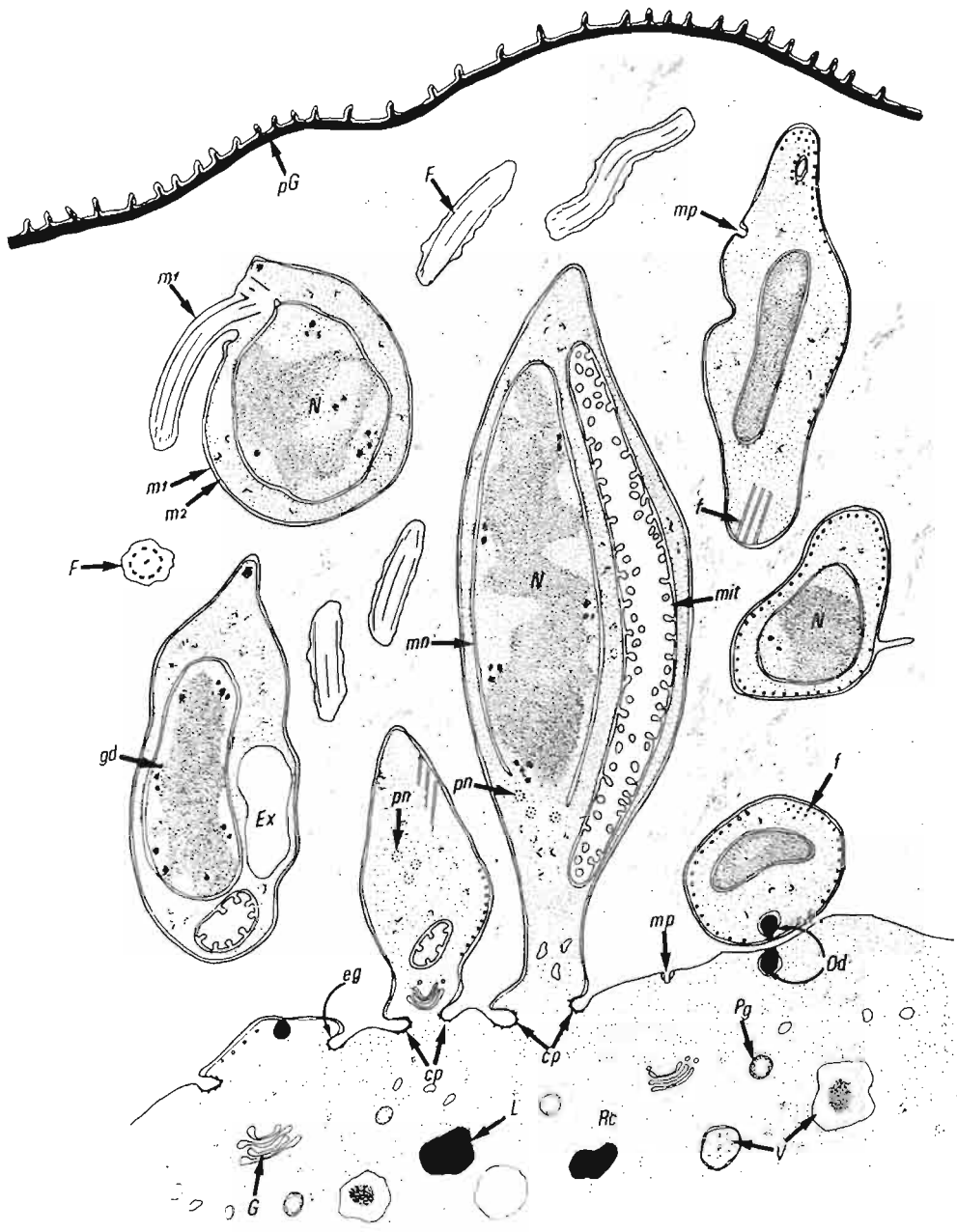


Fig. 1-23: *Aggregata eberthi* from *Sepia officinalis*. Semi-diagrammatic representation of a section through a male gamont showing stages in the development of microgametes. (cp) Peduncular constriction; (eg) granular thickening; (Ex) distended vacuole; (f) subpellicular microtubule; (F) flagellum; (G) dictysome; (gd) dense granules; (L) lipid inclusion; (m1) external membrane; (m2) internal membrane; (mn) nuclear membrane; (mp) micropore; (N) nucleus; (Od) bottle-shaped dense body; (pG) gamont cell wall; (pg) paraglycogen; (pn) nuclear pore; (Rc) cytoplasmic residue; (V) vacuole. (After Porchet-Henneré and Richard, 1970b.)

Eventually, motile male gametes are released into the surrounding tissue and enter the macrogametes in the area where the nucleus touches the pellicle.

Following fertilization the zygote undergoes a reduction division which subsequently triggers a burst of mitotic activity. During sporogony the cytoplasm of the sporont is progressively divided up and a large number of sporoblasts are produced. When finally enveloped by a gelatinous coat the sporoblasts (Fig. 1-24), which now fill the oocyst, are termed spores or sporocysts (Fig. 1-25). In *Aggregata eberthi*, following 2 additional divisions, each sporocyst contains 3 sporozoites measuring 8 to 9 μm.

Mature sporocysts rupture out of the oocyst and are eliminated with the feces. Often entire portions of necrotic gut lining containing intact oocysts are sloughed off and discharged to the exterior. The infection can be experimentally transmitted to crabs by feeding them ripe spores contained in either detrital material contaminated with cuttlefish feces or scraps of cuttlefish intestine. Within a few hours after ingestion, the infective sporozoites are released and move actively about in the lumen of the crab gut. Within 24 h they will penetrate the epithelial lining of the midgut and migrate into the lymphoid tissues

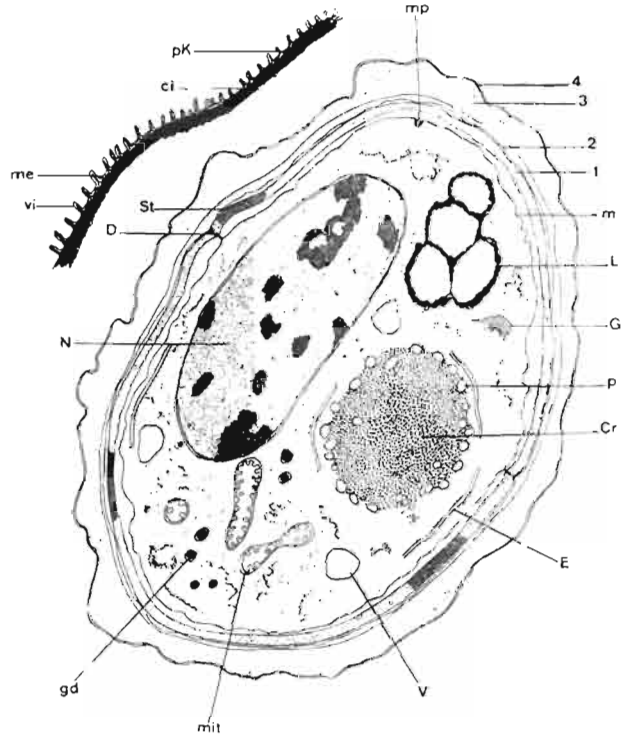


Fig. 1-24: *Aggregata eberthi* from *Sepia officinalis*. Diagrammatic representation of a section through a sporoblast. 1 to 4: Components of the sporoblast envelope. (1) striated layer. (2) elementary membrane, (3) thick outer membrane, (4) outer pellicle composed of two fused membranes. (Ci) Dense internal layer of cyst wall; (Cr) future 'crystalloid'; (D) discharge pore; (E) ergastoplasm; (G) dictyosome; (gd) dense glomerule; (L) lipid vacuole; (me) external limit of cyst wall; (m) internal membrane; (mit) mitochondrian; (mp) micropore; (N) nucleus; (P) paraglycogen; (pK) cyst wall; (St) periodic striation of sporoblast envelope; (V) vacuole; (vi) villi of cyst wall. (After Porchet-Henneré and Richard, 1969.)

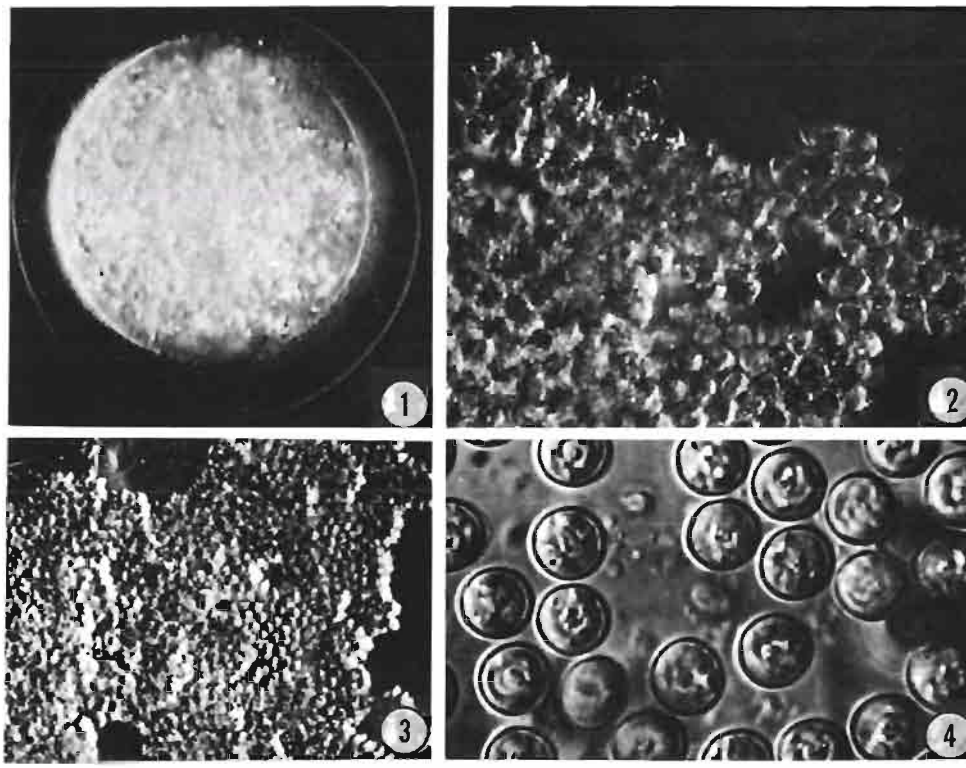


Fig. 1-25: *Aggregata eberthi* in *Sepia officinalis*. Photographs of living coccidians from the intestine of the cuttlefish host. 1: Oocyst (× 400); 2: oocyst, in course of sporulation (× 500); 3: ruptured oocyst showing mammiliform cytoplasmic mass (× 200); 4: mature spores (× 1500). (After Porchet-Henneré and Richard, 1971a.)

of the submucosa. Here they round up and enlarge into meronts. When growth is completed an asexual phase of reproduction begins. During merogony the nucleus divides many times producing a large number of daughter nuclei which come to lie near the surface of the highly convoluted cytoplasm. After the merozoites are released, there is no further development until the crab is eaten by the cuttlefish and the cycle starts over again.

Agents: Microspora

At present 2 species of Microspora are known to parasitize cephalopods. The first was reported by Kalavati and Narasimhamurti (1977). While examining cephalopods for the presence of dicyemids, they discovered that about 5 % of the cuttlefish *Sepia elliptica* were infected with a chytridiopsid microsporidian. *Steinhausia spraguei* (Fig. 1-26) currently is known only from Visakhapatnam, India, where it occurs in the excretory cells of the renal appendages of cuttlefish hosts. As is typical, the parasite completes its entire life cycle within a single host cell. Inside the host cell the parasite undergoes schizogony. Later, when the plasmodium reaches 10 μm in diameter the nuclei pair up and migrate to the periphery of the cell prior to sporogony (Fig. 1-26, 3). Fully developed cysts are spherical to ovoid, measure 40 to 45 μm in diameter and contain 40 to 50 spores (Fig. 1-26, 4).

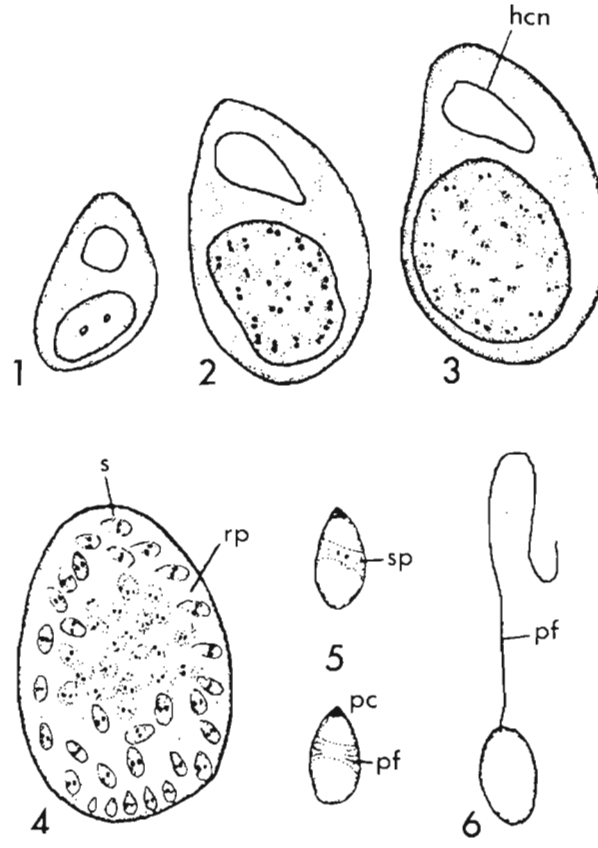


Fig. 1-26: *Steinhausia spraguei* in cells of renal appendages of *Sepia elliptica*. (1) Early intracytoplasmic stage showing two nuclei; (2) multinucleate sporogonial plasmodium showing migration of nuclei to the periphery and pairing; (3) developing cyst showing formation of spores; note hypertrophy of the host excretory cell and nucleus and the displacement of the nucleus; (4) mature cyst found free in the renal fluid; note fully formed spores around the periphery and developing spores in the center of the cyst; (5) fresh spores; (6) spore with extended polar filament. (hcn) Host cell nucleus; (pc) polar cap; (pf) polar filament; (rp) residual protoplasm; (s) spore; (sp) sporoplasm. (After Kalavati and Narasimhamurti, 1977.)

Mature spores are oval in shape and 4.5 to 5.0 μm long (Fig. 1-26, 5). In response to the presence of the parasite the host cell and nucleus undergo considerable hypertrophy.

Transmission of the parasite is accomplished by release of spores from ruptured excretory cells into the renal fluids which are then voided to the exterior of the host. Since *Steinhausia spraguei* infects cells of the renal appendages the spores are most likely swept into the mantle cavity of a new host during respiratory pumping. Upon contact with a suitable epithelium the spore presumably releases the polar filament through which the amoebula invades the cytoplasm of the new host cell (Fig. 1-26, 6). Nagasawa and Nakata (1984) briefly noted the presence of white cysts full of spores in the visceral mass of *Ommastrephes bartrami* collected off Japan. No figures were provided and the authors did not identify the parasite other than its being a microsporidian. Three other squid species, *Berryteuthis magister, Gonatopsis borealis* and *Onychoteuthis borealis-japonica,* were examined and were negative.

The small size and intracellular habits of the Microspora preclude detection in most routine surveys for parasites. Knowledge that microsporidians occur in cephalopods may prompt workers to more carefully examine cuttlefishes, squids and octopods for the presence of infections. In the future, additional species will likely be encountered.

Agents: Ciliophora

With the exception of the dicyemids, ciliates are the most frequently encountered protistan parasites of cephalopods. At least 5 families are parasitic in the renal organs and in the digestive tracts and digestive glands of cuttlefishes, squids and octopuses. However, only a few published studies deal with these unusual forms and many new findings await analysis.

The genus *Chromidina* is restricted to a small group of vermiform ciliates which attach to the appendages within the renal or renal-pancreatic coela of cephalopods. Three species have been described, though a total of 23 species of cephalopods in 20 genera currently are known to harbor chromidinids (Hochberg, 1982a; Table 1-9). In the Mediterranean and English Channel, *C. coronata* (Fig. 1-27, 2) occurs in *Octopus vulgaris, Sepiola rondeleti, Illex coindetti, Eledone cirrhosa,* and *Scaeurgus unicirrhus.* A second species, *C. elegans* (Fig. 1-27, 1), lives in *Sepia elegans, S. orbignyana* and *I. coindetti.* For details see Dobell (1909); Collin (1914b, 1915); Chatton and Lwoff (1928, 1931, 1935); Nouvel (1935a, b, c, 1937, 1945); and Hochberg (1971). A third species, *C. cortezi,* reported from *Pterygioteuthis giardi* in the Gulf of California, Mexico, has been described by Hochberg (1971).

Ciliates, attributed to *Chromidina elegans,* have been reported from *Todarodes sagittatus* and *Octopus salutii* in the Mediterranean (Nouvel, 1945; Hochberg, 1971); from *Loligo* sp. off Russia (Wermel, 1928); and from *Spirula spirula* in the Atlantic Ocean (Jepps, 1931; Clarke, 1970). This material has not been critically examined and compared to the type species and hence the true designations are not known. In the North Pacific Ocean a wide variety of schooling epi- and mesopelagic cephalopods are infected with chromidinid ciliates (see Table 1-9).

Hochberg (1971) surveyed large numbers of small mesopelagic squids for the presence of ciliates. Of 235 *Pterygioteuthis giardi* examined from the Gulf of California (Fig. 1-28), 29 % were infected with *Chromidina cortezi* (Fig. 1-29), whereas the related *P. gemmata,* an endemic of the California current, was never infected. In *P. giardi* larvae measured less than 5 mm mantle length (ML). Males matured at 15 mm ML and attained a maximum size of 25 mm ML. Females matured at 16 mm ML and reached a maximum size of 27 mm ML. The infection was heaviest in adult squid larger than 15 mm ML (Fig. 1-29) Larvae are concentrated in the upper 50 m of the water column. They feed on copepods and were never infected. In the size range from 5 to 15 mm juvenile and post juvenile squid feed on euphausiids and are infected with ciliates.

Characteristically only truly pelagic squids and octopods are infected. Infection of benthic or epibenthic hosts occasionally has been reported but in all such cases the ciliates were found only in octopuses which have planktonic larvae (i.e., *Octopus salutii, O. vulgaris, Scaeurgus unicirrhus,* and *Eledone cirrhosa*) or in sepioids whose young feed in surface waters (i.e., *Sepia elegans, S. orbignyana* and *Sepiola rondeleti*). Larval cephalopods contract the ciliates through association with planktonic crustaceans and bring the infection to the bottom at the time of settlement. Dicyemid infections are

Table 1-9
Chromidina and *Opalinopsis*, ciliate parasites of cephalopods (Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	Locality	Source
ORDER SEPIOIDEA			
Spirula spirula	*Chromidina 'elegans'*	Eastern North Atlantic Ocean (Canary Islands)	Jepps (1931), Clarke (1970)
Sepia elegans	*Chromidina elegans* (= *Benedenia*)	Mediterranean (Italy, Monaco, France)	Foettinger (1881a, b), Gonder (1905), Dobell (1909), Collin (1914b, 1915), Chatton & Lwoff (1931, 1935), Nouvel (1935a, b, 1945)
S. elegans	*Opalinopsis sepiolae*	Mediterranean (Italy)	Gonder (1905)
Sepia officinulis	*Opalinopsis sepiolae*	English Channel (England); Mediterranean (Italy, France)	Dobell (1909), Mohr (unpubl.); Hochberg (1971)
Sepia orbignyana	*Chromidina elegans*	Mediterranean (Italy, Monaco, France)	Dobell (1909), Nouvel (1945)
ORDER SEPIOLIOIDEA			
Heteroteuthis hawaiiensis	*Chromidina* sp. A	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
H. hawaiiensis	*Opalinopsis* sp. I	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Rossia macrosoma	*Opalinopsis 'sepiolae'*	Eastern North Atlantic Ocean (Norway)	Hochberg (1971)
Sepietta oweniana	*Opalinopsis 'sepiolae'*	Mediterranean (France)	Hochberg (1971)
Sepiola atlantica	*Opalinopsis 'sepiolae'*	English Channel (England)	Mohr (unpubl.), Hochberg (1971)
Sepiola rondeleti	*Chromidina coronata*	Mediterranean (France)	Collin (1915)
S. rondeleti	*Opalinopsis sepiolae*	Mediterranean (Italy, Monaco, France)	Foettinger (1881a, b), Gonder (1905), Dobell (1909), Collin (1915)
ORDER TEUTHOIDEA			
Alloteuthis subulata	*Opalinopsis* sp. III	English Channel (England)	Mohr (unpubl.), Hochberg (1971)
Loligo sp.	*Chromidina 'elegans'*	Japan Sea (USSR)	Wermel (1928)

Table 1-9 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER TEUTHOIDEA			
Abralia trigonura	*Chromidina* sp. C	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Abraliopsis brevis	*Chromidina* sp. B	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Abraliopsis falco	*Chromidina* sp. H	Eastern North Pacific Ocean (California, USA)	Hochberg (unpubl.)
Abraliopsis felis	*Chromidina* sp. H	Eastern North Pacific Ocean (Mexico)	Hochberg (1971)
Pterygioteuthis giardi	*Chromidina cortezi*	Gulf of California/Eastern North Pacific Ocean (Mexico)	Hochberg (1971)
Pterygioteuthis microlampas	*Chromidina* sp. F	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Gonatus sp.	*Chromidina* sp. H	Eastern North Pacific Ocean (Oregon, USA)	Hochberg (1971)
Histioteuthis heteropsis	*Chromidina* sp. G	Eastern North Pacific Ocean (California, USA; Mexico)	Hochberg (1971)
H. heteropsis	*Opalinopsis* sp. II	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Ctenopteryx sicula	*Chromidina* sp. D	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Dosidicus gigas	*Chromidina* sp. E	Gulf of California/Eastern North Pacific Ocean (Mexico)	Hochberg (1971)
Illex coindetti	*Chromidina elegans*	Mediterranean (Italy, Monaco, France)	Gonder (1905), Dobell (1909), Nouvel (1945)
I. coindetti	*Chromidina coronata*	Mediterranean (Italy)	Dobell (1909)

Table 1-9 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER TEUTHOIDEA			
Sthenoteuthis oualaniensis (= *Symplectoteuthis*)	*Chromidina* sp. E.	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Todarodes pacificus (= *Ommastrephes sloani-pacificus*)	*Chromidina* sp. G	Western North Pacific Ocean (Japan)	Hochberg (1971)
Todarodes sagittatus (= *Ommatostrephes*)	*Chromidina elegans*	Mediterranean (Monaco, Spain)	Nouvel (1945), Hochberg (1971)
Chiroteuthis calyx	*Chromidina* sp. H	Eastern North Pacific Ocean (California, USA)	Hochberg (unpubl.)
Mastigoteuthis pyroides	*Chromidina* sp. H	Eastern North Pacific Ocean (Mexico)	Hochberg (1971)
ORDER OCTOPODA			
Japatella diaphana	*Chromidina* sp. G	Eastern North Pacific Ocean (California, USA; Mexico)	Hochberg (1971)
Eledone cirrhosa (= *E. Aldrovandii*, *Acantheledone*)	*Chromidina coronata*	English Channel (England); Mediterranean (Italy, France)	Gonder (1905), Nouvel (1935a, 1937, 1945), Rees (1956), Mohr (unpubl.), Hochberg (1971)
Octopus macropus	*Opalinopsis 'octopi'*	Mediterranean (Italy)	Hochberg (1971)
Octopus salutii (= *O. saluzzi*)	*Chromidina 'elegans'*	Mediterranean (Monaco, France)	Nouvel (1945), Hochberg (1971)
Octopus vulgaris	*Chromidina coronata*	Mediterranean (Italy, Monaco, France)	Foettinger (1881a, b), Gonder (1905)
Pteroctopus tetracirrhus (= *Octopus*)	*Opalinopsis octopi*	Mediterranean (Italy, France)	Foettinger (1881a, b), Gonder (1905), Hochberg (1971)
Scaeurgus unicirrhus	*Chromidina 'coronata'*	Mediterranean (France)	Hochberg (1971)

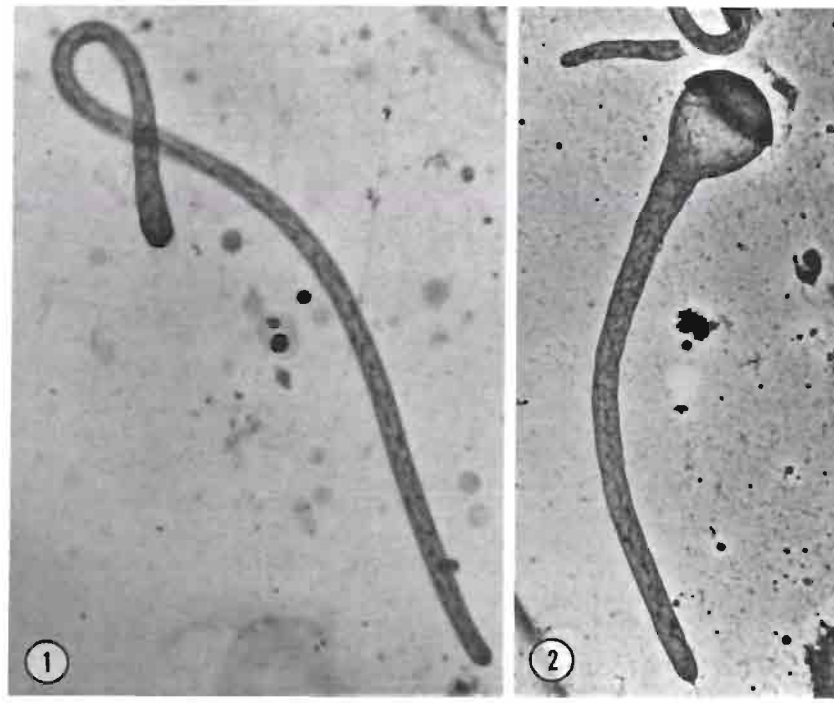


Fig. 1-27: Tropho-tomonts of *Chromidina,* apostome ciliates found in the renal organs of cephalopods. 1: *C. 'elegans'* morph from *Histioteuthis heteropsis.* 2: *C. 'coronata'* morph from *Mastigoteuthis pyrodes*; note crown of elongate cilia. (Original.)

established when juvenile cephalopods take up residence on the sea floor. The ciliates are progressively eliminated as the dicyemids multiply and fill the entire renal habitat (see Table 1-11; Nouvel, 1937, 1945; Hochberg, 1971).

As elucidated by Hochberg (1971, 1982a) the genus *Chromidina* has a 2-host life cycle (Fig. 1-30). Like the better known foettingeriid ciliates, the genus *Chromidina* undergoes a complex polymorphic cycle involving an ordered sequence of distinct phases. Young squids pick up ciliates when they associate with or feed on swarms of pelagic crustaceans, such as euphausiids. At present the method of entry into the host is not known. Within the cephalopod, the stages of the cycle are considerably modified and condensed, compared with the small, ovoid, and less specialized foettingeriids (Chatton and Lwoff, 1935; Bradbury, 1966). In the genus *Chromidina,* the vegetative and divisional phases are combined into long, thin forms known as tropho-tomonts. These vermiform individuals attach to the renal appendages by means of short thigmotactic cilia which cover the anterior end. The remainder of the body, which is actively involved with nutrient uptake and division, hangs free in the fluid-filled coelomic space. Reproduction takes place by unequal, transverse fission or budding at the posterior end of the body.

Two distinct budding patterns are observed, monotomy and palintomy. In young hosts, the ciliates all produce large, single buds, termed apotomites, which resemble the parents (Fig. 1-31). When detached they are transformed directly into second generation tropho-tomonts. By means of this initial budding process the number of ciliates is

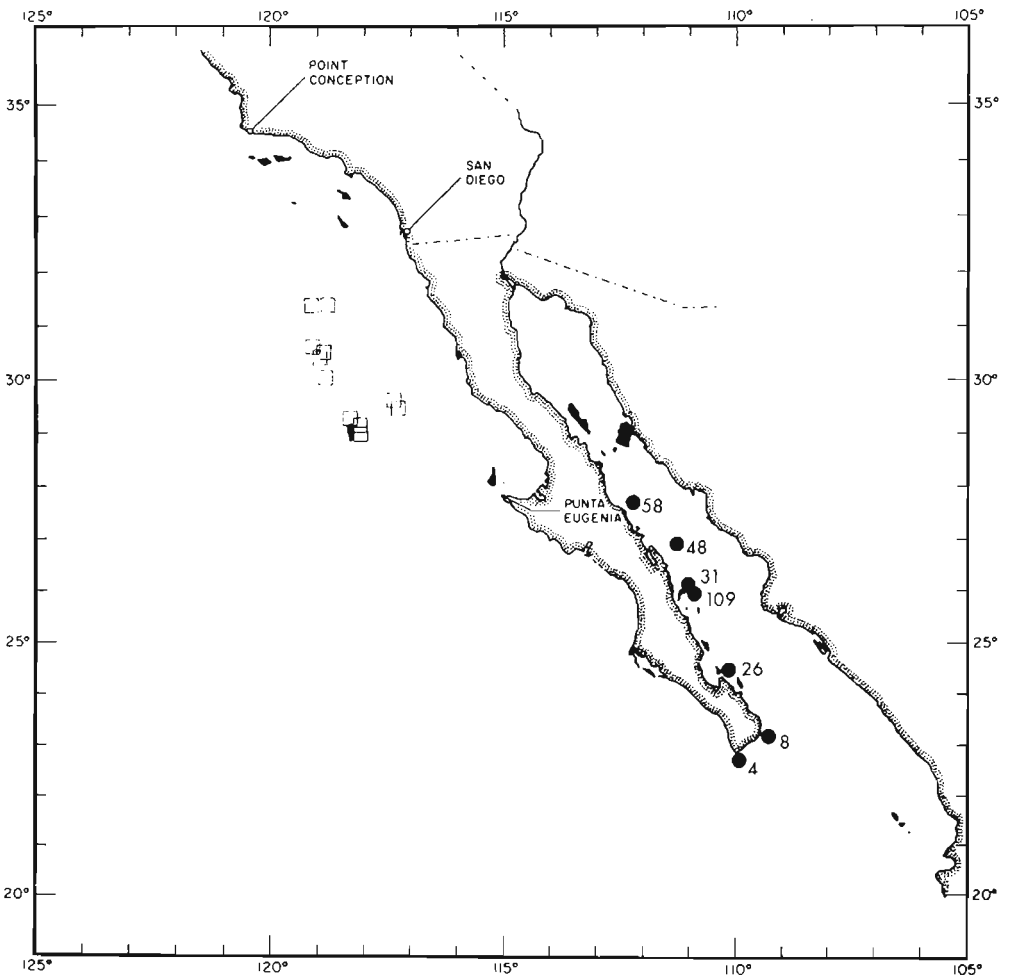


Fig. 1-28: *Chromidina cortezi.* Map showing station localities of *Pterygioteuthis* capture: *P. giardi* (circles) and *P. gemmata* (squares). Black circles: stations where squid hosts were infected with the apostome ciliate. All *P. gemmata* examined were uninfected. Station number indicated next to each circle for reference to Fig. 1-29. (After Hochberg, 1971.)

continually increased within the renal sacs until eventually the renal habitat is saturated with ciliates. The second divisional phase, palintomy, is probably triggered by chemical factors related to the density of parasites or maturation of the host. During palintomy, a multiple fission process takes place which produces long chains of 8, 12, or 24 small buds (Figs 1-32 and 1-33). Tiny, ovoid dispersal stages, termed tomites eventually are formed which bear little resemblance to the parent tropho-tomonts. The tomites exit through the renal pores to the exterior with the passage of urine.

Once in the sea, the ciliates swim about until they contact a euphausiid, copepod or another appropriate crustacean host. The tomites encyst on the mouth parts and setaceous appendages of the crustacean host (Sewell, 1951). Phoronts, or encysted resting stages, of several sizes have been found, indicating that the ciliates undergo a series of growth

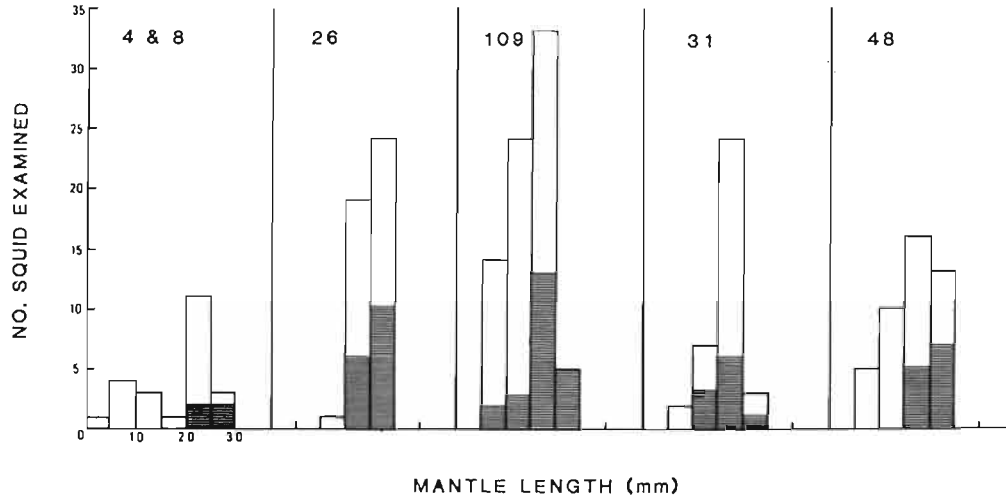


Fig. 1-29: *Chromidina cortezi*. Graph of size class distributions for the squid *Pterygioteuthis giardi* in the Gulf of California, Mexico. Number in upper left of each column refers to station numbers indicated on map in Fig. 1-28. Stations arranged from left to right going up the Gulf of California. Shaded area: squid infected with the apostome ciliate. (After Hochberg, 1971.)

phases. Euphausiids are known to molt every few days. As in other apostome cycles, it is presumed that the ciliates excyst with each molt, feed on exuvial fluids in the cast off moult, grow, and then reencyst on another host individual (Trager, 1957; Bradbury and Trager, 1967; Hochberg, 1982a). Eventually a size is attained which is capable of infecting a cephalopod, and the cycle begins again.

The maximum length of vermiform stages in the cephalopod renal organs ranges from 400 to 2000 μm depending on the species. Two basic body shapes occur. *Chromidina coronata* has an inflated anterior end and a conspicuous crown of elongate cilia, whereas in *C. elegans* and *C. cortezi* the anterior end is not swollen and the ciliary crown is lacking (Fig. 1-27). In other ways the species are almost identical. The infraciliature of the trophotomonts consists of a tight dextral helix, continuous without breaks from the anterior to the posterior pole. Typically 12 to 14 kineties are present (Fig. 1-31, 4 to 6). The macronucleus is an open network of chromatin found throughout the entire body (Fig. 1-33). A tiny spindle-shaped micronucleus is located in the posterior end of the body in the region of the future fission plane (Fig. 1-33, 1). The appearance of trichocycts in the posterior region of the body signals the onset of division. Unlike the foettingeriid ciliates, full grown vegetative stages do not encyst prior to cell division. Mouth, rosette and contractile vacuole, typically found in the foettingeriids, are absent in the vermiform stages within the cephalopod host.

During palintomy the kineties are shortened and straightened with each successive division. The oral field and contractile vacuole develop after detachment from the parent. The appearance of the nuclei also alters markedly during palintomy, as shown by Chatton and Lwoff (1935) and Hochberg (1971). Fully developed tomites range in size from 15 to 30 μm. They are pyriform in shape with a convex dorsal surface and a flat or slightly concave ventral surface. Hovasse (pers. comm.) observed conjugation immediately following release of the tomites.

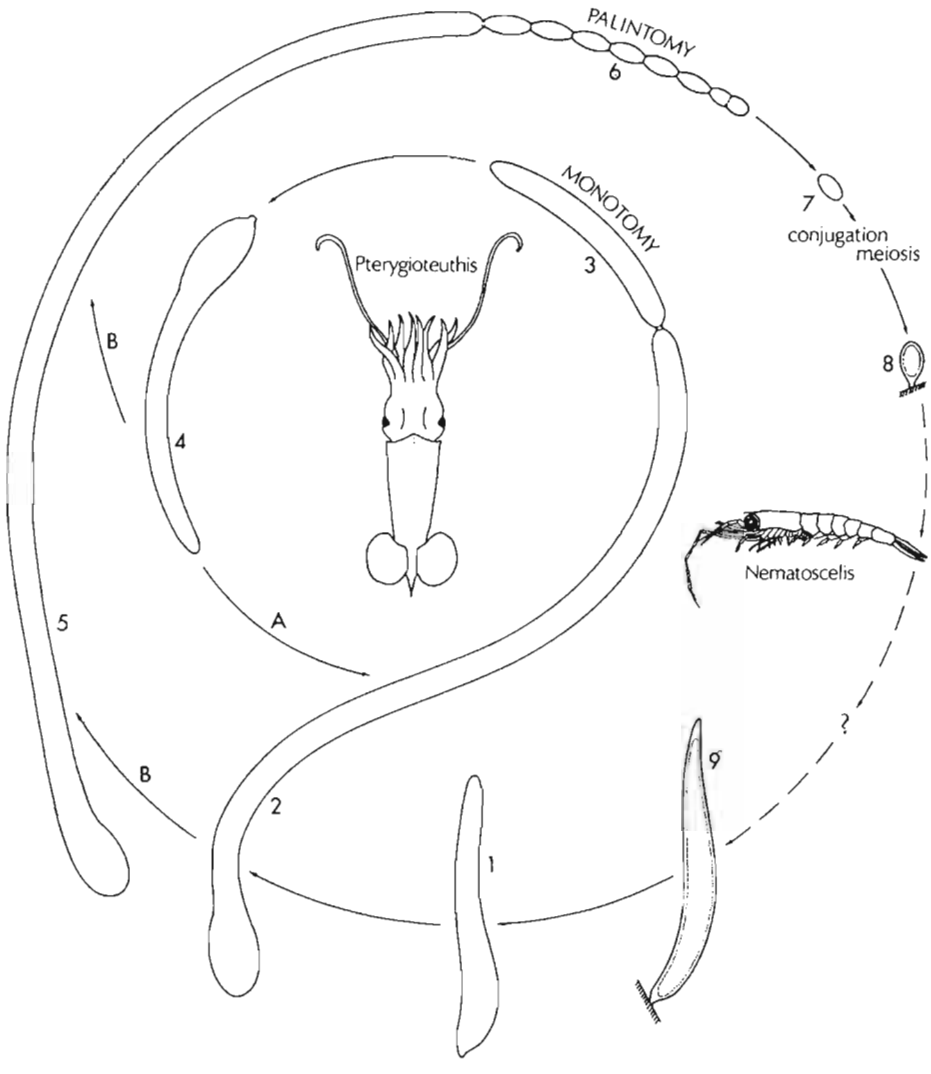


Fig. 1-30: *Chromidina cortezi.* Life cycle of this apostome ciliate in the squid *Pterygioteuthis giardi* and the euphausiid *Nematoscelis difficilis.* (1) Protropho-tomont in renal organs of squid host; (2) primary tropho-tomont; (3) production of apotomite via monotomy (single fission); (4) apotomite; (5) secondary tropho-tomont; (6) production of tomites via palintomy (multiple fission); (7) tomite detached from parent and released into the sea; (8) primary phoront attached to appendages of crustacean host; (9) secondary phoront immediately prior to infection of squid. Density of parasites in squid host: (A) low, (B) high. (After Hochberg, 1982a.)

The tomites of the genus *Chromidina* closely resemble the tomites of many of the crustacean epibionts and it is at this stage that affinities between the apostome families, Foettingeriidae and Chromidinidae, are most evident. This affinity allows comparison of the genus *Chromidina* with the foettingeriids which are considered to be the most primitive of the apostome ciliates. This in turn provides insight into the adaptations which have occurred as a result of an endoparasitic existence (p. 110; Fig. 1–55; Hochberg, 1982a).

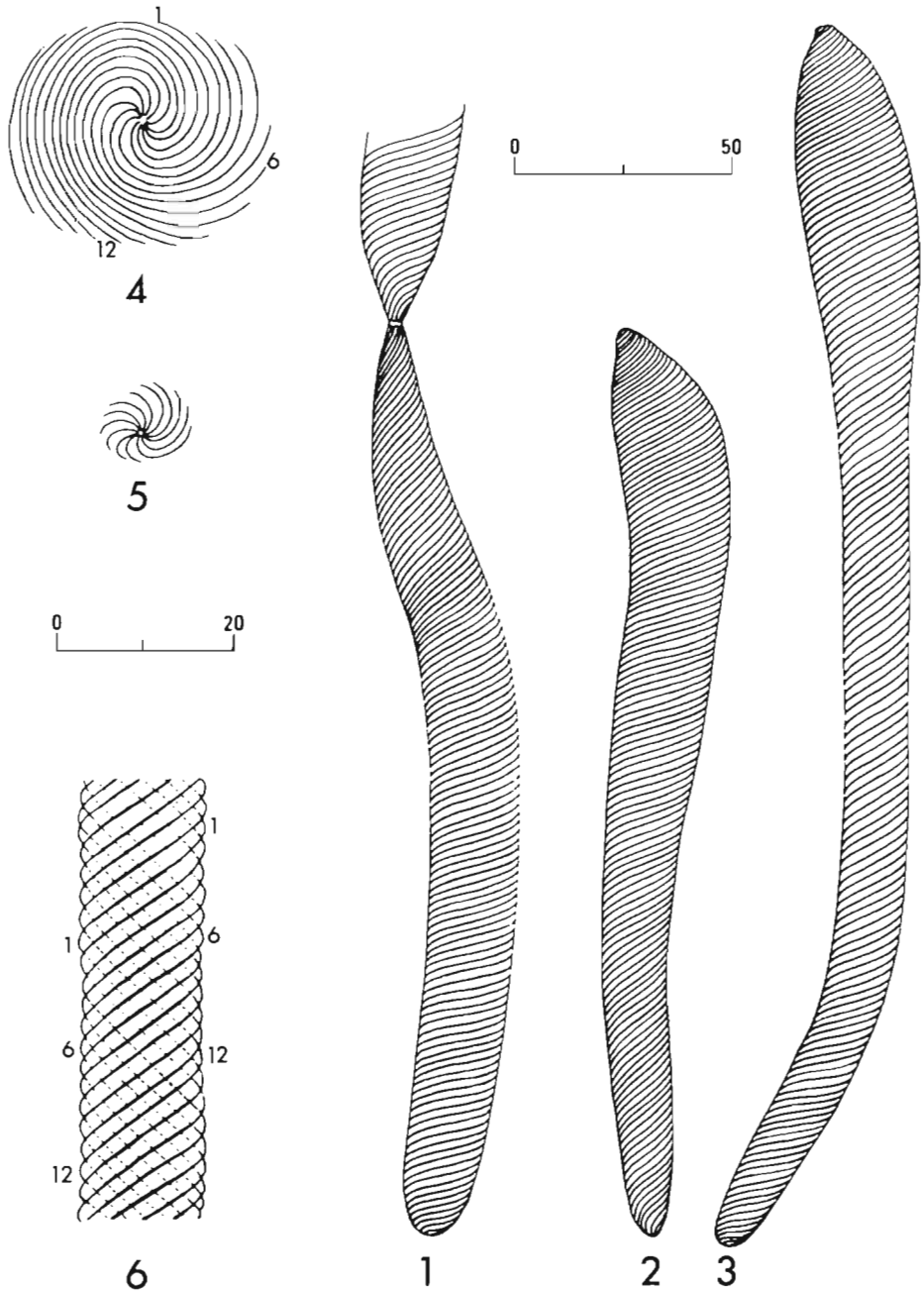


Fig. 1-31: *Chromidina cortezi* from *Pterygioteuthis giardi*. 1 to 3: Details of ciliature of single bud stage attached to parent tropho-tomont, following detachment and subsequent growth. 4 and 5: Details of ciliature at anterior and posterior poles of a full grown tropho-tomont; 12 meridinals indicated. 6: Details of ciliature in the midbody region of a full grown tropho-tomont; note dextral helix formed by the 12 meridinals. Scales in μm.(After Hochberg, 1971.)

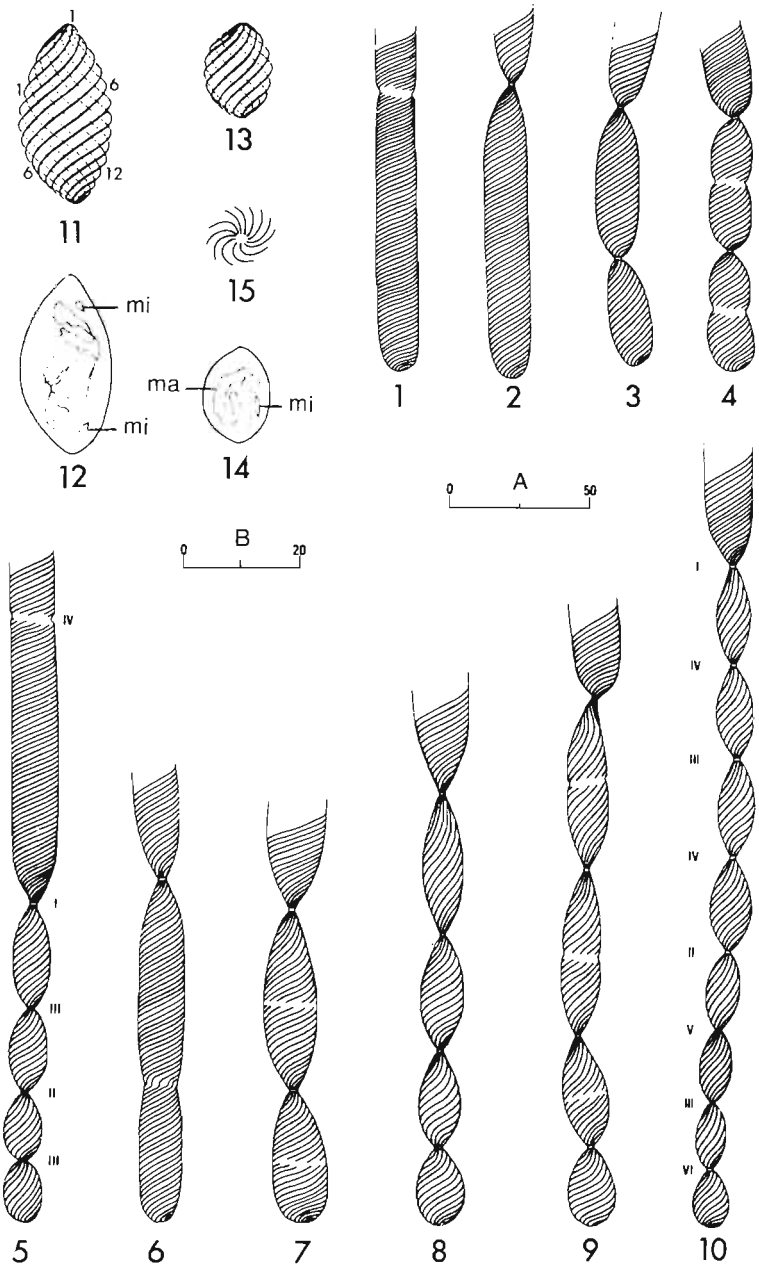


Fig. 1-32: *Chromidina cortezi* from *Pterygioteuthis giardi*. Details of palintomy. 1 to 10: Details of ciliature in 2 different budding patterns which result in the formation of chains of either 4 or 8 protomites; Roman numerals indicate the sequence of budding. 11 to 14: Details of bud stages leading to formation of a protomite, (11, 13) details of ciliature, (12, 14) nuclear details. 15: anterior end of protomite as attached to bud chain showing details of 12 meridinals. (ma) Macronucleus; (mi) micronucleus. Scales in μm. Scale A applies to Figs. 1 to 10; scale B, to Figs. 11 to 15. (After Hochberg, 1971.)

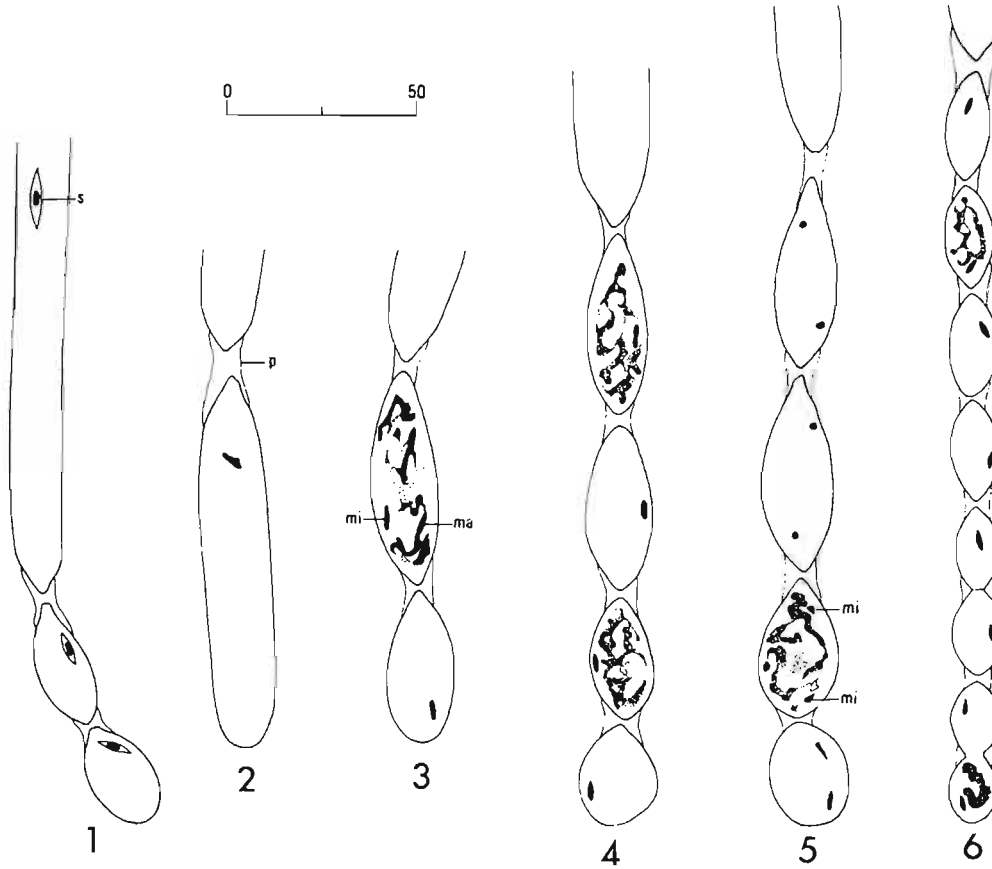


Fig. 1-33: *Chromidina cortezi* from *Pterygioteuthis giardi.* 1: Position of micronucleus in buds and posterior third of tropho-tomont. 2 to 6: Details of nuclear changes which occur during palintomy and the formation of chains of protomites; (2) first-order bud; (3) second-order buds; (4 and 5) third-order buds; (6) fourth-order buds or protomites. (ma) Macronucleus; (mi) micronucleus; (p) pellicle; (s) sheath-surrounding nucleus. Scale in μm. (After Hochberg, 1971.)

Hyptertrophonts of *Chromidina elegans,* measuring up to 5000 μm, occasionally are found. Described by Collin (1914b, 1915) these individuals appear to have penetrated the epithelium of the reno-pancreatic appendages and entered the blood spaces within. There they increase rapidly in size, probably because of high osmotic pressures. The nuclei undergo caryolysis and the cilia are lost. The ciliates, which are thus imprisoned and immobilized, appear degenerative due to attack by phagocytes from the blood of the host.

Small, ovoid infusorians infecting the midgut and digestive glands of cephalopods are placed in the related genus *Opalinopsis.* Two species have been described from the Mediterranean and the English Channel. *Opalinopsis sepiolae* (Fig. 1-34) is reported from *Sepia rondeleti, S. elegans,* and *S. officinalis* (Foettinger, 1881a, b; Gonder, 1905; Dobell, 1909; Collin, 1914b, 1915). What is probably the same species has been observed in *Sepiola atlantica, Sepietta oweniana,* and *Rossia macrosoma. Opalinopsis octopi* has been obtained from *Pteroctopus tetracirrhus* and *Octopus macropus* at Naples and Banyuls (Foettinger, 1881a, b; Hochberg, 1971). Collin (1914a) described a third species of *Opalinopsis* from

Fig. 1-34: *Opalinopsis sepiolae*. Tropho-tomont of this apostome ciliate from digestive organs of *Rossia macrosoma*. (Original.)

the heteropod mollusc, *Carinaria mediterranea,* collected at Villefranche. This latter species has not been studied since first described and should be reexamined. Collin (1914a) promised a review of the genus *Opalinopsis* but it was never forthcoming. Recently several undescribed species of *Opalinopsis* have been found in the genera *Heteroteuthis, Histioteuthis* and *Japatella* off Hawaii and Baja California, Mexico (Table 1-9; Hochberg, 1982b).

Uniformly ciliated tropho-tomonts of the genus *Opalinopsis* move freely through the digestive glands and digestive gland appendages ('liver', 'pancreas', and 'hepatopancreas' of previous authors) of their molluscan hosts. The ciliates attach to the epithelium of the digestive duct or intestine by means of a conical rostrum. In shape, the body is ellipsoid or inversely ovoid, gradually tapering posteriorly. The size varies depending on the species. Cilia are short and arranged in 30 tight helical rows. The macronucleus is a complex, seemingly continuous network located in the medullary zone. Cytostome, rosette and oral ciliature are not present at this stage.

The life cycle of the opalinopsids is incompletely known. Simple, unencysted divisional stages are commonly encountered in the digestive gland. Division is equatorial and monotomic. Long chains of buds are not produced. Conjugation has not been observed. Stages outside the cephalopod are not known.

The taxonomic position of the genera *Opalinopsis* and *Chromidina* has been subject to considerable debate. In the past both genera were typically treated together. An affinity between these 2 genera of highly specialized cephalopod parasites and the apostome ciliates, which typically occur on crustaceans as epibionts, was first proposed by Chatton and Lwoff (1926). Their ideas regarding this relationship were later expanded (1928, 1930). In 1931 Chatton and Lwoff reported morphological stages in the life cycle of the genus *Chromidina* that were very similar to stages in the life cycle of the apostomes, especially the foettingeriids. The extensive monograph by Chatton and Lwoff (1935) provides the only definitive study of the genera *Chromidina, Opalinopsis,* and the apostomes as a whole.

In reviewing the systematic literature Hochberg (1971) pointed out the distinctness of the 2 genera and placed each in its own family. Hochberg also reaffirmed placement of the chromidinids in the Order Apostomatida. The opalinopsids, on the other hand, are regarded as perhaps outside the defined limits of the apostomes (Chatton and Lwoff, 1935; Hochberg, 1971, 1982b). Though widely accepted as a well characterized and relatively homogenous group, the apostomes are still an enigmatic assemblage without definite affinities in the accepted scheme of ciliate evolution (Corliss, 1979).

Young (1972) reported the presence of several types of ciliates in the stomach and cavity surrounding the buccal mass of a single specimen of the bathypelagic octopod *Eledonella pygmaea* (originally identified by Young as *Bolitaena microcotyla*). Hochberg (unpubl.) found similar large ovoid ciliates amid food debris in the stomachs of numerous specimens of the genera *Pterygioteuthis* and *Abraliopsis* examined off Hawaii. These ciliates remain as yet unidentified as to species.

An unusual group of oval, flattened ciliates whose taxonomic affinities are unclear has been found in the renal organs of oceanic squids from the central and eastern North Pacific Ocean. The parasites occur in 30 to 60 % of adult squids and often are found concurrently with species of *Chromidina*. Species of squids in the following genera, *Abraliopsis, Pyroteuthis, Histioteuthis, Gonatopsis,* and *Sthenoteuthis* are infested (Hochberg, unpubl.). At least 5 new species are involved. In the same area Hochberg (unpubl.) found an undescribed mobiline peritrich in the renal sacs of over 30 % of adults in the small enoploteuthid squid *Abraliopsis brevis*.

Agents: Protistae *incertae sedis*

Large protists of unknown taxonomic affinities commonly are found on the gills of a number of species of pelagic cephalopods (McLean and co-authors, 1987). When the mantle cavity of an infected host is opened, large numbers of white to yellow-orange ovoid cysts are easily visible with the naked eye (Fig. 1-35). The cyst-like parasites are attached by a holdfast to the gills and often are partially embedded within small pockets on the surface or between the gill lamellae (Fig. 1-36, 2). Although the holdfast appears to penetrate the gill tissues (Fig. 1-36, 3) there is no evidence of necrosis. The walls of the cyst are convoluted or elaborated into a series of low triangular plates separated by distinct

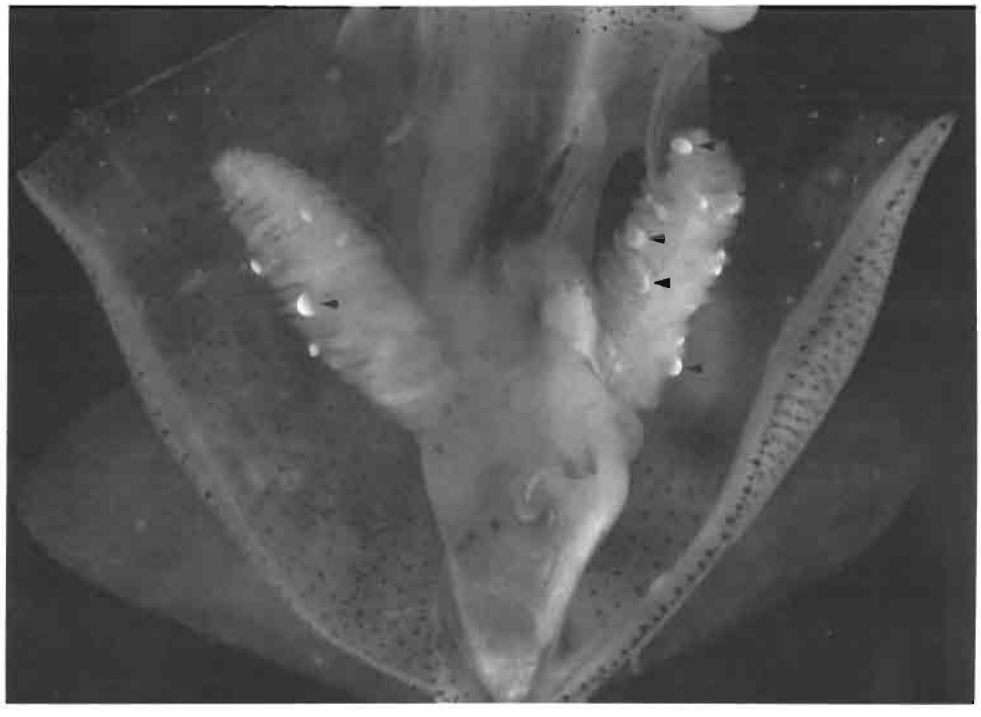


Fig. 1-35: *Hochbergia* sp. from *Abralia trigonura* off Hawaii. Mantle cavity opened to show large protists encysted on gills of host (arrows); note variation in sizes of cysts. (Original.)

grooves (Fig. 1-36, 1). A single large nucleus is centrally located and trichocysts are present, which suggests affinities with dinoflagellates or ciliates (McLean and co-authors, 1987).

A complex of species appears to be represented based on the size and shape of the cyst and the number of plates present. Shinn and McLean (1989) have erected a new genus for the parasite in *Moroteuthis robusta* from the North Pacific Ocean. In *Hochbergia moroteuthensis* the cysts range in length from 1.19 to 1.99 mm, whereas cysts from *Abralia trigonura* and *Histioteuthis dofleini* captured off Hawaii average 1.10 and 0.56 mm in length, respectively. Related parasites have been found in species of the genera *Heteroteuthis, Pyroteuthis, Pterygioteuthis, Chiroteuthis, Ctenopteryx, Octopoteuthis* and *Eledonella* off Hawaii and in *Abraliopsis, Gonatopsis, Gonatus, Berryteuthis, Galiteuthis, Japatella,* and *Vampyroteuthis* off the west coast of North America.

Large excysted stages (1 to 2.5 mm) of what appears to be the same parasite occasionally are encountered in the digestive tracts of *Abralia trigonura* and other oceanic squids off Hawaii (Fig. 1-37). In my unpublished observations the incidence of hosts infected with gill cysts approached 100 % in squids over 10 mm ML, while the excysted stages were found in the caecum of the same host species in less than 50 % of the hosts examined.

A second type of protist was found by R. Young (pers. comm.) while working on the anatomy of *Vampyroteuthis infernalis*. The tiny parasite is attached to the velar filament of

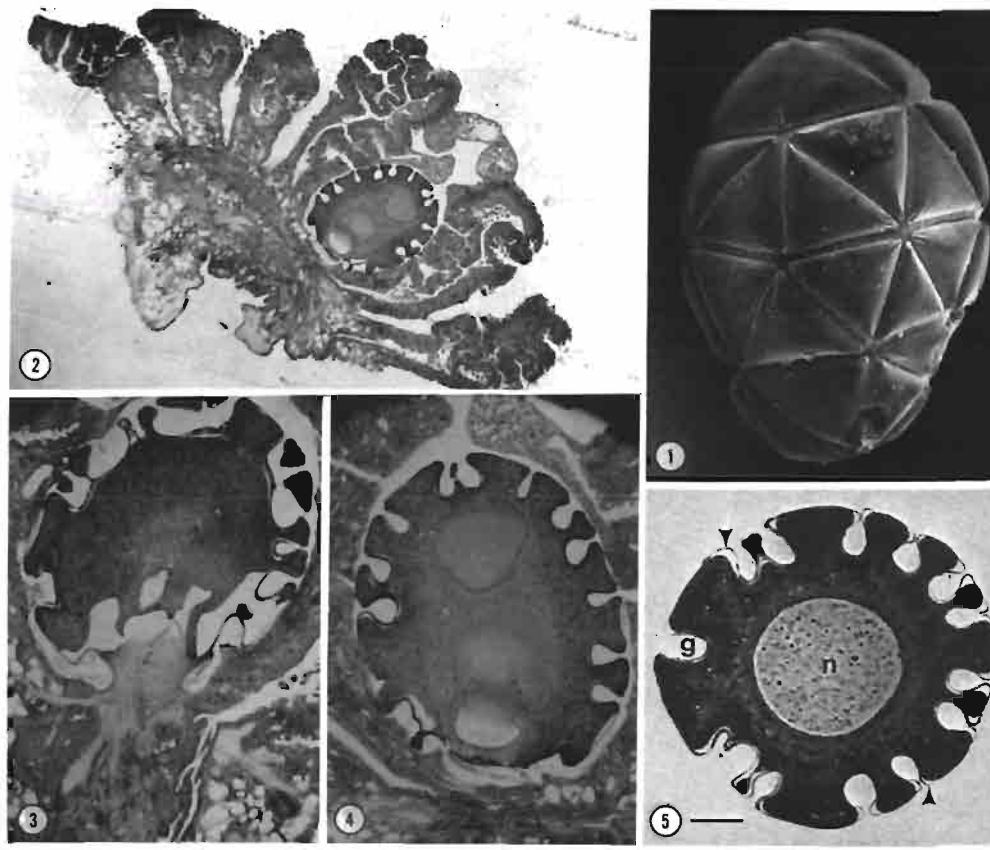


Fig. 1-36: *Hochbergia* spp. from gills of various cephalopod hosts. 1: *Moroteuthis robusta*. 2 to 4: *Abralia trigonura*. 5: *Histioteuthis dofleini*. (1) SEM of protist, note smooth plates and broad groove between plates; (2) section of gills of host with parasite in place; (3) longitudinal section of protist through holdfast region; (4) longitudinal section through middle of cyst; (5) cross section through protist, note lobes of cytoplasm extending into convolutions of cyst wall. Scale bar = 50 μm. (g) Groove between plates; (n) nucleus; arrows: cyst wall. (1 and 5 after McLean and co-authors, 1987; 2 to 4 Original.)

the cephalopod host (Fig. 1-38). From the sections examined there appears to be a suctorial tube that penetrates the epithelial cell of the host. Until additional material is available for study the parasite cannot be further identified.

DISEASES CAUSED BY METAZOANS

Agents: Dicyemida

The dicyemid mesozoans (Fig. 1-39) are a small and puzzling group without definite affinities in the animal kingdom. They exhibit an impressive array of truly unique characters which hold a special curiosity for zoologists. Although first observed by Cavolini (1787) they were first described by Krohn (1839). Later, Erdl (1843) observed that they produced 2 kinds of embryos but it was not until 1849 that von Kölliker provided

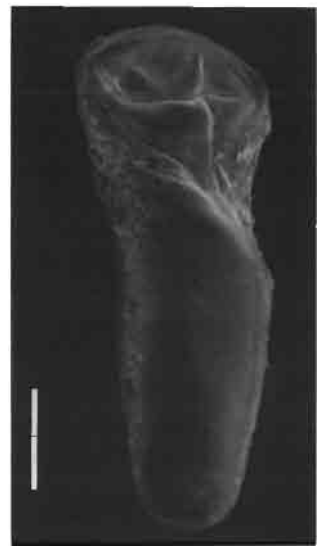


Fig. 1-37: Unidentified protist from caecum of *Abralia trigonura* (SEM). Scale bar = 200 μm. (Original.)

the generic name, *Dicyema,* to denote the presence of 2 stages in the life cycle (1849b). Van Beneden (1876; see also Power, 1877) believed that these simple, cell-constant organisms linked the protozoans and the metazoans and hence proposed the name Mesozoa. The dicyemids, along with the orthonectids, have long been considered classes within the Phylum Mesozoa (see reviews by Braun, 1889–1893; Delage and Hérouard, 1899; Benham, 1901; Garbowski, 1903; Neresheimer, 1908; Borri, 1929; Hyman, 1940, 1959; Mendes, 1940; Stunkard, 1954, 1972, 1982; Dodson, 1956; Czihak, 1958; Grassé, 1961; McConnaughey, 1963, 1968; Ivanov, 1983). The orthonectids parasitize a number of marine invertebrate phyla: Platyhelminthes (turbellarians); Nemertea; Annelida (polychaetes); Mollusca (gastropods, bivalves, but not cephalopods); Echinodermata (crinoids and ophiuroids); and Chordata (ascidians). In light of dissimilar spermatozoon morphologies, internal features and the lack of homologies in stages of life cycles, it is best to treat these two assemblages as separate phyla and to use the term 'Mesozoa' to refer to their grade of organization only (Bresciani, 1971; Hochberg, 1982a; Wirth, 1984). Early investigators related the dicyemids to the protozoans, a position again proposed by Lapan and Morowitz (1974) and supported by DNA analysis. Most students of the group traditionally align the dicyemids with the platyhelminthes as discussed by Stunkard (1954, 1972, 1982) and Gottschalk (1971).

The dicyemids are the most common and characteristic parasites of the excretory organs of cephalopod molluscs. However, aside from their systematics, morphology and embryology, very little is known about this unusual group of organisms. The minute, vermiform organisms attach principally to the renal appendages (Fig. 1-40) where they live and reproduce, doing no apparent harm to the host. In decapods they are found additionally in the reno-pancreatic coelom attached to the digestive duct appendages. In the case of *Dicyemennea brevicephaloides,* vermiform stages are principally located in the pericardium attached to the branchial heart appendages of the host sepiolid, *Rossia*

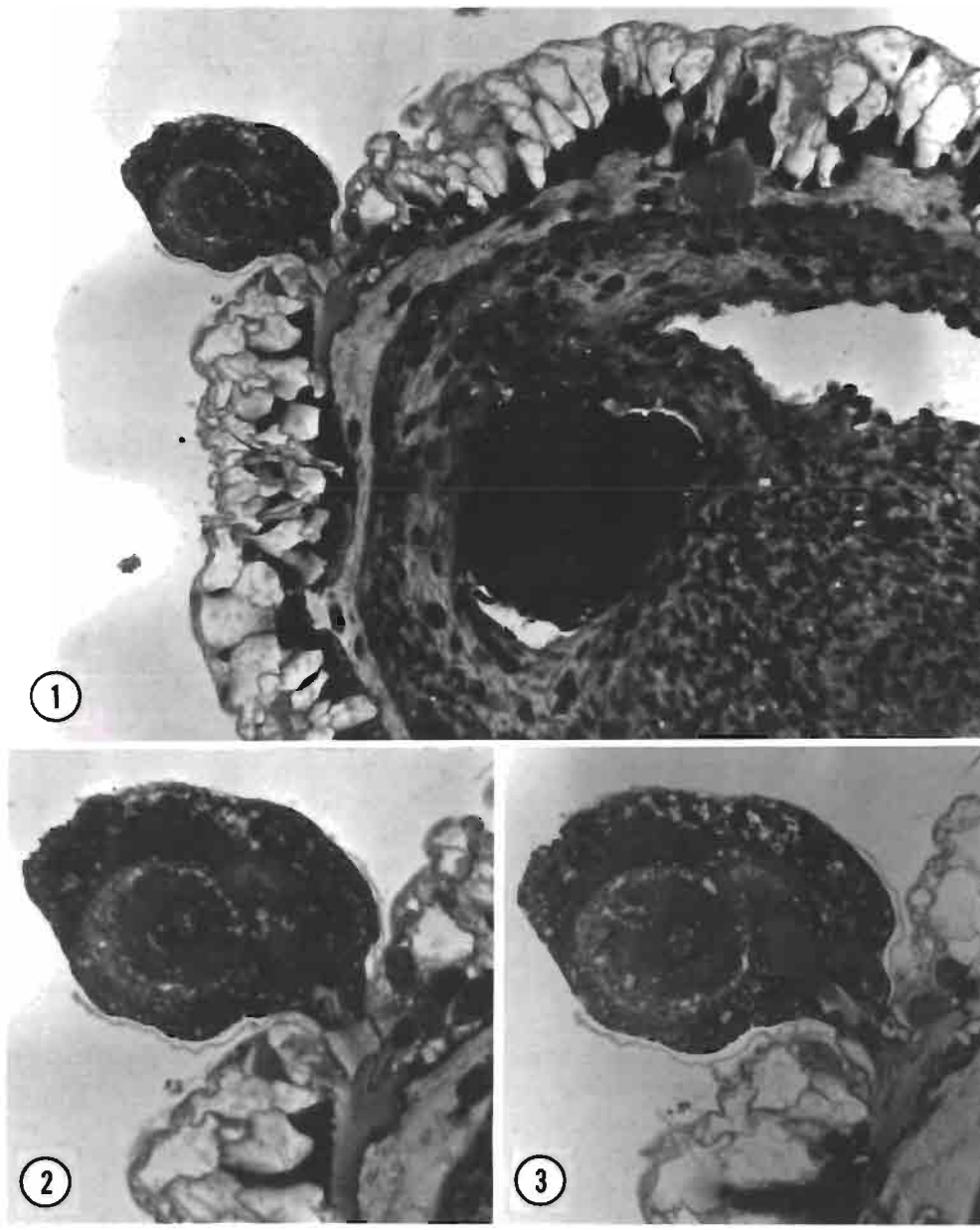


Fig. 1-38: Unidentified protist attached to velar filaments of *Vampyroteuthis infernalis*. 1: Section through velar filament with parasite *in situ*. 2 and 3: longitudinal sections through protist showing 2 views of attachment to host. (Original, provided by R. E. Young.)

pacifica. Other dicyemid species probably occur in this latter site in decapods but simply have been missed in routine dissections. In octopods, the coelomic spaces surrounding the branchial heart appendages are greatly reduced and the peritoneum of the appendages is smooth, hence parasites are unlikely to be present.

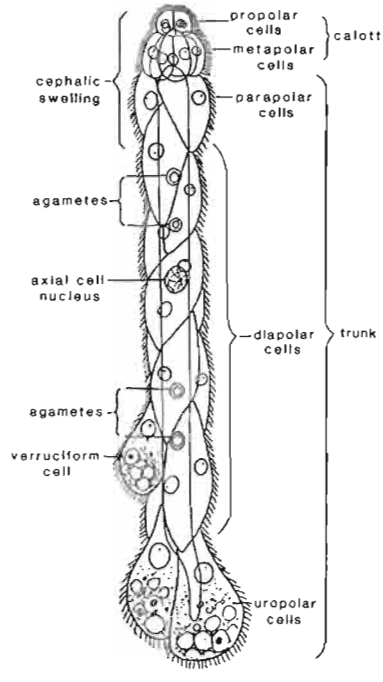


Fig. 1-39: Generalized drawing of a young nematogen stage of a dicyemid with terms that have been applied to various parts of the body. (After McConnaughey, 1951.)

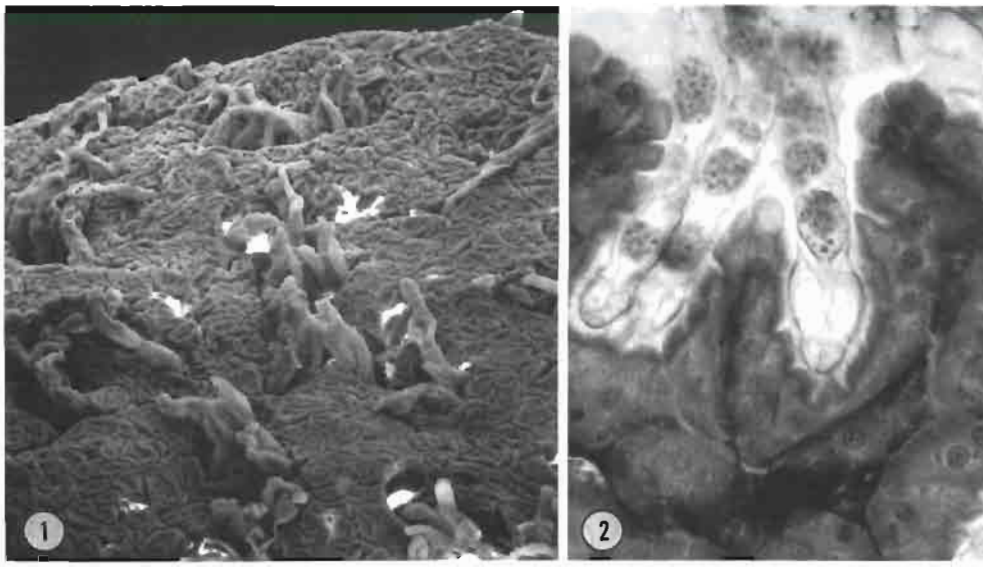


Fig. 1-40: *Dicyema* sp. Under *in situ* conditions in the renal appendages of *Octopus* sp. 1: SEM. 2: Section. (Original.)

The appendages to which the parasites are attached are convoluted, and the epithelial substrate is transport-active and covered with a microvillous brush border. Dense thigmotactic cilia interfinger with the microvillar surface of the appendages to hold the parasites in place. In addition, a viscous mucous envelops the dicyemids and creates an interface between the urine and the excretory structures. Undulations of the body generated by ciliary beat create additional forces directed toward the surface of the excretory appendages. In dicyemids with rounded or pointed calottes the anterior ends are imbedded between the convolutions of the excretory appendages or they may even occupy individual depressions or 'crypts' in the renal surface (Fig. 1-40; Ridley, 1968). Species with flattened calottes attach in sucker fashion directly to the surface of the excretory appendages.

The excretory appendages are surrounded by fluid-filled coelomic spaces into which the dicyemids hang. Periodic and incomplete emptying of these coelomic sacs ensure that the parasites are constantly surrounded by fluids. Ciliary currents continually move the urine past the ruffled uptake surface of the elongate body of the vermiform stages (Figs 1-

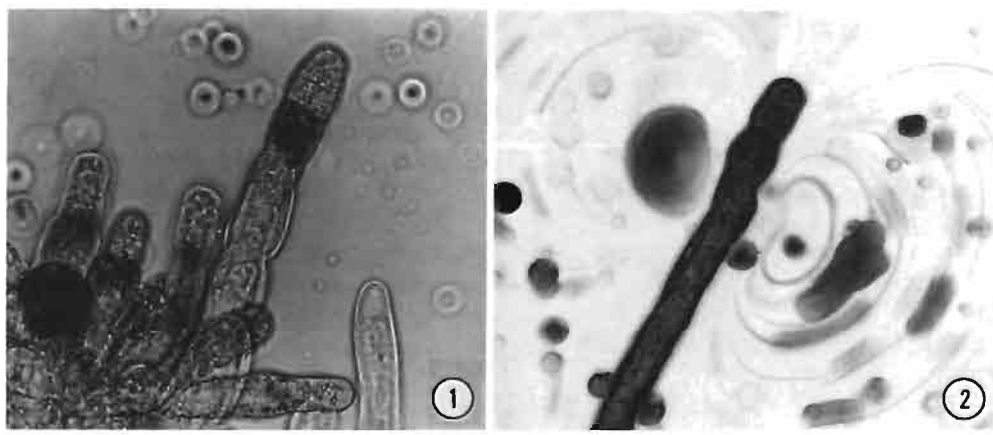


Fig. 1-41: Living dicyemids from renal organs of *Octopus* sp. 1: Cluster of nematogens; note dark, granular parapolar cells below calotte. 2: Nematogen photographed at slow shutter speed to demonstrate ciliary beat which continually moves urine and particulate matter in the renal coelom past the body of the parasite. (Original.)

41 and 1-42). The dicyemids derive all their metabolic requirements from the dissolved nutrients within the excretory fluids. A great deal of study has gone into unraveling the physiology of urine formation and the chemical composition of the urine, but little is known about the actual products utilized by the parasites (for review see Hochberg, 1982a). Since urine from uninfected cephalopods has never been analyzed we do not know what effects the parasites have on the excretory processes nor do we know how they modify the composition of the urine through discharge of their own metabolic wastes.

The dicyemids occupy an interface in the cephalopod host between the excretory fluid and the surface of the excretory appendages. Lapan (1975a) indicated that the dicyemids may facilitate host excretion of NH_3 by contributing to the acidification of the urine. If this is true then they should be termed symbionts and not parasites. This concept holds only for

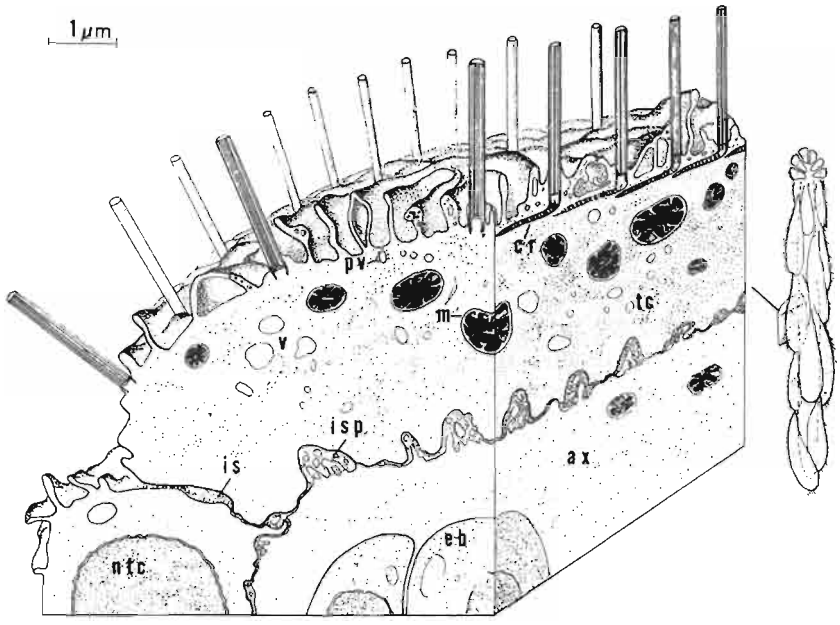


Fig. 1-42: *Pseudicyema truncatum*. Reconstruction of morphology as revealed by electron microscope. (ax) Axial cell; (cr) ciliary rootlet; (eb) embryo in the axial cell; (is) intercellular space between 2 adjacent trunk cells; (isp) intercellular space between a trunk cell and the axial cell; (m) mitochondrion; (ntc) nucleus of a trunk cell; (pv) pinocytosis vesicle; (v) vacuole. (After Bresciani and Fenchel, 1965.)

areas where 100 % of the cephalopod hosts are infested but it does not explain the relationship in areas where potential hosts are uninfected.

More than 40 species of benthic or epibenthic cephalopods, representing at least 15 genera, are currently reported to host dicyemids (Table 1-10). They occur in cuttlefishes, sepiolids, loliginid squids and octopods (both cirrate and incirrate groups). Each cephalopod host species harbors either a single species of dicyemid or, more typically, a complex of species that is distinct at the generic level. As examples: *Octopus rubescens* is host to *Dicyema apollyoni* (= *D. balamuthi*), *Dicyemennea adscita* and *Conocyema adminicula* in the southern part of its distributional range, whereas *D. apollyoni* and *Dicyemennea brevicephala* occur in the northern part (McConnaughey, 1949a; Hochberg, 1971); *O. tehuelchus* harbors *Dicyema australis, Dicyema platycephalum* and *Conocyema marplatensis* (Penchaszadeh, 1968, 1969; Penchaszadeh and Christiansen, 1970); *Benthoctopus magellanicus* is infected with *Dicyema benthoctopi* and *Dicyemennea littlei* (Hochberg and Short, 1970); and *Sepia officinalis* may concurrently host *Dicyemennea gracile, Pseudicyema truncatum* and *Microcyema vespa* (Nouvel, 1947).

In what is now a classic study, Pickford and McConnaughey (1949) distinguished a sibling species complex of 2-spotted octopuses off California on the basis of the dicyemid parasites (Fig. 1-43) and the size of the eggs of the host. The older established name, *Octopus bimaculatus*, applies to the small egg (2 to 3 mm) species which ranges from southern California to Panama. It specifically harbors *Dicyemennea abelis*. Pickford and McConnaughey's new species, *O. bimaculoides*, is a short-range endemic which overlaps

Table 1-10
Dicyemid parasites from cephalopods (Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	Locality	Source
ORDER SEPIOIDEA			
Sepia elegans (= *S. biserialis*)	*Dicyema macrocephalum*	Mediterranean (Italy, Monaco, France)	van Beneden (1876), Whitman (1883), Hartmann (1906), Nouvel (1947)
S. elegans	*Dicyema schulzianum*	Mediterranean (Italy, Monaco, France)	van Beneden (1876), Nouvel (1935a, 1947)
Sepia elliptica	*Dicyema ganapatii*	Western Bay of Bengal (India)	Kalavati and co-authors (1984)
S. elliptica	*Dicyemennea coromadelensis*	Western Bay of Bengal (India)	Kalavati and co-authors (1978)
Sepia esculenta	*'Pseudicyema truncatum'*	Western North Pacific Ocean (Japan)	Nouvel (1947)
Sepia officinalis	*Dicyemennea gracile* (= *Dicyemina Koellikeriana*)	English Channel (England); Mediterranean (Italy, Monaco, France)	Wagener (1857), Whitman (1883), Braun (1887), Nouvel (1933b, 1938b, 1947), Hochberg (unpubl.)
S. officinalis	*Microcyema vespa*	English Channel (France); Mediterranean (Italy, Monaco, France)	van Beneden (1882), Lameere (1916b, 1918b, 1919), Hartmann (1939), Schartau (1940)
S. officinalis	*Pseudicyema truncatum*	Eastern North Atlantic Ocean (France); English Channel (France); Mediterranean (Italy, France, Spain)	Whitman (1883), Nouvel (1933b, 1947)
Sepia orbignyana	*Dicyemennea gracile* (rare)	Mediterranean (France, Spain)	Wagener (1857), Whitman (1883), Nouvel (1933b, 1938b, 1947)
S. orbignyana	*Pseudicyema truncatum*	Mediterranean (Italy, Monaco, France)	Whitman (1883), Nouvel (1933b, 1937, 1947)
ORDER SEPIOLIOIDEA			
Rondeletiola minor	*Dicyema rondeletiolae*	Mediterranean (Italy, Monaco, France)	Nouvel (1944)
R. minor	*Dicyema schulzianum*	Mediterranean (Italy, Monaco, France)	van Beneden (1876), Whitman (1883), Nouvel (1935a, 1944, 1947)
Rossia macrosoma	*Pseudicyema truncatum*	Mediterranean (Italy, Monaco, France)	Whitman (1883), Nouvel (1944, 1947)
Rossia pacifica	*Dicyema acuticephalum*	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1960)

Table 1-10 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER SEPIOLIOIDEA			
Rossia pacifica	*Dicyema caudatum*	Okhotsk Sea (USSR)	Bogolepova-Dobrokhotova (1960)
R. pacifica	*Dicyema oligomerum*	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1962)
R. pacifica	*Dicyemennea brevicephala*	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1962)
R. pacifica	*Dicyemennea brevicephaloides*	Okhotsk Sea (USSR); Japan Sea (USSR)	Bogolepova-Dobrokhotova (1962)
R. pacifica	*D. brevicephaloides*	Eastern North Pacific Ocean (Washington, Oregon, California, USA)	Hoffman (1965), Hochberg (1987)
R. pacifica	*Dicyemennea curta*	Okhotsk Sea (USSR)	Bogolepova-Dobrokhotova (1962)
R. pacifica	*Dicyemennea filiformis*	Okhotsk Sea (USSR)	Bogolepova-Dobrokhotova (1962)
R. pacifica	*D. filiformis* (= *D. parva*)	Eastern North Pacific Ocean (Washington, Oregon, California, USA)	Hoffman (1965), Hochberg (1987, unpubl.)
R. pacifica	*Dicyemennea nouveli* (= *D. californica*)	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1962), Hochberg (unpubl.)
R. pacifica	*Dicyemennea rossiae*	Okhotsk Sea (USSR); Japan Sea (USSR)	Bogolepova-Dobrokhotova (1962)
Sepietta neglecta	*Dicyema rondeletiolae*	Mediterranean (Italy, Monaco, France)	Nouvel (1944)
Sepietta obscura	*Dicyema macrocephalum*	Mediterranean (Italy, Monaco, France)	van Beneden (1876), Whitman (1883), Nouvel (1944, 1947)
Sepietta oweniana	*Dicyema macrocephalum*	Mediterranean (Italy, Monaco, France)	van Beneden (1876, 1878), Whitman (1883), Nouvel (1944, 1947)
S. oweniana	*Dicyema rondeletiolae*	Mediterranean (Italy, Monaco, France)	Nouvel (1944)
Sepiola rondeleti	*Dicyema moschatum*	Mediterranean (Monaco, France)	Whitman (1883), Nouvel (1944, 1947)
Sepiola steenstrupiana	*Dicyema macrocephalum*	Mediterranean (Italy, Monaco, France)	van Beneden (1876), Whitman (1883), Nouvel (1944, 1947)

Table 1-10 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER SEPIOLIOIDEA			
Sepiola steenstrupiana	*Dicyema microcephalum*	Mediterranean (Italy, Monaco, France)	Whitman (1883), Nouvel (1944, 1947)
ORDER TEUTHOIDEA			
Loligo opalescens	*Dicyemennea nouveli*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1959)
Loligo sp.	*Dodecadicyema loligoi*	Western Bay of Bengal (India)	Kalavati and Narasimhamurti (1980)
Photololigo duvauceli (= *Loligo*)	*Dicyema nouveli*	Western Bay of Bengal (India)	Kalavati and co-authors (1984)
Sepioteuthis lessoniana	*Dicyema orientale*	Western North Pacific Ocean (Japan)	Nouvel and Nakao (1938), Nouvel (1947)
ORDER OCTOPODA			
Grimpoteuthis glacialis	*Dicyemennea discocephala*	Antarctic Ocean (Orkney Islands)	Hochberg and Short (1983)
Bathypolypus sponsalis	*Pleodicyema delamarei*	Mediterranean (Spain)	Nouvel (1961)
Benthoctopus magellanicus	*Dicyema benthoctopi*	Western South Atlantic Ocean (Falkland Islands)	Hochberg and Short (1970)
B. magellanicus	*Dicyemennea littlei*	Western South Atlantic Ocean (Falkland Islands)	Short and Hochberg (1970)
Eledone cirrhosa (= *E. Aldrovandi*)	*Dicyemennea eledones* (= *D. Mülleri*)	Eastern North Atlantic Ocean (Sweden, Norway); English Channel (France); Mediterranean (Italy, France)	Wagener (1857), Claparede and Lachmann (1861), Whitman (1883), Braun (1887), Hartmann (1906), Nouvel (1933b, 1935b, 1937, 1938b, c, 1947)
E. cirrhosa	*Dicyemennea lameerei*	English Channel (France)	Nouvel (1932a, 1933b, 1938b, 1947)
Eledone moschata	*Dicyema moschatum* (= *Dicyemella Wageneri*)	Mediterranean (Italy, France)	van Beneden (1876), Whitman (1883), Hartmann (1906), Nouvel (1938b, 1947)
E. moschata	*Dicyemennea eledones*	Mediterranean (Italy, France)	Claparede (1857), Wagener (1857), Whitman (1883), Nouvel (1947)

Table 1-10 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER OCTOPODA			
Octopus bimaculatus	*Dicyemennea abelis*	Eastern North Pacific Ocean (California, USA; Mexico)	McConnaughey (1949a), Pickford and McConnaughey (1949)
O. bimaculatus	*Dicyemennea granularis*	Eastern North Pacific Ocean (California, USA; Mexico)	McConnaughey (1949a), Pickford and McConnaughey (1949)
Octopus bimaculoides (= *O.* sp.)	**Dicyema acciaccatum*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1949a)
O. bimaculoides (= *O.* sp.)	**Dicyema acheroni*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1949a)
O. bimaculoides	*Dicyema sullivani*	Eastern North Pacific Ocean (California, USA; Mexico)	McConnaughey (1949a, b, 1951, 1960), Pickford and McConnaughey (1949)
O. bimaculoides (= *O.* sp.)	**Dicyemennea abasi*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1949a)
O. bimaculoides (= *O.* sp.)	**Dicyemennea abbreviata*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1949a)
O. bimaculoides	*Dicyemennea californica*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1941, 1949a, 1951), Pickford and McConnaughey (1949)
O. bimaculoides	*Dicyemennea granularis*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1949a, 1951), Pickford and McConnaughey (1949)
Octopus briareus	*Dicyema briarei*	Gulf of Mexico (Florida, USA)	Short (1961)
Octopus californicus	*Dicyemennea nouveli* (= *D. californica*)	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1963), Hochberg (unpubl.)
O. californicus	*Dicyemodeca dogieli*	Okhotsk Sea (USSR)	Bogolepova (1957)
Octopus defilippi	*Dicyema microcephalum*	Mediterranean (Italy, France)	Whitman (1883), Nouvel (1947)

* Host identity unknown, ocellate species reported by McConnaughey (1949) to be *Octopus bimaculoides*

Table 1-10 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER OCTOPODA			
Octopus dofleini (= *O.* sp., *O. apollyon*/ *hongkongensis*)	*Dicyemodeca deca* (= *Conocyema*)	Eastern North Pacific Ocean (Washington, Oregon, USA)	McConnaughey (1957)
O. dofleini	*Dicyemennea abreida*	Eastern North Pacific Ocean (Washington, Oregon, USA)	McConnaughey (1957)
O. dofleini	*Dicyemennea nouveli*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1959)
O. dofleini	*D. nouveli* (= *D. californica*)	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1963), Hochberg (unpubl.)
O. dofleini	*Dicyemodeca dogieli*	Okhotsk Sea (USSR)	Bogolepova (1957)
Octopus joubini	*Dicyema apalachiensis*	Gulf of Mexico (Florida, USA)	Short (1962)
O. joubini	*Dicyema hypercephalum*	Gulf of Mexico (Florida, USA)	Short (1962)
Octopus macropus	*Dicyema paradoxum* (= *D. clausianum*)	Mediterranean (Italy, Monaco, France)	von Kölliker (1849b), van Beneden (1876), Whitman (1883), Nouvel (1933b, 1947), Stunkard (1948)
Octopus maorum	*Dicyema knoxi*	Western South Pacific Ocean (New Zealand)	Short (1971)
O. maorum	*Dicyema maorum*	Western South Pacific Ocean (New Zealand)	Short (1971)
O. maorum	*Dicyemennea kaikouriensis*	Western South Pacific Ocean (New Zealand)	Short and Hochberg (1969)
Octopus membranaceus (= *O. fangsiao*)	*Dicyema misakiensis*	Western North Pacific Ocean (Japan)	Nouvel (1947)
Octopus minor (= *O. variabilis*)	*Dicyemodeca dogieli*	Okhotsk Sea (USSR); Japan Sea, (USSR)	Bogolepova (1957)

Table 1-10 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER OCTOPODA			
Octopus ochotensis	*Dicyemennea nouveli* (= *D. californica*)	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1963), Hochberg (unpubl.)
O. ochotensis	*Dicyemodeca dogieli*	Okhotsk Sea (USSR); Japan Sea (USSR)	Bogolepova (1957)
Octopus rubescens (= *O.* sp., *O. apollyon*)	*Conocyema adminicula* (= *Dicyemodeca sceptrum*)	Eastern North Pacific Ocean (California, USA; Mexico)	Wheeler (1897), McConnaughey (1949a), Hochberg (unpubl.)
O. rubescens (= *O. apollyon*)	*Dicyema apollyoni* (= *D. coluber*, *D. balamuthi*)	Eastern North Pacific Ocean (California, USA; Mexico)	Wheeler (1897), Nouvel (1947), McConnaughey (1949a), Hochberg and Fields (1980), Hochberg (unpubl.)
O. rubescens (= *O.* sp.)	*Dicyemennea adscita* (= *D. whitmanii*)	Eastern North Pacific Ocean (California, USA; Mexico)	Wheeler (1897), McConnaughey (1949a, 1951), Hochberg (unpubl.)
O. rubescens (= *O. apollyon*)	*Dicyemennea brevicephala*	Eastern North Pacific Ocean (California, USA; Mexico)	McConnaughey (1941, 1949a, 1951), Hochberg (unpubl.)
O. rubescens (= *O.* sp.)	*Dicyemennea nouveli*	Eastern North Pacific Ocean (California, USA)	McConnaughey (1959), Hochberg (unpubl.)
Octopus salutii (= *O. saluzzi*)	*Dicyemennea eledones*	Mediterranean (Italy, France)	Wagener (1857), Whitman (1883), Hartmann (1906), Nouvel (1935b, 1937, 1947)
Octopus tehuelchus	*Conocyema marplatensis*	Western South Atlantic Ocean (Argentina)	Penchaszadeh and Christiansen (1970)
O. tehuelchus	*Dicyema australis*	Western South Atlantic Ocean (Argentina)	Penchaszadeh (1969)
O. tehuelchus	*Dicyema platycephalum*	Western South Atlantic Ocean (Argentina)	Penchaszadeh (1968)
Octopus vulgaris	*Conocyema polymorpha*	Mediterranean (Italy, Monaco, France)	van Beneden (1882), Hartmann (1939), Nouvel (1932b, 1947)

Table 1-10 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER OCTOPODA			
Octopus 'vulgaris'	*Dicyema acuticephalum*	Western North Pacific Ocean (Japan)	Nouvel and Nakao (1938), Nouvel (1947), Bogolepova-Dobrokhotova (1960)
O. 'vulgaris'	*Dicyema aegira*	Western North Atlantic Ocean (Florida, USA); Gulf of Mexico (Florida, USA)	McConnaughey and Kritzler (1952), Short and Damian (1966, 1967)
O. 'vulgaris'	*Dicyema bilobum*	Gulf of Mexico (Florida, USA)	Couch and Short (1964)
O. vulgaris (= *O.* sp., *O. rugosus*)	*Dicyema megalocephalum*	Eastern North Atlantic Ocean (Mauritania)	Nouvel (1934, 1947)
O. 'vulgaris'	*Dicyema misakiense*	Western North Pacific Ocean (Japan)	Nouvel and Nakao (1938), Nouvel (1938c, 1947)
O. vulgaris (= *O.* sp., *O. rugosus*)	*Dicyema monodi*	Eastern North Atlantic Ocean (Mauritania)	Nouvel (1934, 1947)
O. vulgaris (= *O.* sp., *O. rugosus*)	*Dicyema paradoxum* (= *D. clausianum*)	English Channel (France); Mediterranean (Monaco, France); Eastern North Atlantic Ocean (Mauritania)	von Kölliker (1849b), van Beneden (1876), Whitman (1883), Nouvel (1932b, 1933b, 1934, 1947)
O. 'vulgaris'	*Dicyema typoides* (= *D. typus*)	Western North Atlantic Ocean (Florida, USA); Gulf of Mexico (Florida, USA)	McConnaughey and Kritzler (1952), Short (1964)
O. vulgaris	*Dicyema typus*	English Channel (France, England); Eastern North Atlantic Ocean (France); Mediterranean (Italy, Monaco, France)	van Beneden (1876), Nouvel (1932b, 1946, 1947)
O. vulgaris	*Dicyemennea lameerei*	English Channel (France); Eastern North Atlantic Ocean (France); Mediterranean (Italy, Monaco, France)	Nouvel (1932a, b, 1933b, 1947)
Pareledone charcoti	*Dicyemennea antarcticensis*	Antarctic Ocean (Antarctica)	Short and Hochberg (1970), Hochberg (unpubl.)
Pareledone turqueti	*Dicyemennea antarcticensis*	Antarctic Ocean (Antarctica)	Short and Hochberg (1970)

Table 1-10 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER OCTOPODA			
Robsonella australis	*Dicyema robsonellae*	Western South Pacific Ocean (New Zealand)	Short (1971)
R. australis	*Dicyemennea rostrata*	Western South Pacific Ocean (New Zealand)	Short and Hochberg (1969)
Unidentified octopod	*Dicyema caudatum*	Okhotsk Sea (USSR)	Bogolepova-Dobrokhotova (1960)
Unidentified octopod	*Dicyema madrasensis*	Western Bay of Bengal (India)	Kalavati and co-authors (1984)
Unidentified octopod	*Dicyema octopusi*	Western Bay of Bengal (India)	Kalavati and co-authors (1984)
Unidentified octopod	*Dicyema oligomerum*	Okhotsk Sea (USSR)	Bogolepova-Dobrokhotova (1960, 1963)
Unidentified octopod	*Dicyemennea antarcticensis*	Antarctic Ocean (Antarctica)	Short and Hochberg (1970)
Unidentified octopod	*Dicyemennea brevicephaloides*	Okhotsk Sea (USSR)	Bogolepova-Dobrokhotova (1962)
Unidentified octopod	*Dicyemennea dogieli*	Bering Sea (USSR)	Bogolepova-Dobrokhotova (1962)
Unidentified octopod	*Dicyemennea eltanini*	Antarctic Ocean (Orkney Islands)	Short and Powell (1969)
Unidentified octopod	*Dicyemennea filiformis*	Okhotsk Sea (USSR)	Bogolepova-Dobrokhotova (1962)
Unidentified octopod	*Dicyemennea longinucleata*	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1962)
Unidentified octopod	*Dicyemennea nouveli* (= *D. californica*)	Japan Sea (USSR)	Bogolepova-Dobrokhotova (1963), Hochberg (unpubl.)
Unidentified octopod	*Dicyemodeca dogieli*	Okhotsk Sea (USSR); Japan Sea (USSR)	Bogolepova (1957), Bogolepova-Dobrokhotova (1960)

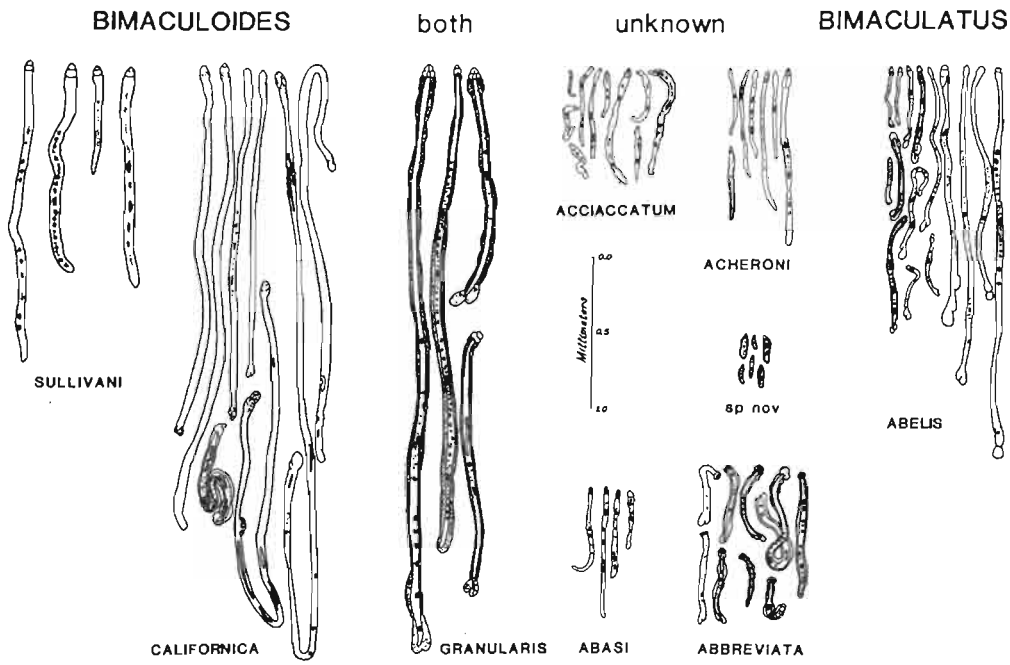


Fig. 1-43: Dicyemids present in the 2-spotted octopus complex off California (USA) and Baja California (Mexico). Two species are restricted to *Octopus bimaculoides,* 1 species occurs only in *O. bimaculatus,* and 1 species occurs in both host species. Five species have been reported from an unknown ocellate host which is now thought to be *O. bimaculoides.* (Original; assembled from figures in McConnaughey, 1949a, 1951.)

with *O. bimaculatus* in the northern part of the range. *O. bimaculoides* has large eggs (10 to 15 mm) and is host to *Dicyema sullivani* and *Dicyemennea californica.* A single species, *Dicyemennea granularis,* occurs in both hosts. An additional five species were recorded by McConnaughey (1949a) from an unknown ocellate octopus in Balboa Bay, California (USA). These latter 5 species have not been found since and the host specimens were not saved but they are believed to be *O. bimaculoides* since this octopus more typically resides in mud flat areas along the California coast.

Rossia pacifica provides one of the best examples of a host infected by a wide diversity of dicyemids on both sides of the North Pacific Ocean (Fig. 1-44; see also Table 1-10). Nine species have been described from *Rossia pacifica* (Bogolepova, 1957; Bogolepova-Dobrokhotova, 1960, 1962, 1963; Hoffman, 1965, Hochberg, 1987). Such a high diversity is very unusual and suggests that a complex of host species may be involved. In the Atlantic Ocean, 9 species also have been described from *Octopus vulgaris* (Table 1-10). Recent evidence suggests that a 2-species complex is involved. The type species occurs in the Mediterranean and eastern North Atlantic and a second distinct species is found in the western North Atlantic and the Gulf of Mexico. Resolution of complex cephalopod taxonomy problems involving cryptic or sibling species may be clarified or resolved by a critical examination of host specific dicyemid parasites.

Dicyemids parasitize only benthic or epibenthic cephalopods, although the distribution is by no means universal. In temperate and polar waters adult, benthic cephalopods

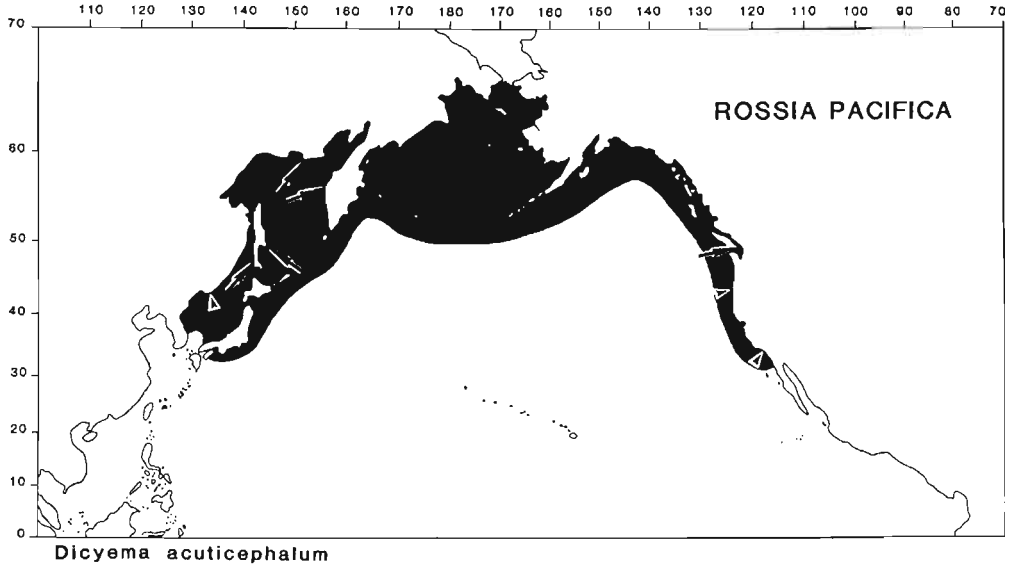


Fig. 1-44: *Rossia pacifica*. Distribution of this sepiolid with an indication of the species of dicyemids and localities of collection (arrows) in the Western (Okhotsk Sea and Japan Sea) and Eastern North Pacific Ocean. (Original.)

generally are 100 % infected. It is exceptional for an octopus to be uninfected in these areas (Raabe, 1933). In the tropics and off oceanic islands no cephalopods have been reported to be infected (see Koshida and co-authors, 1986). In subtropical regions the infection rates are variable and range from 10 to 20 %. The reasons behind these distribution patterns are not known and need to be explored in more detail.

Initial infections normally occur in very young animals, either immediately following hatching, in cephalopods with demersal juveniles, or following settlement to the bottom, in those host species with planktonic larval stages. Dicyemids have never been encountered in holopelagic or oceanic cephalopods. McConnaughey (1959) reported a species of *Dicyemennea* in *Loligo opalescens*, and Aldrich (1964) reported a single dicyemid in a single specimen of *Illex illecebrosus*. These reports always have been suspect until Kalavati and Narasimhamurti (1980) and Kalavati and co-authors (1984) described several new dicyemids from 2 species of *Loligo* collected in the Bay of Bengal (India) thus confirming the presence of dicyemids in neritic squids (Table 1–10).

Nouvel (1947) and McConnaughey (1949a) reviewed the dicyemids and hosts known up to that time. A number of species have been described subsequently from a variety of geographical localities; East coast of Russia (Bogolepova, 1957; Bogolepova-Dobrokhotova, 1960, 1962); France (Nouvel, 1961); Florida (USA), and the Gulf of Mexico (McConnaughey and Kritzler, 1952; Short, 1961, 1962, 1964; Couch and Short, 1964);

West coast of North America (McConnaughey, 1949b, 1957, 1959, 1960; Hoffman, 1965); Argentina (Penchaszadeh, 1968, 1969; Penchaszadeh and Christiansen, 1970); New Zealand and the Antarctic (Short and Hochberg, 1969, 1970; Short and Powell, 1969; Hochberg and Short, 1970, 1983; Short, 1971).

Genera are determined by the number and orientation of cells in each tier of the

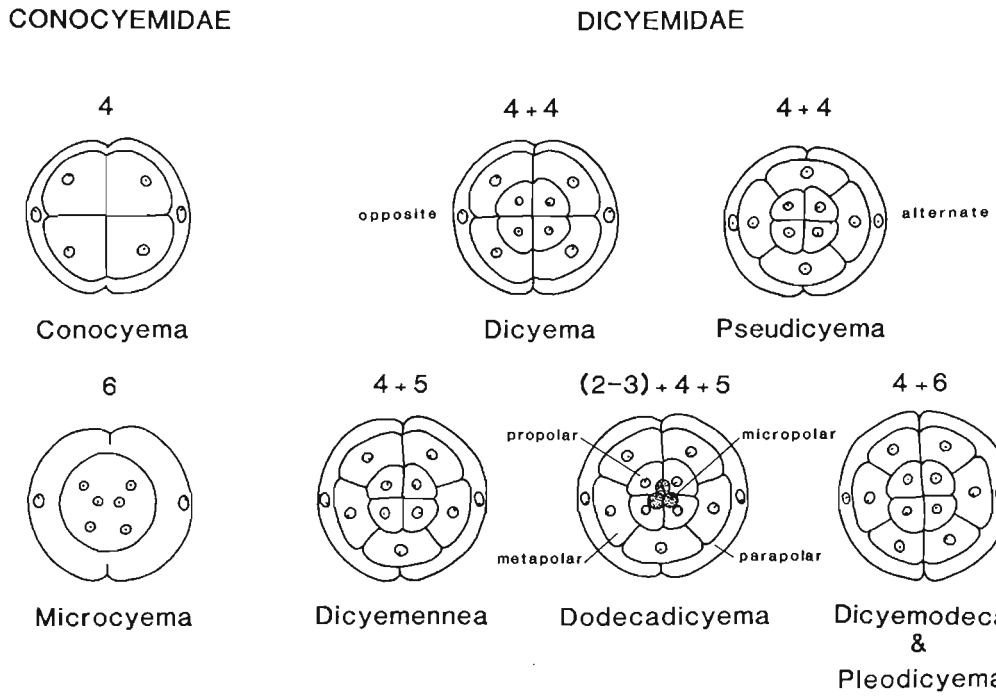


Fig. 1-45: Enface views of the calottes of genera in the 2 families of Dicyemida. Numbers above each drawing indicate variation in number of cells comprising the propolar and metapolar tiers. (Original.)

calotte (Fig. 1-45), the presence or absence of abortive axial cells (Nouvel, 1936) and the presence or absence of syncytial stages. Eight genera are currently recognized and placed in 2 families — Dicyemidae: *Dicyema* (35 species), *Pseudicyema* (1), *Dicyemennea* (25), *Dicyemodeca* (1), *Pleodicyema* (1), and *Dodecadicyema* (1); Conocyemidae: *Conocyema* (1 species), and *Microcyema* (1). At least 2 and possibly 3 new genera await description.

In the Family Dicyemidae the genera *Dicyema, Dicyemennea* and *Dicyemodeca* are easily identified by the respective presence of 4, 5 or 6 metapolar cells in the calotte. In all cases 4 propolar and 2 parapolar cells are present (Fig. 1-45). In the genus *Pseudicyema* cells of the propolar tier alternate with cells of the metapolar tier whereas in the genus *Dicyema* the orientation of cells in the calotte is opposite. The status and validity of *Pseudicyema* have not been resolved but the genus should probably be considered a junior synonym of *Dicyema*. In addition, *Pleodicyema* is regarded as a synonym of *Dicyemodeca*. The number of metapolar cells is the same in both genera and only the shapes of the cephalic swelling differ. In *Pleodicyema* the cephalic region is bluntly rounded whereas in *Dicyemodeca* it is flattened. On the basis of shape differences seen in other genera (Fig. 1-46) this character difference does not warrant generic separation (Hochberg, unpubl.).

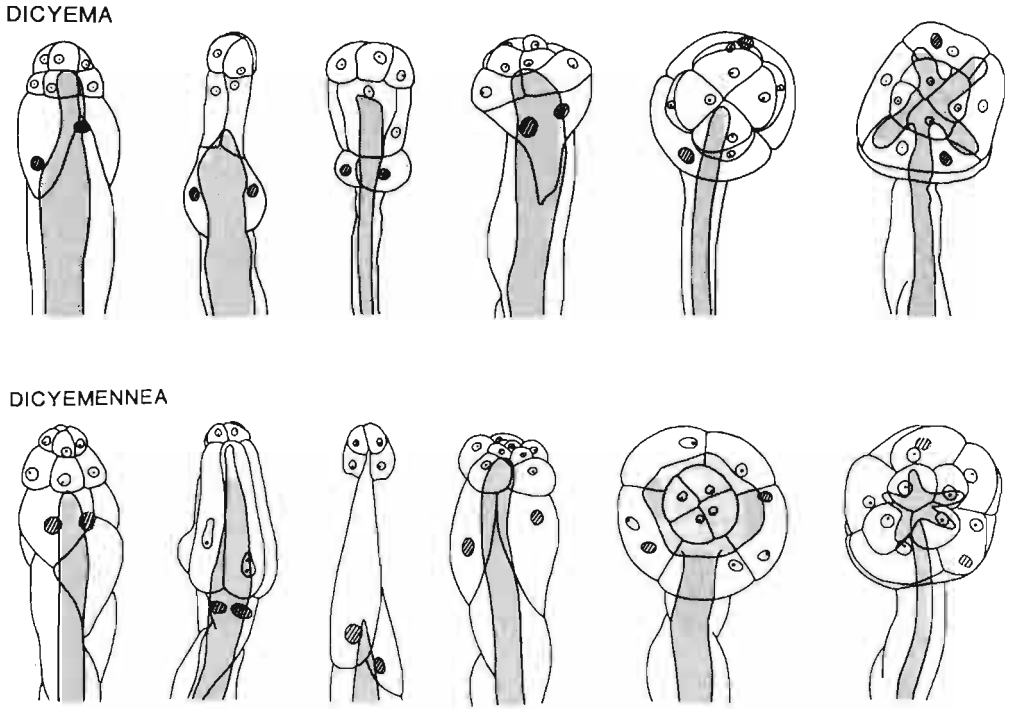


Fig. 1-46: Variations in size and shape of the cephalic swelling (propolar, metapolar and parapolar cells) and the anterior extension of the axial cells in the genera *Dicyema* and *Dicyemennea*. The nuclei of the parapolar cells are cross-hatched and the axial cells are shaded. (Original.)

The newly erected genus *Dodecadicyema* (Fig. 1-45) resembles *Dicyemennea* in the presence of five metapolar cells. However, *Dodecadicyema* is unusual in the presence of 2 or 3 cells which are located on top of the propolar tier. These cells are termed micropolars (Hochberg, unpubl.) and appear to be distinct enough to warrant separate genetic status.

There is considerable confusion regarding generic designations in the family Conocyemidae and, in fact, the family needs to be extensively reexamined. The adults of *Conocyema polymorpha* (Fig. 1-47) are irregular in shape and lack external cilia. Twelve somatic cells are present. The 4 large cells in the calotte appear to represent metapolar cells but no propolar cells are evident. Of the remaining cells, 2 parapolar cells and 6 trunk cells are present. Unusually shaped cuneiform embryos (Fig. 1-47, 4) are present in nematogens and not the more typical vermiform embryos. Three other species have been placed in the genus *Conocyema* but, strictly speaking, they do not belong there because they all have distinct tiers of both propolar and metapolar cells and typical vermiform embryos. *C. deca* belongs in the genus *Dicyemodeca,* whereas *C. adminicula* and *C. marplatensis* both have 5 metapolar cells and hence should be placed in the genus *Dicyemennea* (Hochberg, unpubl.).

Microcyema vespa (Fig. 1-48) is another unusual conocyemid found in *Sepia officinalis*. Adults are irregular in shape, lack external cilia and the somatic cells are fused into a syncytium in which the cell boundaries are difficult or impossible to make out. Ten

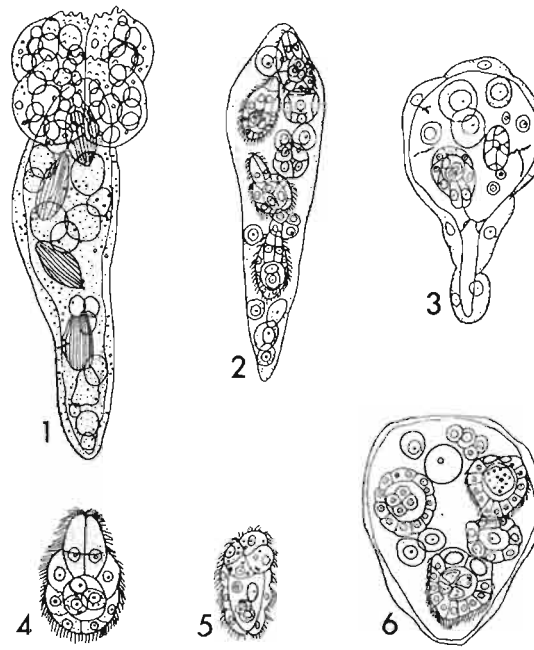


Fig. 1-47: *Conocyema polymorpha*. 1 to 3: Nematogens with cuneiform embryos. 2: Axial cell of nematogen after treatment with acetic acid. 4: Cuneiform embryo. 5: Young nematogen before cilia are lost. 6: Rhombogen with infusoriform embryos. (From van Beneden, 1882; after Grassé, 1961.)

somatic nuclei are present. Posterior to the cephalic swelling 2 nuclei represent trunk cells. A cluster of 6 nuclei collectively represent the propolar and metapolar cells of the calotte and 2 more nuclei equal the parapolar cells. A distinctive shaped Wagener's embryo is present in the axial cell of the parent nematogen (Fig. 1-48, 1)

To date 68 species of dicyemids have been described (Table 1-10). If we add to this the undescribed species in several collections and the number of potential host species still to be examined, it is possible to project a total of about 200 species in the phylum. Species are characterized by the size of the adult and vermiform embryo stages, the number of cells comprising the body, the shape of the cephalic swelling, the anterior extension of the axial cell, the presence or absence of verruciform cells and the morphology of the infusoriform larvae. Recent descriptions of new species from a number of new host genera has greatly expanded our ideas about the morphological characteristics of the phylum as well as helped to define the limits of geographic distribution and host specificity.

Close examination of the dicyemids reveals a simple structure. In the adult vermiform stages, called nematogens and rhombogens, a single internal, axial cell runs almost the entire length of the body (Fig. 1-39). The total length of the adult vermiform stages ranges from 250 to 10,000 μm, depending on the species. Reproductive products are relegated to the interior of the axial cell of the parent, which functions as a nurse or follicular cell providing both protection and nourishment for the germ cells and developing embryos. The axial cell is surrounded by a jacket of 14 to 40 large ciliated cells, called somatic or trunk cells. The number of cells in the jacket is species specific. The anterior end is modified into a calotte, by which the parasite attaches to the host renal tissue. The calotte

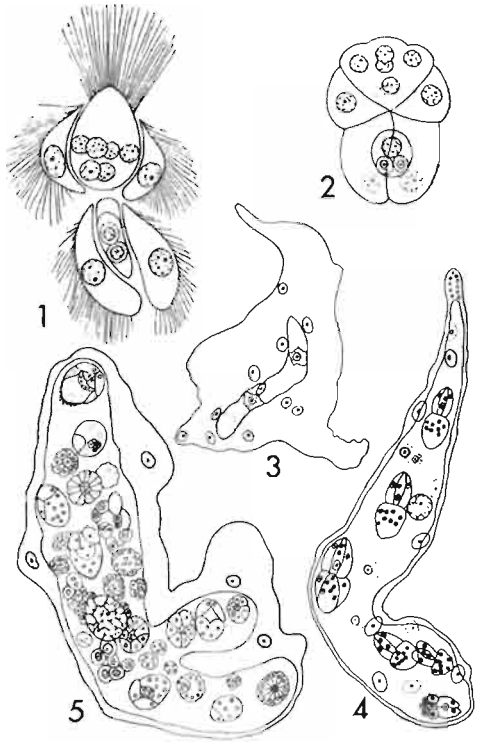


Fig. 1-48: *Microcyema vespa*. 1: Wagener embryo. 2: Embryo from a primary nematogen at the time of eclosion (cilia not represented). 3: Young amoebiform primary nematogen. 4: Adult primary nematogen with Wagener embryos. 5: Rhombogen with 2 infusorigens and infusoriform embryos in various stages of development. (From Lameere, 1916b, 1918a; after Grassé, 1961.)

is covered by short stiff thigmotactic cilia which interdigitate with the brush border of the epithelial cells of the excretory appendages. The shape of the calotte varies a great deal, depending on the species (Fig. 1-46). There is no trace of a differentiated digestive, circulatory, nervous, respiratory, glandular or excretory system. No muscles, sensory receptors, or skeletal elements are present. In fact, nothing comparable to organs, tissues or glands is observed.

The infusoriform, or dispersal stage, is morphologically the most complex stage in the life cycle, and yet, it is remarkably similar from species to species. It has been described in detail by Nouvel (1933a, 1948, 1961) and Short and Damian (1966). Mature larvae are ovoid. All species are ciliated posteriorly and most have 2 large refringent bodies anteriorly. When present, the refringent bodies may account for up to half the weight of the larvae. The dense refractile material which fills the apical cells is now known to be a highly hydrated magnesium salt of inositol hexaphosphate (Lapan, 1975b) and not uric acid as indicated by Nouvel (1948).

Infusoriform larvae range in length from 25 to 50 μm and have a total of either 37 or 39 cells. Internally there is an urn cavity filled with 4 large cells, each containing 1 or 2 smaller germinal cells (Nouvel, 1938a). A relatively large nucleus and the intracellular location of these small cells indicate they are probably germinal cells which give rise to the next

generation. Recent fine structure studies by Bresciani (1971) and Bresciani and Fenchel (1965, 1967); Ridley (1968, 1969); and Matsubara and Dudley (1976a, b) have helped to clarify and resolve many observations on both the vermiform and infusoriform stages in the life cycle.

The life cycle (Fig. 1-49) has been a subject of controversy and, in spite of extensive study, it is still incompletely known (see papers by Lankester 1873; Whitman, 1883; Koeppen, 1892; Wheeler, 1899b; Hartmann, 1904, 1906, 1925; Lameere, 1905–1923; Mesnil and Caullery, 1905a, b; Stunkard, 1937, 1954; Gersch, 1938a, b, 1941a, b; Nouvel, 1947, 1948; McConnaughey, 1951; McConnaughey and McConnaughey, 1954; Hochberg,

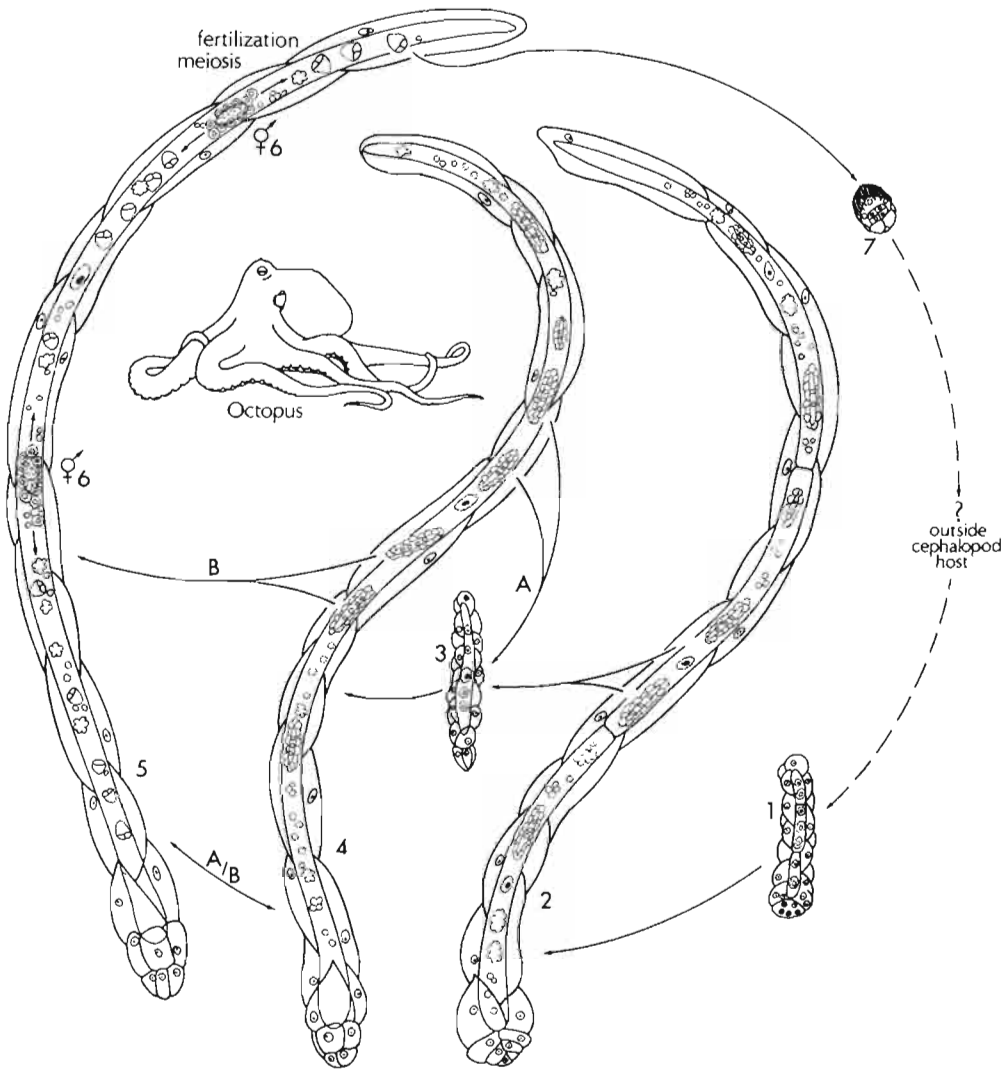


Fig. 1-49: Life cycle of a dicyemid mesozoan. (1) Larval stem nematogen; (2) stem nematogen; (3) vermiform embryo; (4) nematogen; (5) rhombogen; (6) infusorigen; (7) infusoriform released from parent. Density of parasites: (A) low; (B) high. (After Hochberg, 1982a.)

1982a). In its simplest expression it consists of an alternation of essentially isomorphic, parent generations. The embryos of all known stages develop within the axial cell of the parent until they are released through rupture of the parent's body wall. Cleavage is determinant, and a definite cell number is attained early in development. Subsequent growth is by cell enlargement.

The morphological simplicity of the dicyemids is such as to allow mapping of every individual cell during development (Fig. 1-50; Lameere, 1919; McConnaughey, 1938; Bogomolov, 1970; Lapan and Morowitz, 1975). Individual cells are of a size amenable to micromanipulation and several morphogenetic markers are readily apparent. Lapan and Morowitz (1975) maintained dicyemids in vitro in an axenic medium over 3 months. The possibility of culturing dicyemids makes them excellent candidates for use in studying morphogenesis and cytodifferentiation. The early work by Nouvel (1929a, b, 1931) on cytoplasmic contents needs to be repeated with modern technology.

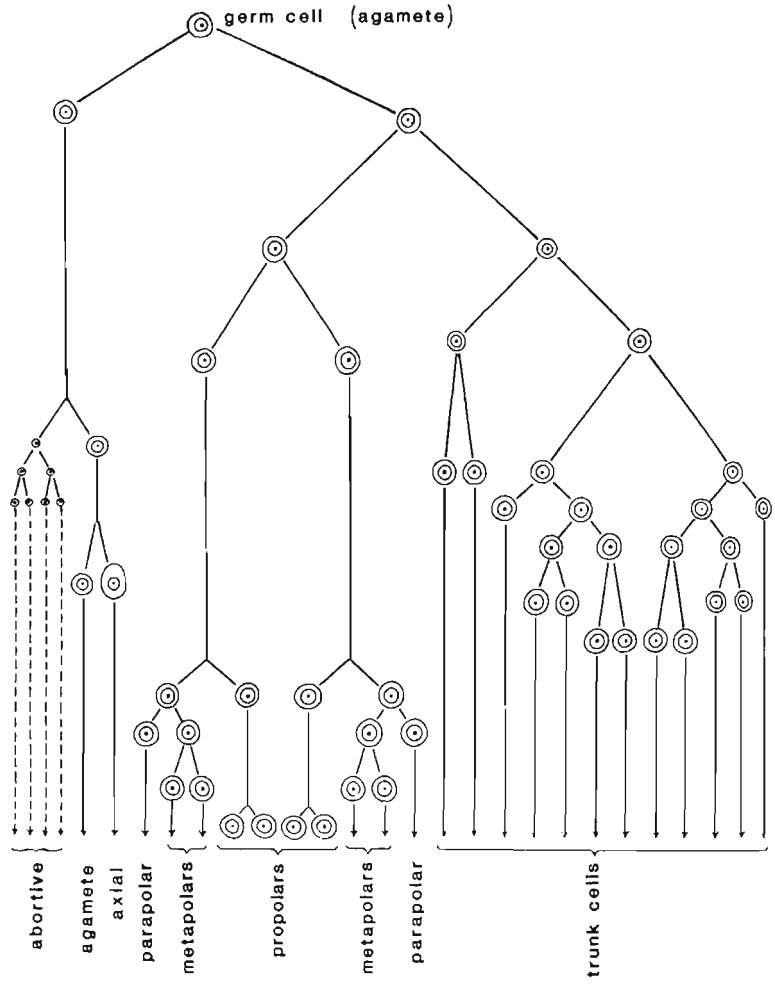


Fig. 1-50: *Pseudicyema truncatum*. Cell lineage and chronology of divisions in the development of the primary nematogen. (From Lamcere. 1919; after Grassé, 1961.)

The mode of entry into the host and the initiation of the infection is not known. Lapan and Morowitz (1972) proposed that germinal cells from the urn of the infusoriform could directly infect the circulatory system of the host and from there penetrate into the kidneys. However, they did not present evidence or experimental data to support their contention. The earliest known stage observed in juvenile cephalopods is termed a larval stem nematogen. It is only rarely encountered and has been described in only a few species. This stage differs from the typical adult vermiform stages principally in having 2 or 3 axial cells

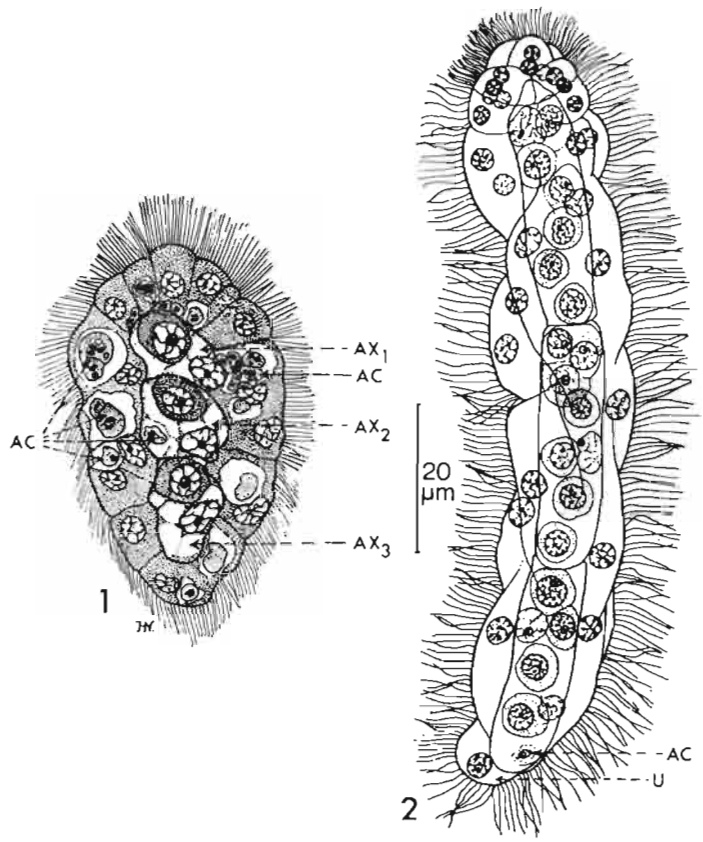


Fig. 1-51: Larval stem nematogens. 1: *Microcyema vespa*. 2: *Dicyema schulzianum*. (AC) Abortive cell residue; (AX) axial cells 1 to 3; (U) uropolar cell. Scale in µm. (After Nouvel, 1937.)

instead of the usual 1 (Fig. 1-51). Subsequently, however, the stem nematogens produce vermiform embryos which have only 1 axial cell.

The stage of the dicyemid cycle appears to depend on the maturity of the host. Immature hosts harbor mixed populations of stem nematogens and nematogens (Table 1-11), all of which contain elongate vermiform embryos (Fig. 1-52) in their axial cells. The embryos develop asexually from agametes (axoblasts) and resemble the parent nematogens by the time they are released. Constant proliferation of daughter nematogens eventually results in an enormous population of dicyemids which fills the renal organs of the cephalopod host.

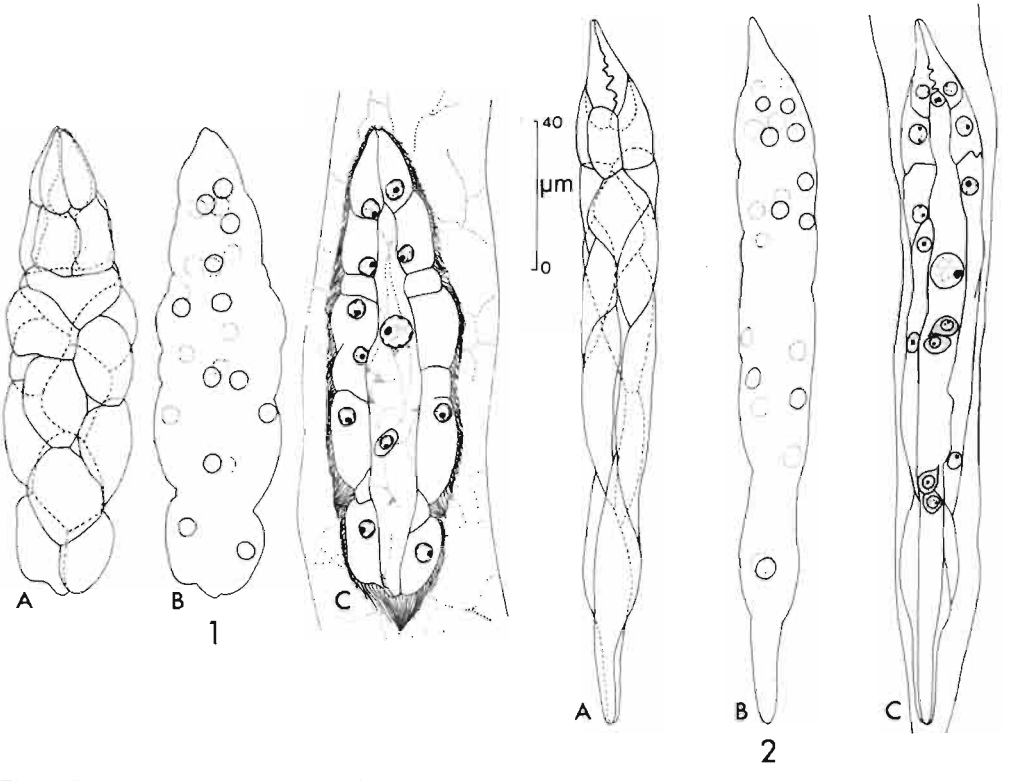


Fig. 1-52: Details of mature vermiform embryos from axial cells of nematogens. 1: *Dicyemennea kaikourensis*. 2: *D. rostrata*. Note presence of abortive axial cell in 2C. (A) Peripheral cell outlines; (B) positions of peripheral cell nuclei; (C) optical section. Scale in µm. (After Short and Hochberg, 1969.)

In older hosts the adult vermiform stage is called a rhombogen. In the axial cell of this parent stage the vermiform embryos are replaced by gamete-producing infusorigens and infusoriform larvae. Long a subject of controversy, the hermaphroditic infusorigen has been described as either an individual or a gonad. The infusorigen (Fig. 1-53) consists of a nearly spherical axial cell which contains all the developmental stages leading to mature spermatozoa, and a jacket composed of oogonia and oocytes. Amoeboid spermatozoa emerge from the axial cell and penetrate periphally located oocytes (Austin, 1964; Short and Damian, 1967; McConnaughey, 1983a, b). The resulting zygotes develop into ovoid embryos which, when full grown, are termed infusoriform larvae (Fig. 1-54; Sponholtz, 1964). After breaking out of the parent body, the infusoriforms escape from the renal environment with the passage of the urine. The fate of this dispersal stage and the phase(s) of the cycle which occur(s) outside the cephalopod host are still a mystery. Several authors have suggested that the infusoriform larvae or their released germinal cells must infect a secondary benthic host since they are not attracted to young cephalopods (Nouvel, 1947; McConnaughey, 1951; Stunkard, 1954). On the other hand, Lapan and Morowitz (1975) recovered dicyemids in the renal organs of *Sepia* reared from eggs in isolated aquaria and exposed only to infusoriform larvae. This indicates that an intermediate host may not be necessary.

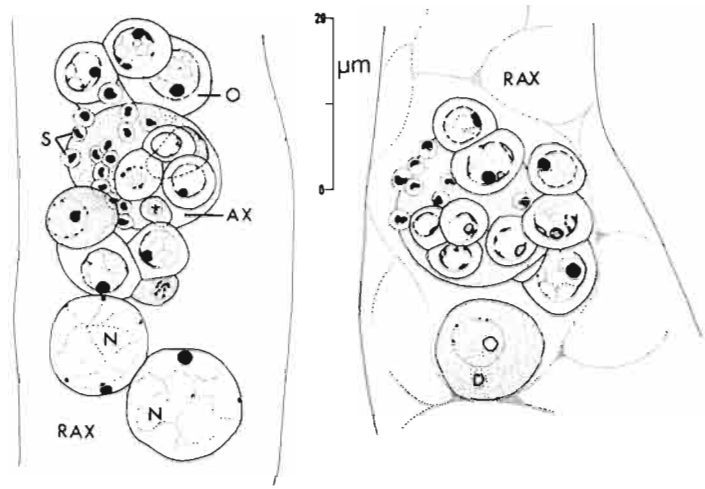


Fig. 1-53: *Dicyemennea kaikouriensis*. Details of 2 infusorigens. (AX) Axial cell; (D) degenerating oocyte; (N) nucleus and paranucleus of rhombogen axial cell; (O) oocyte; (RAX) axial cell of rhombogen; (S) amoebid spermatozoa. Scale in μm. (After Short and Hochberg, 1969.)

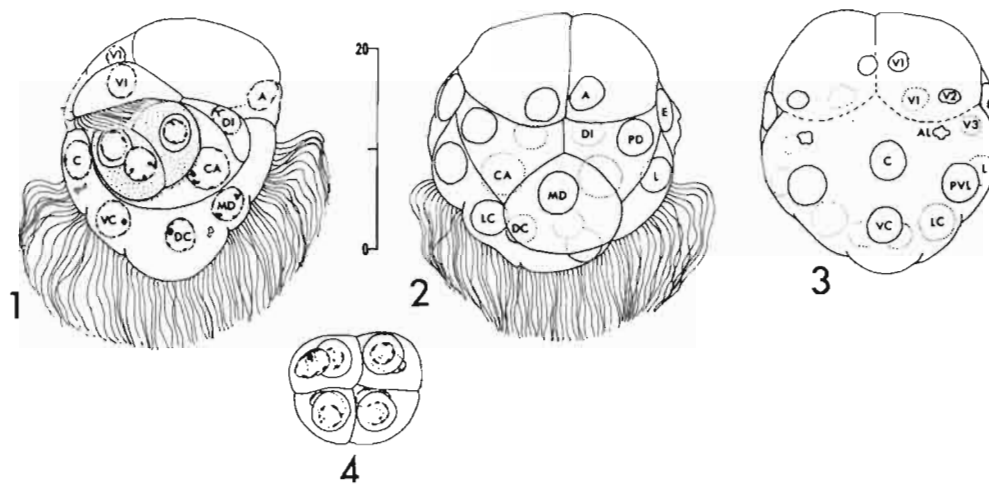


Fig. 1-54: *Dicyemennea rostrata*. Details of infusoriform larva. 1: Side view, optical section. 2: Dorsal view. 3: Ventral view to show position of certain nuclei. 4: Urn cells. Designation of cells (in some cases only shown by nuclei): (A) apical; (AL) anterior lateral; (CA) capsule; (C) couvercle; (DC) dorsal caudal; (DI) dorsal internal; (E) enveloping; (L) lateral; (LC) lateral caudal; (MD) median dorsal; (PD) paired dorsal; (PVL) posterior ventral lateral; (VC) ventral caudal; (VI) ventral internal; (V1) first ventral; (V2) second ventral; (V3) third ventral. Scale in μm. (After Short and Hochberg, 1969.)

Twice during the course of an infection the parasites undergo a change of phase. The initial infective phase is brief, and when the stem nematogens are spent they disappear and are replaced by nematogens. As the cycle progresses all nematogens are eventually transformed into rhombogens, during which stage gametic reproduction takes place. In octopods, the transition from nematogens to rhombogens is prolonged and a mixture of

Table 1-11
Number of *Eledone cirrhosa* infected with *Chromidina coronata* and/or *Dicyemennea eledones* based on weight of host. Stages in the life cycle of *D. eledones* at different host weights indicated (After Nouvel, 1937)

Number of *E. cirrhosa*	*E. cirrhosa* (body weight in g)								
	1.5–2	2–3	3–4	4–5	5–6	6–8	8–10	10–13	13+
Negative for *Chromidina* and *Dicyemennea*	28	14	4	2	0	0	0	0	0
Only with *Chromidina*	32	4	0	0	2	0	0	0	0
With *Chromidina* & *Dicyemennea*	0	6	6	3	1	3	1	2	0
Only with *Dicyemennea*	0	16	10	20	8	8	5	5	11
Total	*60*	*40*	*20*	*25*	*11*	*11*	*6*	*7*	*11*
Dicyemennea life cycle stages		◄— Stem Nematogen —►							
		◄— Primary Nematogen —►							
								◄— Rhombogen —►	

stages is often found (Table 1-11; Nouvel, 1937; Hochberg, 1971), whereas in cuttlefishes a rapid metamorphosis is completed at the time of sexual maturation of the host (Nouvel, 1933b). Because the shift in phase is particularly evident in adult cephalopods, most authors have suggested that the hormonal flux associated with host maturation acts as a trigger. However, at the time of transition the renal organs are maximally crowded with parasites. Lapan and Morowitz (1975) demonstrated that population pressure or crowding may be the key factor which initiates the shift from the nematogen to rhombogen phase.

Both dicyemid mesozoans and chromidinid ciliates live in the excretory organs of cephalopods. Concurrent infections rarely occur since the hosts of these two parasites are normally spatially isolated. *Chromodina* species typically infect oceanic cephalopods which never contact the bottom, whereas the dicyemids are known from exclusively benthic or epibenthic hosts. Overlaps occur only in situations where planktonic larval stages of otherwise bottom dwelling cephalopods, such as *Eledone cirrhosa,* become infected with species of *Chromidina* before they settle to the bottom and pick up dicyemids (Table 1-11; Nouvel, 1937). The exploitation of the excretory organs of cephalopods by these 2 unusual vermiform parasites, thus, is facilitated and maintained by the habits of the hosts and the spatial separation of the infective stages. In the absence of competition, adaptation to the selective pressures within the excretory environment has favored the convergence of both form and reproductive strategy. In addition to the sizes and shapes of all stages being nearly similar, both parasites exhibit a diphasic life cycle which is remarkably well adapted to the requirements of their endoparasitic existence (Fig. 1-55; Hochberg, 1982a).

Agents: Platyhelminthes

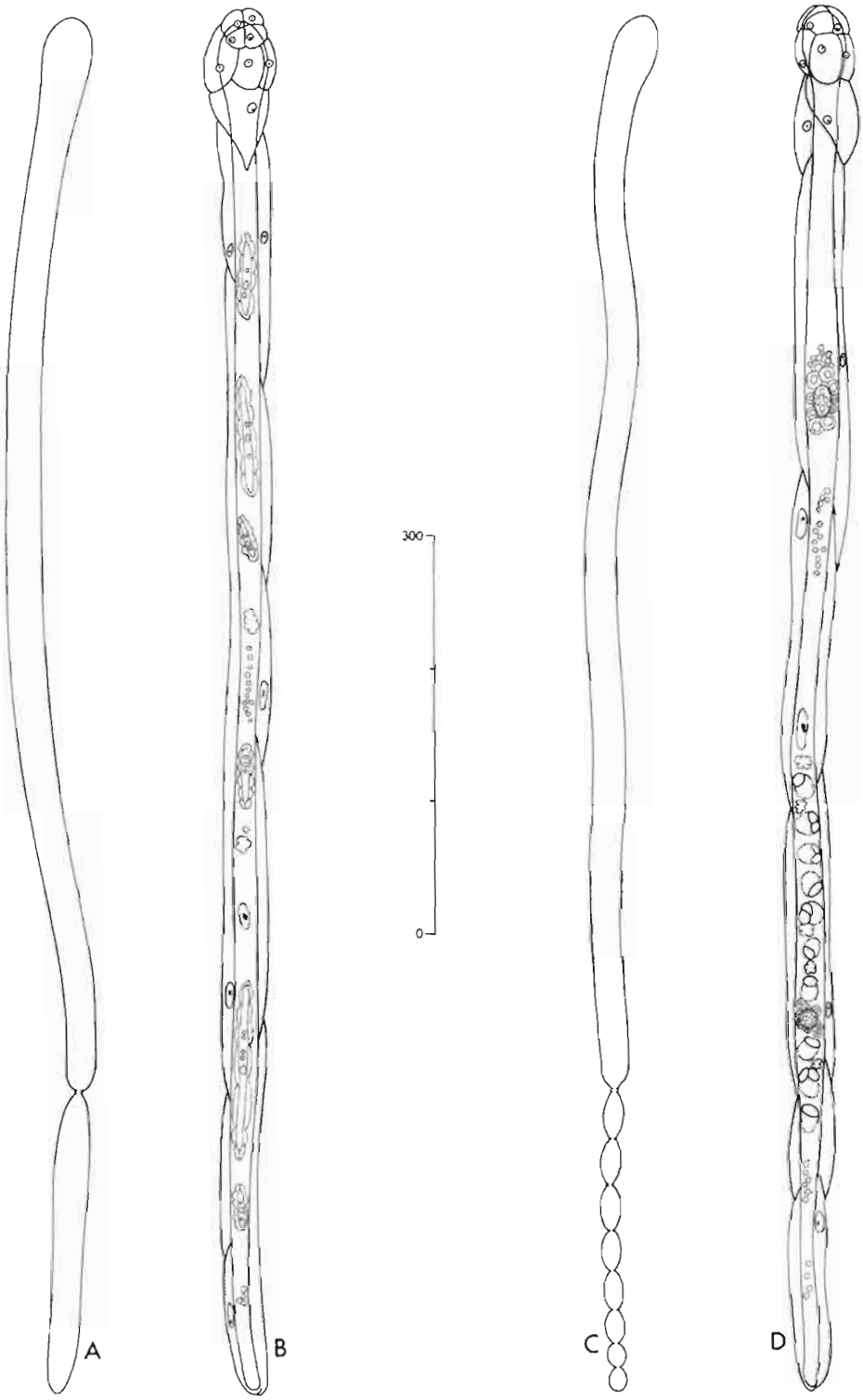
Monogenea

Several monogeneans have been described from cephalopods. These forms are reviewed or figured by Sproston (1946), Palombi (1949), Dollfus (1958) and Bychowsky (1961).

Delle Chiaje (1822) recorded *Polystoma loliginum* from *Loligo vulgaris* in the vicinity of Naples, Italy. In 1841, he related that Krohn had discovered a similar monogenean in the vena cava of *Sepia officinalis*. Later, Diesing (1850) described *Solenocotyle chiaje,* which is now considered to be a synonym of *P. loliginum*. The existence of this species is the center of considerable controversy, as described by Dollfus (1913). This unusual endoparasitic worm is reported to infect the large blood vessels of at least two cephalopod hosts as mentioned above.

Immature specimens of a monogenean were collected at Woods Hole, Massachusetts on an unidentified squid (probably either *Loligo* or *Illex*). This worm was originally assigned to the genus *Erpocotyle* by Price (1942). Though transferred to the genus *Squalonchocotyle* by Sproston (1946), Yamaguti (1963) later reassigned the original genus name. Until more material is available the exact generic placement and specific name remain in doubt.

Fig. 1-55: Convergence of chromidinid ciliates and dicyemid mesozoans. 1 and 2: Parasites from young cephalopods. 3 and 4: Parasites from mature hosts. 1 and 3: *Chromidina cortezi* from *Pterygioteuthis giardi*; 2 and 4: *Dicyema apollyoni* from *Octopus rubescens*. Scale in μm. (After Hochberg, 1982a.)


A
B
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0
C
D

Two species of the gyrodactylid genus *Isancistrum* have been found on adult *Alloteuthis subulata* (in older literature referred to as *Loligo media*, see Jaeckel, 1958) captured in the North Sea and the English Channel off France and England. *I. loliginis* (Fig. 1-56, 2), originally described by Beauchamp (1912), is now known to occur in small numbers in the mantle cavity and on the gills of the squid host (see also Sprehn, 1933;

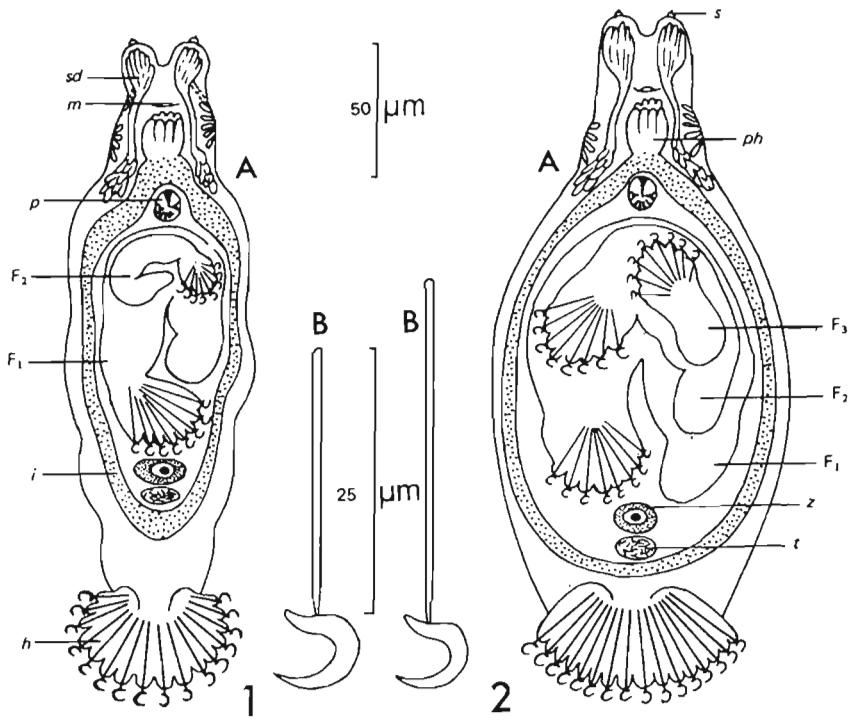


Fig. 1-56: *Isancistrum* spp. from *Alloteuthis subulata*. 1: *Isancistrum subulatae;* (A) dorsal view; (B) marginal hook. 2: *Isancistrum loliginis;* (A) dorsal view; (B) marginal hook. (F_1 to F_3) Successive embryonic generations; (H) haptor; (I) intestine; (M) mouth; (P) penis; (PH) pharynx; (S) sensilla; (SD) sticky-gland ducts; (T) testis; (Z) zygote. Scale in µm. (After Llewellyn, 1984.)

Llewellyn, 1974, 1975, 1979, 1984; Anon., 1976). The second species, *I. subulatae* (Fig. 1-56, 1), recently described by Llewellyn (1984) lives in very large numbers on the arms and tentacles of *Alloteuthis* (Fig. 1-57). The worms are tiny (averaging less than 0.25 mm long), transparent and exceedingly difficult to detect even by a trained observer. As typical for the gyrodactylids both species are viviparous and lack a free swimming stage. In addition to microhabitat differences the species can be separated on the basis of size, shape, length of hook handles and the maximum number of daughter generations contained in the uterus. *I. loliginis* is robust bodied, retains a maximum number of 3 embryos and lives in the sheltered habitat of the mantle cavity, whereas *I. subulatae* is slended bodied, contains only 2 embryos and lives in exposed locations on the external surfaces of the squid body.

The haptor of both species bears 16 identical hooks arranged in an arc (Fig. 1-56). Beauchamp's (1912) original count of 15 hooks is erroneous. The handle of each hook is

Fig. 1-57: *Isancistrum subulatae* on arm and suckers of *Alloteuthis subulata* (SEM). Scale = 500 μm. (After Llewellyn, 1984.)

contained within a muscular sleeve. The hooks can be extended and rapidly withdrawn, effectively gaffing the host. The hooks probably do not normally penetrate below the epidermis and appear to cause no damage to the host squid (Llewellyn, 1984). Locomotion is leech-like aided by sticky-gland secretions from the anterior end of the body and the haptor hooks at the posterior. Llewellyn (1984) considers *Isancistrum* to be an epidermal browser.

Unlike other gyrodactylids, the genus *Isancistrum* lacks hamuli or large median anchors which suggest a primitive condition. Malmberg (1974a, b) concluded on the basis of the protonephridial system that *Isancistrum* is not a primitive gyrodactylidean genus but most likely invaded and successfully adapted to cephalopods which lived in the same habitat as fishes. Llewellyn (1984) postulated that viviparity is acquired later in the evolution of monogeneans than the acquisition of the hamuli. Hence, he also concluded that following secondary invasion of cephalopod hosts the isancistrines lost the large median hooks.

Llewellyn (1984) reported that larger squids were more likely to be parasitized by monogeneans than smaller squids (Fig. 1-58). *Alloteuthis* less than 60 mm ML were lightly

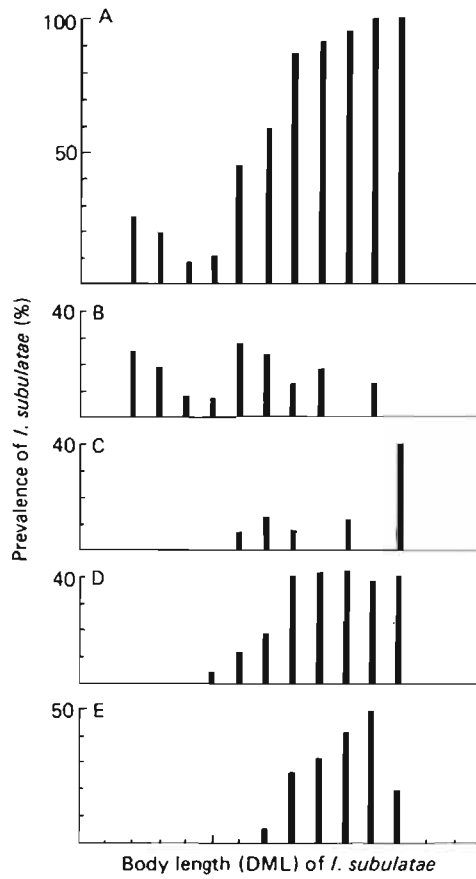


Fig. 1-58: *Isancistrum subulatae*. Incidence and intensity of infections on *Alloteuthis subulata* of various mantle lengths. A: All intensities. B: 1 to 9 parasites host^{-1}. C: 10 to 99. D: 100 to 999. E: 1000. Number = 199. Mantle length scale in intervals of 10 mm. (After Llewellyn, 1984.)

infected (about 15 % with less than 10 worms host^{-1}) whereas squid 100 mm ML or larger were heavily infected (95 to 100 % with densities in excess of 100 worms host^{-1}). Due to the mechanics of viviparity, a single founder isancistrine on a young host could lead to a large population of parasites on an older host.

In a series of simple experiments Llewellyn (1984) demonstrated that the invasion of new hosts takes place through direct contact of adults. The transfer likely occurs at the time of mating. His studies also indicated that the survival and perpetuation of *Isancistrum* is dependent on overlapping generations of *Alloteuthis*. Most loliginid species are thought to mate, spawn and die at one time with cohorts of the same size and age. However, the English Channel collections of *Alloteuthis subulata* exhibited a distinct polymodal distribution in which the opportunity for transfer of worms between members of different age groups was a distinct possibility (Llewellyn, 1984).

Llewellyn (1984) also investigated the possibility of *Isancistrum* transferring to host species other than loliginids. When small *Sepiola atlantica* were placed together with a heavily infected *Alloteuthis* transfers were effected on 3 separate occasions. Whether

opportunities for such transfers occur in nature or whether *Isancistrum* can multiply on a 'foreign' host cephaloped is not known. Squids with heavy infections of over 5000 isancistrines appear to exhibit no obvious pathogenetic symptoms (Llewellyn, 1984). However, in the presence of stress or in combination with other pathogens such large numbers could be potentially harmful. Llewellyn's work indicates that problems might arise from the introduction of monogenean infections into high density mariculture situations.

Llewellyn (1984) reported the presence of unidentified isancistrines on the arms and gills of *Loligo vulgaris* and the gills of *L. forbesi*. And, Paperna (pers. comm.) indicated that loliginids in the Red Sea are commonly infected with monogeneans. These reports may represent new species. As more cephalopods, especially loliginids, are critically examined for parasites monogeneans may prove to be very common.

Digenea

Until recently cephalopods attracted little attention as potential hosts for digenetic trematodes. However, reviews by Overstreet and Hochberg (1975) and Gaevskaya (1977b) point out that almost 20 species of digeneans have been recovered from a total of nearly 30 species of cephalopod hosts (see Tables 1-12 and 1-13). Cephalopods are parasitized by either larval stages (metacercaria) or adults and, hence, act as second intermediate, paratenic or final hosts but never as first intermediate hosts.

The most characteristic and quantitatively the most important group of digeneans which infect oceanic squids are the larval didymozoids. Several morphs are recognized (Fig. 1-59) which may represent distinct species. These differ in body dimensions, sucker size, shape of the intestine and the presence or absence of a thick walled stomach. In the majority of cases it has not been possible to associate these metacercarial stages with specific identified adult worms, hence, in the literature most are collectively lumped under the names *'Monilicaecum'* and *'Torticaecum'* (Yamaguti, 1942) or given a type designation. For a list of hosts and didymozoid parasites see Table 1-12.

Cephalopods acquire didymozoids by eating infected invertebrates and fishes. The metacercaria migrate from the digestive system to their final location between the outer connective tissue covering and the inner muscle layer of the stomach or caecum. In large ommastrephid squids the highest densities of didymozoid cysts are clustered in the anterior part of the stomach around the main blood vessel (Filippova, 1974; Gaevskaya and Nigmatullin, 1981; Naidenova and co-authors, 1981). The metacercaria constantly move about in thin-walled oval cysts. There is little or no evidence of host tissue reaction.

Judging from variations in worm size and number of caecal chambers (Fig. 1-59) didymozoid metacercaria grow inside the squid hosts. The metacercaria appear to survive for only a short time in each host individual. Once the worms reach a maximum size in a given host they die, the cyst wall dissolves and the larvae are macerated. In *Sthenoteuthis pteropus* the incidence and intensity of infection with didymozoids peaks in small squid that are 2 to 3 months old (3 to 10 cm ML) after which the indices gradually decrease (Fig. 1-60). In squid that are 6 to 8 months old (20 cm ML) large numbers of dead worms are evident. The duration of life of metacercaria within species of *Sthenoteuthis* proably does not exceed 4 to 6 months (Gaevskaya and Nigmatullin, 1981; Naidenova and co-authors, 1981).

The life cycle of the didymozoids is complex and is thought to involve 3 to 4 hosts

Table 1-12
Didymozoidae from cephalopod hosts. Incidence and intensity of infections in relation to host species and localities of capture
(Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Loliolopsis chiroctes	*Monilicaecum* sp.	24	30–83	62.5	2–8	Gulf of California (Mexico)	Hochberg (1969), Overstreet and Hochberg (1975)
Lolliguncula brevis	Didymozoid	14	28–32	7	1	Gulf of Mexico (Mississippi, USA)	Hochberg (unpubl.)
Abraliopsis falco	*Monilicaecum* sp. A	13	11–32	77	1–8	Eastern and Central North Pacific Ocean; Gulf of California (Mexico)	Hochberg (1969), Overstreet and Hochberg (1975)
Abraliopsis felis	*Monilicaecum* sp. A	38	8–51	10	1–5	Eastern North Pacific Ocean	Hochberg (1969), Overstreet and Hochberg (1975)
Pterygioteuthis gemmata	*Monilicaecum* sp. A	37	7–37	2.7	2	Eastern North Pacific Ocean	Hochberg (1969), Overstreet and Hochberg (1975)
Pterygioteuthis giardi	*Monilicaecum* sp. A	234	7–27	16	1–5	Gulf of California (Mexico)	Hochberg (1969), Overstreet and Hochberg (1975)
Pholidoteuthis boschmai	Didymozoid	32	95–250	6.2	1–16	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Tetronychoteuthis dussemieri	Didymozoid	2	143, 230	50	25	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Dosidicus gigas	*Monilicaecum* sp. A	14	7–206	71	1–15	Gulf of California (Mexico)	Hochberg (1969), Overstreet and Hochberg (1975)
D. gigas	Didymozoid	223	150–360	5	ni	Eastern South Pacific Ocean	Gaevskaya and co-authors (1982)
D. gigas	Didymozoid	403	50–430	7.4	1–30	Eastern South Pacific Ocean	Gaevskaya and co-authors (1983)
D. gigas	Didymozoid	410	20–400	ni	ni	Eastern Pacific Ocean (2° N–22° S, 82–87° W)	Gaevskaya and co-authors (1987)

Table 1-12 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Eucleoteuthis luminosa	Didymozoid	ni				ni	Gaevskaya (1977b)
E. luminosa	Didymozoid	29	150–220	6.9	7–13	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Hyaloteuthis pelagica	Didymozoid	34	16–72	79.4	to 102	Atlantic Ocean	Gaevskaya and Nigmatullin (1977), Nesis and Nigmatullin (1979)
Illex coindeti	*Monilicaecum* sp. C (= *Pleorchis* sp.)	ni				Mediterranean (Monaco, France)	Nouvel (in Wirz, 1958), Dollfus (1971)
I. coindeti	Didymozoid	ni				Atlantic Ocean	Gaevskaya (1977b), Gaevskaya and Nigmatullin (1977)
Illex illecebrosus	Didymozoid	ni				Atlantic Ocean	Nesis (1967), Gaevskaya and Nigmatullin (1977)
Ommastrephes bartrami	Didymozoid type IV	60	100–760	1.9	to 1000	North Atlantic Ocean	Gaevskaya and Nigmatullin (1976b, 1977, 1978)
O. bartrami	*Monilicaecum* (= didy. type III)	42	120–450	3.0	ni	South Atlantic Ocean	Gaevskaya and Nigmatullin (1974, 1978), Gaevskaya (1976)
O. bartrami	Didymozoid	35	160–405	91	1–250	Western South Atlantic Ocean	Gaevskaya and co-authors (1986a)
O. bartrami	Didymozoid type IV	ni	ni	100	1–200	Indian Ocean	Belyaeva (1979)
O. bartrami	Didymozoid	184	150–450	7.8	1–70	Western North Pacific Ocean	Kurochkin and Solov'eva (1982)
O. bartrami	Didymozoid	16	160–360	100	9–613	Eastern South Pacific Ocean	Gaevskaya and co-authors (1983)
O. bartrami	Didymozoid	7	240–360	85.7	6–1500	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)

Table 1-12 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Ornithoteuthis antillarum	Didymozoid	62	20–130	ni	to 120	Atlantic Ocean	Gaevskaya and Nigmatullin (1977), Nesis and Nigmatullin (1979)
Sthenoteuthis oualaniensis	Didymozoid	3	15–61	31	140	Eastern Central Pacific Ocean	Fields and Gauley (1972)
S. oualaniensis (= *Symplectoteuthis*)	*Monilicaecum*	ni	ni	13.2 (♀) 6.2 (♂)	ni	Western North Pacific Ocean	Tung (1976), Okutani and Tung (1978)
S. oualaniensis	Didymozoid	80	70–320	7.4	1–30	Eastern South Pacific Ocean	Gaevskaya and co-authors (1983)
S. oualaniensis	Didymozoid	60	ni	100	ni	Tropical Indian Ocean	Filippova (1974)
S. oualaniensis	Didymozoid type III	ni	ni	100	1–200	Indian Ocean	Belyaeva (1979)
S. oualaniensis	*Monilicaecum*	303	ni	80–95	1–2000	Indian Ocean and Red Sea	Naidenova and co-authors (1981)
Sthenoteuthis pteropus	Didymozoid (sexually mature)	2262	20–800	.05	1	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1978, 1981)
S. pteropus	Didymozoids (includes 2 species)	ni ni	100–150 350–500	85–90 19	ni ni	Tropical Atlantic Ocean	Gaevskaya (1977a)
S. pteropus	Didymozoid	2262	200–500	65	1–20,000	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1977a, 1981)
S. pteropus	*Torticaecum*	1039	to 800	12.5	ni	Eastern Central Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1978), Naidenova and Zuev (1978a, b)
Todarodes angolensis	Didymozoid	ni				Atlantic Ocean	Gaevskaya and Nigmatullin (1977)
T. angolensis	Didymozoid	18	200–295	22	1–5	Western South Atlantic Ocean	Gaevskaya and co-authors (1986a)
Todarodes sagittatus	Didymozoid	ni				Eastern South Atlantic Ocean	Reimer (1974)

Table 1-12 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Todaropsis eblanae	Didymozoid	ni				Mediterranean (France)	Hochberg (unpubl.)
T. eblanae	Didymozoid	ni				Atlantic Ocean	Gaevskaya and Nigmatullin (1977)
Thysanoteuthis rhombus	Didymozoid	ni				ni	Gaevskaya (1977b)

ni: Not indicated

Table 1-13
Digenetic trematodes from cephalopod hosts (except for didymozoids). Incidence and intensity of infections in relation to host species and localities of capture (Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER SEPIOIDEA							
Sepia officinalis	*Derogenes varicus* Hemiuridae	12	105–146	58	1–9	English Channel (England)	Reimer (1974), Overstreet and Hochberg (1975)
S. officinalis	*Gonocercella sepiocola* Hemiuridae	ni				English Channel	Reimer (1974, 1975a)
S. officinalis	Distome	ni				English Channel (France)	Vaullegeard (1896), Dollfus (1958)
Sepia sp.	Distome	ni				ni	Gros (1847), Dollfus (1958)
ORDER SEPIOLIOIDEA							
Rossia molleri	Mature digenean Hemiuridae	ni				Arctic Ocean (Canada)	Overstreet and Hochberg (1975)
Rossia sublaevis	Immature digenean Hemiuridae	ni				Western North Atlantic Ocean (Newfoundland, Canada)	Overstreet and Hochberg (1975)
Sepiola aurantiaca	Unident. digenean	ni				English Channel (England)	Overstreet and Hochberg (1975)
ORDER TEUTHOIDEA							
Alloteuthis subulata	Unident. digenean	ni				English Channel (England)	Overstreet and Hochberg (1975)
Loligo forbesi	*Hirudinella ventricosa* Hirudinellidae	ni				Tropical Atlantic Ocean	Gaevskaya (1977b)
Lolliguncula brevis	*Lecithochirium microstomum* Hemiuridae	138	50–87	8.7	1–2	Gulf of Mexico (Mississippi, USA)	Overstreet and Hochberg (1975)

Table 1-13 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Lolliguncula brevis	*Lepocreadium* sp. Lepocreadiidae	14	23–28	7	1	Gulf of Mexico (Mississippi, USA)	Hochberg (unpubl.)
Berryteuthis magister	*Derogenes varicus* Hemiuridae	1204	100–360	0.2	1	Bering Sea (USSR)	Avdeeva and co-authors (1982)
Ctenopteryx sicula	? *Elytrophallus* sp.	ni				Central North Pacific Ocean	Hochberg (unpubl.)
Illex illecebrosus	*Distomum* sp. (immature)	ni				Western North Atlantic Ocean (Newfoundland, Canada)	Stafford (1907)
Ommastrephes sp.	Unident. digenean (adult)	ni				ni	Pelseneer (1928), Clarke (1966)
Sthenoteuthis oualaniensis	*Hirudinella ventricosa* Hirudinellidae	ni				Indian Ocean & Red Sea	Belyaeva (1979), Naidenova and co-authors (1981, 1985)
Sthenoteuthis pteropus	*H. ventricosa* Hirudinellidae	2262	20–500	0.07	1–2	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1976a, 1981, 1983), Gaevskaya (1977a)
S. pteropus	*H. ventricosa* Hirudinellidae	883	ni	ni	ni	Eastern Central Atlantic Ocean	Naidenova and Zuev (1978b)
Todarodes sagittatus	*Distoma todari*	ni				Mediterranean (Italy)	Delle Chiaje (1822, 1841)
Chiroteuthis veranyi	*Lepocreadium album* (= *Cercaria setifera*) Lepocreadiidae	ni				Meditterranean (Italy)	Dollfus (1958), Rebecq (1965)
ORDER OCTOPODA							
Japatella heathi	? *Elytrophallus* sp. Hemiuridae	24	11–90	4	1	Gulf of California (Mexico)	Overstreet and Hochberg (1975)
Argonauta argo	*Accacoelium pelagiae* (= *Distoma Kollikerii*) Accacoelidae	ni				Mediterranean (Italy)	see Overstreet and Hochberg (1975)

Table 1-13 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER OCTOPODA							
Argonauta nouryi	*Dinurus barbata* Hemiuridae	13	28–45	30	1	Eastern Tropical Pacific Ocean	Hochberg and Overstreet (unpubl.)
Argonauta sp.	*Distomum dactylipherum*	ni				Indian Ocean	see Overstreet and Hochberg (1975)
Eledone cirrhosa	Unident. digenean (immature)	ni				Mediterranean (Monaco)	Dollfus (1958)
Octopus briareus	*Stephanochasmus* sp. Stephanochasmidae	2	50–70	100	5, 8	Straits of Florida (Florida, USA)	Overstreet and Hochberg (1975)
Octopus maorum	*Lecithochirium* sp. Hemiuridae	1	15	100	7	Western South Pacific Ocean (New Zealand)	Overstreet and Hochberg (1975)
O. maorum	*Plagioporus maorum* Opecoelidae	3	ni	66	6, 52	Western South Pacific Ocean (New Zealand)	Allison (1966)
O. maorum	*P. maorum*	11	ni	27	1–30	Western South Pacific Ocean (New Zealand)	Short and Powell (1968)
Octopus vulgaris	*Distoma octopodis*	ni				Mediterranean (Italy)	Delle Chiaje (1822, 1829, 1841)
O. vulgaris	*Ptychogonimus megastoma* Ptychogonimidae	ni				Mediterranean (Italy)	Palombi (1942)
O. vulgaris	*Proctoeces maculatus* Fellodistomidae	ni	ni	50	ni	Indian Ocean (South Africa)	Smale and Buchan (1981)
Robsonella australis	*Plagioporus maorum* Opecoelidae	32	ni	3	1	Western South Pacific Ocean (New Zealand)	Short and Powell (1968)

ni: Not indicated

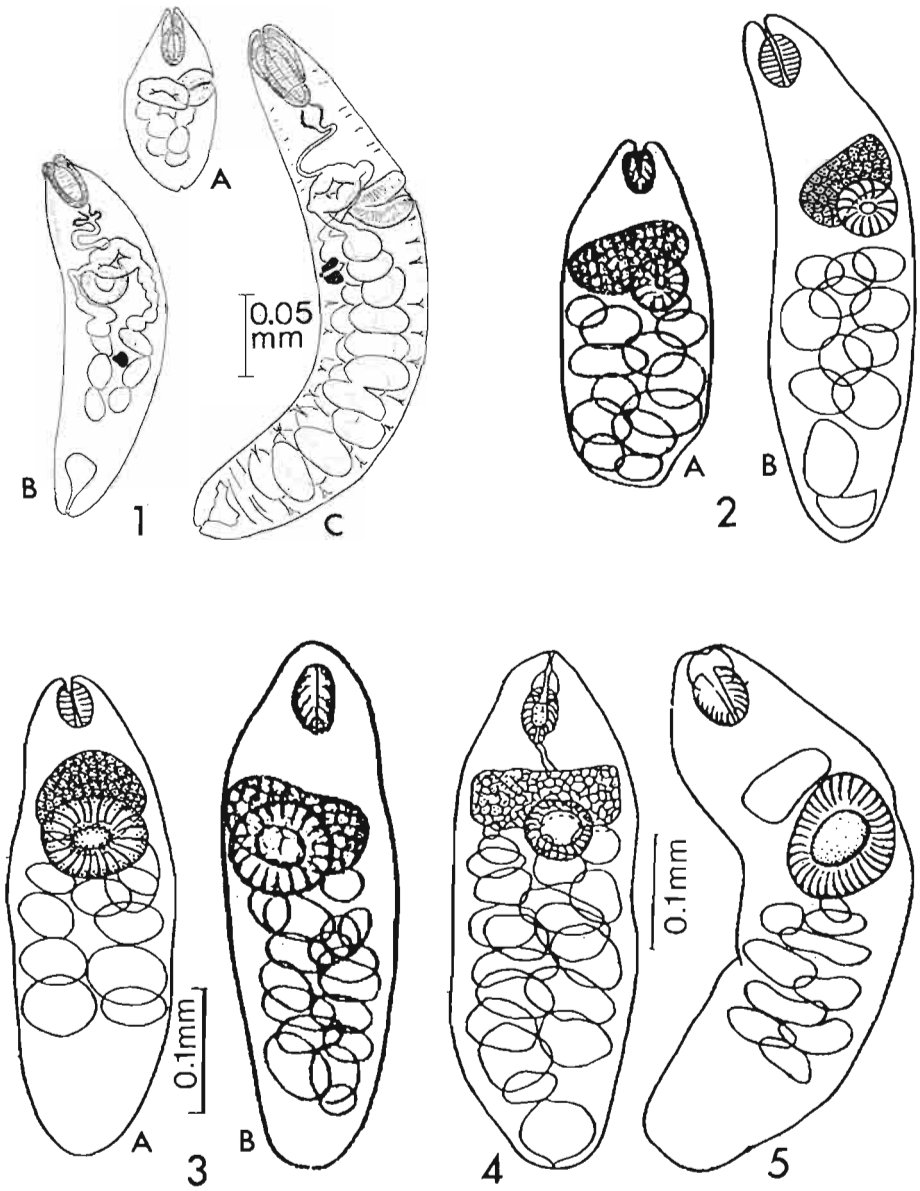


Fig. 1-59: Larval didymozoids from cephalopods. 1: *Monilicaecum* sp. (A and C) from *Abraliopsis falco;* (B) from *Pterygioteuthis giardi;* (A) recently hatched individual, dorsolateral view, note absence of testicular anlagen; (B) medium-size individual, dorsal view; (C) large individual, dorsolateral view. 2: Didymozoid type I larva (small ventral sucker), ventral views; (A) from *Sthenoteuthis oualaniensis;* (B) from *S. pteropus.* 3: Didymozoid type II larva (large ventral sucker), ventral views; (A) from *S. pteropus;* (B) from *S. oualaniensis.* 4: Didymozoid type III larva (*'Monilicaecum'*) from *Ommastrephes bartrami,* ventral view. 5: Didymozoid type IV larva from *O. bartrami,* ventral view. (1 after Overstreet and Hochberg, 1975; 2A and 3A after Gaevskaya, 1977a; 2B and 3B after Gaevskaya, 1977b; 4 and 5 after Gaevskaya and Nigmatullin, 1976b.)

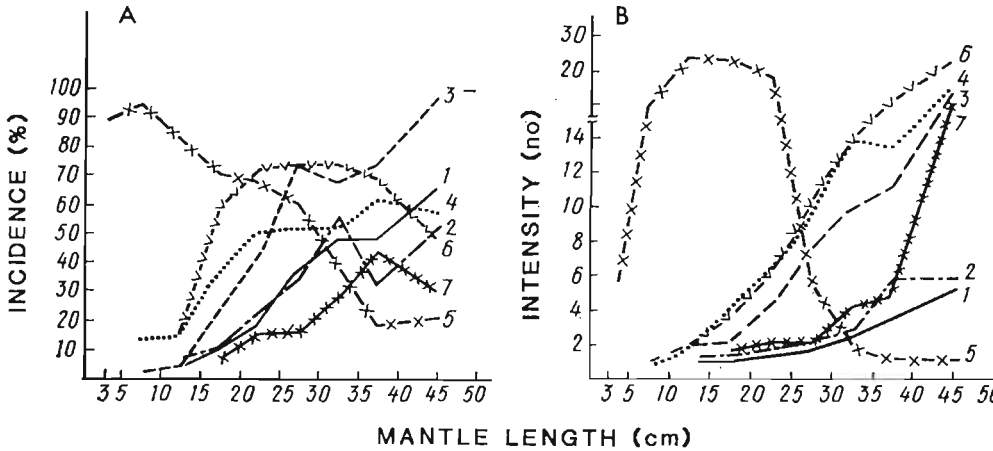


Fig. 1-60: Changes in incidence (A) and intensity (B) of parasite infections of the ommastrephid *Sthenoteuthis pteropus* in relation to size. (1) *Anisakis simplex*; (2) *Phyllobothrium* sp. 1; (3) *Tentacularia coryphaenae*; (4) *Nybelinia lingualis;* (5) didymozoid gen. and sp. 1; (6) *Porrocaecum* sp. 1 (stomach); (7) *Porrocaecum* sp. 2 (mantle). (After Gaevskaya and Nigmatullin, 1981.)

(Nikolaeva, 1965, 1981; Yamaguti, 1970). No cycle has been worked out experimentally and at present it is impossible to correlate developmental stages with mature stages. Most likely the didymozoid life cycles will be similar to those known for related hemiurids (Køie and Lester, 1985).

Cycles in which oceanic cephalopods are involved probably have 4 obligatory host stages. Pelagic mollusks, such as heteropods, pteropods and ptenoglossans, are likely candidates for first intermediate hosts. In preliminary studies, Gaevskaya (pers. comm., see also Gaevskaya and Nigmatullin, 1977) found 3 % of 50 *Cavolina* sp. infected with trematode stages resembling didymozoid partenits. Cystophorous cercaria released into the plankton infect members of copepod genera such as *Paracalanus* and *Calanus,* which serve as the principal second intermediate hosts (Madhavi, 1968; Reimer and co-authors, 1971, 1975). A wide diversity of small predatory invertebrates and fishes feed on copepods. Whether these organisms serve as second or third intermediate hosts has not been determined. Included here are such planktonic invertebrates as hydrozoan medusae and siphonophores (genera *Obelia, Phialidium, Leuckartiara, Dimophyes, Halistemma, Aglantha, Abylopsis*), ctenophores (*Pleurobrachia*), polychaete annelids (*Tomopteris*), euphausiid and cirriped crustaceans (*Euphausia, Lepas*), and chaetognaths (*Sagitta, Pterosagitta, Spadella*) (Dollfus, 1960a, 1963; Cable and Nahhas, 1962; Madhavi, 1968; Reimer and co-authors, 1971, 1975; Shimazu, 1978; Slankis and Shevchenko, 1974). Cephalopods which serve as third intermediate hosts or transport hosts include small mesopelagic vertically migrating genera (*Pterygioteuthis, Abraliopsis*) and large epipelagic forms (*Sthenoteuthis,* etc.). The larval didymozoids from fishes have been described by a number of authors (see especially Fischthal and Kuntz, 1964; Nikolaeva, 1965, 1970; Fischthal and Thomas, 1968; Kurochkin and Nikolaeva, 1978, and Køie and Lester, 1985).

Small mesopelagic squids (species of *Pterygioteuthis, Abraliopsis*) and neritic squids (*Loliolopsis*) generally have a lower incidence of didymozoids (less than 50 %) and a lower intensity (1 to 10 worms host^{-1}) of infection than the much larger epipelagic squid

(*Sthenoteuthis, Todarodes,* etc.), where the incidence may reach 90 to 100 % and the intensity 200 to 2000 worms host^{-1} (Table 1-12). Large oceanic squids concentrate metacercarial cysts and play a key role in the transfer of didymozoids to the fourth or final host, fishes such as tunas, scombroids, xiphoids. In the Indian Ocean, for example, adult didymozoids are found in almost 100 % of the wahoo and 50 % of the tuna, with intensities of about 100 cysts fish^{-1} (Nikolaeva and Dubina, 1978).

In studies by Gaevskaya and co-workers on the ommastrephid *Sthenoteuthis pteropus* (see especially Gaevskaya and Nigmatullin, 1981), young up to 10 cm ML have been shown to prey on copepods, other small crustaceans, chaetognaths, heteropods and fry of a number of fishes. Low-level infections with didymozoids and other helminths occur at this time. As the squids increase in size they gradually begin feeding on small pelagic fishes (mainly myctophids) and squids (mainly enoploteuthids), thus continuing to increase the numbers of didymozoid metacercaria. This results in the peak intensities of infection observed in squids with mantle lengths of 10 to 25 cm (Fig. 1-60). Adults larger than 30 to 35 cm ML feed mainly on larger fishes and squids and do not continue to acquire didymozoids (Gaevskaya, 1979; Gaevskaya and Nigmatullin, 1981). The principal squid predators and the final hosts for didymozoids, fishes such as tunas, dorados, and swordfishes, target on young ommastrephids in the size range from 5 to 20 cm ML, which ensures optimal infection of final hosts.

At least 5 genera of metacercaria are known from octopods. Overstreet and Hochberg (1975) described a single specimen of an unidentified species of *Elytrophallus* from the stomach of 1 of 24 *Japatella heathi* examined in the Gulf of California, Mexico. Specimens of *Stephanochasmus* (Fig. 1-61, 6) were encysted in the mantle cavity of *Octopus briareus* in Florida (USA) (McSweeney, pers. comm.; see also Overstreet and Hochberg, 1975). The latter is noteworthy because it is one of only a few digeneans known to infect its cephalopod host by active cercarial invasion rather than through ingestion of the metacercaria. Smale and Buchan (1981) discovered immature stages of the fellodistomid *Proctoeces maculatus* in 50 % of the stomachs of *O. vulgaris* off the Natal coast of South Africa. Adults of the genus *Proctoeces* typically infect gastropods, bivalves and fishes. *O. vulgaris* is postulated to serve as an intermediate host for this worm.

Immature stages of the hemiurid *Dinurus barbata* repeatedly have been found free in the funnel and mantle cavity of female *Argonauta nouryi* (Hochberg and Overstreet, unpubl.). Other species of *Argonauta* examined were uninfected. The infection does not appear to be accidental since the incidence is over 30 %. In the Mediterranean, *A. argo*, a large and easily identified epipelagic octopod repeatedly has been reported as an accidental host for *Accacoelium pelagiae*. Consult Overstreet and Hochberg (1975) for a review of the literature on the digenean parasites of *Argonauta*.

Metacercaria of *Hirudinella ventricosa* (Fig. 1-61, 4) were first reported from the flying squid *Sthenoteuthis pteropus* by Gaevskaya and Nigmatullin (1975). Infections of this ommastrephid in the Atlantic have also been noted in Gaevskaya (1977a), Gaevskaya and Nigmatullin (1981) and Naidenova and Zuev (1978b). *H. ventricosa* has additionally been recorded from *Loligo forbesi* in the tropical Atlantic, and *S. oualaniensis* in the Indian Ocean and Red Sea (Gaevskaya, 1977b; Belyaeva, 1979; Naidenova and co-authors, 1981, 1985). The large immature worms were found singly in the coelomic cavity. Only squids measuring 100 mm ML or larger were infected. In all cases less than 2 % of the squids examined were infected and never more than 2 worms were found per host. Gaevskaya

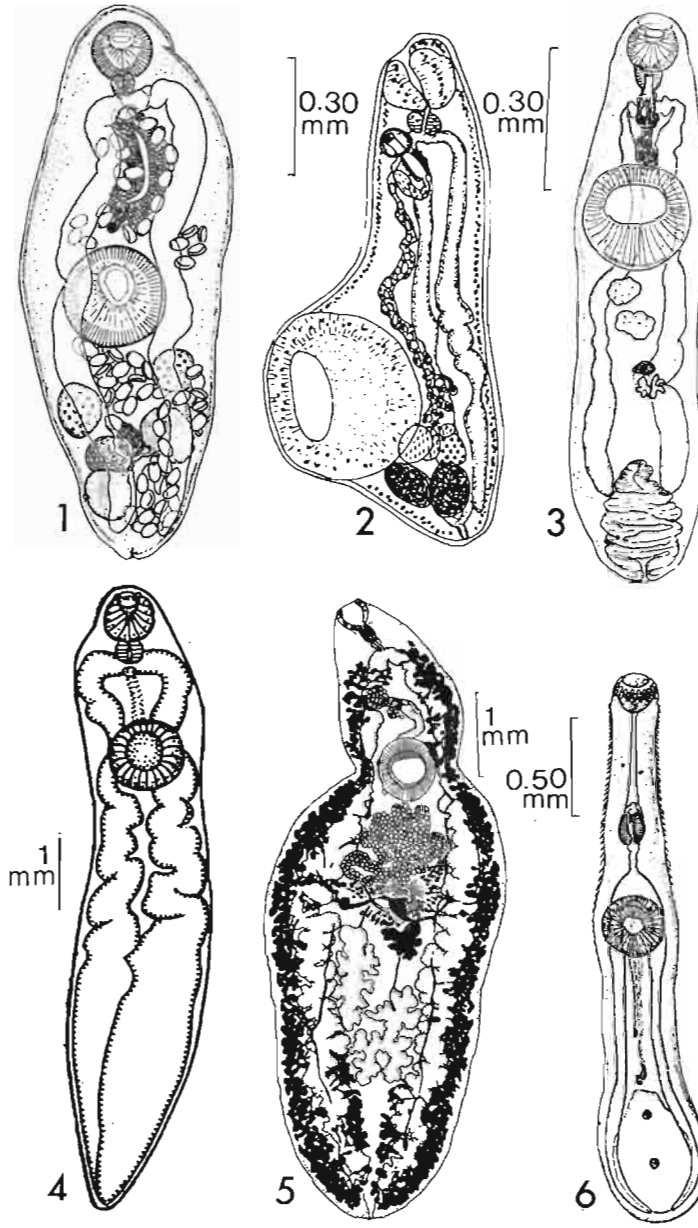


Fig. 1-61: Digeneans from cephalopods. 1: *Derogenes varicus* from *Sepia officinalis*, dorsal view. 2: *Gonocercella sepiocola* from *S. officinalis*, lateral view. 3: *Lecithochirium microstomum* from *Lolliguncula brevis*, ventral view. 4: *Hirudinella ventricosa* from *Sthenoteuthis pteropus*, ventral view. 5: *Plagioporus maorum* from *Octopus maorum*, ventral view. 6: *Stephanochasmus* sp. from *O. briareus*, ventral view. Scales in mm. (1, 3 and 6 after Overstreet and Hochberg, 1975; 2 after Reimer, 1975a; 4 after Gaevskaya, 1977a; 5 after Short and Powell, 1968.)

and Nigmatullin (1981) consider squids to be reservoir hosts for hirudinellid trematodes. Infections occur passively when squids feed on crustaceans and small fishes. Adults of *H. ventricosa* are parasitic in the stomachs of large, carnivorous, marine teleosts, principally scombroids and dorados.

A number of immature, progenetic, and even a few sexually mature, adult digeneans have been reported. Two derogenids, *Derogenes varicus* (Fig. 1-61, 1) and *Gonocercella sepiocola* (Fig. 1-61, 2), occur in *Sepia officinalis* (Reimer, 1974, 1975a; Overstreet and Hochberg, 1975). The worms found in *S. officinalis* by Gros (1847) and Vaullegeard (1896) were probably *D. varicus*. *G. sepiocola* is not well known but *D. varicus* is considered by some to be the most widely distributed of all marine parasites. *D. varicus* occurs worldwide and has been reported from a great diversity of fishes and invertebrate hosts. In Plymouth, England, *S. officinalis* appear to be initially infected at a size of 100 to 110 mm ML. Of the *S. officinalis* examined over 100 mm ML, more than 80 % were infected. The intensity of the infection increased with increasing host size to a maximum of 9 worms ind.$^{-1}$ (Hochberg, unpubl.).

Køie (1979) reviewed the life cycle of *Derogenes varicus* (Fig. 1-62) and redescribed several of its stages. Redia and cystophorous cercariae develop within the first intermediate host, gastropods of the genus *Natica*. When released, the free swimming cercariae

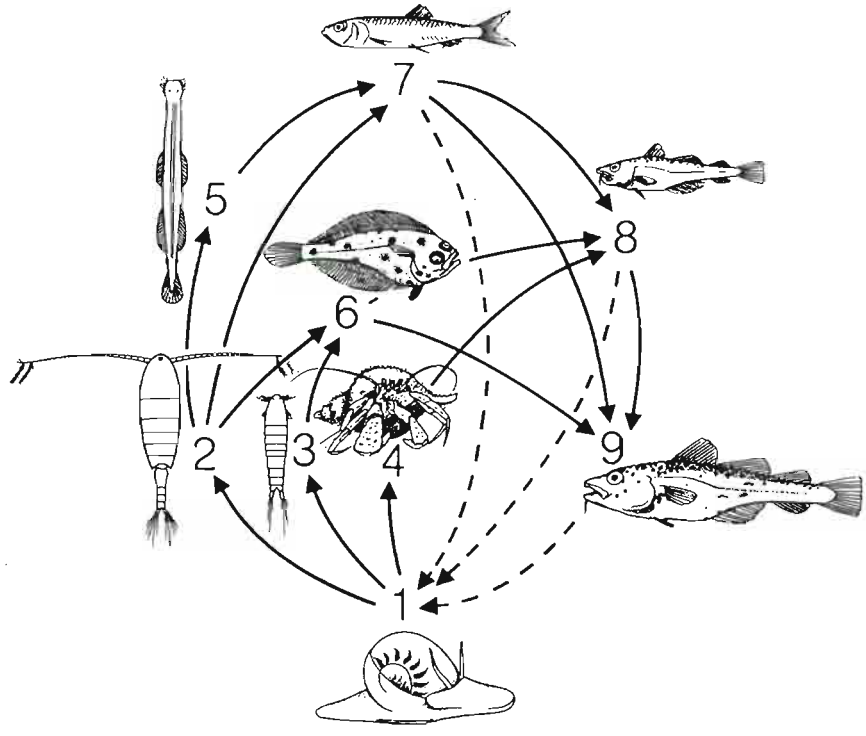


Fig. 1-62: Life cycle of *Derogenes varicus*. (1) *Natica*; (2) calanoid copepod; (3) harpacticoid copepod; (4) crustaceans, e.g., hermit crabs, decapods, etc.; (5) *Sagitta*; (6) small fishes, e.g., gobies and juveniles of other fishes; (7) planktivorous fishes, e.g., salmon, herring, juvenile gadids; (8) *Sepia officinalis* and other benthophagous and piscivorous fishes, e.g., gadids and flatfishes; (9) piscivorous and teuthivorous fishes, e.g., large cod, etc. (After Køie, 1979.)

enter copepods and develop into metacercaria. When the copepods are ingested by larger crustaceans and chaetognaths the metacercaria may mature into adult worms, though usually the cuttlefish *S. officinalis* and a variety of fishes are regarded as normal final hosts to the adult stage. Immature and even egg-bearing progenetic worms may be transferred from one fish to another or to a cuttlefish.

In New Zealand, the allocreadiid *Plagioporus maorum* (Fig. 1-61, 5) commonly infects *Octopus maorum* and occasionally occurs in *Robsonella australis* (Allison, 1966; Short and Powell, 1968). Typically, 40 % or more of these octopods are infected. The renal sacs and adjacent areas may contain up to 50 or more worms. The presence of sexually mature, adult worms indicate that these octopods can be regarded as final hosts and not merely intermediate hosts.

With the exception of the cases discussed above most reports of trematodes in cephalopods are discoveries of single hemiurids, accacoelids and hirudinellids (Table 1-13). Typically, the prevalence of infection is low. As a result, cephalopods are not thought to play an important role in the life cycle of most digeneans. In the majority of cases, cephalopods probably function simply as paratenic hosts which acquire infections when they eat the same intermediate hosts normally consumed in large numbers by teleosts which serve as the final hosts. As an example, in Mississippi (USA), Overstreet and Hochberg (1975) reported *Lecithochirium microstomum* (Fig. 1-61, 3) in only 10 % of the *Lolliguncula brevis* examined. Thus, these 'accidental' occurrences are due to ecological similarities between cephalopods and fishes in the marine environment.

Aspidogastrea

Aspidogastrid trematodes typically have a single molluscan host in their life cycle (Rhode, 1972). Though the definite hosts normally are gastropods or bivalves, there is a single cephalopod record. Smale and Buchan (1981) reported an unidentified species of *Lobatostoma* in the stomachs of 2 *Octopus vulgaris* collected off the Natal coast of South Africa. However, the authors stated that most likely this is an accidental infection in which the worms were ingested with their prey. In their study the dominant prey item and the presumed definitive host for the aspidogastrean was the mussel *Perna perna*.

Cestoda

With one possible exception, adult cestodes have never been reported from cephalopods. However, a diversity of larval and post-larval stages repeatedly have been described from decapods and octopods. This diversity indicates that cephalopods are important as intermediate or paratenic hosts for cestodes which mature in elasmobranchs and fishes, and are transferred from host to host through the food chain. Cestodes are excellent parasites to use as tags for studying trophic interactions in the marine environment.

Two orders of cestodes are commonly represented in cephalopods, the Tetraphyllidea and Trypanorhynchidea. Adults in both groups parasitize the digestive tracts of sharks, skates, and rays. Life cycles have not been completed for either of these 2 orders although several possible patterns have been postulated. At least 2 and sometimes 3 intermediate hosts and as many morphological forms of the parasite are involved (Anantaraman, 1963; Mudry and Daily, 1971; Euzet, 1979; Overstreet, 1983). In general terms, eggs, each containing a ciliated larval stage, are discharged from the vertebrate definitive host with

the feces. Once in the sea the eggs are ingested by crustaceans, especially copepods and euphausiids. In the first intermediate host the oncospheres (= hexacanths) penetrate the intestine and undergo metamorphosis in the haemocoel to form procercoids. When the copepods are ingested by teleost fishes the procercoids develop into solid-bodied post-larvae or plerocercoids. Recent evidence suggests that, at least in the tetraphyllideans, small planktivorous fishes serve as additional obligatory intermediate hosts between the crustacean and fish hosts (Overstreet, 1983). Cephalopods are thought to pick up post-larval stages by feeding on either crustaceans or small fishes. The cycle is completed when predaceous elasmobranchs feed on prey containing infective post-larvae. Trypanorhynch post-larvae are not directly comparable to tetraphyllidean plerocercoids and hence some authors, such as Dollfus (1942), have proposed the term plerocercus for the equivalent life cycle stage. The term metacestode is used by many authors to refer to all post-larval stages between oncosphere and adult. Therefore, in the above discussions it would replace the words procercoid, plerocercoid, and plerocercus.

In tetraphyllidean cestodes the scolex characteristically bears 4 large leaf-like flaps or bothridia. Plerocercoids of the genus *Phyllobothrium* (Fig. 1-63) occur free or attached in the stomach, caecum and rectum of host cephalopods. Though the genus was reviewed by Williams (1968), the species reported from cephalopods are not well known and the genus still needs extensive study. *Phyllobothrium loliginis* (Fig. 1-63, 1) is the most common species encountered in cephalopods. Originally described by Leidy (1887) from *Illex illecebrosus*, this cestode has been reported in a number of species of loliginids *(Loligo)* and ommastrephids *(Illex, Todarodes, Todaropsis)*. The squids in these genera all occur in the neritic zone on both sides of the North Atlantic. Linton (1922) and later Stunkard (1977) indicated that the species *P. tumidum* may be identical to *P. loliginis*, in which case all host records may be referred to the one cestode species (see also Linton, 1922; Guiart, 1933; Stevensen, 1933; Dollfus, 1936, 1958; Squires, 1957; Euzet, 1959; Stunkard, 1977).

In France, *Sepia officinalis* is infected by *Phyllobothrium lactua* (Dollfus, 1958). In the Mediterranean, *Todarodes sagittatus* is infected with *P. dohrnii*, and in the Baltic Sea *Eledone moschata* harbors *P. pusillus* (see Siebold, 1850; Dollfus, 1936). The latter 2 species originally were placed in the genus *Orygmatobothrium* but are now considered to belong to the genus *Phyllobothrium*. In addition to those listed above, specimens referred to *Phyllobothrium*, but not identified to species, have been recovered from a wide diversity of cephalopod hosts, especially in the Atlantic and Indian Oceans (Tables 1-14 and 1-15; Figs 1-63 and 1-64; MacGinitie and MacGinitie, 1949; Dollfus, 1958, 1964; Brown and Threlfall, 1968a;Threlfall, 1970; Gaevskaya and Nigmatullin, 1975, 1978, 1981; Gaevskaya, 1976, 1977a, 1978; Naidenova and Zuev, 1978a; Naidenova and co-authors, 1981). The plerocercoids of *Phyllobothrium* that occur in commercially important squids (i.e., loliginids and ommastrephids) are large, often reaching lengths of 10 to 35 mm. Though typically located in the digestive tract, upon death of the host the worms actively migrate throughout the body. The movement of large plerocercoids, especially into the body cavity, lowers the commercial value of the fishery product considerably. Freezing squids immediately upon capture prevents this problem from occurring.

Large oceanic and neritic squids acquire *Phyllobothrium* larvae through ingestion of planktonic invertebrates, small teleost fishes and small squids. As a rule, the plerocercoids in ommastrephids and loliginids are larger than *Phyllobothrium* larvae in bony fishes and other species of small squids. The cestodes undergo growth and development in the squids

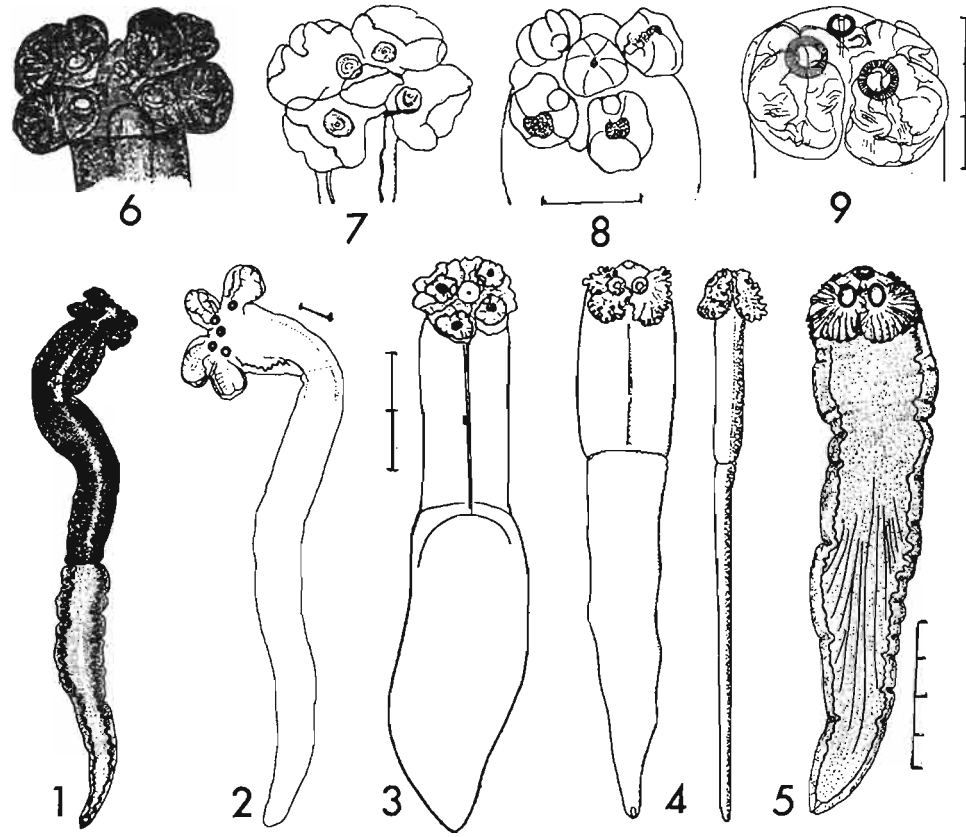


Fig. 1-63: Larval tetraphyllidean metacestodes from cephalopods. *Phyllobothrium* plerocercoids from the stomachs of a variety of hosts. 1 and 6: *P. loliginis* from *Heteroloiligo* (= *Loligo*) *pealei*; (6 scolex). 2: *P. caudatum* from *Todarodes pacificus*. 3 to 5 and 8: *Phyllobothrium* sp. from *Loligo vulgaris*; (8 scolex). 4: Dorsal and lateral views of same individual. 7 and 9: Scolices from species of *Illex*; (7 from *I. illecebrosus*, 9 from *I. coindetii*). Scale bars in mm. (1 and 6 after Stunkard, 1977; 2 after Nagasawa and Nakata, 1984; 3 and 8 after Dollfus, 1931; 4 after Joyeux and Dollfus, 1931; 5 after Guiart, 1933; 7 after Brown and Threlfall, 1968a; 9 after Dollfus, 1964.)

and strobilization may even occur in the largest plerocercoids. As a result, large squids are considered to function as either reservoir or transport hosts or more likely as obligatory intermediate hosts between small nektonic fishes and squids and the next stage in the life cycle. Elasmobranchs, many of which feed on squids, are the final hosts for the genus. However, larval cestodes in large squids also may be transferred to cetaceans rather than directly to the final hosts. Cysticercoids of *Phyllobothrium* repeatedly have been found encysted in the blubber and intestines of porpoises and other cetaceans such as pilot whales of the genus *Globicephala,* which feed exclusively on squid genera such as *Illex* and *Loligo*.

Representatives of the genus *Dinobothrium* have been reported from a few species of squids in the Mediterranean and on both sides of the Atlantic Ocean. Stunkard (1977) found *D. septaria* (Fig. 1-65, 7) encapsulated in the walls of the digestive tracts of *Heteroloiligo (= Loligo) pealei*. Species of *Illex, Todaropsis* and *Sepia* harbor either *D. plicitum* (Fig. 1-65, 4) or an as yet undesignated species of *Dinobothrium* (Dollfus,

Table 1-14
Tetraphyllidean and pseudophyllidean cestodes in cephalopods other than ommastrephids (Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	Locality	Source
ORDER SEPIOIDEA			
Sepia elegans	*Phyllobothrium* sp.	Mediterranean (Monaco, France)	Dollfus (1958, 1964), Nouvel (in Dollfus, 1958)
S. elegans	*Scolex pleuronectis trilocularis*	Mediterranean (Monaco)	Nouvel (in Dollfus, 1964)
S. elegans	*Scolex* sp.	English Channel (England)	Mohr (unpubl.)
Sepia officinalis (= *S. filliouxi*)	*Calliobothrium filicolle* (= *Scolex polymorphus*)	Mediterranean (Italy, France)	Monticelli (1885), Brumpt (1913), Pixell-Goodrich (1914), Dobell (1925), Dollfus (1958), Hochberg (unpubl.)
S. officinalis	*Dinobothrium plicitum*	English Channel (France)	Dollfus (1958), Reimer (1974)
S. officinalis (= *S. filliouxi*)	*Marsupiobothrium* sp. (= *Scolex pleuronectis bilocularis*)	Mediterranean (Monaco)	Nouvel (in Dollfus, 1964), Reimer (1975c)
S. officinalis	*Phyllobothrium* cf. *lactuca*	English Channel (France)	van Beneden (1850), Dollfus (1958, 1964)
S. officinalis	*Scolex pleuronectis* (= *S. sepiae-officinalis*, *S. polymorphus bilocularis*)	English Channel (Belgium, England, France); Eastern North Atlantic Ocean (France)	Diesing (1850, 1863), van Beneden (1850), Wagener (1854), Vaullegeard (1896)
S. officinalis (= *S. filliouxi*)	*Scolex pleuronectis trilocularis* (= *S. polymorphus trilocularis*)	English Channel (France)	Cuenot (1927), Dollfus (1936, 1958, 1964), Legendre (in Dollfus 1964), Reimer (1974)
S. officinalis	*Scolex pleuronectis unilocularis* (= *S. polymorphus unilocularis*)	Mediterranean (France)	Théodoridès (in Dollfus, 1964)
ORDER SEPIOLIOIDEA			
Rossia macrosoma	*Scolex pleuronectis* (= *Monostoma sepiola, Cysticercus sepiolae, S. polymorphus*)	Mediterranean (Italy)	Delle Chiaje (1830), Monticelli (1892), Dollfus (1936, 1958)

Table 1-14 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER SEPIOLIOIDEA			
Sepiola atlantica	*Scolex pleuronectis unilocularis*	Mediterranean (Monaco)	Nouvel (in Dollfus, 1964)
S. atlantica	*S. p. trilocularis* (= *S. polymorphus*)	Mediterranean (Monaco); English Channel (France)	Dollfus (1936, 1958, 1964), Nouvel (in Dollfus, 1964)
Sepiola rondeleti	*Scolex pleuronectis bilocularis* (= *Cysticercus sepiolae, Pseudoscicus longicollis, S. dibothrius, S. bilobatus, S. polymorphus*)	Mediterranean (Italy)	Delle Chiaje (1829), Polonio (1860a, b), Dollfus (1936, 1958)
ORDER TEUTHOIDEA			
Alloteuthis subulata	*Scolex pleuronectis* (= *S. de Bavay*)	English Channel (England)	Mohr (unpubl.)
Heterololigo pealei (= *Loligo*)	*Ceratobothrium xanthocephalum* (= *Scolex bothriis bilocularis*)	Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
H. pealei	*Dinobothrium septaria*	Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
H. pealei	*Phyllobothrium loliginis* (= *P. tumidum*)	Western North Atlantic Ocean (Connecticut, Massachusetts, USA; Newfoundland, Canada)	Linton (1922), Stevenson (1933), Squires (1957), Stunkard (1977)
H. pealei	*Scolex pleuronectis*	Western North Atlantic Ocean (Connecticut, Massachusetts, USA; Newfoundland, Canada)	Stunkard (1977)
Loligo forbesi	*Phyllobothrium* sp.	English Channel (England)	Mohr (unpubl.), Hochberg (unpubl.)
Loligo opalescens	*Pelichnibothrium speciosum* (= *Scolex pleuronectis bilocularis*)	Eastern North Pacific Ocean (California, USA)	Riser (1949, 1956a), Dailey (1969)
L. opalescens	Phyllobothrid type 4	Eastern North Pacific Ocean (California, USA)	Riser (1949, 1956a)
L. opalescens	Unident. pseudophyllidean	Eastern North Pacific Ocean (California, USA)	Dailey (1969)

Table 1-14 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER TEUTHOIDEA			
Loligo vulgaris (= *L. loligo*)	*Bothriocephalus loliginis* (= *Dibothrium gracile*)	Mediterranean (Italy)	Delle Chiaje (1829), Diesing (1850), Dollfus (1958)
L. vulgaris (= *L. loligo*)	*Echeneibothrium* sp. (= *Scolex de Bavay, S. polymorphus unilocularis*)	Eastern North Atlantic Ocean (France)	Beauchamp (1912), Bavay (in Dollfus, 1923), Legendre (in Cuenot, 1927), Dollfus (1929, 1936, 1958, 1964)
L. vulgaris (= *L. loligo*)	*Phyllobothrium loliginis* (= *P. tumidum*)	Eastern North Atlantic Ocean (France)	Cuenot (in Dollfus, 1929), Herubel (in Guiart, 1933), Guiart (1933), Joyeux and Dollfus (1931), Dollfus (1958), Legendre (in Dollfus, 1958)
L. vulgaris (= *L. loligo*)	*Phyllobothrium* sp. (= *Scolex polymorphus*)	Mediterranean (Italy); Eastern North Atlantic Ocean (France)	Dobell (1925), Dollfus (1929, 1936, 1958)
L. vulgaris	*Scyphophyllidium pruvoti* (= *Diplobothrium pruvoti*)	Eastern North Atlantic Ocean (France)	Guiart (1933), Dollfus (1936, 1958, 1964)
Loligo sp.	*Pelichnibothrium* sp.	Western North Pacific Ocean (Japan)	Yamaguti (1934)
Lolliguncula brevis	*Scolex pleuronectis unilocularis*	Gulf of Mexico (Mississippi, USA)	Hochberg (unpubl.)
Lycoteuthis diadema	*Scolex pleuronectis*	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Abralia trigonura	*Scolex pleuronectis unilocularis*	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Abraliopsis brevis	*Pelichnibothrium caudatum* (= *Scolex pleuronectis bilocularis*)	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
A. brevis	*Scolex pleuronectis unilocularis*	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Abraliopsis falcopsis	*Scolex pleuronectis unilocularis*	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)

Table 1-14 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER TEUTHOIDEA			
Abraliopsis felis	*Pelichnibothrium caudatum* (= *Scolex pleuronectis bilocularis*)	Eastern North Pacific Ocean (Oregon, USA)	Hochberg (unpubl.)
Enoploteuthis higginsi	*Scolex pleuronectis unilocularis*	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Pterygioteuthis gemmata	*Scolex pleuronectis unilocularis*	Eastern North Pacific Ocean (Mexico)	Hochberg (1971)
P. gemmata	Unident. pseudophyllidean	Eastern North Pacific Ocean (Mexico)	Hochberg (unpubl.)
Pterygioteuthis giardi	*Scolex pleuronectis unilocularis*	Gulf of California (Mexico)	Hochberg (1971)
Pterygioteuthis microlampas	*Scolex pleuronectis unilocularis*	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Octopoteuthis nielseni	*Pelichnibothrium caudatum* (= *Scolex pleuronectis bilocularis*)	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
O. nielseni	*Scolex* sp. C	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Berryteuthis magister	*Phyllobothrium* sp.	Western North Pacific Ocean (Kuriles, USSR); Bering Sea (USSR)	Avdeeva and co-authors (1982)
Pholidoteuthis boschmai	*Scolex pleuronectis*	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Tetronychoteuthis dussemieri	*Scolex pleuronectis*	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Chiroteuthis picteti	*Pelichnibothrium caudatum*	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
C. picteti	*Scolex* sp. C	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Galiteuthis sp.	Unident. pseudophyllidean	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)

Table 1-14 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER TEUTHOIDEA			
Liocranchia reinhardti	*Pelichnibothrium caudatum* (= *Scolex pleuronectis bilocularis*)	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
L. reinhardti	*Scolex pleuronectis unilocularis*	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Megalocranchia fisheri	*Pelichnibothrium caudatum* (= *Scolex pleuronectis bilocularis*)	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
M. fisheri	*Scolex pleuronectis unilocularis*	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
ORDER VAMPYROMORPHA			
Vampyroteuthis infernalis	*Scolex pleuronectis*	Eastern North Pacific Ocean (Mexico)	Hochberg (unpubl.)
ORDER OCTOPODA			
Eledone cirrhosa (= *E. Aldrovandi*)	*Phyllobothrium* sp.	Mediterranean (France)	Euzet (1959)
E. cirrhosa (= *E. Aldrovandi*)	*Scolex pleuronectis unilocularis* (= *S. polymorphus*)	English Channel (England, France); Mediterranean (Italy, Monaco, France)	Pixell-Goodrich (1914), Dobell (1925), Dollfus (1958, 1964), Nouvel (in Dollfus, 1964), Hochberg (unpubl.)
Eledone moschata	*Acanthobothrium* sp. (= *Scolex pleuronectis trilocularis*)	Mediterranean (Monaco, France)	Dollfus (1923, 1936, 1964), Nouvel (in Dollfus, 1964)
E. moschata	*Orygmatoscolex pusillum* (= *Scolex acanthobothrium musteli; O. musteli*)	Baltic Sea (Poland); Mediterranean (France)	van Beneden (1850), von Siebold (1850, 1851), Diesing (1854), Dollfus (1936, 1958)
E. moschata	*Phyllobothrium* sp. (= *Scolex pleuronectis*)	Mediterranean (France)	Euzet (1959)
E. moschata	*Scolex pleuronectis unilocularis* (= *S. polymorphus unilocularis*)	Mediterranean (Monaco)	Nouvel (in Dollfus, 1964)

Table 1-14 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER OCTOPODA			
Eledone moschata	*Scolex pleuronectis quadrilocularis* (= *S. polymorphus eledones-moschatae*)	Baltic Sea (Poland); Mediterranean (Italy, Monaco)	von Siebold (1850), Diesing (1863), Dobell (1925), Dollfus (1958, 1964)
Octopus cyanea	Unident. cestode	Central North Pacific Ocean (Hawaii, USA)	Devaney (1981), van Heukelem (1983)
Octopus joubini	*Scolex pleuronectis quadrilocularis*	Gulf of Mexico (Florida to Mississippi, USA)	Cake (1976)
Octopus macropus	*Scolex pleuronectis*	Mediterranean (Italy)	Dobell (1925)
Octopus salutii	*Scolex pleuronectis*	Mediterranean (France)	Hochberg (unpubl.)
Octopus vulgaris	*Acanthobothrium* sp. (= *Scolex pleuronectis*)	English Channel (France)	Dollfus (1923, 1936)
O. vulgaris	*Phyllobothrium* sp. (= *Scolex pleuronectis*)	Mediterranean (France)	Euzet (1959)
O. vulgaris (= *O. octopodia, Polypus octopus*)	*Scolex pleuronectis* (= *S. polymorphus; S. de Bavay*)	Mediterranean (Italy, France); Eastern North Atlantic Ocean (France)	Rudolphi (1819), Dobell (1925), Cuenot (1927)
O. 'vulgaris'	*Scolex pleuronectis bilocularis* (= *S. polymorphus*)	Western North Pacific Ocean (Japan)	Yamaguti (1934)
Pteroctopus tetracirrhus	*Scolex pleuronectis*	Mediterranean (France)	Hochberg (unpubl.)
Scaeurgus unicirrhus	*Scolex pleuronectis*	Mediterranean (France)	Hochberg (unpubl.)
Ocythoe tuberculata	*Scolex* sp.	Mediterranean (Italy)	Dobell (1925)

Table 1-15
Cestodes in ommastrephid squids. Incidence and intensity of infections in relation to host species and localities of capture
(Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Dosidicus gigas	*Dinobothrium* sp.	223	150–360	44.6	10–25,000 (7000–8000)	Eastern South Pacific Ocean (0–17° S, 85–86° W)	Gaevskaya and co-authors (1982)
D. gigas	*Dinobothrium* sp.	403	70–430	73	1–50,000 (500–2000)	Eastern South Pacific Ocean (0–22° S)	Gaevskaya and co-authors (1983)
D. gigas	*Pelichnibothrium speciosum* (= *Scolex pleuronectis bilocularis*)	ni				Eastern North Pacific Ocean (California, USA)	Riser (1949, 1956a), Fields (1965)
D. gigas	*Phyllobothrium* sp.	ni	ni	2.9	1	Eastern South Pacific Ocean (0–17° S, 85–86° W, Chile)	Gaevskaya and co-authors (1982), Dollfus (1964)
D. gigas	*Phyllobothrium* sp. type 4	ni				Eastern North Pacific Ocean (California, USA)	MacGinitie and MacGinitie (1949), Riser (1949)
D. gigas	*Phyllobothrium* sp.	410	20–400	ni	ni	Eastern Pacific Ocean (2° N–22° S, 82–87° W)	Gaevskaya and co-authors (1987)
D. gigas	*Phyllobothrium* sp.	380	30–380	1.1	1–5	Eastern South Pacific Ocean (0–21° S, 83–106° W)	Shukhgalter (1985)
D. gigas	*Scolex pleuronectis unilocularis*	ni				Eastern North Pacific Ocean (Mexico)	Hochberg (unpubl.)
D. gigas	*S. p. unilocularis*	410	20–400	ni	ni	Eastern Pacific Ocean (2° N–22° S, 82–87° W)	Gaevskaya and co-authors (1987)
D. gigas	*S. pleuronectis*	380	30–380	66.5	to 63,000 (1000 to 10,000)	Eastern South Pacific Ocean (0–21° S, 83–106° W)	Shukhgalter (1985)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Dosidicus gigas	*Nybelinia* sp. (= *Tetrarhynchus* sp.)	ni				Eastern North Pacific Ocean (California, USA)	MacGinitie and MacGinitie (1949)
D. gigas	*Tentacularia coryphaenae*	223	150–360	24	1–4	Eastern South Pacific Ocean (0–17° S, 85–86° W)	Gaevskaya and co-authors (1982)
D. gigas	*T. coryphaenae*	410	20–400	ni	ni	Eastern Pacific Ocean (2° N–22° S, 82–87° W)	Gaevskaya and co-authors (1987)
D. gigas	*T. coryphaenae*	380	30–380	7.5	1–5	Eastern South Pacific Ocean (0–21° S, 83–106° W)	Shukhgalter (1985)
Eucleoteuthis luminosa	*Grillotia* sp.	1	132	100	1	Central South Atlantic Ocean (15° S, 7° W)	Zuev and co-authors (1975)
E. luminosa	*Scolex pleuronectis*	29	150–220	96.6	ni	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
E. luminosa	*Nybelinia* sp.	29	150–220	ni	ni	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Illex argentinus	*Pelichnibothrium* sp.	132	ni	ni	ni	Western South Atlantic Ocean (Argentina)	Threlfall (1970)
I. argentinus	*Phyllobothrium* type I	377	60–360	25	1–31	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a), Shukhgalter (1986a)
I. argentinus	*P.* type II	377	60–360	70	1–31	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a), Shukhgalter (1986a)
I. argentinus	*P.* type III	377	60–360	5	1	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a), Shukhgalter (1986a)
I. argentinus	*Phyllobothrium* sp.	132	ni	50.8	ni	Western South Atlantic Ocean (Argentina)	Threlfall (1970)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Illex coindeti	*Dinobothrium 'plicitum'*	ni				Eastern North Atlantic Ocean (France); English Channel (England)	Dollfus (1958, 1964), Legendre (in Dollfus, 1964), Gaevskaya and Nigmatullin (1975, 1978) Mohr (unpubl.)
I. coindeti	*Phyllobothrium tumidum*	ni				Eastern North Atlantic Ocean (France)	Dollfus (1958, 1964), Euzet (1959), Legendre (in Dollfus, 1964)
I. coindeti	*Phyllobothrium* sp.	34	ni	32	ni	Eastern North Atlantic Ocean (Ireland)	Gaevskaya and Nigmatullin (1975, 1978)
I. coindeti	*Scolex pleuronectis* (= *S. polymorphus*)	ni				Eastern North Atlantic Ocean (France); English Channel (England)	Dollfus (1958, 1964), Mohr (unpubl.), Legendre (in Dollfus, 1964)
I. coindeti	*S. pleuronectis*	1880	ni	0.4	ni	Eastern North Atlantic Ocean (Africa)	Gaevskaya and Nigmatullin (1975, 1978)
I. coindeti	*Nybelinia yamagutii*	1880	ni	0.05	ni	Eastern North Atlantic Ocean (Africa)	Gaevskaya and Nigmatullin (1975, 1978)
Illex illecebrosus	*Ceratobothrium xanthocephalum* (= *Scolex bothriis bilocularis*)	ni				Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
I. illecebrosus (= *Ommastrephes*)	*Dinobothrium septaria* (= *D. plicitum, Thysanocephalum crispum*)	ni				Western North Atlantic Ocean (Newfoundland, Canada; Massachusetts, USA)	Linton (1897), Frost and Thompson (1932), Guiart (1933), Dollfus (1936), Squires (1957), Aldrich (1964), Mercer (1965), Brown and Threlfall (1968a), Gaevskaya and Nigmatullin (1975, 1978)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Illex illecebrosus	*Dinobothrium septaria* (= *D. plicitum*)	802	140–319	18.7	ni	Western North Atlantic Ocean (Newfoundland, Canada; Massachusetts, USA)	Brown and Threlfall (1968a, b)
I. illecebrosus	*Pelichnibothrium speciosum*	802	140–319	0.1	ni	Western North Atlantic Ocean (Newfoundland, Canada)	Brown and Threlfall (1968a, b)
I. illecebrosus	*Phyllobothrium loliginis* (= *Taenia loliginis*, *Tetrabothrium loliginis*)	ni				Western North Atlantic Ocean (Massachusetts, USA)	Leidy (1887, 1890), Linton (1897, 1922), Stunkard (1977)
I. illecebrosus	*P. loliginis*	ni				Eastern North Atlantic Ocean (France)	Guiart (1933)
I. illecebrosus	*Phyllobothrium* sp.	76	ni	49	ni	Eastern North Atlantic Ocean (Ireland)	Gaevskaya and Nigmatullin (1975, 1978)
I. illecebrosus	*Phyllobothrium* sp.	802	140–319	39.6	1–41	Western North Atlantic Ocean (Newfoundland, Canada)	Brown and Threlfall (1968b)
I. illecebrosus	*Phyllobothrium* sp.	ni				Western North Atlantic Ocean (Newfoundland, Nova Scotia, Canada; Massachusetts, USA)	Leidy (1890), Linton (1922), Frost and Thompson (1932), Squires (1957), Aldrich (1964), Mercer (1965), Gaevskaya and Nigmatullin (1975, 1978), Nesis (1967)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Illex illecebrosus	*Scolex pleuronectis* (= *S. polymorphus*)	802	140–319	0.1	ni	Western North Atlantic Ocean (Newfoundland, Canada; Massachusetts, USA)	Brown and Threlfall (1968a), Gaevskaya and Nigmatullin (1975, 1978), Stunkard (1977)
I. illecebrosus	*Lacistorhynchus tenue*	ni				Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
I. illecebrosus	*Nybelinia bisulcata*	ni				Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
I. illecebrosus	*Nybelinia* sp.	802	140–319	0.1	ni	Western North Atlantic Ocean (Newfoundland, Canada)	Brown and Threlfall (1968a), Gaevskaya and Nigmatullin (1975, 1978)
I. illecebrosus	*Otobothrium crenacole*	ni				Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
I. illecebrosus	*Tentacularia coryphaenae*	ni				Western North Atlantic Ocean (Florida, USA)	Threlfall and co-authors (1971)
Martialia hyadesi	*Phyllobothrium* sp.	21	260–305	47.6	1–4	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
Nototodarus sloani	*Nybelinia* sp.		[see Table 1-22]			Western South Pacific Ocean and Tasman Sea (New Zealand)	Smith and co-authors (1981)
Ommastrephes bartrami	*Phyllobothrium* sp.	60	100–760	1.9	ni	Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1976b)
O. bartrami	*Phyllobothrium* sp.	42	120–450	3.0	ni	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1976b)
O. bartrami	*Phyllobothrium* sp.	184	150–450	84.2	1–225	Western North Pacific Ocean (Japan)	Kurochkin and Solov'eva (1982)
O. bartrami	*Scolex pleuronectis*	35	160–405	55.6	1–500	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML * (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Ommastrephes bartrami	*Scolex pleuronectis*	7	240–360	14.2	2	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
O. bartrami	*Nybelinia lingualis*	60	100–760	25	1–2	Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1974, 1975, 1976b, 1978), Gaevskaya (1976)
O. bartrami	*N. lingualis* (= *N. africana* f. *ommastrephes*)	42	120–450	6.1	1–2	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1974, 1976b)
O. bartrami	*N. lingualis*	ni	ni	20	2–16	Indian Ocean	Belyaeva (1979)
O. bartrami	*Nybelinia surmenicola*	184	150–450	40.4	1–25	Western North Pacific Ocean (Japan)	Kurochkin and Solov'eva (1982)
O. bartrami	*Nybelinia* sp.	ni				Indian Ocean	Belyaeva (1979)
O. bartrami	*Tentacularia coryphaenae*	35	160–405	25	1–2	Western South Atlantic Ocean (37–47° S)	Gaevskaya (1976), Gaevskaya and co-authors (1986a)
O. bartrami	*T. coryphaenae*	60	100–760	5.7	ni	Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1974, 1975, 1976b, 1978)
O. bartrami	*T. coryphaenae*	42	120–450	19	ni	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1976b)
O. bartrami	*T. coryphaenae*	ni	ni	20	1–10	Indian Ocean	Belyaeva (1979)
Ommastrephes caroli	*Tentacularia coryphaenae*	ni				Western North Atlantic Ocean (Grand Banks)	Threlfall and co-authors (1971)
Ornithoteuthis antillarum	*Nybelinia* sp.	ni	ni	92	ni	Atlantic Ocean	Nesis and Nigmatullin (1979)
Sthenoteuthis oualaniensis	*Dinobothrium* sp.	80	70–320	26	10–25,000	Eastern South Pacific Ocean	Gaevskaya and co-authors (1983)
S. oualaniensis	*Phyllobothrium* sp.	303	ni	seldom	ni	Indian Ocean and Red Sea	Naidenova and co-authors (1981, 1985)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Sthenoteuthis oualaniensis	*Phyllobothrium* sp.	82	70–280	6.0	1–5	Eastern South Pacific Ocean (0–21° S, 83–106° W)	Shukhgalter (1985)
S. oualaniensis	*Scolex pleuronectis unilocularis*	ni				Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
S. oualaniensis	*S. pleuronectis*	82	70–280	ni	to 5000 (100–1000)	Eastern South Pacific Ocean (0–21° S, 83–106° W)	Shukhgalter (1985)
S. oualaniensis	Unident. pseudophyllidean	ni				Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
S. oualaniensis	*Christianella* sp.	80	70–320	1	1	Eastern South Pacific Ocean (0–22° S, 83–106° W)	Gaevskaya and co-authors (1983)
S. oualaniensis	*Nybelinia lingualis*	ni	ni	50	2–16	Indian Ocean	Belyaeva (1979)
S. oualaniensis	*N. lingualis*	303	ni	90	1–200+	Indian Ocean and Red Sea	Naidenova and co-authors (1981, 1985)
S. oualaniensis	*Nybelinia* sp.	ni				Indian Ocean	Belyaeva (1979)
S. oualaniensis	*Nybelinia* sp. A.	ni				Central North Pacific Ocean	Hochberg (unpubl.)
S. oualaniensis	*Tentacularia coryphaenae*	ni	ni	30	1–17	Indian Ocean and Red Sea	Belyaeva (1979)
	T. coryphaenae	303	ni	65	ni	Indian Ocean and Red Sea	Naidenova and co-authors (1981, 1985)
	T. coryphaenae	82	70–280	12.1	1–5	Eastern South Pacific Ocean (0–21° S, 83–106° W)	Shukhgalter (1985)
Sthenoteuthis pteropus (= *Ommastrephes*)	*Phyllobothrium* type I	2262	20–500	25	1–25 (1–5)	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1978, 1981), Gaevaskaya (1977a, 1978)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Sthenoteuthis pteropus (= *Ommastrephes*)	*Phyllobothrium* type II	2262	20–500	0.7	1–3	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1978, 1981), Gaevskaya (1977a, 1978), Naidenova and Zuev (1978b)
S. pteropus	*Scolex pleuronectis bilocularis*	ni				Tropical Atlantic Ocean	Naidenova and Zuev (1978b)
S. pteropus	*Hepatoxylon trichiuri*	2262	20–500	0.2	1	Tropical Atlantic Ocean	Gaevskaya (1977a), Gaevskaya and Nigmatullin (1981)
S. pteropus	*Nybelinia lingualis* (= *N. lingualis* var. 1, *N.* f. *typica*, N. sp.)	2262	20–500	47	1–250 (1–20)	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1978, 1981), Gaevskaya (1977a)
S. pteropus	*N. lingualis*	883	120–500	94.4	1–204	Eastern Central Atlantic Ocean (20–32° W)	Naidenova (1978), Naidenova and Zuev (1978b)
S. pteropus (= *Ommastrephes*)	*Nybelinia yamagutii*	2262	20–500	38	1–20 (1–5)	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1978, 1981), Gaevskaya (1977a), Naidenova (1978), Naidenova and Zuev (1978b)
S. pteropus (= *Ommastrephes*)	*Tentacularia coryphaenae*	2262	20–500	46	1–62 (1–15)	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975, 1978, 1981)
S. pteropus	*T. coryphaenae*	883	120–500	49.6	1–55	Eastern Central Atlantic Ocean (20–32° W)	Naidenova (1978), Naidenova and Zuev (1978b)
Todarodes angolensis	*Phyllobothrium* sp.	ni	250+	2.6	ni	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1978)
T. angolensis	*Phyllobothrium* sp.	18	200–295	40	1–7	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
T. angolensis	*Tentacularia coryphaenae*	ni	250+	1.3	ni	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1978)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Todarodes angolensis	*Tentacularia coryphaenae*	18	200–295	16.7	1–2	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
Todarodes pacificus (= *Ommastrephes sloani pacificus*)	*Pelichnibothrium speciosum*	*500*	*ni*	*14.6*	*1–5*	*Japan Sea (USSR)*	*Kurochkin (1972)*
T. pacificus	*P. speciosum* (= *Scolex polymorphus bilocularis*)	ni				Western North Pacific Ocean (Japan)	Yamaguti (1934)
T. pacificus	Unident. pseudophyllidean	ni				Western North Pacific Ocean (Japan)	Nagasawa and Nakata (1984)
T. pacificus	*Nybelinia surmenicola*	500	ni	22	1–10	Japan Sea (USSR)	Kurochkin (1972)
T. pacificus (*O. s. spacificus*)	*N. surmenicola* (= *Tetrarhynchus*)	ni				Western North Pacific Ocean (Japan)	Okada (in Dollfus, 1929), Yamaguti (1934), Dollfus (1942)
Todarodes sagittatus (= *Ommastrephes, Loligo todarus*)	*Phyllobothrium dohrni* (= *Orygmatobothrium Dohrni, Scolex phyllobothrii*)	ni				Mediterranean (Italy)	Monticelli (1888), Dollfus (1936, 1958)
T. sagittatus	*Phyllobothrium loliginis*	ni				Mediterranean (Italy, France)	Guiart (1933), Harant (in Wirz, 1958), Dollfus (1958)
T. sagittatus	*Phyllobothrium* sp. (= *P.* type I, *P.* type II)	254	ni	0.4	ni	Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1978)
T. sagittatus	*Scolex pleuronectis*	ni				Eastern North Atlantic Ocean (off Africa)	Reimer (1974)
T. sagittatus	*Scolex* sp. (= *Cysticercus sepiolae, Monostoma loliginum, M. todari*)	ni				Mediterranean (Italy)	Delle Chiaje (1830, 1841), Dollfus (1936), Monticelli (1892)

Table 1-15 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Todarodes sagittatus	*Hepatoxylon todari* (= *Dibothriorhynchus todari*)	ni				Mediterranean (Italy)	Delle Chiaje (1822, 1829, 1841), Dollfus (1958)
T. sagittatus	*Nybelinia lingualis* (= *Amphistoma loliginis*)	ni				Mediterranean (Italy)	Delle Chiaje (1830, 1841), Dollfus (1958)
T. sagittatus	*Nybelinia* sp.	ni				Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1978)
T. sagittatus	*Tentacularia coryphaenae*	ni				Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1978)
T. sagittatus	*Tetrarhynchus* sp.	ni				Mediterranean (Italy)	Dollfus (1936)
Todaropsis eblanae	*Dinobothrium plicitum* (= *D.* sp.)	ni				Eastern North Atlantic Ocean (France)	Dollfus (1936, 1958), Legendre (in Dollfus, 1964)
T. eblanae	*Phyllobothrium tumidum* (= *P. loliginis*)	ni				Eastern North Atlantic Ocean (France)	Dollfus (1936, 1958), Legendre (in Dollfus, 1964)
T. eblanae	*Phyllobothrium* sp. I type 2	96	ni	5.2	ni	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1978)
T. eblanae	*Scolex pleuronectis*	96	ni	2.8	ni	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1978)

ni: Not identified (): Average intensity

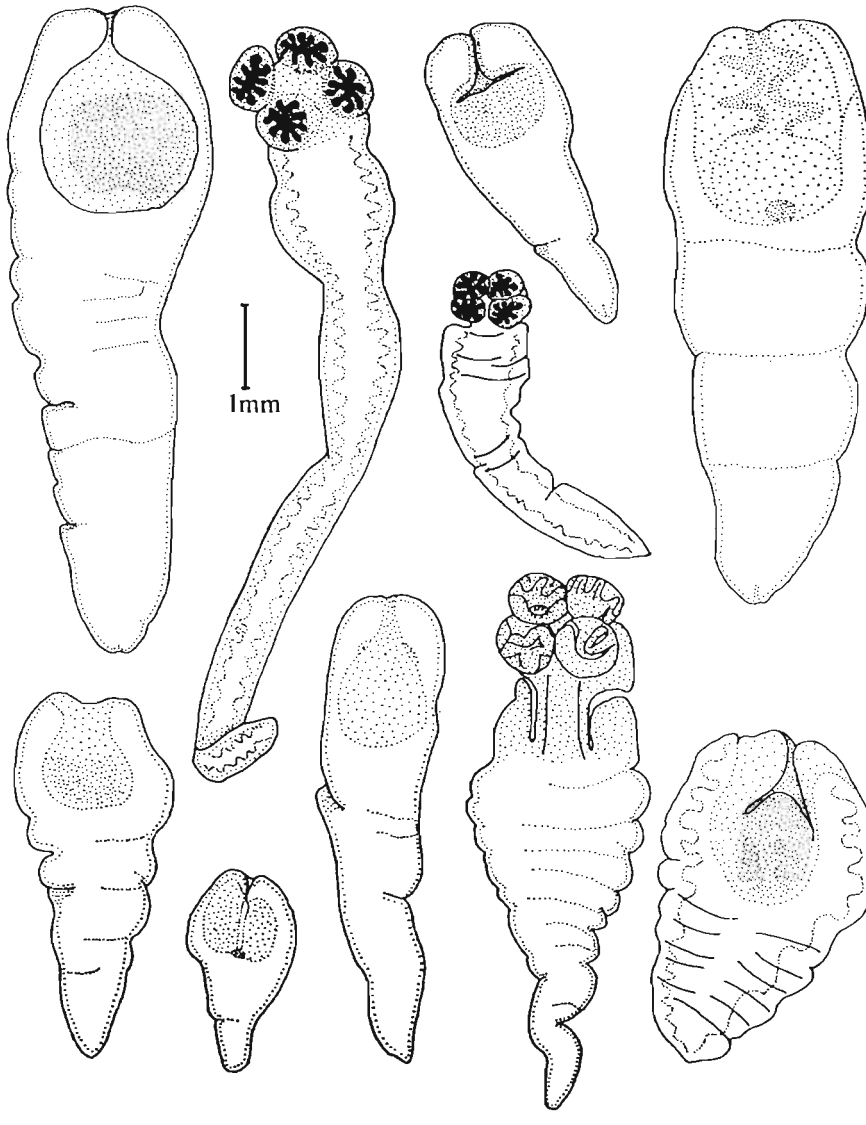


Fig. 1-64: Larval tetraphyllidean cestodes. Range of sizes and shapes of plerocercoids of *Phyllobothrium* sp. from *Illex argentinus*; note in most cases the scolices are invaginated. (After Threlfall, 1970.)

1936, 1958, 1964; Squires, 1957; Mercer, 1965; Brown and Threlfall, 1968a; Gaevskaya and Nigmatullin, 1975, 1978). Stunkard (1977) indicated the strong possibility that *D. septaria* and *D. plicitum* are conspecific. In the eastern South Pacific Ocean 26 % of *Sthenoteuthis* and 73 % of *Dosidicus* examined had *Dinobothrium* larvae, often in excess of 10,000 worms host^{-1} (Gaevskaya and co-authors, 1983). Dinobothrids are known to mature in large, oceanic selacians such as species of *Cetorhinus* and *Carcharodon*.

Details of the relationships between cestodes and cephalopods have been investigated in relatively few cases and then only in ommastrephid squids which are commercially

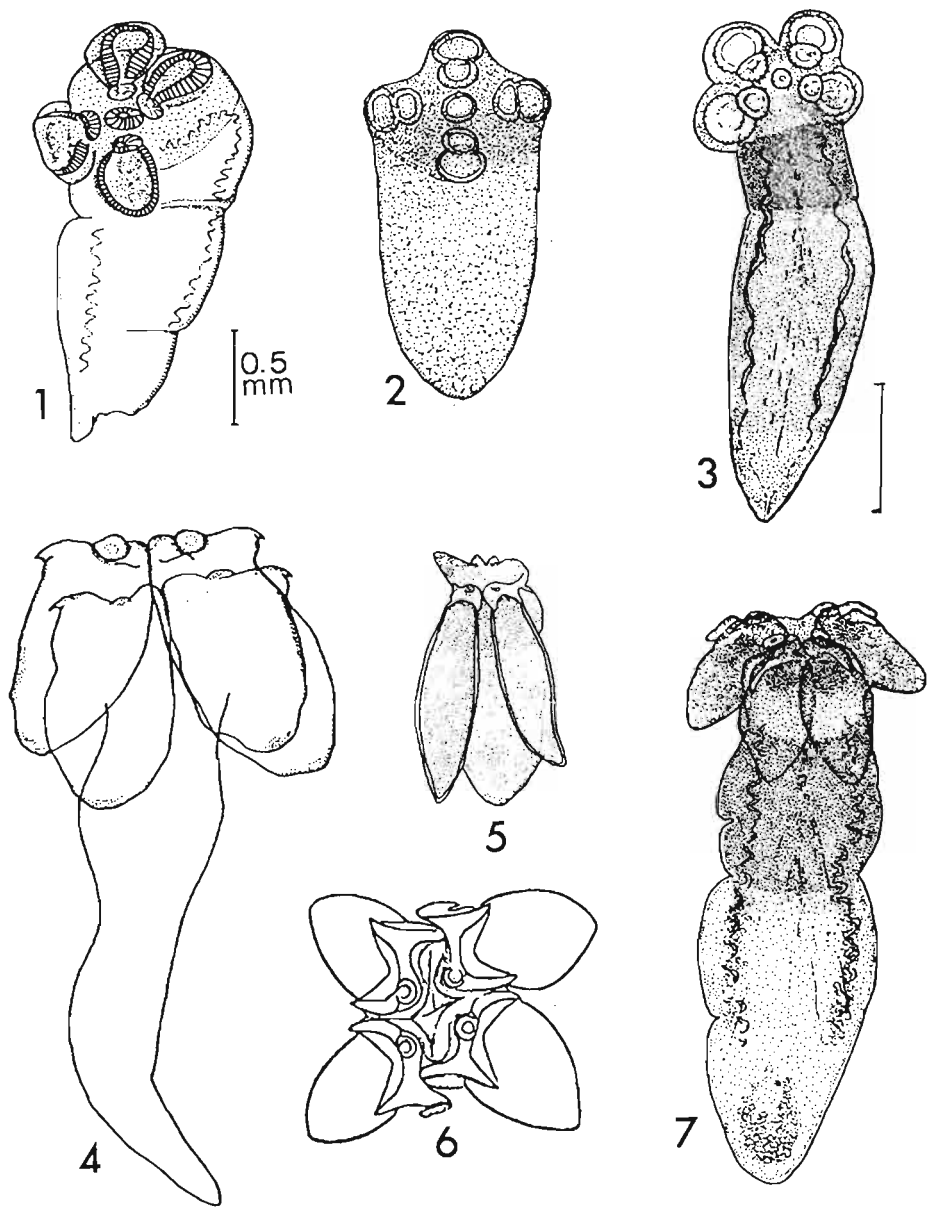


Fig. 1-65: Larval tetraphyllidean metacestodes from cephalopods. Plerocercoids from a variety of hosts. 1: *Pelichnobothrium speciosum* from *Illex argentinus*. 2 and 3: *Ceratobothrium xanthocephalum* from *Heterololigo* (= *Loligo*) *pealei*. 4: *Dinobothrium plicitum* from *Illex illecebrosus*. 5: scolex of *Thysanocephalum* (probably equals *Dinobothrium plicitum*) from *Illex illecebrosus*, lateral view. 6: scolex of *Dinobothrium plicitum* from *Todaropsis eblanae*, en face view. 7: *Dinobothrium septaria* from *Illex illecebrosus*. Scale bars in mm. (1 after Threlfall, 1970; 2, 3, and 7 after Stunkard, 1977 (5 from Linton, 1897); 4 after Brown and Threlfall, 1968a; 6 after Joyeux and Baer, 1936.)

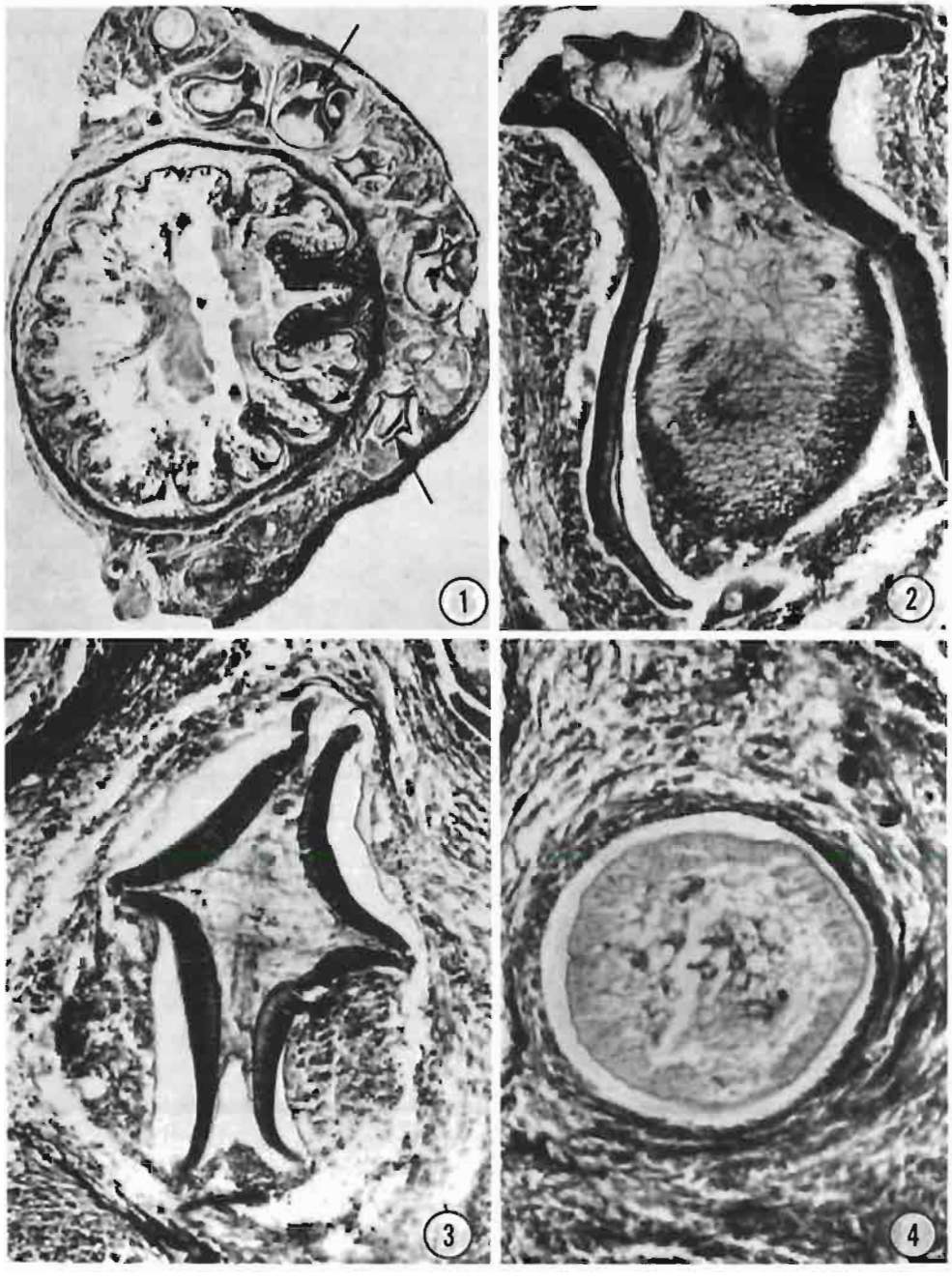


Fig. 1-66: *Dinobothrium plicitum*. Sections of the intestine of *Illex illecebrosus* showing encysted specimens of the tetraphyllidean plerocercoid. 1: Cross section of the intestine with encysted cestodes (arrows). 2: Longitudinal section of an encysted plerocercoid. 3: Cross section through a scolex. 4: Cross section through a cyst containing the cestode. (After Brown and Threlfall, 1968a.)

important. Most reports simply document the presence of cestodes, the site of infection and list the geographic locality of collection. Only in the last 20 years have quantitative studies been initiated which address the age, size and sex of the hosts and provide information on number of hosts examined, percentage of squids infected and number of parasites recovered per host.

The northern short-finned squid *Illex illecebrosus* is medium-sized and lives along the coasts of the western and northern North Atlantic. This ommastrephid is commonly fished commercially by both Canada and the United States. Seasonal northward/inshore and southward/offshore migrations take place which can be connected with environmental conditions, feeding and reproductive cycles.

The cestode parasites of *Illex illecebrosus* have been extensively studied by Squires (1957), Mercer (1965) and Brown and Threlfall (1968a, b). Of 802 squid examined by Brown and Threlfall an overall total of almost 50 % were infected with larval tetraphyllideans. In 1966 and 1967 39 to 40 % were infected with plerocercoids of *Phyllobothrium*,

Table 1-16
Monthly variation in intensity of infection by cestode parasites in *Illex illecebrosus*, July to September, 1966 and July to November, 1967 (After Brown and Threlfall, 1968b)

Infection	July		August		September		October	November
	1966	1967	1966	1967	1966	1967	1967	1967
No. squid infected	10	52	111	74	76	1	71	19
% squid infected	66.7	96.3	46.1	74.0	56.7	5.5	34.6	38.7
No. squid not infected	5	2	130	26	58	17	130	30
% squid not infected	33.3	3.7	53.9	26.0	43.3	94.5	65.4	61.3
Total	*15*	*54*	*241*	*100*	*134*	*18*	*205*	*49*

whereas only 15 to 21 % were infected with *Dinobothrium*. The indices of infection varied significantly on a seasonal or monthly basis and from year to year (Table 1-16). The fluctuations are due principally to the short life cycle of the host squid (12 months from hatching to spawning) and to the structure of the squid populations with several spawning periods and thus several age classes per year (Brown and Threlfall, 1968b).

The months of peak infection are July and August (Table 1-16). There is an increase in the incidence of infection in *Dinobothrium* with increasing mantle length (Fig. 1-67; Table 1-17), whereas with *Phyllobothrium* the incidence tends to level off or decline slightly with increasing size and age of host (Table 1-18; Mercer, 1965). Squid smaller than 130 mm ML do not appear to harbor tetraphyllidean parasites. Mercer (1965) postulated that *Illex illecebrosus* becomes infected with cestodes while feeding in offshore waters around the Grand Banks. Tetraphyllidean larvae have been reported in copepods, euphausiids and other planktonic invertebrates which are preyed upon by *I. illecebrosus* (Dollfus, 1936; Reimer, 1975b, c; Shimazu, 1978, 1982; Kagei, 1979b).

Brown and Threlfall (1968b) found no differences in the indices of infection between male and female squid in 1966. However, in 1967 a significant difference was observed. The infection in males peaked early in the sampling period and then dropped to zero in September. This was followed by a second peak in the number of squid infected and a

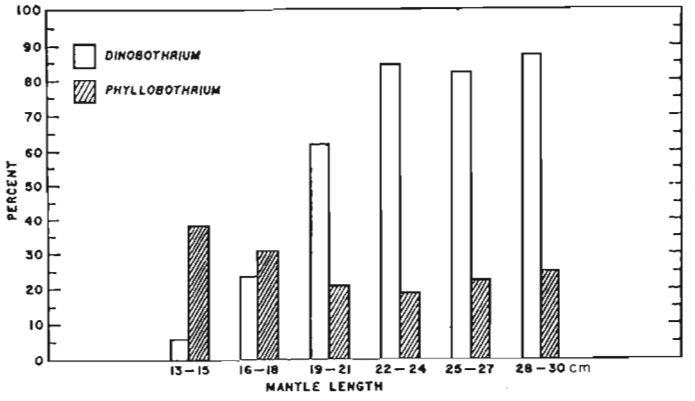


Fig. 1-67: Percentage of *Illex illecebrosus* infected with plerocercoids of *Phyllobothrium* and *Dinobothrium*, May to November, 1952. (After Squires, 1957.)

Table 1-17
Percentage infection with *Dinobothrium plicitum* as a function of increasing mantle length in *Illex illecebrosus*, 1966 and 1967 (After Brown and Threlfall, 1968b)

Mantle length intervals (mm)	No. squid infected		Total sampled		% infected	
	1966	1967	1966	1967	1966	1967
140–159	0	0	9	0	0	0
160–179	0	0	14	5	0	0
180–199	3	6	41	33	7.3	18.1
200–219	13	23	123	102	10.5	22.4
220–239	21	16	120	69	7.5	23.1
240–259	18	22	63	98	28.5	22.4
260–279	7	15	13	68	53.8	22.0
280–299	3	7	3	33	100.0	21.5
300–319	0	1	1	4	0	25.0

1966, $P > 0.005$; 1967 $P = 0.10$–0.05

second low when no parasites were found. A third but smaller peak occurred in late October. Although the same pattern was evident in females only two peaks were observed.

With respect to numbers of parasites, less than 40 plerocercoids of *Phyllobothrium* were found with an average of 6.6 worms per *Illex illecebrosus*. In contrast, in the case of *Dinobothrium*, often more than 100 plerocercoids were found per host (Fig. 1-66). The heaviest densities of *Dinobothrium* larvae were encysted in the caecum, though in more than 50 % of the parasitized squid additional body organs were infected. The number and combinations of organs infected differed considerably during the 2 years of study (Table 1-19; Brown and Threlfall, 1968b).

Illex argentinus is another abundant ommastrephid squid which lives in neritic waters off South America. At least 3 distinct types or growth stages of *Phyllobothrium* plerocercoids have been distinguished in *I. argentinus* on the basis of the shape of the bothridia, the length of the worms, and the incidence of infection (Gaevskaya and co-authors, 1986a;

Table 1-18
Percentage infection with cestode plerocercoids as a function of increasing mantle length in *Illex illecebrosus*, 1966 and 1967 (After Brown and Threlfall, 1968b)

Mantle length intervals (mm)	No. squid infected		Total sampled		% infected	
	1966	1967	1966	1967	1966	1967
140–159	1	0	9	0	11.1	0.0
160–179	4	0	14	5	28.6	0.0
180–199	13	17	41	33	31.7	51.5
200–219	58	79	123	102	47.2	77.5
220–239	61	38	120	69	50.9	55.1
240–259	32	39	63	98	50.8	30.6
260–279	10	24	13	68	76.9	35.3
280–299	4	12	6	33	66.7	36.4
300–319	1	4	1	4	100.0	100.0

1966, $P = 0.500$–0.250, 1967 $P > 0.005$

Table 1-19
Sites involved in multiple infections by *Dinobothrium plicium* in *Illex illecebrosus*, 1966 and 1967 (After Brown and Threlfall, 1968b)

Sites infected	No. infected		% infected	
	1966	1967	1966	1967
Intestine – caecum	17	43	44.7	95.6
Intestine – kidney mesentery	1	0	2.6	0.0
Intestine – mesenteries	0	1	0.0	2.2
Esophagus – mesenteries	1	0	2.6	0.0
Caecum – mesenteries	1	1	2.6	2.2
Intestine – caecum – mesenteries	4	0	10.6	0.0
Intestine – caecum – stomach	5	0	13.3	0.0
Intestine – caecum – esophagus	2	0	5.2	0.0
Caecum – esophagus – stomach	1	0	2.6	0.0
Intestine – caecum – esophagus – stomach	1	0	2.6	0.0
Intestine – caecum – esophagus – mesenteries	1	0	2.6	0.0
Intestine – caecum – esophagus – stomach – mesenteries	4	0	10.6	0.0
Total	*38*	*45*		

Shukhgalter, 1986a). Type I larvae have smooth, oval bothridia, are 0.6 to 0.9 mm long and occur in 25 % of the hosts examined by the Russian workers. Type II larvae (Fig. 1-64) have wavy-edged bothridia, range in size from 1 to 26 mm and occur in 70 % of the hosts. Type III larvae have scalloped-edged bothridia, range in length from 1 to 21 mm but occur in only 5 % of the host squids. In the North and South Atlantic Ocean the overall incidence of infection with *Phyllobothrium* plerocercoids in *I. argentinus* ranges from 48 % (Shukhgalter, 1986a) to 51 % (Threlfall, 1970), in *I. illecebrosus* it averages 50 % (Brown and Threlfall, 1968b). However, the plerocercoids appear to be distinctly different in these 2 host species (Table 1-20; Threlfall, 1970).

Table 1-20
Comparison of *Phyllobothrium* plerocercoids from *Illex illecebrosus* and *I. argentinus*. Measurements in mm (After Threlfall, 1970)

Morphological criteria	*Illex illecebrosus*	*Illex argentinus*
Length	10.0–36.0	0.8–10.5
Width	–	0.8– 2.9
Bothridial length	1.30	0.41–0.74
Accessory suckers, diameter	0.32	0.21–0.27
Apical sucker, diameter	0.46	0.19–0.27

In winter, 8 % of schooling *Illex argentinus* in the size range of 6 to 10 cm ML are infected. Only small Type I worms are found throughout the digestive tract. The incidence and intensity of infection increases to over 50 % in squid with mantle lengths of 16 to 20 cm. Approximately 80 % of adult squid with mantle lengths over 30 cm are infected. In the largest squids plerocercoids aggregate in the rectum, and large worms (Types II and III) principally are present. Small larvae are obtained when small *I. argentinus* feed on pelagic invertebrates. Large plerocercoids are ingested when adults feed on small bony fishes and squids. Transitional sizes come from growth of small larvae in the squid hosts. Depending on age and size, *I. argentinus* may function as either secondary or tertiary intermediate hosts in the life cycle of *Phyllobothrium*.

The flying squid *Sthenoteuthis pteropus* is a large predator which dominates the epipelagic realm of the tropical Atlantic Ocean. In studies of this important oceanic ommastrephid Gaevskaya and Nigmatullin (1981) found, in contrast to the situation in inshore forms such as *Illex*, that an overall average of 25 % were infected with plerocercoids of the genus *Phyllobothrium*. The incidence increased with increasing host age and size, although the number of plerocercoids per host remained approximately the same (Fig. 1-60).

The genus *Pelichnibothrium* is represented by 2 species, though some workers consider the genus to be monotypic (Yamaguti, 1959). Originally described from California (USA) by Riser (1949, 1956a), *P. speciosum* and *P. caudatum* occur in *Dosidicus gigas* and *Loligo opalescens* respectively. Off California, the digestive glands and intestines of several *D. gigas* were massively infected with very large plerocercoids, up to 15 cm long (MacGinitie and MacGinitie, 1949). *P. speciosum* (Fig. 1-65, 1) also has been recovered off Japan in *Loligo* sp. (Yamaguti, 1934), off Newfoundland (Canada) in *Illex illecebrosus* (Brown and Threlfall, 1968a), and off Argentina in *I. argentinus* (Threlfall, 1970). Adult worms have been recovered from the blue shark *Prionace glauca*, the opah *Lampris regia*, and the bluefin tuna *Thunnis thynnis* (Yamaguti, 1934). Larval stages have been reported from the euphausiid *Thysanoessa longipes* off Japan (Shimazu, 1975a; Kagei, 1979b).

Loligo vulgaris is known to harbor larval cestodes of several additional genera. *Diplobothrium pruvoti* was described in detail by Guiart (1933). Dollfus (1936) later reclassified the species and placed it in the genus *Scyphophyllidium* (Fig. 1-69, 1). According to Dollfus (1958) the true identity of the second species, *Bothriocephalus loliginis*, originally named by Delle Chiaje (1829), is still an enigma.

The genus *'Scolex'* (Figs 1-68 and 1-69) is a heterogenous assemblage in which tetraphyllidean plerocercoids of uncertain affinity are placed. Several distinct types of

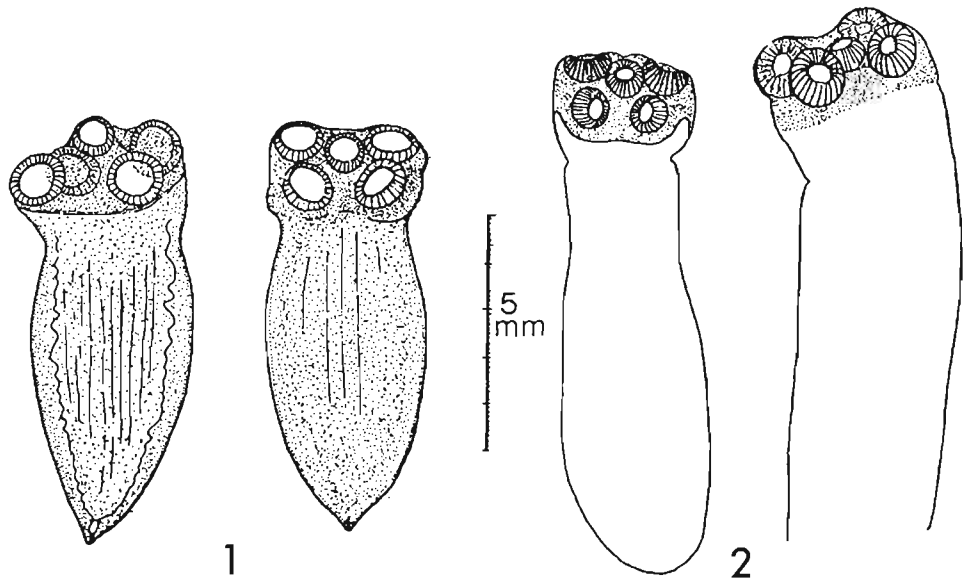


Fig. 1-68: Larval tetraphyllidean metacestodes. *Echeneibothrium* sp. (formerly referred to as *Scolex de Bavay* or *S. polymorphus unilocularis*) from *Loligo vulgaris*. 1: from the stomach wall; 2: from the gonads. (After Dollfus, 1929.)

'Scolex' larvae have been described from some 30 species of cephalopods (Tables 1-14 and 1-15) but most cannot be assigned to a specific genus or species (Dollfus, 1964). The unilocular *'Scolex de Bavay'* (Fig. 1-68) from the stomach and gonads of *Loligo vulgaris* (= *L. loligo*) is now placed in the genus *Echeneibothrium* (Dollfus, 1964). Wagener's *'Scolex bothriis bilocularis'* was found in *L. pealei* by Stunkard (1977) and tentatively identified as *Ceratobothrium xanthocephalum* (Fig. 1-65, 2 and 3). Adult cestodes of the genus *Ceratobothrium* infect the spiral valve of shark genera such as *Glaeocerdo, Lamna* and *Isurus*.

'Scolex pleuronectis' represents a complex of species, the members of which probably belong to genera such as *Phyllobothrium, Monorygma* or *Acanthobothrium* (Euzet, 1959; Cake 1976; Avdeeva and Avdeev, 1980). Depending on the number of suckers in the bothridia, subspecific types have been designated as *'unilocularis'*, *'bilocularis'*, *'trilocularis'*, or *'quadrilocularis'* (Figs 1-68 and 1-69). Other unidentified tetraphyllidean larvae are referred to by the name *'S. polymorphus'* or simply *'Scolex* sp.'. The literature in this area is confused and since descriptions of various larval forms are often inadequate, I have simply tabled information where it is known (Table 1-14 and 1-15). In all cases descriptive details and live history studies are critically needed. For additional information see Dollfus (1923, 1958, 1964), Euzet (1959), Brown and Threlfall (1968a), Gaevskaya and Nigmatullin (1975, 1978), Cake (1976), Stunkard (1977), Naidenova and Zuev (1978b), Avdeeva and Avdeev (1980).

Larval cestodes tenatatively identified as pseudophyllideans occasionally have been found in oceanic cephalopods (Tables 1-14 and 1-15). The rare occurrence indicates that cephalopods are probably accidental or paratenic hosts. Pseudophyllideans could be picked up by cephalopods through ingestion of pelagic invertebrates or fishes. Collard

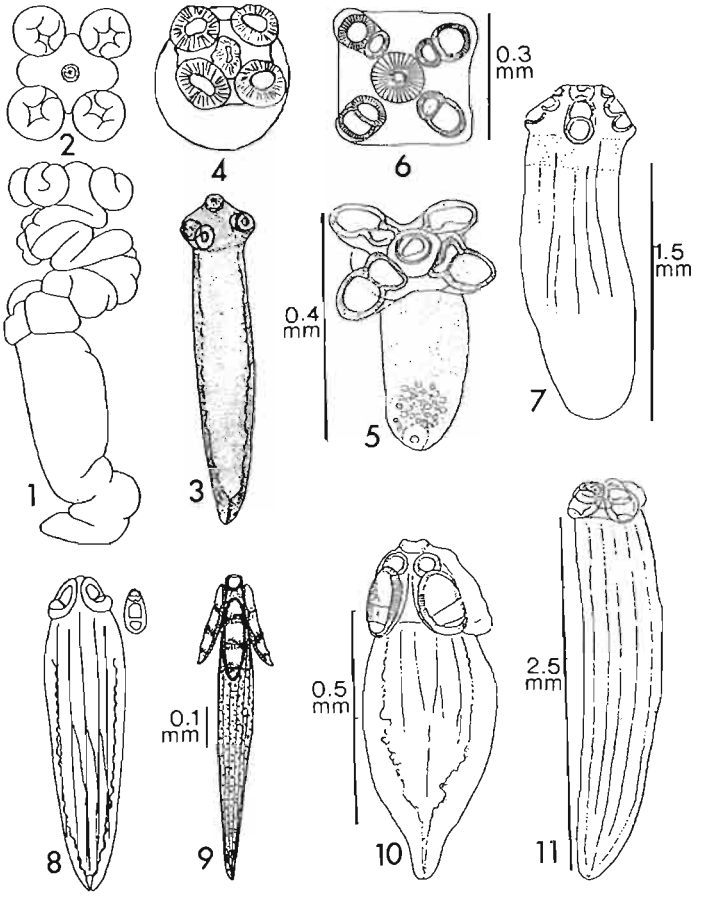


Fig. 1-69: Larval tetraphyllidean metacestodes from cephalopods. Plerocercoids from a variety of hosts. 1 and 2: *Scyphophyllidium pruvoti* from *Loligo vulgaris*; (2) apical view of scolex. 3 and 4: *Scolex pleuronectis unilocularis*; (3) from *Heterololigo* (= *Loligo*) *pealei*; (4) from *Pterygioteuthis giardi*; apical view of scolex. 5 to 7: *S. pl. bilocularis* from *Sepia officinalis*; (6) apical view of scolex. 8: *S. pl. trilocularis* from *Octopus vulgaris*. 9 to 11: *S. pl. quadrilocularis;* (9) from *Octopus joubini*; (10) from *Eledone moschata*; (11) from *Sepia officinalis*. (1 and 2 after Guiart, 1933; 3 after Stunkard, 1977; 4 after Hochberg, unpubl.; 5 to 7, 10 and 11 after Dollfus, 1964; 8 after Dollfus, 1923; 9 after Cake, 1976.)

(1970) reported the presence of unidentified larval pseudophyllideans in midwater myctophid fishes and Reimer (1975d) found similar forms in the mesopelagic fish *Ceratoscopelus maderensis*. Little is known about the biology or life history of pseudophyllidean species infecting oceanic hosts.

Some larval cestodes recovered from cephalopods resemble tetrabothriids which, as adults, occur in all orders of sea birds and in some cetaceans and pinnipeds. The family Tetrabothriidae appears to be closely related to the Tetraphyllideans, as indicated by larval morphology and development, and live cycle patterns (Avdeev and Avdeeva, 1986; Hoberg, 1987a, b). Planktonic crustaceans are thought to be first intermediate hosts, and cephalopods and fishes are second intermediate or paratenic hosts. Many marine

homoetherms, especially pelagic sea birds, feed extensively on cephalopods, and cephalopods may play an important role in the transmission of tetrabothriids. Many of the unidentified 'tetraphyllidean' and 'pseudophyllidean' plerocercoids in squids should be critically reexamined to see if they may in fact be tetrabothriids. As the diets of sea birds are more carefully studied and the cephalopod prey identified it may well be possible to define more accurately the role of cephalopods in tetrabothriid life histories.

Cestodes in the Order Trypanorhynchidea possess 4 tentacles armed with hooks and thus are easily identified. Larval stages are typically encapsulated in tough, fibrous cysts in the walls of the stomach and caecum, in the intestinal mesentaries, or in the gonads of cephalopod hosts. Cephalopods appear to function merely as paratenic hosts, acquiring larval trypanorhynchs by feeding directly on euphausiids and other crustaceans or on small teleost fishes, which also commonly serve as hosts. Trypanorhynchs mature only in the intestine of elasmobranch fishes.

The widely distributed genus *Nybelinia* is the most commonly encountered trypanorhynch in cephalopods (Tables 1-15 and 1-21). *N. lingualis* (Fig. 1-70,1) has been reported from a diversity of hosts in the Mediterranean, Atlantic and Indian Oceans: *Sepia officinalis, Loligo vulgaris, Ommastrephes bartrami, Sthenoteuthis oualaniensis, S. pteropus, Eledone moschata,* and *Octopus vulgaris* (Cuenot 1927; Dollfus, 1929, 1936, 1958, 1964; Pintner, 1930; Gaevskaya, 1976; Gaevskaya and Nigmatullin, 1976b; Naidenova and Zuev, 1978b; Belyaeva, 1979; Naidenova and co-authors, 1981, 1985). The older literature mentions *Amphistoma loliginis* and *Fasciola barbata* (= *F. loliginis*) from *Loligo vulgaris;* in both cases these worms are probably *N. lingualis* (see Dollfus, 1942, 1958).

In large ommastrephid squids the indices of infection by *Nybelinia lingualis* typically exceed 90 %, and more than 200 plerocercoids may be found in a single squid. In contrast the infections in fishes seldom exceed 40 %, and as a rule the intensity is less than 30 plerocercoids fish^{-1}. Squids acquire small numbers of *N. lingualis* by feeding on the first intermediate hosts, pelagic crustaceans such as copepods and euphausiids (Slankis and Shevchenko, 1974). When large squids feed on second intermediate hosts, such as small fishes and other squids, the intensity of the infection is greatly magnified through secondary accumulation of plerocercoids (Figs 1-60 and 1-71; Naidenova and co-authors, 1981, 1985). In fact the main source of infection may be through repeated ingestion of second and not first intermediate hosts. The final hosts of *N. lingualis* are carcharhinid, isurid and other sharks that ingest squids both alive or dead following spawning. In the life cycle cephalopods function as transport or reservoir hosts between small teleosts and selacians.

Off Japan another tentaculariid, *Nybelinia surmenicola* (Fig. 1-79, 8 and 9) has been reported in *Todarodes pacificus* and *Berryteuthis magister* by Dollfus (1929, 1930, 1942), Yamaguti (1934), Kurochkin (1972), and Nagasawa and Nakata (1984). The incidence in *T. pacificus* may reach 22 %. Larval stages of *N. surmenicola* have been found in the euphausiids *Thysanoessa longipes* and *Euphausia pacifica* (Shimazu, 1975a; Kagei, 1979b) and adult stages in the salmon shark *Lamna ditropis* (Shimazu, 1975b). Salmon serve as transport or paratenic hosts for this tentaculariid cestode.

Heterololigo (= Loligo) pealei harbors both *Nybelinia bisulcata* (Fig. 1-70, 6) and *N. yamagutii* (Fig. 1-70, 5). The latter species also is found in *Sthenoteuthis pteropus* and *Illex coindetti* (Gaevskaya and Nigmatullin, 1975, 1978; Gaevskaya, 1977a; Stunkard, 1977). A number of undetermined or undescribed species of *Nybelinia* have been reported from

Table 1-21
Trypanorhynch cestodes in cephalopods other than ommastrephids (Original; compiled from the sources indicated)

Host species	Parasites	Locality	Source
ORDER SEPIOIDEA			
Sepia officinalis	*Christianella minuta* (= *Tetrarhynchus* sp.)	English Channel (France)	Dicquemare (1783), van Benden (1870), Dollfus (1936, 1942, 1958)
S. officinalis	*Grillotia* sp.	English Channel	Dollfus (1958), Reimer (1974)
S. officinalis (= *S. filliouxi*)	*Nybelinia lingualis* (= *Tetrarhynchus lingualis, T. megabothrium, T. sepiae, T. macrobothrium, T. bisulcatus*)	Adriatic (Italy); Mediterranean (Italy, France); English Channel (Belgium, France, England); Eastern North Atlantic Ocean (France)	Dicquemare (1783), Rudolphi (1819), Gros (1847), von Siebold (1850, 1851), Wagener (1854), van Beneden (1870), Parona (1887, 1894), Vaullegeard (1899, 1901), Cuenot (in Dollfus, 1929), Pintner (1930), Dollfus (1923, 1929, 1958), Mohr (unpubl.)
ORDER SEPIOLIOIDEA			
Heteroteuthis hawaiiensis	*Nybelinia* sp. B	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Sepiella japonica (= *S. maindroni*)	*Nybelinia surmenicola* (= *N.* cf. *bisulcata*)	Western North Pacific Ocean (Japan)	Yamaguti (1934), Dollfus (1942), Nagasawa and Nakata (1984)
ORDER TEUTHOIDEA			
Heterololigo pealei (= *Loligo*)	*Lacistorhynchus tenue*	Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977, 1981)
H. pealei	*Nybelinia bisulcata*	Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
H. pealei	*Nybelinia yamagutii*	Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
H. pealei	*Otobothrium crenacolle*	Western North Atlantic Ocean (Massachusetts, USA)	Stunkard (1977)
Loligo vulgaris (= *L. loligo*)	*Nybelinia lingualis* (= *Amphistoma loliginis; Fasciola barbata, F. loliginis*)	Eastern North Atlantic Ocean (France, Greenland); Baltic Sea (Sweden); Mediterranean (Italy)	Linné (1761, 1767), Fabricius (1780), Delle Chiaje (1830, 1841); Cuenot (in Dollfus, 1929), Dollfus (1929, 1958)

Table 1-21 (continued)

Host species	Parasites	Locality	Source
ORDER TEUTHOIDEA			
Loligo sp.	*Nybelinia lingualis*	Mediterranean (Italy, France)	Pintner (1930), Dollfus (1958)
Abralia trigonura	*Nybelinia* sp. A.	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Abraliopsis brevis	*Nybelinia* sp. A.	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Enoploteuthis higginsi	*Nybelinia* sp. A.	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Octopoteuthis nielseni	*Nybelinia* sp. A.	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
O. nielseni	*Nybelinia* sp. B.	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Moroteuthis robusta	*Nybelinia* sp.	Eastern North Pacific Ocean (California, USA)	Riser (1949)
Berryteuthis magister	*Nybelinia surmenicola* (= *N.* sp.)	Western North Pacific Ocean (Japan, USSR); Bering Sea (USSR)	Avdeeva and co-authors (1982), Nagasawa and Nakata (1984)
Gonatus 'fabricii'	*Nybelinia* sp.	Eastern North Pacific Ocean (British Columbia, Canada)	Riser (1941, in Dollfus, 1964)
Lepidoteuthis grimaldi	*Nybelinia* sp.	Eastern North Atlantic Ocean	Clarke and Maul (1962)
Architeuthis dux	*Hepatoxylon trichiuri*	Western North Atlantic Ocean (Newfoundland, Canada)	Pippy and Aldrich (1969)
Architeuthis sp.	Unident. tetrarhynchid	Eastern South Atlantic Ocean (South Africa)	Perez-Gandaras and Guerra (1978)
Histioteuthis dofleini	*Nybelinia* sp. A.	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Liocranchia reinhardti	*Nybelinia* sp. A.	Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)

Table 1-21 (continued)

Host species	Parasites	Locality	Source
ORDER OCTOPODA			
Chunioteuthis unguiculatus	*Tentacularia coryphaenae*	Western North Atlantic Ocean (Newfoundland, Canada)	Mercer (in Dollfus, 1967)
Eledone cirrhosa (= *E. aldrovandi*)	*Nybelinia lingualis*	Mediterranean (Monaco)	Nouvel (in Dollfus, 1942), Dollfus (1942, 1958, 1964)
Octopus bimaculatus	*Eutetrarhynchus* sp.	Eastern North Pacific Ocean (Mexico)	Riser (1949), Dollfus (1964)
Octopus joubini	*Eutetrarhynchus* sp.	Gulf of Mexico (Mississippi, USA)	Cake (1976)
Octopus vulgaris	*Nybelinia lingualis* (= *Tetrarhynchus bisulcatus*, *T. megabothrium*)	English Channel (France); Mediterranean (Italy, France)	Redi (1684), Vaullegeard (1899, 1901), Pintner (1930), Dollfus (1936, 1958, 1964)
Unident. *Octopus*	*Nybelinia lingualis*	Mediterranean (Italy, France)	Pintner (1930), Dollfus (1958)
Unident. *Octopus*	*Nybelinia* sp.	Western Indian Ocean (Andaman Is.)	Adam (1938)

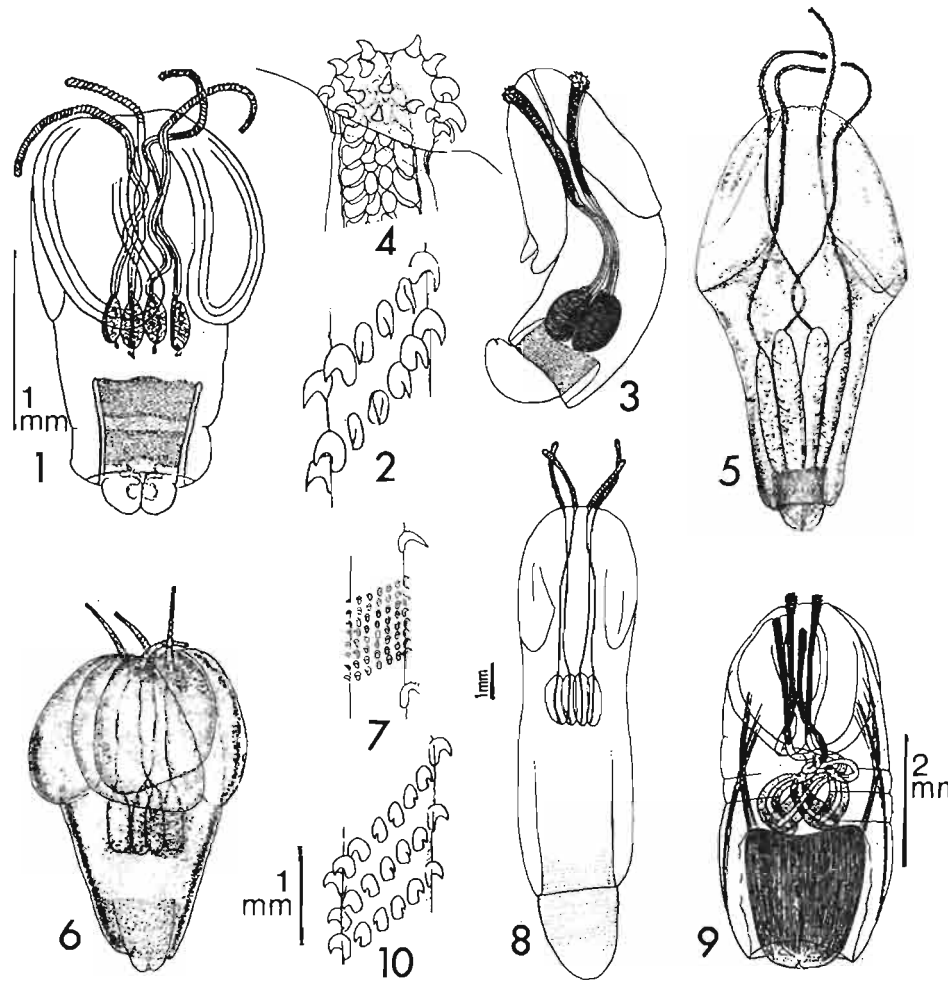


Fig. 1-70: Larval tetrarhynchidean metacestodes from cephalopods. *Nybelinia* plerocerci from a variety of hosts. 1 and 2: *Nybelinia lingualis* from *Sepia officinalis*; (2) arrangement of hooks on section of tentacle. 3 and 4: *N.* sp. from *Octopus* sp.; (4) arrangement of hooks on section of tentacle. 5: *N. yamagutii* from *Heterololigo* (= *Loligo*) *pealei*. 6 and 7: *N. bisulcata* from *H. pealei*; (7) arrangement of hooks on section of tentacle. 8: *N. surmenicola* from *Berryteuthis magister*. 9 and 10: *N. surmenicola* from *Todarodes pacificus*; (10) arrangement of hooks on a section of tentacle. (1, 2, 9 and 10 after Dollfus, 1929; 3 and 4 after Adam, 1938; 5 to 7 after Stunkard, 1977; 8 after Nagasawa and Nakata, 1984.)

species of *Sepiella, Lepidoteuthis, Moroteuthis, Illex, Ommastrephes, Sthenoteuthis* and *Gonatus* (see Tables 1-15 and 1-21; Yamaguti, 1934; Riser, 1949; Clarke and Maul, 1962; Brown and Threlfall, 1968a; Gaevskaya, 1977a; Gaevskaya and Nigmatullin, 1978; Belyaeva, 1979). Off Hawaii, 2 forms of *Nybelinia* commonly are encountered in pelagic squids (Hochberg, unpubl.). The first type typically is embedded in the digestive tracts of species of *Abralia, Abraliopsis, Enoploteuthis, Octopoteuthis, Histioteuthis, Sthenoteuthis* and *Liocranchia*. The second type is encysted either in the digestive gland or in the ventral mantle musculature of the oceanic sepiolid *Heteroteuthis hawaiiensis*.

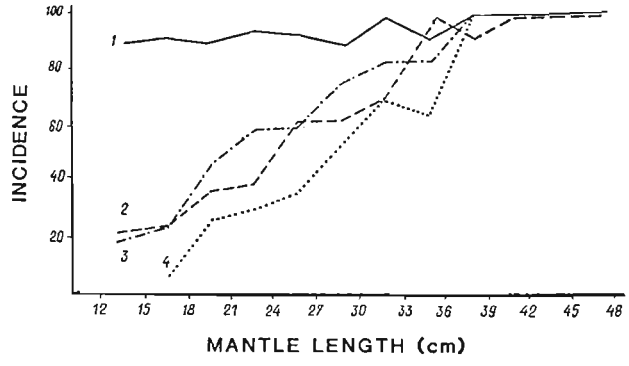


Fig. 1-71: *Sthenoteuthis pteropus*. Mantle-length-related changes in incidence of infection in the eastern Central Atlantic Ocean. 1: *Nybelinia lingualis*. 2: *Porrocaecum (= Phocanema)*. 3: *Tentacularia coryphaenae*. 4: *Anisakis simplex*. (After Naidenova and Zuev, 1978b.)

Smith and co-authors (1981) utilized parasites in combination with morphological and electrophoretic studies to demonstrate the presence of 2 distinct species of the commercially important arrow squid *Nototodarus* off New Zealand. The prevalence and intensity of infestations with *Nybelinia* sp. are directly correlated with the distribution of the 2 squid species. Eastern and southern populations have no cestode parasites, whereas the western population has high infestation rates even in small squids (Table 1-22). The use of parasites, especially cestodes, as biological indicators of species diversity is important in fishery situations where separate quotas must be established to effectively manage cephaloped resources.

The presence of helminths not only is dependent on the species of host but the geographical region where the host lives. Ommastrephids in the eastern South Pacific Ocean lack cestodes of the genus *Nybelinia* as do populations of the squid *Nototodarus* in specific locations off New Zealand (Smith and co-authors, 1981; Gaevskaya and co-authors, 1983), yet throughout the Atlantic and Indian Oceans this larval cestode is commonly encountered. The parasites of *Todaropsis eblanae* were studied by Gaevskaya and Nigmatullin (1975) off the coasts of Africa. Of 647 squid examined off northwest Africa, none were infected, whereas off southwest Africa 13.5 % of 96 squids were infected with cestode and nematode larvae.

Tentacularia coryphaenae (Fig. 1-72, 1 and 2) has been recovered from a number of species of *Illex, Ommastrephes, Sthenoteuthis* and *Todarodes*, and from the finned octopod *Chunioteuthis* (Tables 1-15 and 1-21; Yamaguti, 1959; Dollfus, 1967; Threlfall and co-authors, 1971; Gaevskaya, 1976, 1977a; Gaevskaya and Nigmatullin, 1976b, 1978; Naidenova and Zuev, 1978b; Belyaeva, 1979; Naidenova and co-authors, 1981, 1985; Gaevskaya and co-authors, 1986a). Naidenova and co-authors found larval *T. coryphaenae* in 65 % of the *Sthenoteuthis oualaniensis* examined in the Indian Ocean. The mobile plerocercoids typically are embedded in the internal organs and mantle cavity or in the musculature of the mantle wall, particularly at the posterior end. In *S. oualaniensis* the worms measure 3 to 8 mm long, whereas in *O. bartrami* in the Atlantic Ocean the larvae range in length from 8 to 14 mm. In *S. pteropus* plerocercoids first appear in squids measuring 8 to 10 cm ML. At mantle lengths of 14 to 18 cm the indices of infection increase sharply and adult squid, over 40 cm ML, may be 100 % infected (Figs 1-60 and 1-71;

Table 1-22
Prevalence and intensity of infestation of *Nototodarus* spp. with *Anisakis simplex* larvae and *Nybelinia* sp. post larvae. Prevalence expressed as percentage infestation (Inf.) and intensity (Int.) as mean number of parasites per specimen (n, no. of samples) (After Smith and co-authors, 1981)

Host mantle length (cm)	Eastern & Southern group						Western group					
	Chatham Rise Mar 1979			Auckland Is. & Stewart I. Feb 1980			Karamea Bight & Tasman Bay Feb 1980			Tasman Bay & Egmont Apr 1980		
	n	Inf. (%)	Int.	n	Inf. (%)	Int.	n	Inf. (%)	Int.	n	Inf. (%)	Int.
Anisakis simplex												
4–8	0	–	–	0	–	–	0	–	–	7	0.0	0.00
8–12	0	–	–	21	0.0	0.00	0	–	–	25	8.0	0.08
12–16	10	20.0	0.20	11	0.0	0.00	0	–	–	14	28.6	0.43
16–20	65	50.8	1.05	43	4.7	0.16	3	0.0	0.00	22	31.8	1.32
20–24	21	85.7	2.71	32	9.4	0.13	11	45.5	1.36	18	55.6	2.44
24–28	1	100.0	3.0	18	50.0	1.11	83	77.1	2.31	17	76.5	3.76
28–32	0	–	–	44	84.1	3.07	58	81.0	6.24	17	94.1	26.6
32–36	0	–	–	15	93.3	2.47	5	100.0	5.20	8	100.0	24.3
Nybelinia sp.												
0–4	0	–	–	0	–	–	0	–	–	2	0.0	0.00
4–8	0	–	–	0	–	–	0	–	–	13	15.4	0.77
8–12	0	–	–	21	0.0	0.00	0	–	–	36	55.6	2.08
12–16	10	0.0	0.00	11	0.0	0.00	0	–	–	29	82.8	5.90
16–20	65	0.0	0.00	43	0.0	0.00	3	100.0	9.33	19	94.7	9.32
20–24	21	0.0	0.00	32	0.0	0.00	11	100.0	12.64	15	93.3	18.57
24–28	1	0.0	0.00	18	0.0	0.00	88	100.0	19.17	14	100.0	14.07
28–32	0	–	–	44	0.0	0.00	61	100.0	27.64	11	100.0	30.64
32–36	0	–	–	15	0.0	0.00	7	100.0	48.43	3	100.0	13.31

Gaevskaya and Nigmatullin, 1981). As a rule only 1 to 2 plerocercoids occur in a given host individual, although in *S. pteropus* the intensity increases considerably with age. The first intermediate hosts of *T. coryphaenae* are planktonic crustaceans and the second are a wide diversity of teleost fishes and cephalopods. The final hosts are sharks. *T. coryphaenae* is widely distributed in the Atlantic Ocean.

Several other genera of trypanorhynchs are known from cephalopods. Stunkard (1977) provisionally identified *Lacistorhynchus tenue* (Fig. 1-72, 6) and *Otobothrium crenacolle* (Fig. 1-72, 9) from *Heterololigo pealei*. Van Beneden (1870) found a post-larva of *Christianella minuta* in *Sepia officinalis*, though Dollfus (1958) doubts the validity of this earlier identification. *Dibothriorhynchus todari*, originally described by Delle Chiaje (1829, 1841) from *Todarodes sagittatus*, was transferred to the genus *Hepatoxylon* by Yamaguti (1959). A second species of *Hepatoxylon*, *H. trichiuri* (Fig. 1-72, 5) has been reported from *Sthenoteuthis pteropus* in the Atlantic Ocean and from an *Architeuthis dux* stranded in Newfoundland (Canada) (Pippy and Aldrich, 1969; Gaevskaya, 1977a).

Octopods generally harbor a distinct assemblage of trypanorhynch genera. Riser (1949, in Dollfus, 1964) identified a specimen of *Eutetrarhynchus* (Fig. 1-72, 7) from *Octopus bimaculatus* in California (USA), and Cake (1976) described a similar parasite from *O. joubini* in the Gulf of Mexico. In France and Italy, *O. vulgaris* harbors both *Tetra-*

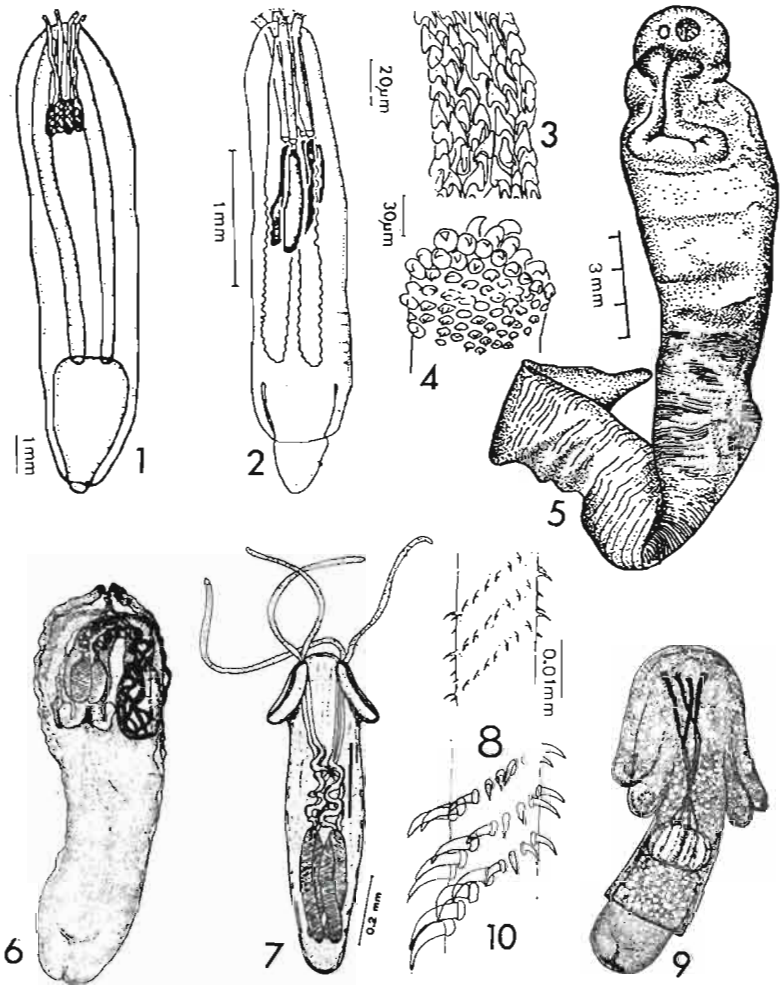


Fig. 1-72: Larval tetrarhynchidean metacestodes from a variety of cephalopod hosts. 1 to 4: *Tentacularia coryphaenae;* (1) from *Ommastrephes bartrami;* (2) from *Illex illecebrosus;* (3) arrangement of hooks at base of tentacle; (4) arrangement of hooks at tip of tentacle. 5: *Hepatoxylon trichiuri* from *Architeuthis dux*. 6: *Lacistorhynchus tenue* from *Heterololigo* (= *Loligo*) *pealei*. 7 and 8: *Eutetrarhynchus* sp. from *Octopus joubini*. (8) arrangement of hooks on tentacle. 9 and 10: *Otobothrium crenacolle* from *Heterololigo* (= *Loligo*) *pealei;* (10) arrangement of hooks on tentacle. (1 after Gaevskaya, 1976; 2 to 4 after Threlfall and co-authors, 1971; 5 after Pippy and Aldrich, 1969; 6, 9 and 10 after Strunkard, 1977; 7 and 8 after Cake, 1976.)

bothriorhynchus octopodidae and *Tetrarhynchus megabothrium* (Redi, 1684; Diesing, 1850; Vaullegeard, 1899; Mingazzini, 1904). According to Dollfus (1958), *T. megabothrium* may represent a species of *Nybelinia*. Adam (1938) figured an unidentified species of *Nybelinia* from an unidentified octopus taken off the Andaman Islands in the Indian Ocean.

Off Hawaii, specimens of what appear to be an adult cestode have been observed in *Octopus cyanea* (Devaney, 1981). Scolices have never been recovered, hence the identifi-

cation of these worms is not known. The cestodes appear to live in the circulatory system of the host and often extend down the arms and throughout the body in the major blood vessels. In massive infections the worms may fill the entire body cavity of the host octopus. The death of several laboratory-reared octopuses was attributed to the presence of infections of this parasite (van Heukelem, 1983, and pers. comm.).

Agents: Acanthocephala

In a recent review of their investigations, Gaevskaya and Nigmatullin (1983) indicated that 2 to 3 species of mature acanthocephalans are regularly encountered in cephalopods, though they are narrowly host specific. In at least 3 other instances accidental infections have been reported. The presence of acanthocephalans in cephalopods is unusual since adults of this entirely parasitic phylum typically infect only vertebrate hosts.

Gaevskaya and Nigmatullin (1976a) first encountered, and Gaevskaya (1977a) later described, the palaeacanthocephalan *Neorhadinorhynchus atlanticus* from the flying squid *Sthenoteuthis pteropus* captured south of the Gulf of Guinea in a limited geographic region of the Atlantic Ocean. Similar forms have also been recovered from the same host in the central and north Atlantic (Naidenova and Zuev, 1978b; Hochberg, unpubl.). *N. atlanticus* (Fig. 1-73, 1) inhabits the lumen of the stomach. The acanthocephalan typically infects *S. pteropus* larger than 150 to 170 mm ML. The onset of infection coincides with the time when the squid switch feeding habits to a diet composed predominantly of fishes. Although the incidence of infection reaches a maximum of 20 % in squids measuring 250 to 300 mm ML, the number of worms continues to accumulate with increased age and growth. Means of 10 to 30 and a maximum of 110 acanthocephalans have been reported from single hosts (Gaevskaya and Nigmatullin, 1981).

Mature males and females of this species are similar in size and measure 6 to 13 mm ML. The proboscis is armed with 12 longitudinal rows of hooks, each with 16 to 17 hooks. All other species described in the genus live as adults in fishes in the Pacific Ocean (Golvan, 1969). This is the first record of the genus in the Atlantic Ocean and the first from a cephalopod host. Since these small cavisomatids attain sexual maturity in cephalopods, Gaevskaya proposed that *Sthenoteuthis pteropus* may function as a final host in this case and not simply a paratenic or transfer host. In the developmental cycle of acanthocephalans, stages normally infective to fishes are found in crustaceans and hence could be ingested by squids, or the infection could be obtained when squid feed on fishes at a later stage in their life cycle.

Gaevskaya (1977a), Naidenova and Zuev (1978b), and Gaevskaya and Nigmatullin (1981) recovered a second species of acanthocephalan from Atlantic specimens of *Sthenoteuthis pteropus* (Fig. 1-73, 2). The unidentified worms were found in the mantle cavity where they penetrated and were attached to the muscular wall of the mantle. The long, thin, white worms occurred in only 0.02 % of the host squid and typically only 1 to 2 worms were present. Mature males measure 68 to 75 mm long, whereas mature females are longer than about 20 mm. The proboscis is short and covered with tiny hooks which merge with the spines covering the anterior third of the body. In mature females the uterus often is packed with numerous oval, embryonated eggs (Fig. 1-73, 2C). The worms are unusual in that the embryonic larva is completely covered with spines (holoechinate), whereas the egg resembles the hemiechinate type in possessing 1 thin external membrane

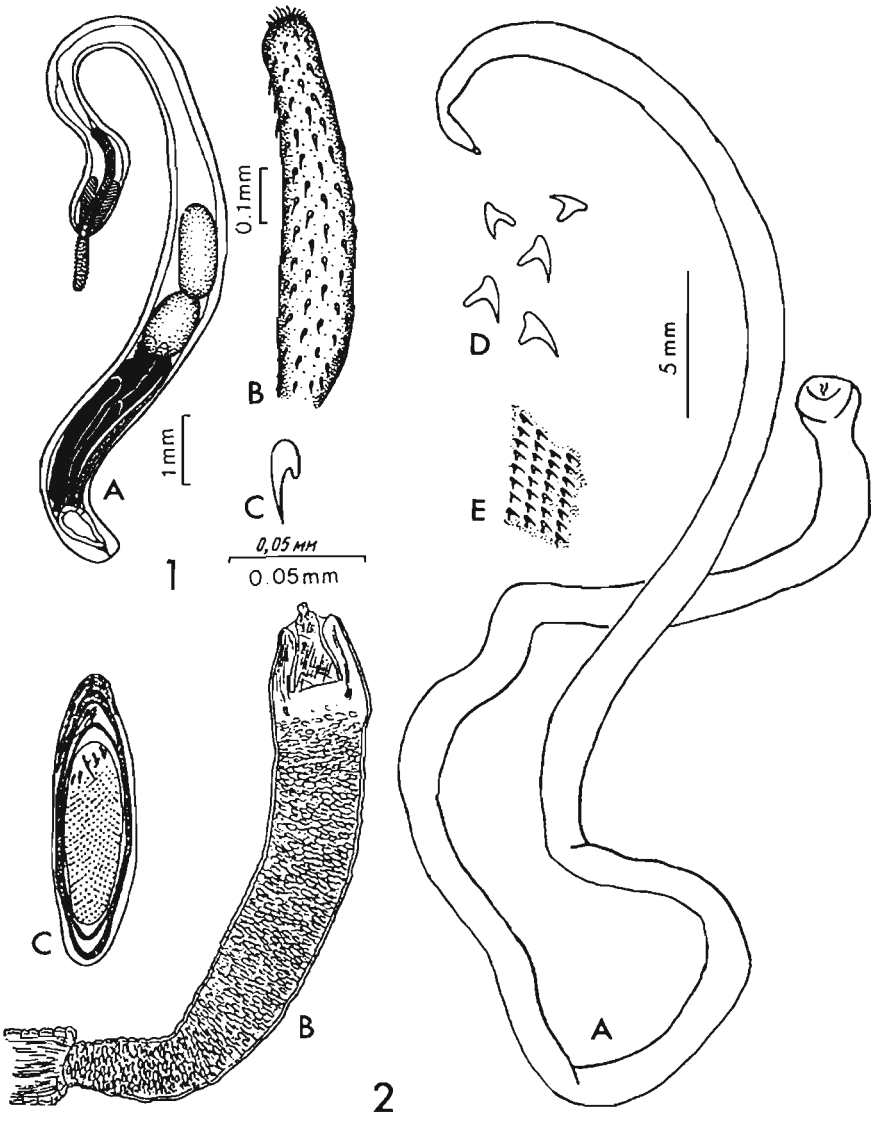


Fig. 1-73: Acanthocephalans from *Sthenoteuthis pteropus*. 1: *Neorhadinorhynchus atlanticus;* (A) adult male; (B) proboscis; (C) proboscis hook. 2: Unidentified acanthocephalan; (A) adult male; (B) anterior end; (C) embryonated egg; (D) proboscis hooks; (E) proboscidial sheath hooks. (1 after Gaevskaya, 1977a; 2 after Naidenova and Zuev, 1978b.)

and 2 thick internal membranes. Unfortunately, the lack of sufficient well-fixed material makes it impossible to further describe or determine the systematic placement of this species. Naidenova and Zuev (1978b) suggested that the species is a remnant of the ancient parasitic fauna of *Sthenoteuthis*.

Long thread-like acanthocephalans similar to those found in *Sthenoteuthis pteropus* were reported by Naidenova and co-authors, (1981, 1985) in 3% of the *S. oualaniensis* examined from the Indian Ocean. The long (10 to 20 cm) worms were observed in the

pericardial coelom attached to the mantle wall or digestive gland. Both mature and immature stages were obtained. Due to poor preservation the material was not described.

Naidenova and co-authors (1981, 1985) reported the presence of a second unidentified species of acanthocephalan in *Sthenoteuthis oualaniensis* in the Indian Ocean. In 2 instances they found fairly high numbers (12 and 18) of small (10 to 13 mm) worms in the stomachs of large squid. An unidentified species of *Rhadinorhynchus* was reported by Kurochkin and Solov'eva (1982) to occur in *Ommastrephes bartrami* from the northwest Pacific Ocean. In the Bering Sea south to the Kurile Islands, Avdeeva and co-authors (1982) examined over 1200 specimens of the commander squid *Berryteuthis magister* for parasites. In 3 instances they encountered a single immature stage of the echinorhynchid *Echinorhynchus cotti*. Figures and further information were not provided on any of these 3 species. In all cases the acanthocephalans probably were accidentally ingested along with food items.

Biological and ecological information relating to the acanthocephalans found in cephalopods is not available. Although numerous samples of large, muscular bodied ommastrephids have been examined for parasites, acanthocephalans have been encountered infrequently. In all likelihood acanthocephalans will not be found in the many small, fragile or gelatinous species of pelagic squids. However, as more loliginids, gonatids, sepiids, and octopodids are examined acanthocephalans will, without a doubt, more frequently be encountered.

Agents: Nematoda

Larval nematodes are commonly encountered in many species of cuttlefishes, squids and octopuses. However, little information is available other than records of presence or absence (Table 1-23). The abundant literature is complicated by a variety of unresolved taxonomic and nomenclatural problems (Smith and Wooten, 1978). In fact, larval nematodes of marine animals, both fishes and invertebrates, are in need of critical review.

In the older cephalopod/parasite literature several species are briefly mentioned or figured. For the most part these worms are inadequately described and, hence, a modern taxonomic designation cannot be applied. However, it is of interest to list these worms because the hosts are known and future investigators may some day be able to re-examine the host cephalopods and fit the pieces of the puzzle together.

Ascaris todari was reported to occur in *Ommastrephes bartrami* and *Todarodes sagittatus* in Naples (Delle Chiaje, 1829; Schuurmans-Stekhoven, 1935). A second species, *A. moschata,* was described by Stossich (1897; see Dollfus, 1958) from *Eledone moschata,* also from Italy. *Filaria loliginis* was described by Delle Chiaje (1829) from *Loligo vulgaris* captured in the vicinity of Naples. The same nematode was found by Grimpe (in Schuurmans-Stekhoven, 1935) in the mantle cavity and ovaries of *Alloteuthis subulata* near Helgoland (southern North Sea). Wülker (1930) presumed this worm to be a larval ascaridoid. Dujardin (1845) mentioned the presence of *F. piscium* in *Sepia officinalis.* This is probably the same nematode that Gros (1847) observed encysted in the stomach lining of an unidentified species of *Sepia* (Dollfus, 1958).

Although nematode genera are relatively easy to distinguish, only a few that occur in cephalopods have been positively determined. The majority of the nematodes identified are larval ascaridoids. Several genera are represented: *Porrocaecum* (Family Ascaridae); *Acanthocheilus* (Family Acanthocheilidae); and *Anisakis, Contracaecum, Hysterothy-*

Table I-23
Nematode parasites from cephalopod hosts. Incidence and intensity of infections in relation to host species and localities of capture
(Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER SEPIOIDEA							
Sepia elegans	Unident. larva	ni				Mediterranean (Monaco)	Nouvel (in Dollfus, 1958)
Sepia officinalis	Unident. larva	ni				Baltic Sea (USSR)	Gros (1847)
S. officinalis	*Filaria piscium*	ni				English Channel (France)	Dujardin (1845), Dollfus (1958)
S. officinalis	*Anisakis* sp.	ni				English Channel	Reimer (1974)
S. officinalis	*Contracaecum* sp.	ni				English Channel	Reimer (1974)
S. orbignyana	Unident. larva	ni				Mediterranean (Monaco)	Nouvel (in Dollfus, 1958)
ORDER SEPIOLIOIDEA							
Sepiola atlantica	Unident. larva	ni				Mediterranean (Monaco)	Nouvel (in Dollfus, 1958)
ORDER TEUTHOIDEA							
Alloteuthis subulata	*Filaria loliginis*	ni				North Sea (Germany)	Wülker (1930), Grimpe (in Schuurmans-Stekhoven 1935), Dollfus (1958)
Heterololigo bleekeri (= *Doryteuthis*)	*Anisakis physeteris* (= *A.* type II)	ni				Western North Pacific Ocean (Japan)	Kato and co-authors (1968), Oshima (1972)
Loligo opalescens	philometroid	26	ni	3.8	1	Eastern North Pacific Ocean (California, USA)	Dailey (1969)
L. opalescens	Unident. larva	26	ni	3.8	1	Eastern North Pacific Ocean (California, USA)	Fields (1965), Dailey (1969)
Loligo vulgaris	*Filaria loliginis*	ni				Mediterranean (Italy)	Delle Chiaje (1829), Dollfus (1958)
L. vulgaris	*Anisakis* sp.	ni				English Channel	Reimer (1974)
L. vulgaris	*Contracaecum* sp.	ni				English Channel	Reimer (1974)
+*Loliolopsis diomedeae*	*Spinitectus* sp.	ni				Gulf of California (Mexico)	Hochberg (unpubl.)
Lolliguncula brevis	*Hysterothylacium reliquens* (= *Thynnascaris* sp.)	ni				Gulf of Mexico (Mississippi, USA)	Norris and Overstreet (1976), Deardorff and Overstreet (1981), Hochberg (unpubl.)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Photololigo singhalensis (= *Doryteuthis*)	*Spinitectus* sp.	100	126–130	2	ni	Indian Ocean	Pinchukov and Makarova (1979)
Lycoteuthis diadema	*Anisakis simplex* (= *A.* sp. 1)	26	47–120	3.8	1	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
+*Abralia trigonura*	Unident. larva	ni				Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
+*Abraliopsis brevis*	*'Raphidascaris'* sp.	ni				Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
+*Abraliopsis felis*	Unident. larva	ni				Eastern North Pacific Ocean (California, USA)	Hochberg (unpubl.)
+*Enoploteuthis higginsi*	Unident. larva	ni				Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
+*Pterygioteuthis giardi*	Unident. larva	ni				Gulf of California (Mexico)	Hochberg (unpubl.)
Moroteuthis robusta	*Contracaecum* type II	2	735, 960	100	3, 4	Western North Pacific Ocean (Kuriles, USSR)	Bagrov (1982)
M. robusta	*Contracaecum* type II	ni				Eastern North Pacific Ocean (California, USA)	Hochberg (unpubl.)
Onychoteuthis banksi	Unident. larva	ni				Mediterranean (Monaco)	Nouvel (in Dollfus, 1958)
Onychoteuthis borealijaponica	*Anisakis simplex*	7	207–311	1	3	Western North Pacific Ocean (off Japan)	Bagrov (1982)
Lepidoteuthis grimaldi	*Anisakis* sp.	ni				Eastern North Atlantic Ocean	Clarke and Maul (1962)
Berryteuthis magister	*Anisakis simplex* (= *A.* sp. I)	1204	100–360	13.6	ni	Western North Pacific Ocean (Kuriles, USSR); Bering Sea (USSR)	Avdeeva and co-authors (1982)
B. magister	*A. simplex* (= *A.* sp. I)	244	120–320	22.5	1–4	Bering Sea (USSR)	Bagrov (1982)
B. magister	*A. simplex* (= *A.* sp. I)	902	162–342	9.4	1–2	Western North Pacific Ocean (Kuriles, USSR)	Bagrov (1982)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Berryteuthis magister	*Hysterothylacium reliquens* (= *Thynnascaris* sp.)	1204	100–360	0.08	1	Western North Pacific Ocean (Kuriles, USSR); Bering Sea (USSR)	Avdeeva and co-authors (1982)
Pholidoteuthis boschmai	*Contracaecum* sp. 1	32	92–250	9.3	1–3	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
P. boschmai	Spirurate larva	32	92–250	0.03	1	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Histioteuthis bonellii (*H. bonelliana*)	Unident. larva	ni				Mediterranean (France)	Dollfus (1958)
Dosidicus gigas	*Anisakis* sp.	223	150–360	5	1–5	Eastern South Pacific Ocean (0–17° S, 85–86° W)	Gaevskaya and co-authors (1982)
D. gigas	*Anisakis* sp.	403	70–430	32	1–5	Eastern South Pacific Ocean (0–22° S, 83–106° W)	Gaevskaya and co-authors (1983)
D. gigas	*Anisakis* sp.	410	20–400	ni	ni	Eastern Pacific Ocean (2° N–22° S, 82–87° W)	Gaevskaya and co-authors (1987)
D. gigas	*Porrocaecum* sp.	223	150–360	61.2	1–14	Eastern South Pacific Ocean (0–17° S, 85–86° W)	Gaevskaya and co-authors (1982)
D. gigas	*Porrocaecum* sp.	403	70–430	33	1–14	Eastern South Pacific Ocean (0–22° S, 83–106° W)	Gaevskaya and co-authors (1983)
D. gigas	*Porrocaecum* sp.	410	20–400	ni	ni	Eastern Pacific Ocean (2° N–22° S, 82–87° W)	Gaevskaya and co-authors (1987)
Eucleoteuthis luminosa	*Contracaecum* sp. 1	29	150–220	86.3	1–14	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
E. luminosa	Spirurate larva	29	150–220	31	1–3	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
Illex argentinus	*Anisakis simplex* (= *A.* sp. I)	377	60–360	1.4	1–2	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Illex argentinus	*Anisakis physeteris* (= *A.* sp. II)	377	60–360	7.4	1–2	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986)
I. argentinus	*Hysterothylacium* sp. (= *Contracaecum* sp.)	377	60–360	4.9	1–2	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986)
I. argentinus	*Porrocaecum* sp.	377	60–360	0.9	1–2	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986)
I. argentinus	*Anisakis* sp.	132	ni	1.5	1	Western South Atlantic Ocean (Argentina)	Threlfall (1970)
I. argentinus	Unident. larva	132	ni	0.7	1	Western South Atlantic Ocean (Argentina)	Threlfall (1970)
Illex coindetii	*Porrocaecum* sp.	34	ni	3	ni	Eastern North Atlantic Ocean (off Ireland)	Gaevskaya and Nigmatullin (1975)
I. coindetii	*Porrocaecum* sp.	1880	ni	0.3	ni	Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975)
I. coindetii	Unident. larva	ni				Eastern North Atlantic Ocean (France)	Dollfus (1958)
Illex illecebrosus	*Anisakis simplex* (= *A.* sp. 1)	ni				Western North Atlantic Ocean (Nova Scotia, Newfoundland, Canada)	Gaevskaya and Nigmatullin (1975), Mercer (1965)
I. illecebrosus	*Contracaecum* sp.	802	ni	1.6	1–2	Western North Atlantic Ocean-(Newfoundland, Nova Scotia, Canada)	Brown and Threlfall (1968a), Gaevskaya and Nigmatullin (1975)
I. illecebrosus	*Porrocaecum* sp.	ni				Western North Atlantic Ocean (Newfoundland, Canada)	Mercer (1965)
I. illecebrosus	Unident. larvae	1503	100–300	1.3	1	Western North Atlantic Ocean (Newfoundland, Canada)	Squires (1957)
Nototodarus sloani	*Anisakis simplex*		[see Table 1–22]			Western South Pacific Ocean and Tasman Sea (New Zealand)	Brunsdon (1956), Smith and co-authors (1981)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Nototodarus sloani	*Contracaecum* sp. IA	ni				Western South Pacific Ocean and Tasman Sea (New Zealand)	Brunsdon (1956), Hurst (pers. comm.)
N. sloani	*Contracaecum* sp. IIA	ni				Western South Pacific Ocean and Tasman Sea (New Zealand)	Brunsdon (1956)
Ommastrephes bartrami (= *Stenoteuthis*)	*Ascaris todari*	ni				Mediterranean (Italy)	Delle Chiaje (1829), Schuurmans-Stekhoven (1935), Dollfus (1958)
O. bartrami	*Anisakis simplex*	133	220–418	3.3	1–11	Western North Pacific Ocean (off Japan)	Bagrov (1982)
O. bartrami	*A. simplex* (= *A.* sp. I)	184	150–450	7.6	1–2	Western North Pacific Ocean (off Japan)	Kurochkin and Solov'eva (1982)
O. bartrami	*A. simplex* (= *A.* sp. I)	35	160–405	5.7	1	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
O. bartrami	*A. simplex* (= *A.* sp. I)	7	240–360	14.2	12	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)
O. bartrami	*Anisakis physeteris*	133	220–418	0.7	ni	Western North Pacific Ocean (off Japan)	Bagrov (1982)
O. bartrami	*A. physeteris* (= *A.* sp. II)	184	150–450	ni	ni	Western North Pacific Ocean (off Japan)	Kurochkin and Solov'eva (1982)
O. bartrami	*A. physeteris* (= *A.* sp.)	54	ni	50	1–6	Central Atlantic Ocean (6° N–40° S, 54° W–17° E)	Gaevskaya (1974)
O. bartrami	*A. physeteris* (= *A.* sp. I (II))	60	100–760	19.2	1–2	Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1976b)
O. bartrami	*A. physeteris* (= *A.* sp. II)	35	160–405	11	1	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
O. bartrami	*Contracaecum* sp.	7	240–360	42.8	12–102	South Pacific Ocean (37–41° S, 78–103° W)	Shukhgalter and Polozhayev (1987)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Ommastrephes bartrami	*Contracaecum* type I	133	220–418	3.7	1–11	Western North Pacific Ocean (off Japan)	Bagrov (1982)
	Contracaecum type I	ni				Indian Ocean	Belyaeva (1979)
O. bartrami	*Contracaecum* type II	133	220–418	9.8	1–11	Western North Pacific Ocean (off Japan)	Bagrov (1982)
O. bartrami	*Contracaecum* type II	ni				Indian Ocean	Belyaeva (1979)
O. bartrami	*Hysterothylacium* sp. (= *Thynnascaris* sp.)	184	150–450	25.5	1–13	Western North Pacific Ocean (off Japan)	Kurochkin and Solov'eva (1982)
O. bartrami	*Porrocaecum* type I	60	100–760	96	1–1500 (2–20)	Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1976b)
O. bartrami	*Porrocaecum* type I	42	120–450	72	1–35	South Atlantic Ocean (off Africa, Argentina)	Gaevskaya (1976), Gaevskaya and Nigmatullin (1975, 1976b)
O. bartrami	*Porrocaecum* type II	60	100–760	50	1–7	Eastern North Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975, 1976b)
O. bartrami	*Porrocaecum* type II	42	120–450	28.5	2–27	South Atlantic Ocean (off Africa, Argentina)	Gaevskaya (1976), Gaevskaya and Nigmatullin (1975, 1976b)
O. bartrami	*Porrocaecum* sp.	35	160–405	42	1–2000	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a, b)
O. bartrami	*Porrocaecum* sp.	ni	ni	62	1–21	Eastern South Pacific Ocean (0–22° S, 83–106° W)	Gaevskaya and co-authors (1983)
O. bartrami	*Porrocaecum* sp.	ni				Indian Ocean	Belyaeva (1979)
O. bartrami	*Spinitectus* sp.	35	160–405	2.8	4	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
Ornithoteuthis antillarum	*Porrocaecum* sp.	62	22–132	ni	ni	Eastern Central Atlantic Ocean (off Africa)	Nesis and Nigmatullin (1979)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Sthenoteuthis oualaniensis	*Anisakis* sp.	80	70–320	21	1–6	Eastern South Pacific Ocean (0–22° S, 83–106° W)	Gaevskaya and co-authors (1983)
S. oualaniensis	*Anisakis* sp.	ni				Indian Ocean	Belyaeva (1979)
S. oualaniensis	*Anisakis* sp.	303	ni	0.9	1–2	Indian Ocean	Naidenova and co-authors (1981, 1985)
S. oualaniensis	*Anisakis* sp.	19	ni	3.8	1–2	Red Sea	Naidenova and co-authors (1981, 1985)
S. oualaniensis	*Contracaecum* sp. 1	ni				Indian Ocean	Belyaeva (1979)
S. oualaniensis	*Contracaecum* sp. 2	ni				Indian Ocean	Belyaeva (1979)
S. oualaniensis	*Porrocaecum* type I & II	303	ni	24.1	ni	Indian Ocean	Naidenova and co-authors (1981, 1985)
S. oualaniensis	*Porrocaecum* type I & II	19	ni	57	1–108	Red Sea	Naidenova and co-authors (1981, 1985)
S. oualaniensis	*Porrocaecum* sp.	80	70–320	26	1–30	Eastern South Pacific Ocean (0–22° S, 83–106° W)	Gaevskaya and co-authors (1983)
S. oualaniensis	*Porrocaecum* sp.	ni				Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Sthenoteuthis pteropus (= *Ommastrephes*)	*Anisakis physeteris* (= *A.* sp. 2_2)	1039	ni	25.3	ni	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975)
S. pteropus	*A. physeteris* (= *A.* sp. (II))	2200	ni	ni	1–6	Tropical Atlantic Ocean	Gaevskaya (1977a)
S. pteropus	*A. physeteris* (= *A.* sp. I (II))	2262	20–500	23	1–13 (1–3)	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1981)
S. pteropus	*A. physeteris* (= *A.* sp.)	833	ni	39.1	1–18 (2)	Eastern Central Atlantic Ocean (20–32° W)	Naidenova (1978); Naidenova and Zuev (1978b)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Sthenoteuthis pteropus (= *Ommastrephes*)	*Anisakis physeteris* (= *A.* sp.)	26	ni	38	1–6	Central Atlantic Ocean (6° N–40° S, 54° W–17° E)	Gaevskaya (1974)
S. pteropus (= *Ommastrephes*)	*Porrocaecum* type I	1039	ni	73.6	ni	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975)
S. pteropus	*Porrocaecum* type I	2262	20–500	64	1–120 (5–20)	Tropical Atlantic Ocean	Gaevskaya (1977a), Gaevskaya and Nigmatullin (1981)
S. pteropus	*Porrocaecum* type I (= *Phocanema* sp. type 1)	833	ni	54.2	1–28	Eastern Central Atlantic Ocean (20–32° W)	Naidenova (1978), Naidenova and Zuev (1978b)
S. pteropus (= *Ommastrephes*)	*Porrocaecum* type II	1039	ni	26.4	ni	Tropical Atlantic Ocean	Gaevskaya and Nigmatullin (1975)
S. pteropus	*Porrocaecum* type II	2262	20–500	14	1–94 (1–8)	Tropical Atlantic Ocean	Gaevskaya (1977a), Gaevskaya and Nigmatullin (1981)
S. pteropus	*Porrocaecum* type II (= *Phocanema* sp. type II)	[see above for combined totals]				Eastern Central Atlantic Ocean (20–32° W)	Naidenova (1978), Naidenova and Zuev (1978b)
Todarodes angolensis	*Anisakis simplex* (= *A.* type I)	18	200–295	11	1	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
T. angolensis	*Anisakis physeteris* (= *A.* type II)	18	200–295	11	1	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
T. angolensis	*Anisakis* sp.	ni	250+	1.3	ni	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975)
T. angolensis	*Porrocaecum* sp.	[see above for combined totals]				Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975)
T. angolensis	*Porrocaecum* sp.	18	200–295	22.2	1–4	Western South Atlantic Ocean (37–47° S)	Gaevskaya and co-authors (1986a)
Todarodes pacificus	*Anisakis simplex* (= *A.* type I)	500	ni	0.6	1	Japan Sea (USSR)	Kurochkin (1972)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Todarodes pacificus	*Anisakis simplex* (= *A.* type I)	101	ni [see also Fig. 1–76]	50	3.3*	Western North Pacific Ocean (Japan)	Oshima (1972)
T. pacificus	*A. simplex* (= *A.* type I)	93	75–130	2.1	1–4	Western North Pacific Ocean (USSR)	Bagrov (1982)
T. pacificus (= *Ommastrephes sloanei*)	*A. simplex* (= *A.* type I)	167	ni	15.6	0.3*	Western North Pacific Ocean (Japan)	Shiraki (1974)
T. pacificus (= *O. sloanei*)	*A. simplex* (= *A.* type I)	103	ni	25.2	0.59*	Western North Pacific Ocean (Japan)	Shiraki (1974)
T. pacificus (= *O. sloani pacificus*)	*A. simplex* (= *A.* type I)	755	ni	42	1.7*	Japan Sea (Japan)	Ono (1976)
T. pacificus	*A. simplex* (= *A.* type I)	ni	ni	74.1	1–19	Western North Pacific Ocean (Japan)	Okumura (1967)
T. pacificus (= *Ommastrephes sloani pacificus*)	*A. simplex* (= *A.* type I)	ni				Western North Pacific Ocean (Japan)	Kobayashi and co-authors (1966), Nishimura and co-authors (1966), Yamaguchi (1966, 1968), Honda and co-authors (1967), Yokogawa and Yoshimura (1967), Ichihara and co-authors (1968), Kato and co-authors (1968), Koga and co-authors (1968), Kuwabata and co-authors (1968), Orihara and co-authors (1968), Yamaguchi and co-authors (1968), Hara (1969), Kagei (1969), Koyama and co-authors (1969), Kosugi and co-authors (1970), Saito and co-authors (1970), Hirabayashi and co-authors (1971), Hiraoki and Hirayama (1974), Nagasawa and Nakata (1984)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
T. pacificus (= *O. sloanei*)	*Anisakis physeteris* (= *A.* type II)	103	ni	1	0.01*	Western North Pacific Ocean (Japan)	Shiraki (1974)
T. pacificus	*A. physeteris* (= *A.* type II)	93	75–130	1.1	1	Western North Pacific Ocean (off Japan)	Bagrov (1982)
T. pacificus	*A. physeteris* (= *A.* type II)	ni				Western North Pacific Ocean (Japan)	Kato and co-authors (1968), Koga and co-authors (1968), Hiraoki and Hirayama (1974), Nagasawa and Nakata (1984)
T. pacificus	*Hysterothylacium reliquens* (= *Contracaecum* spp.)	2	170, 245	50	1	Western North Pacific Ocean (Kuriles, USSR)	Bagrov (1982)
T. pacificus	*H. reliquens* (= *Contracaecum* spp.)	93	75–130	12.9	1–4	Western North Pacific Ocean (off Japan)	Bagrov (1982)
T. pacificus (= *O. sloanei*)	*H. reliquens* (= *Contracaecum* type B)	167	ni	14.9	0.3*	Western North Pacific Ocean (Japan)	Shiraki (1974)
T. pacificus (= *O. sloanei*)	*H. reliquens* (= *Contracaecum* type B)	103	ni	10.7	0.13*	Western North Pacific Ocean (Japan)	Shiraki (1974)
T. pacificus	*H. reliquens* (= *Contracaecum* sp., A-type)	ni				Western North Pacific Ocean (Japan)	Kikuchi and co-authors (1969, 1972), Koyama and co-authors (1969), Kosugi and co-authors (1970), Oshima (1972), Oshima (1972), Nagasawa and Nakata (1984)
T. pacificus	*Pseudoterranova decipiens* (= *Terranova* sp.)	ni				Western North Pacific Ocean (Japan)	Orihara and co-authors (1968)

Table 1-23 (continued)

Cephalopod hosts	Parasites	No. hosts examined	ML (mm) range	Incidence (%)	Intensity	Locality	Source
ORDER TEUTHOIDEA							
Todarodes pacificus	*Contracaecum* sp. (A-type)	ni				Western North Pacific Ocean (Japan)	Kagei and co-authors (1970b), Kikuchi and co-authors (1972), Oshima (1972)
T. pacificus	*Acanthocheilus* sp.	500	ni	0.2	1	Japan Sea (USSR)	Kurochkin (1972)
T. pacificus	*Raphidascaris* sp.	500	ni	0.4	1–3	Japan Sea (USSR)	Kurochkin (1972)
T. sagittatus (= *Loligo todarus*, *Ommatostrephes*)	*Ascaris todari*	ni				Mediterranean (Italy)	Delle Chiaje (1829), Schuurmans-Stekhoven (1935), Dollfus (1958)
T. sagittatus	*Anisakis simplex*	ni				North Sea (Norway)	Berland (1961)
T. sagittatus	*A. simplex*	ni				Mediterranean (France)	Hochberg (unpubl.)
T. sagittatus	*Contracaecum* sp.	ni				Eastern North Atlantic Ocean (Morocco)	Reimer (1974)
Todaropsis eblanae	*Anisakis simplex* (= *A.* sp. 1)	96	ni	7.2	ni	Eastern South Atlantic Ocean (off Africa)	Gaevskaya and Nigmatullin (1975)
+*Chiroteuthis picteti*	Unident. larva	ni				Central North Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
ORDER VAMPYROMORPHA							
+*Vampyroteuthis infernalis*	Unident. larva	ni				Eastern North Pacific Ocean (California, USA)	Young (pers. comm.)
ORDER OCTOPODA							
+*Japatella diaphana*	Unident. larva	ni				Eastern North Pacific Ocean (California, USA)	Hochberg (unpubl.)
Eledone cirrhosa (= *E. aldrovandi*)	Unident. larva	ni				Mediterranean (Monaco)	Nouvel (in Dollfus, 1958)
Eledone moschata	*Ascaris moschata* (adult)	ni				Mediterranean (Italy)	Dollfus (1958)
+*Octopus rubescens*	Unident. larva	ni				Eastern North Pacific Ocean (California, USA)	Hochberg (unpubl.)

+ New host records, * Index of abundance, () Average intensity

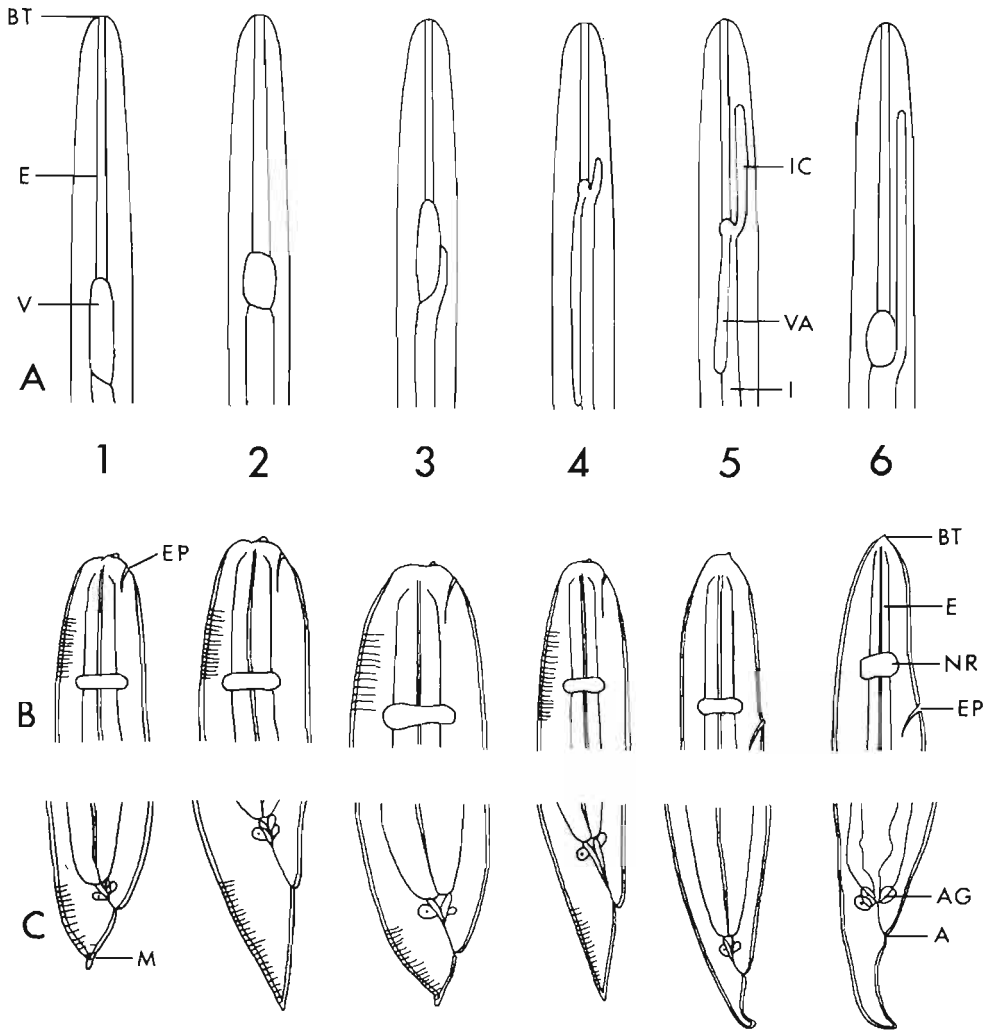


Fig. 1-74: Schematic diagrams of larval anisakid nematodes isolated from cephalopods. 1: *Anisakis simplex* (= *A.* type I). 2: *A. physeteris* (= *A.* type II). 3: *Pseudoterranova decipiens* (= *Terranova* type A). 4: *Contracaecum* type A. 5: *Hysterothylacium reliquens* (= *Contracaecum* type B and *Thynnascaris*). 6: *Porrocaecum* type I (= *Phocanema* type I). (Row A) anterior ends; (Row B) anterior extremeties; (Row C) posterior extremeties. (A) Anus; (AG) anal gland; (BT) boring tooth; (E) esophagus; (EXP) excretory pore; (I) intestine; (IC) intestinal caecum; (M) mucron; (NR) nerve ring; (V) ventriculus; (VA) ventriculus appendix. (1, 2, 4 to 6 [Row A] after Koyama and co-authors, 1969; 1, 2, 4 to 6 [Rows B and C], after Shiraki, 1974; 3 [Rows B and C] after Gaevskaya, 1976.)

lacium, *Pseudoterranova*, and *Raphidascaris* (Family Anisakidae) (Fig. 1-74). For reviews of the morphology and taxonomy of these nematodes see Koyama and co-authors (1969), Oshima and co-authors (1969), Davey (1971), Oishi and co-authors (1971), Shiraki (1974), and Hurst (1984a). Cannon (1977) remarked that species of *Anisakis* and *Pseudoterranova* (= *Terranova*) typically are found in hosts which feed on plankton and nekton, whereas those of *Contracaecum* and *Hysterothylacium* (= *Thynnascaris*) occur principally in

bottom feeders. In general this fits with feeding habit patterns of cephalopods examined to date but would be worthy of further careful study.

Specific identification of larval ascaridoids is difficult because few, if any, of the characters used for adult identification are present. Several classification schemes have been developed which use a type (I, II; A, B, etc.) designation rather than a species designation. In the absence of detailed descriptions, measurements and adequate figures it is often impossible to compare larval types with each other, much less to connect them to known adults. This problem has considerably complicated an in depth review of the nematode parasites in cephalopods.

Medium sized inshore or neritic ommastrephid squid genera such as *Todarodes, Illex* and *Ornithoteuthis* rarely harbor *Porrocaecum* (Type I) larvae (Fig. 1-74, 6), whereas a high percentage (often 50% or more) of the larger offshore or oceanic genera *Dosidicus, Ommastrephes* and *Sthenoteuthis,* are infected (Gaevskaya, 1974, 1976, 1977a; Gaevskaya and Nigmatullin, 1975, 1976a, b, 1978; Naidenova and Zuev, 1978b; Nesis and Nigmatullin, 1979; Gaevskaya and co-authors 1982, 1983, 1986b). In the Indian Ocean and Red Sea *Porrocaecum* larvae occur in both *Ommastrephes* and *Sthenoteuthis* (Belyaeva, 1979; Naidenova and co-authors, 1981). Of the ommastrephids examined in the Indian Ocean by Naidenova and co-authors 75 to 95% had small (3 to 5 mm), transparent 'Type I' larvae encysted in connective tissue capsules on the external walls of the stomach, while 30 to 50% had large (20 to 30 mm) 'Type II' worms encysted in the internal wall of the mantle. In *S. pteropus* from the Atlantic Ocean the incidence of *Porrocaecum* larvae peaks in squid of 20 to 40 mm ML and then declines, whereas the number of worms present in each squid continues to increase with increasing age and size of host (Figs 1-60 and 1-71). These worms are considered by many workers to be successive stages in the development of the same species, although the lack of intermediate sizes between small and large larvae is confusing.

The life cycle of *Porrocaecum* species, as is typical of oceanic ascaridoids, involves 4 host stages. It is characterized by a general lack of host specificity at each parasitic stage and is intimately associated with host food webs. The first intermediate hosts are planktonic crustaceans, especially euphausiids. Second intermediate hosts are a diversity of small teleost fishes, and third ones are large ommastrephid squids. The final hosts are teuthophagous bill fishes and marine mammals.

Hurst (1984a) reviewed the taxonomic confusion surrounding larval nematodes currently recognized as *Pseudoterranova decipiens* (Figs 1-74, 3 and 1-75, F-J). Off Japan this nematode occasionally has been found in *Todarodes pacificus* where it was previously identified as *Terranova* (Orihara and co-authors, 1968; Oshima, 1972). A number of the *Phocanema* and *Porrocaecum* records in Table 1-23 need to be critically reexamined to see if they should be placed in the genus *Pseudoterranova.*

Contracaecum (Type B) (Fig. 1-74, 5) larvae are commonly noted in the muscles of *Todarodes pacificus* off Japan (Kikuchi and co-authors, 1969, 1972; Shiraki, 1969, 1974; Kagei and co-authors, 1970a, b; Kosugi and co-authors, 1970; Oshima, 1972). In their review, Norris and Overstreet (1976) considered this worm to be a member of the genus *Thynnascaris.* Deardorff and Overstreet (1981) transferred it to the genus *Hysterothylacium.* Both publications list *H. reliquens* as occurring in *Lolliguncula brevis* off Mississippi in the Gulf of Mexico. Brunsdon (1956) found *Contracaecum* larvae in stomach and mesenteries of *Nototodarus sloani* off New Zealand but a positive identification has not been made (Hurst, pers. comm.).

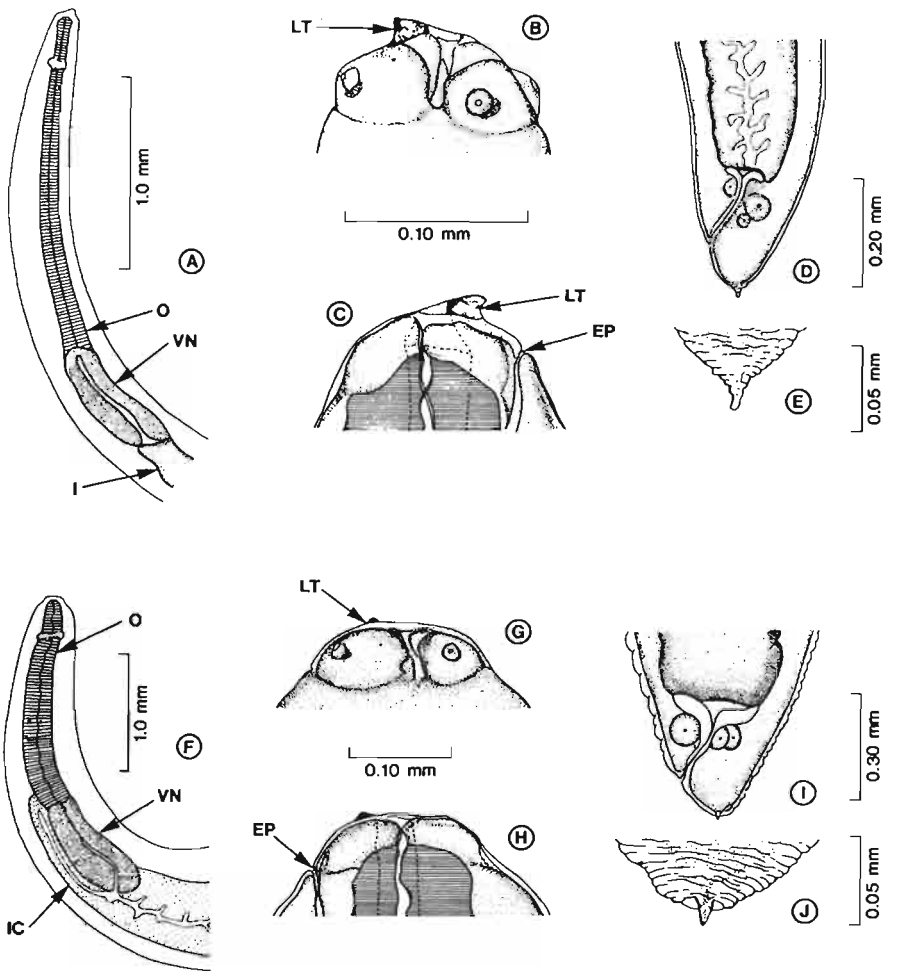


Fig. 1-75: Stage III larvae of *Anisakis simplex* (A–E) and *Pseudoterranova decipiens* (F–J). (A, F) Anterior, lateral view; (B, G) head, lateral view; (C, H) head, sagittal view; (D, I) posterior, lateral view; (E, J) enlargement of posterior extremity showing mucron. (EP) Excretory pore; (I) intestine; (IC) intestinal caecum; (LT) larval tooth; (O) oesophagus; (VN) ventriculus. (After Hurst, 1984a.)

Bagrov (1982) examined squids in the North Pacific Ocean for anisakid larvae (Table 1-23). Two types of *Contracaecum* were found. Small 'Type I' worms, 5 to 8 mm in length, were localized in the stomach wall of *Ommastrephes bartrami* while larger (15 to 25 mm) 'Type II' larvae commonly were recovered from the mantle musculature of *O. bartrami, Moroteuthis robusta* and *Todarodes pacificus*. Large white-colored lesions were evident in the mantle at the site of penetration in all 3 hosts. These lesions measured 2 to 10 mm deep by 20 to 25 mm long and were visible to the naked eye. Off Japan, *Contracaecum* (Type II) larvae also were found in the mantle of *T. pacificus* by Oshima (1972). The distribution of *Contracaecum* and *Anisakis* larvae in the bodies of squids studied by Bagrov (1982) is presented in Table 1-23.

In Norway, Berland (1961) was the first to note the presence of *Anisakis* larvae

encysted in the ventricle of *Todarodes sagittatus* (see also Pippy and Banning, 1975). Throughout Japan, third-stage larval anisakids of 2 distinct species have commonly been recovered by a number of investigators from *T. pacificus* and more rarely from *Heterololigo (= Doryteuthis) bleekeri* (Kobayashi and co-authors, 1966; Okumura, 1967; Kato and co-authors, 1968; Koga and co-authors, 1968; Orihara and co-authors, 1968; Kagei 1969, 1970; Kosugi and co-authors, 1969; Koyama and co-authors, 1969; Oishi and co-authors, 1969; Kagei and co-authors, 1970a; Kurochkin, 1972; Oshima, 1972). A number of other species of cephalopods have been examined in the Orient and all were found to be negative. Type I larvae are currently recognized as *A. simplex* (Figs 1-74, 1 and 1-75, A to F) and Type II larvae as *A. physeteris* (Fig. 1-74, 2). The majority of these worms occur in circular cysts in the secretory portions of the visceral organs, and in the lining of the mantle cavity, although they also may be found in the mantle musculature. In *Sthenoteuthis pteropus* both the incidence and intensity of *A. simplex* increase with increasing size and age of the host squid (Figs 1-60 and 1-71).

Marked seasonal differences in prevalence and intensity of infection of *Anisakis simplex* in *Todarodes pacificus* are evident in studies off Japan (for review see Oshima, 1972). During spring and summer the squid migrate northward along the coast of Japan to the main growing areas around Hokkaido and off Tohoku. During the northward migration incidence and intensity of the *Anisakis* infection are low (Fig. 1-76). The indices of infection increase dramatically while on the feeding grounds and continue to increase during the southward migration in fall and winter to the spawning grounds in the southwestern Sea of Japan. The diet at the time the nematode infections increase is principally euphausiids, which are known to harbor larval anisakids (Oshima and co-authors, 1968, 1969; Smith, 1971; Shimazu and Oshima, 1972; Kagei, 1974, 1979a; Hurst, 1984b).

Anisakis larvae were observed by Clarke and Maul (1962) in a specimen of

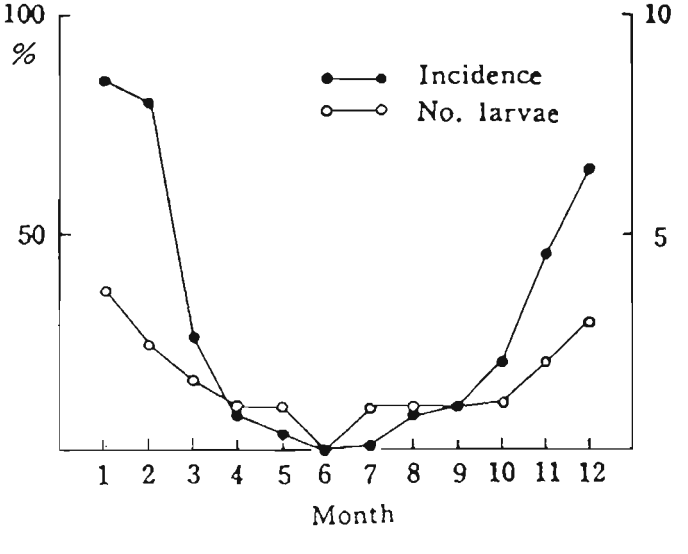


Fig. 1-76: Seasonal variation in incidence and intensity of *Anisakis simplex* (Type I larvae) in *Todarodes pacificus* off Japan. (After Kagei, 1969.)

Lepidoteuthis grimaldi captured in the Atlantic and by Threlfall (1970) in *Illex argentinus* off Mar del Plata, Argentina. Belyaeva (1979) recovered *Anisakis simplex* (Type I) larvae in *Sthenoteuthis oualaniensis* and *Ommastrephes bartrami* in the Indian Ocean. Gaevskaya and Nigmatullin (1975) reported *A. simplex* (Type I) larvae in *Todaropsis eblane* and *Todarodes angolensis* off southwest Africa and in *S. pteropus* in several areas of the tropical Atlantic. *A. physeteris* (Type II) larvae occurred in 2 to 20% of the *O. bartrami* examined in the Atlantic by Gaevskaya and co-workers. Normally, only 1 large (20 mm) pink worm occurred per host, in the lumen of the ovary or testis (Gaevskaya, 1976).

Oshima (1972), Smith and Wootten (1978) and Hurst (1984b) have reviewed the life

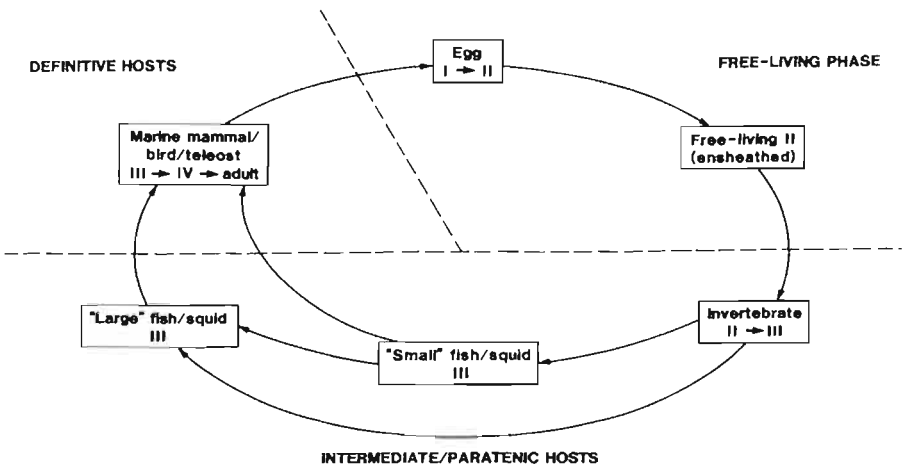


Fig. 1-77: Generalized life cycle of marine anisakids (larval stages in Roman numerals). (After Hurst, 1984b.)

cycle of *Anisakis* (Fig. 1-77). Adult worms are present in the stomachs of many cetaceans, especially small toothed whales, and a few pinnipeds. Embryonated eggs are shed to the exterior with the feces. Following a single molt within the egg, ensheathed second-stage larvae emerge in the sea water. The larvae are preyed upon by euphausiid crustaceans. Upon ingestion, the larval nematodes migrate into the haemocoel of the crustacean. Third-stage larvae develop following ensheathment and another molt in the haemocoel of the first intermediate host. The prevalence of infection in euphausiids is very low but fishes and squids concentrate larvae as they feed on many hundreds or thousands of euphausiids during their life time. In these second intermediate hosts the third-stage larvae penetrate the alimentary tract and encyst in the organs of the body cavity or in the muscles. Advanced third-stage larvae can be serially passed through the oceanic food chain without additional molts occurring. This further concentrates the larvae in a wide diversity of predatory fishes and squids. Squids probably function as obligatory paratenic or transport hosts in the cycle. The cycle is completed when third-stage larvae are consumed by marine mammals. Attaching to the stomach wall, the nematodes undergo 2 more molts, grow and eventually develop into sexually mature adults.

In certain areas of the world — such as Japan, Korea, California, Britain, and Scandinavia — where uncooked fishes and squids are eaten, anisakiasis is an important

human health problem (Otsuru and co-authors, 1965; Oshima, 1966, 1972; Okumura, 1967; Oyanagi, 1967; Otsuru, 1968a, b; Oishi and co-authors, 1969; Myers, 1975; Cheng, 1976; Williams and Jones, 1976; Smith and Wootten, 1978). Human infections, attributed to larval ascaridoid nematodes, are characterized by small ulcers or lesions, particularly in the stomach. This disease is typically transmitted through fishes, though squids, especially *Todarodes pacificus*, serve an equally important role (Okumura, 1967; Oshima, 1972; Doi, 1973). Experimental evidence is lacking to positively link the larval nematodes in cephalopods with pathological symptoms in man, but most clinical parasitologists hold the opinion that species of larval anisakids, normally infective to marine mammal or bird final hosts, may be infective to humans if they ingest raw or partially cooked squids. The numerous reports of larval ascaridoids makes this an area of potential concern, especially when considering the increased harvest of squids throughout the world.

Larvae of *Anisakis simplex* repeatedly have been used as biological indicators of stock heterogeneity by fishery biologists. Most such studies have assessed populations of fishes and not cephalopods. Off New Zealand, Smith and co-authors (1981) used *A. simplex* and the trypanorhynch cestode *Nybelinia* sp. to help document the existence of 2 sympatric species of the arrow squid *Nototodarus* (Table 1-22).

Nematodes in groups other than ascaridoids occasionally have been observed in cephalopods but they are not common (Table 1-23). An unidentified philometroid was taken from the coelomic washings of *Loligo opalescens* in California, USA (Dailey, 1969). Unidentified larvae of the habronematoid nematode *Spinitectus* occur in the stomachs of *Ommastrephes bartrami* in the South Atlantic Ocean (Gaevskaya and co-authors, 1986a), *Photololigo (= Doryteuthis) singhalensis* in the Indian Ocean (Pinchukov and Makarova, 1979) and *Loliolopsis diomedeae* in the Gulf of California (Hochberg, unpubl.). In all cases mentioned here the nematodes have been found in less than 2% of the squids examined.

Unidentified nematodes have been recovered on numerous occasions from caphalopods (Table 1-23). Nouvel (in Dollfus, 1958) working in Monaco, found nematodes encysted in the mantle of *Onychoteuthis banksi* and *Sepia orbignyana*, in the stomach of *S. elegans*, and in the rectum of *Sepiola atlantica* and *Eledone cirrhosa*. In France, Dollfus (1958) recovered nematodes from the musculature of *Histioteuthis bonelliana* and from the stomach of *Illex coindeti*. Off California and Hawaii (USA), I have observed larval nematodes encysted in the digestive tracts of oceanic squid and octopod genera such as *Abralia, Abraliopsis, Enoploteuthis, Pterygioteuthis, Sthenoteuthis, Chiroteuthis, Japatella*, and *Vampyroteuthis*.

Agents: Annelida

Clitellata ('Hirudineans')

Three species of hirudineans have been recovered from cephalopods, in all cases from *Octopus dofleini*. All are piscicolids which have very small posterior suckers and commonly attach to arthropods. Leeches normally obtain blood meals from fishes although some species have been reported to feed on crustaceans. Many of the species which feed on fishes eventually leave to deposit cocoons on hard shelled invertebrates such as crustaceans, pycnogonids, and bivalves (Overstreet, 1983). The association with octopuses appears to be temporary and may or may not involve feeding. Transfer most likely occurs when cephalopods feed on crustaceans.

Borovitzkaya (1949) described *Crangonobdella achmerovi* from *Octopus dofleini* captured in the Okhotsk Sea near the Kurile Islands. According to Epshtein (1961, 1962) this species is synonymous with *C. murmanica* which parasitizes the shrimp *Sclerocrangon boreas* (see also Uspenskaia, 1963), and the fish *Myoxocephalus scorpius*. The worm is widely distributed in Arctic waters having been reported in Greenland, Alaska and Russia as well as in the Okhotsk and Bering Sea. A second species, *Osterobdella papillata* (Fig. 1-78) was described by Burreson (1977) from *O. dofleini* and the black rockfish *Sebastes melanops* collected off Oregon (USA). Little is known about the biology of these 2 species.

A species identified as *Johanssonia arctica* (Burreson, pers. comm.) has been found on *Octopus dofleini* off California (USA). This latter species commonly attaches to deep-sea pycnogonids (i.e., species of *Nymphon* and *Colossendeis*) and decapod crustaceans (i.e., species of *Chionoecetes, Paralithodes,* and *Hyas*) and is also reported to infest fishes (i.e., species of *Anarhichas* and *Gadus*). *J. arctica* is circumpolar in distribution, occurring throughout the Arctic Ocean, as far south as California (USA) in the eastern Pacific Ocean. For a review of this species see Meyer and Khan (1979). Khan and Emerson (1981) elucidated the surface topography of *J. arctica* by scanning electron microscopy.

Polychaeta

Polychaetes are not commonly recognized as symbionts of cephalopods. Clark (1956) and Cheng (1967) reviewed the polychaete annelids which live in the gelatinous egg masses of neritic loliginid squids (Fig. 1-79, 9). *Capitella capitata ovincola* (Fig. 1-79, 3 and 4) was described from the egg fingers of *Loligo opalescens* off California (USA) (Hartman, 1947, 1961). Hartman (1959) later described a second subspecies, *C. c. floridana* (Fig. 1-79, 2), obtained from the eggs of an unidentified loliginid squid collected off Florida, USA. Off France, the egg masses of *Loligo vulgaris* harbor 2 additional species. Boletzky and Dohle (1967) named *C. hermaphrodita* (Fig. 1-79, 1), and Harant and Jecklin (1933) identified *Capitomastus minimus*.

At present these small capitellids are known only from benthic egg masses of species of *Loligo*. They have not been encountered in the egg masses of any other cephalopod genera. The worms live in compact clumps which irregularly penetrate the gelatinous matrix of the squid egg masses. Individuals are intertwined with one another but each is sheathed in a thin flexible mucoid tube. High density infestations of the egg masses of *Loligo opalescens* have been observed on the spawning grounds off California (USA). Several worms, each about 60 mm long, may inhabit a single egg finger (MacGinitie and MacGinitie, 1949; McGowan, 1954; Fields, 1965).

Harant and Jecklin (1933) postulated that *Capitomastus minimus* secretes an enzyme which dissolves the capsular membrane of squid eggs and makes them suitable for food. Species of *Capitella,* on the other hand, appear to feed only on the jelly in which the eggs are imbedded and apparently do not harm the developing embryos. In *Capitella c. ovincola,* the worms infest egg masses at the time they are laid on the bottom. Development time for *Loligo opalescens* embryos is about 30 days. The worms become sexually mature and reproduce about the time the squid larvae hatch; at this time all stages of development to maturity are present (MacGinitie and MacGinitie, 1949; Fields, 1950, 1965; McGowan, 1954). Boletzky and Dohle (1967) reported that the eggs (Fig. 1-79, 9) of *C. hermaphrodita* developed in 5 days. However, they were not able to rear the trochophore larvae (Fig. 1-79, 10 and 11) to complete the life cycle.

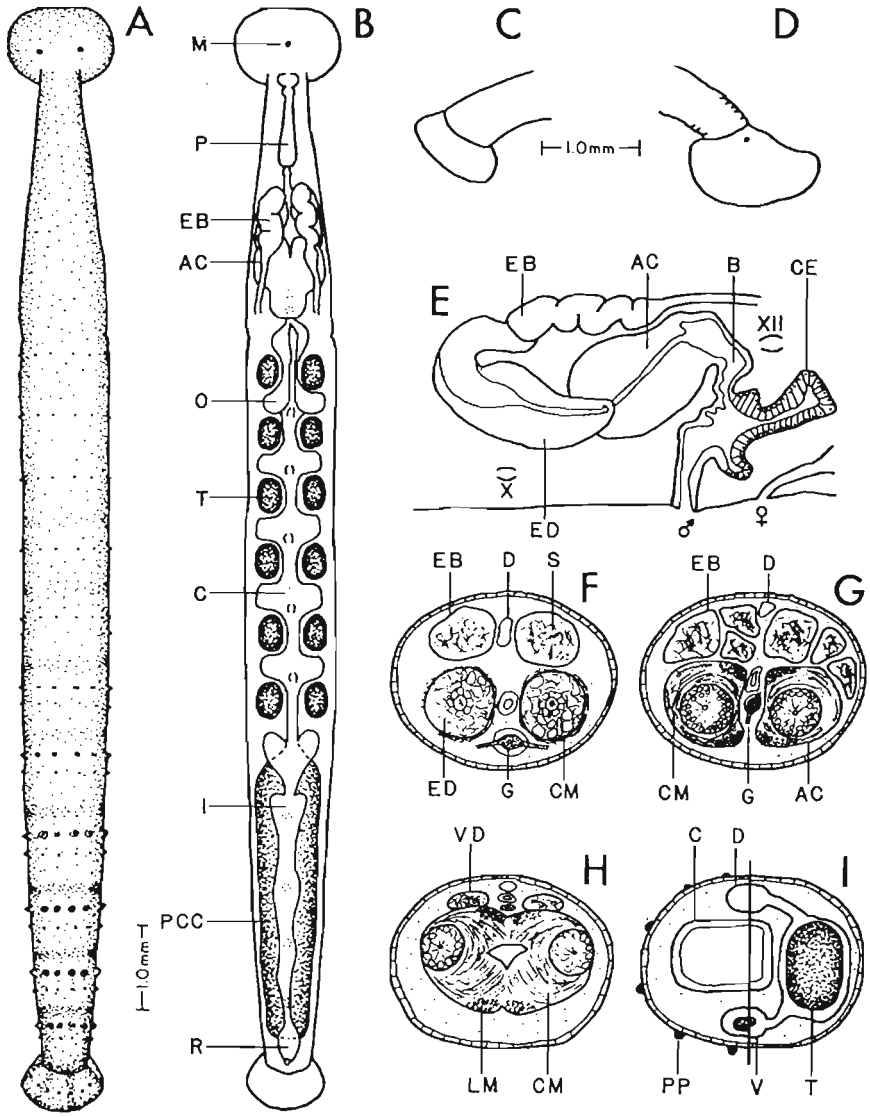


Fig. 1-78: *Ostreobdella papillata* from *Octopus dofleini*. A: Holotype from *Sebastes melanops*, dorsal view. B: Reconstruction of reproductive and digestive systems, ventral view. C: Posterior sucker, lateral view. D: Anterior sucker, lateral view. E: Diagrammatic view of male reproductive tract, lateral view; F: Diagrammatic view of transverse section of ganglion X; G: Diagrammatic view of transverse section of ganglion XI; H: Diagrammatic view of transverse section in posterior portion of segment XI; I: Diagrammatic view of coelomic system, left: segmentally, right: intersegmentally. (AC) Atrial cornu; (B) bursa; (C) crop; (CE) columnar epithelium of posterior bursal pouch; (CM) circular muscle; (D) dorsal sinus; (EB) ejaculatory bulb; (ED) ejaculatory duct; (G) ganglion; (I) intestine; (LM) longitudinal muscle; (M) mouth; (O) ovisac; (P) proboscis; (PCC) posterior crop caecum; (PP) papilla; (R) rectum; (S) sperm; (T) testisac; (V) ventral sinus; (VD) vas deferens. (After Burreson, 1977.)

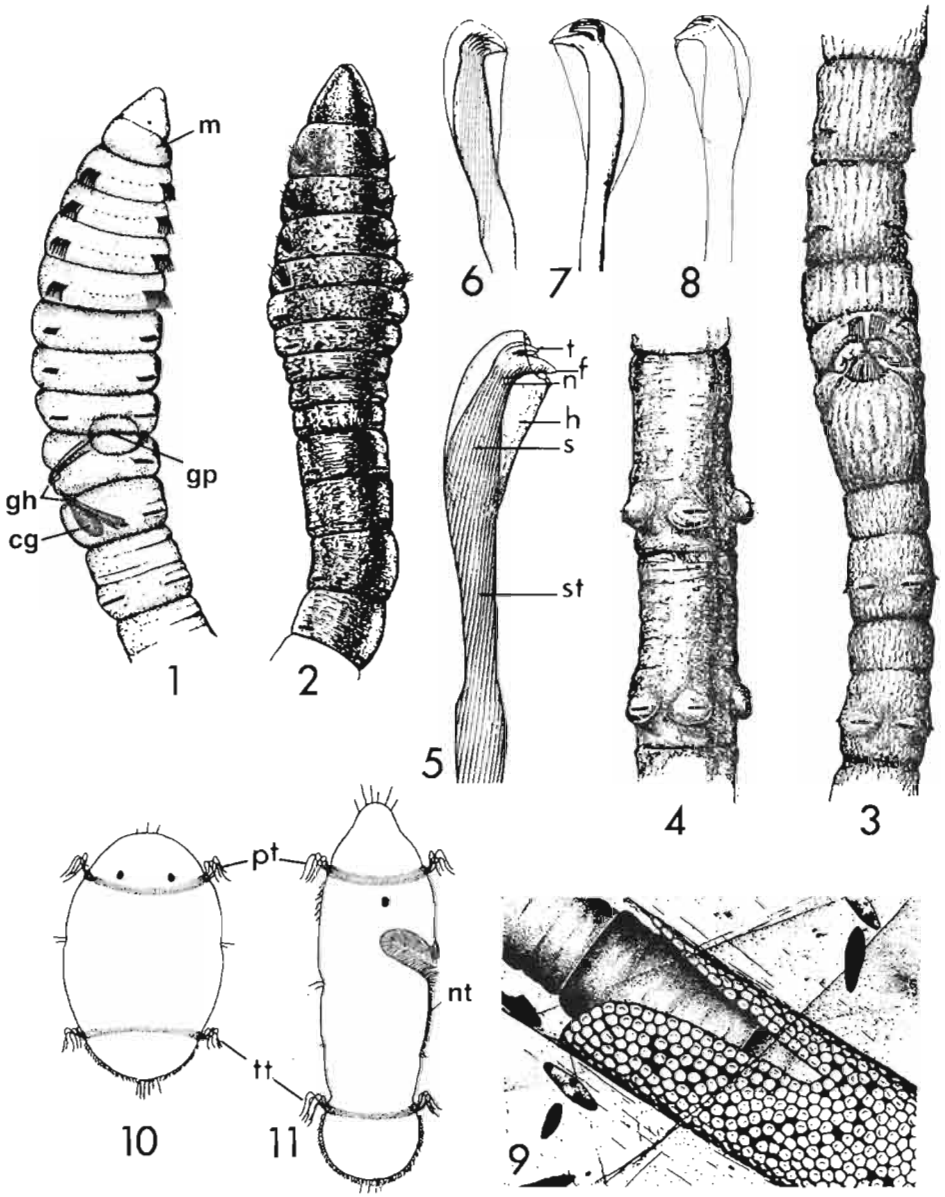


Fig. 1-79: *Capitella* spp. (Polychaeta: Capitellidae) associated with egg masses of loliginid cephalopods. 1, 6, 9 to 11: *Capitella hermaphrodita* from *Loligo vulgaris;* (1) anterior end, lateral view, (6) abdominal hooded hook, lateral view, (9) edge of the polychaete egg mass with the adult worm inside, semi diagrammatic, (10) trochophore larva, day of hatching, (11) trochophore larva, 6 days post hatching. 2 and 7: *C. capitata floridana* from unidentified loliginid egg mass; (2) anterior end, dorsal view, (7) abdominal hooded hook, lateral view. 5: Abdominal hooded hook of *C. capitata* labeled to indicate the parts. 3, 4 and 8: *C. c. ovincola* from *Loligo opalescens;* (3) last 4 thoracic and first 2 abdominal segments of male, dorsal view, (4) twenty-first and twenty-second segments, dorsolateral view, (8) abdominal hooded hook, lateral view. (cg) Copulatory gland; (f) fang; (gh) genital hooks; (gp) genital pore; (h) hood; (m) mouth; (n) neck; (pt) prototroch; (nt) neurotroch; (s) shoulder; (st) shaft; (t) teeth; (tt) teletroch. (1, 6, 9 to 11 after Boletzky and Dohle, 1967; 2, 5 and 7 after Hartman, 1959; 3, 4 and 8 after Hartman, 1947.)

Though these capitellids are most similar to micropredators and not parasites, the degree of host and substrate specificity and the nature of the synchrony of life histories indicate a complex and highly specialized symbiotic interaction.

Agents: Arthropoda/Crustacea

Few published reports treat the crustaceans associated with cephalopods. Seventeen species of copepods, 1 branchiuran, 1 amphipod and 3 isopods have been reported from cephalopods. Crustaceans occur principally in the mantle cavity and on the gills of cephalopod hosts though they may also move about over the external surfaces of the body, head and arms. Other potential parasitic arthropods, such as mites and pycnogonids, and crustaceans, such as barnacles, are not known to infect cephalopods. For reviews see Pelseneer (1929), Monod and Dollfus (1932), Dollfus (1958), and Gotto (1979). For a summary of crustacean parasites see Table 1-24.

Branchiura

The branchiurans are small copepod-like crustaceans which are external parasites of teleost fishes. However, Argilas (1936) discovered a single species, *Argulus arcassonensis* (Fig. 1-80), on the skin of *Sepia officinalis* (formerly referred to as *S. filliouxi*). The argulid, originally described by Cuenot (1912) from a diversity of fishes, has, thus far, only been reported from Arcachon, France (see Cuenot, 1927). Like other ectoparasitic crustaceans this species is dorsoventrally flattened and has developed modifications to enhance the efficiency of attachment and feeding. The second maxillae are greatly enlarged and modified as suckers to aid in attaching to the skin of the host and the mouth parts are adapted for piercing and sucking the blood and body fluids of the host. There are no recent reports on *A. arcassonensis* and nothing is known about its biology or ecology with relation to the cuttlefish host.

Copepoda

The copepods associated with cephalopods do not form a systematic unity. The majority of species have been classified with the poecilostome cyclopoids and harpacticoids but caligoids are also represented. Most of the species are commensals and not true parasites (i.e., they do not injure the host) though in some cases the relationships are highly host specific.

Members of 4 genera of caligoid copepods are reported from cephalopods. Tiny 'tadpole-like creatures' originally discovered by Smith (1887) on *Nautilus pompilius*, are now known to be caligid copepods of the monotypic genus *Anchicaligus* (Ho, 1979). Ho (1980) redescribed the single species, *A. nautili* (Fig. 1-81), which had not been studied in detail since the time of Stebbing (1900). *A. nautili* is the only caligid known to parasitize a deep-water molluscan host. All the nearly 400 other species in the family Caligidae infect coastal or oceanic fishes. In addition to *N. pompilius* the copepod infects *N. macromphalus*. The incidence may approach 100 %. The copepod is distributed throughout the range of both hosts in the Indo-Pacific. Little is known about the biology of the parasite. In his letters from New Guinea, Willey (1896) reported that *A. nautili* attaches to the gills and moves around in the mantle cavity. Haven (1972) indicated that the 'commensal copepod' was common inside the funnel and on the inner surfaces of the ala infundibulae of *N.*

Table 1-24
Crustacean parasites of cephalopods (Original; compiled from the sources indicated)

Cephalopod hosts	Parasites	Locality	Source
ORDER NAUTILOIDEA			
Nautilus pompilius	*Anchicaligus nautili* Copepoda – Caligidae	Western Tropical Pacific Ocean (Palau, Philippines)	Willey (1896), Stebbing (1900), Haven (1972), Ho (1980)
ORDER SEPIOIDEA			
Sepia elegans	*Pennella varians* Copepoda – Pennellidae	Mediterranean (Algeria)	Rose and Hamon (1953)
S. elegans	*Codonophilus* sp. (= *Meinertia*) Malacostraca – Cymothoidae	Mediterranean (France)	Dollfus (in Wirz, 1958)
Sepia esculenta	*Doridicola sepiae* (= *Lichomolgus*) Copepoda – Lichomolgidae	Western North Pacific Ocean (Japan)	Izawa (1976)
Sepia officinalis	*Pennella varians* Copepoda – Pennellidae	Mediterranean (Italy, Algeria)	Wierzejski (1877), Rose and Hamon (1953), Rose and Vaissière (1953)
S. officinalis	*Doridicola longicauda* (= *Sepicola, Lichomolgus, Metaxymolgus*) Copepoda – Lichomolgidae	Eastern North Atlantic Ocean (Holland, France); English Channel (England); Mediterranean (Italy, France)	Claus (1860), Wierzejski (1877), Stossich (1880), Graeffe (1902), Pesta (1909), Cuenot (1927), Stock (1956, 1959, 1960, 1964), Ho (1983)
S. officinalis (= *S. filliouxi*)	*Argulus arcassonensis* Branchiura – Argulidae	Eastern North Atlantic Ocean (France)	Argilas (1936)
ORDER TEUTHOIDEA			
Alloteuthis subulata	*Pennella varians* Copepoda – Pennellidae	English Channel (England)	Hochberg (unpubl.)
Heterololigo pealei (= *Loligo*)	*'Aegathoa oculata'* (= *A. loliginea*) Malacostraca – Cymothoidae	Western North Atlantic Ocean (Connecticut, USA); Caribbean (Virgin Islands); Gulf of Mexico	Harger (1878), Richardson (1905)
Loligo vulgaris	*Pennella varians* Copepoda – Pennellidae	Mediterranean (Italy, Algeria)	Wierzejski (1877), Rose and Hamon (1953), Rose and Vaissière (1953)
Loligo sp.	*Nerocila orbignyi* Malacostraca – Cymothoidae	Western South Atlantic Ocean (Argentina)	Szidat (1955)

Table 1-24 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER TEUTHOIDEA			
Abraliopsis felis	Unident. isopod larva Malacostraca	Central Pacific Ocean (Hawaii, USA)	Hochberg (unpubl.)
Todarodes pacificus	*Caligus orientalis* Copepoda – Caligidae	Japan Sea (USSR)	Kurochkin (1972), Kurochkin and Kazachenko (1975)
T. pacificus	*Caligus* sp. Copepoda – Caligidae	Japan Sea (USSR)	Kurochkin (1972)
T. pacificus	*Lepeophtheirus* sp. Copepoda – Caligidae	Japan Sea (USSR)	Kurochkin (1972)
Todarodes sagittatus	*Doridicola* cf. *agilis* (= *Lichomolgus*) Copepoda – Lichomolgidae	Mediterranean (Spain)	Stock (1960)
Todaropsis eblanae	*Pennella varians* Copepoda – Pennellidae	Mediterranean (Spain)	Stock (1960)
ORDER OCTOPODA			
Opisthoteuthis californiana	*Cholidyella breviseta* Copepoda – Tisbidae	Western North Pacific Ocean (Japan, USSR)	Avdeev (1986)
Unident. cirrate	*Cholidyella intermedia* (= *Cholidya*) Copepoda – Tisbidae	Eastern North Atlantic Ocean (Faroe/Shetland Islands)	Bresciani (1970)
Bathypolypus salebrosus	*Octopinella tenacis* Copepoda – Tisbidae	Western North Pacific Ocean (USSR)	Avdeev (1986)
Benthoctopus ergasticus (= *Polypus*)	*Cholidya polypi* (= *Cholydia*) Copepoda – Tisbidae	Eastern North Atlantic Ocean (Ireland)	Farran (1914)
Benthoctopus fuscus	*Cholidyella nesisi* Copepoda – Tisbidae	Western North Pacific Ocean (Japan)	Avdeev (1986)
Benthoctopus hokkaidensis	*Octopinella tenacis* Copepoda – Tisbidae	Western North Pacific Ocean (USSR)	Avdeev (1986)
Benthoctopus profundorum	*Cholidyella nesisi* Copepoda – Tisbidae	Western North Pacific Ocean (Japan)	Avdeev (1986)
B. profundorum	*Octopinella tenacis* Copepoda – Tisbidae	Western North Pacific Ocean (USSR)	Avdeev (1986)

Table 1-24 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER OCTOPODA			
Eledone moschata	*Pennella varians* Copepoda – Pennellidae	Mediterranean (Italy)	Wierzejski (1877)
Graneledone boreopacifica	*Brescianiana rotundata* Copepoda – Tisbidae	Western North Pacific Ocean (Japan, USSR)	Avdeev (1982a)
G. boreopacifica	*Cholidyella incisa* Copepoda – Tisbidae	Western North Pacific Ocean (Japan, USSR)	Avdeev (1982a)
Octopus briareus	*Octopicola superbus antillensis* Copepoda – Lichomolgidae	Straits of Florida (Florida, USA)	Stock and co-authors (1963)
Octopus cyanea	*Octopicola regalis* Copepoda – Lichomolgidae	Western Central Pacific Ocean (Marshall Islands, New Caledonia)	Humes (1974)
O. cyanea (= *O. cornutus*)	*Octopicola stocki* Copepoda – Lichomolgidae	Western Indian Ocean (Madagascar)	Humes (1963), Humes and Stock (1973)
Octopus longispadiceus	*Octopinella tenacis* Copepoda – Tisbidae	Western North Pacific Ocean (Japan)	Avdeev (1986)
Octopus 'vulgaris'	*Octopicola superbus antillensis* Copepoda – Lichomolgidae	Caribbean (Barbados, Curaçao); Gulf of Mexico (Florida, USA)	Stock and co-authors (1963)
O. vulgaris	*Octopicola superbus superbus* Copepoda – Lichomolgidae	English Channel (France); Mediterranean (France)	Delamare Deboutteville and co-authors (1957), Humes (1957), Bocquet and Stock (1960), Gotto (1962), Laubier (1966)
Octopus sp.	*Octopinella tenacis* Copepoda – Tisbidae	Western North Pacific Ocean (Japan)	Avdeev (1986)
Pareledone charcoti	*Tripartisoma ovalis* Copepoda – Tisbidae	Antarctic Ocean (Ross Sea)	Avdeev (1983)
P. charcoti	*Yunona marginata* (= *Iunona*) Copepoda – Tisbidae	Antarctic Ocean (Ross Sea)	Avdeev (1983)
Pareledone harrisoni	*Tripartisoma ovalis* Copepoda – Tisbidae	Antarctic Ocean (Ross Sea)	Avdeev (1983)
P. harrisoni	*Tripartisoma trapezoidalis* Copepoda – Tisbidae	Antarctic Ocean (Ross Sea)	Avdeev (1983)

Table 1-24 (continued)

Cephalopod hosts	Parasites	Locality	Source
ORDER OCTOPODA			
Pareledone harrisoni	*Yunona marginata* (= *Junona*) Copepoda – Tisbidae	Antarctic Ocean (Ross Sea)	Avdeev (1983)
Pareledone turqueti	*Tripartisoma ovalis* Copepoda – Tisbidae	Antarctic Ocean (Ross Sea)	Avdeev (1983)

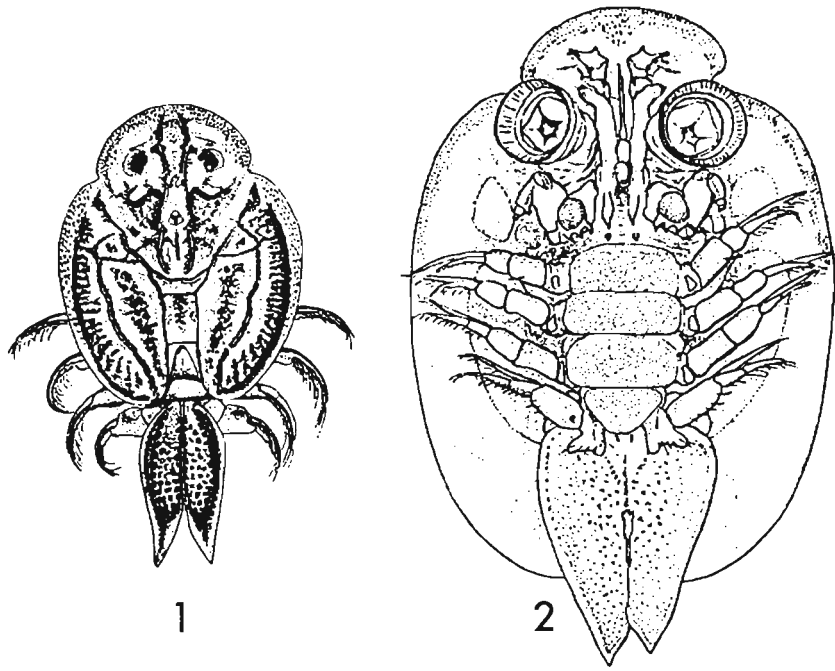


Fig. 1-80: *Argulus arcassonensis* from *Sepia officinalis*. 1: Male, dorsal view. 2: Female, ventral view. (After Cuenot, 1912.)

pompilius in the Philippines. Willey and others have noted that when nautiluses are placed in containers of water, the copepods emerge in large numbers from the mantle cavity and actively swim about. Although not completely known for *A. nautili,* the life cycle of some caligid copepods involves an intermediate host to which a series of chalimus larval stages are attached. Caligid copepods are known to kill fishes held in captivity. Hence, this parasite could potentially present problems in aquariums which maintain displays of live *Nautilus* species.

Kurochkin (1972) reported the presence of 3 species of the genera *Caligus* and *Lepeophtheirus* within the mantle cavities of 9 of 500 (1.8 %) *Todarodes pacificus* examined near Vladivostock (USSR). Kurochkin and Kazachenko (1975) in a paper on the attachment of caligids to humans indicated that one of the above species was *C. orientalis*, a common ectoparasite of a wide variety of fishes. Squids are temporary hosts to which the copepods are briefly attached. Most likely the parasites drop off quickly and do not feed.

Larval stages of the pennellid *Pennella varians* (Fig. 1-82) have been repeatedly noted on the gills of *Eledone moschata, Sepia officinalis, S. elegans, Loligo vulgaris* and *Todaropsis eblanae* (Wierzejski, 1877; Rose and Hamon, 1953; Rose and Vaissière, 1953). All published reports indicate that only cephalopods from the Mediterranean are infected with this copepod. Originally described by Steenstrup and Lutken (1861), adults of this parasite typically occur on a variety of fishes. The presence of *P. varians* on cephalopods has been contested by Stock (1960). However, a specimen which I recovered from the gills of *Alloteuthis subulata* off Plymouth, England, was identified positively as a male *Pennella* varians (Ho, pers. comm.).

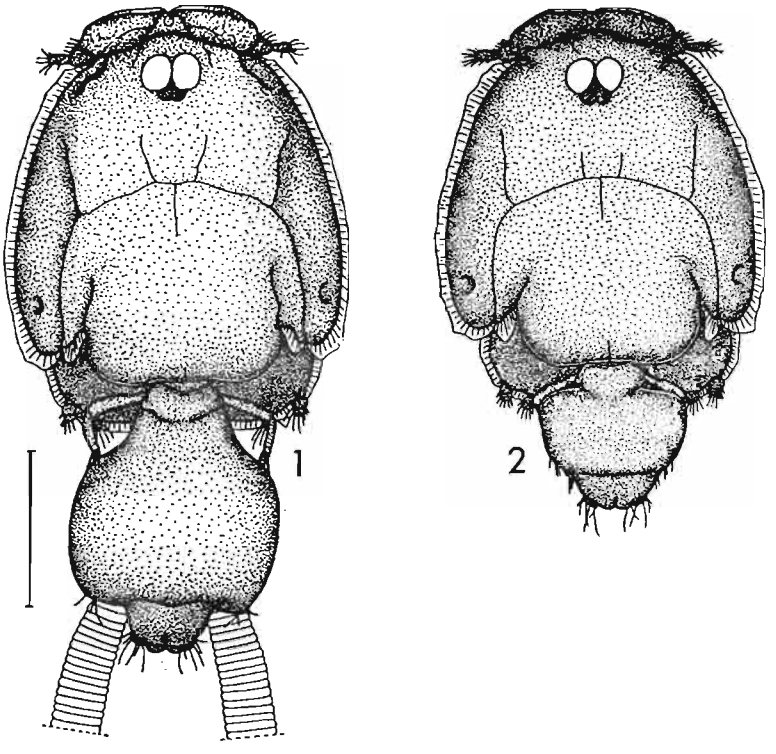


Fig. 1-81: *Anchicaligus nautili* from *Nautilus pompilius*. 1: Female, dorsal view. 2: Male, dorsal view. Scale = 1 mm. (After Ho, 1980.)

The recent work by Avdeev (1982a, b, 1983, 1986) has greatly expanded our knowledge of the harpacticoid copepods associated with deep-sea octopods. All species so far described are placed in the family Tisbidae. Unlike many other harpacticoids which live as symbionts on invertebrates but do not differ significantly from free-living forms (Soyer, 1968), the species associated with octopods often are modified and clearly adapted to a parasitic existence. Farran (1914) provided the first description of a harpacticoid parasitic on a cephalopod. Females of *Cholidya polypi* (often misspelled *Cholydia,* Fig. 1-83, 3) were obtained from the inner surface of the arm web of *Benthoctopus ergasticus*. The host was captured in deep water (1220 to 1400 m) off the coast of Iceland. Mature females are characterized by a single ovisac which contains a relatively few large eggs.

Avdeev (1982a) erected the genus *Cholidyella* to contain a complex of species including *C. intermedia* (Fig. 1-83, 6) first described by Bresciani (1970). Bresciani's species was found in the mantle cavity and on the gills of a unidentified finned octopod collected in 380 to 400 m off Britain in the channel between the Faroe and Shetland Islands. *C. breviseta* (Fig. 1-84, 3 to 6) occurs on the gills of another deep-water cirrate octopod, *Opisthoteuthis californiana*, in the northwest Pacific Ocean (Avdeev, 1986). In the same area, Avdeev (1986) reported *C. nesisi* (Fig. 1-84, 7 and 8) from the gills of *Benthoctopus profundorum* and *B. fuscus* collected at depths ranging from 1000 to 1500 m. *C. incisa* (Fig. 1-84, 1 and 2) and the closely related *Brescianiana rotundata* (Fig. 1-83, 1 and 2) were both found on the gills of *Graneledone boreopacifica* collected off the Pacific coast of Honshu and the Kuriles at depths ranging from 1240 to 1500 m. The incidence of

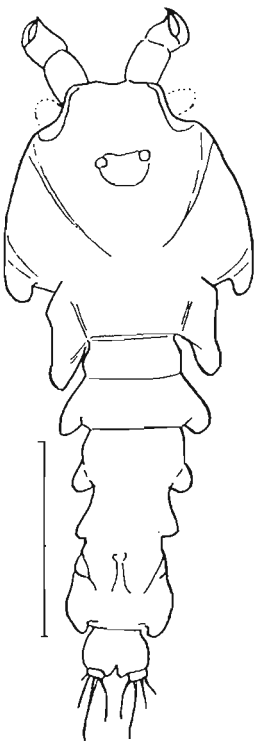


Fig. 1-82: *Pennella varians*. Larval stage of lerneascerid copepod from gills of *Todaropsis eblanae* in the Mediterranean. Scale = 0.25 mm. (After Stock, 1960.)

infection in *G. boreopacifica* was 100 % with an intensity of infection between 2 and 115 copepods host^{-1}.

Another closely related genus, *Tripartisoma,* was erected by Avdeev (1983) to contain 2 species which parasitize octopods of the genus *Pareledone* in depths ranging from 85 to 390 m in the Ross Sea (Antarctica). *T. trapezoidalis* (Fig. 1-83, 5) occurred on the ventral surface of the head of 2 of 13 *P. harrisoni*, whereas *T. ovalis* (Fig. 1-83, 4) was found on the gills of 1 of 3 *P. turqueti,* 1 of 13 *P. harrisoni* and 2 of 7 *P. charcoti.* The above 4 genera are relegated to Boxshall's (1979) subfamily, the Cholidyinae.

Avdeev (1983) placed his new genus *Yunona* in the subfamily Tisbinae. Currently known only from the Antarctic, *Y. marginata* (Fig. 1-85, 3) has been found on the gills of 2 species of *Pareledone* hosts. Of the animals examined by Avdeev (1983), 5 of 7 *P. charcoti* and 1 of 13 *P. harrisoni* were infected. The intensity was low with 1 to 7 female copepods host^{-1}. Avdeev (1986) erected a second genus, *Octopinella,* for a species which parasitizes the gills of a wide diversity of octopods in the Northwestern Pacific Ocean. *O. tenacis* (Fig. 1-85, 1 and 2) lives on *Benthoctopus hokkaidensis, B. profundorum, Bathypolypus salebrosus, Octopus longispadiceus* and an unidentified species of the genus *Octopus*. The work by Avdeev indicates that octopods possess a rich and varied fauna of parasitic harpacticoid copepods. However, nothing has been reported about the biology or life histories of the copepods nor their effects on their octopod hosts.

The lichmologids are highly mobile poecilostomatoid copepods which actively move

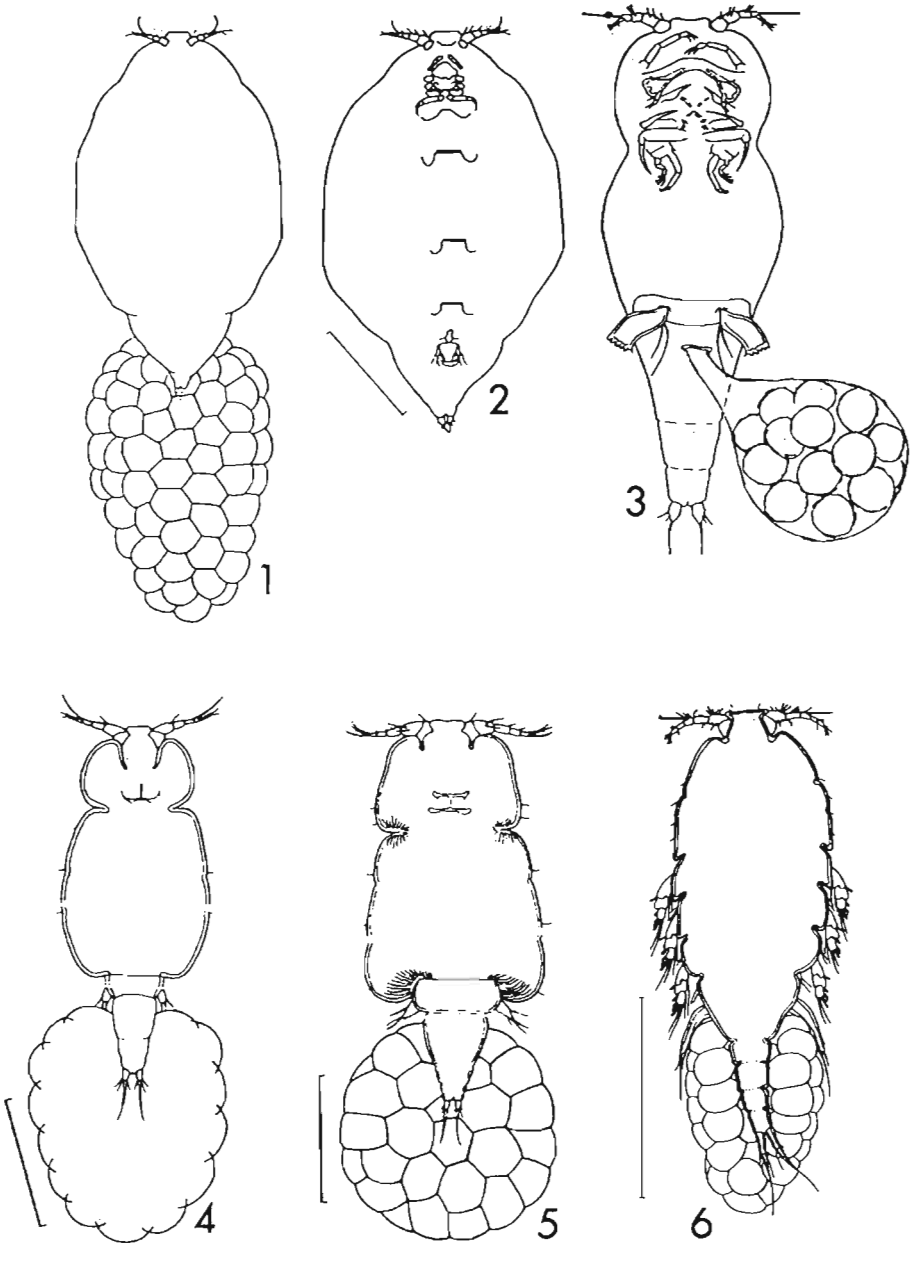


Fig. 1-83: Harpacticoid copepods. Family Tisbidae (Cholidyinae), from octopods. 1 and 2: *Brescianiana rotundata* from *Graneledone boreopacifica*; (1) female, dorsal view; (2) male, ventral view. 3: *Cholidya polypi* from *Benthoctopus ergasticus*; female, dorsal view. 4: *Tripartisoma ovalis* from *Pareledone turqueti*; female, dorsal view. 5: *T. trapezoidalis* from *P. harrisoni*; female, dorsal view. 6: *Cholidyella intermedia* from unidentified cirrate octopod; female, dorsal view. 1, 2 and 6: Scales = 0.5 mm; 4 and 5: scales = 0.3 mm. (1 and 2 after Avdeev, 1982a; 3 after Farran, 1914; 4 and 5 after Avdeev, 1983; 6 after Bresciani, 1970.)

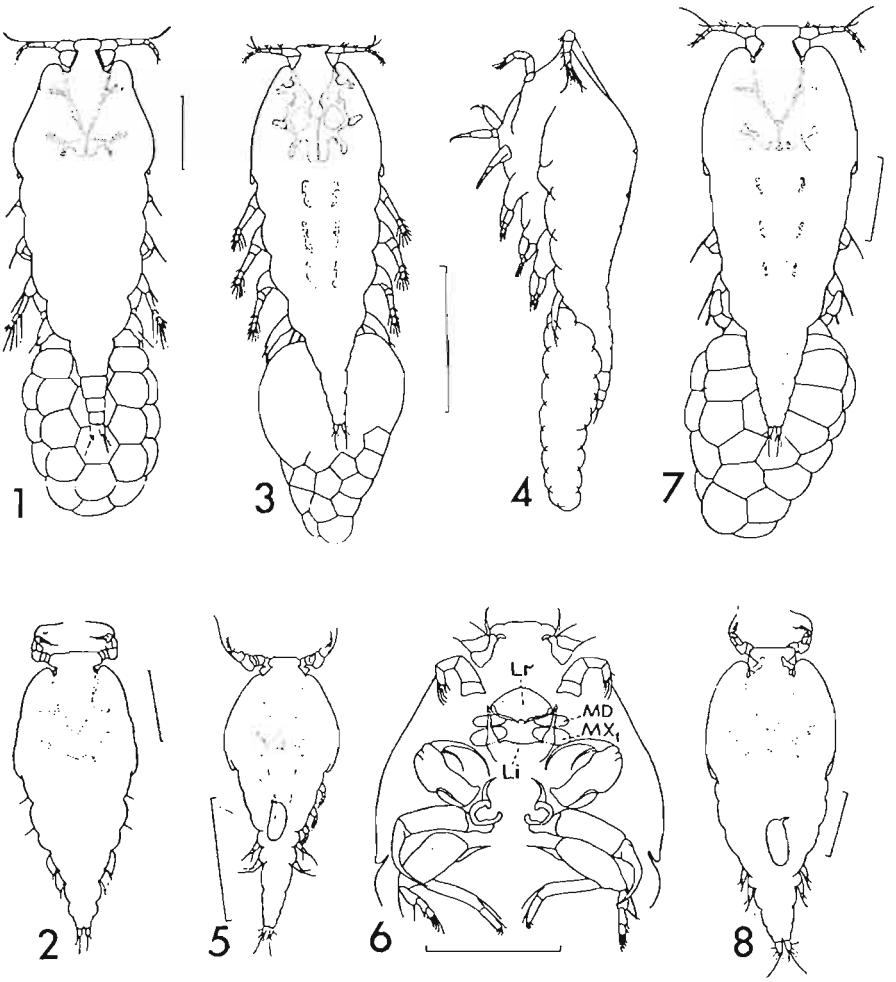


Fig. 1-84: *Cholidyella* spp. (Harpacticoida: Tisbidae: Cholidyinae) from octopods. 1 and 2: *Cholidyella incisa* from *Graneledone boreopacifica*; (1) female, dorsal view; (2) male, dorsal view. 3 to 6: *C. breviseta* from *Opisthoteuthis californiana*; (3) female, dorsal view; (4) female, lateral view; (5) male, dorsal view; (6) female, rostral, oral and postoral region; ventral view. 7 and 8: *C. nesisi* from *Benthoctopus profundorum*; (7) female, dorsal view; (8) male, dorsal view. 1, 5 to 7: Scales = 0.2 mm; 2 and 8 = 0.1 mm; 3 and 4 = 0.5 mm. (1 and 2 after Avdeev, 1982a; 3 to 8 after Avdeev, 1986.)

about over the surface of invertebrate hosts feeding on mucus. In their review of the family, Humes and Stock (1973) and Ho (1983) discussed the species known to live on cephalopods. *Doridicola longicauda* (Fig. 1-86, 1 and 2) is found on the gills and in the mantle cavity of *Sepia officinalis* wherever this species of cuttlefish occurs (Claus, 1860; Wierzejski, 1877; Pesta, 1909; Cuenot, 1927; Stock, 1956, 1960). Originally described as *Sepicola longicauda* the copepod underwent a bewildering series of name changes, including *Lichomolgus sepicola* and *Metaxymolgus longicauda*, before being stabilized in the genus *Doridicola* (Humes and Stock, 1983). Stock (1956) reported that he found hundreds of copepods of both sexes on nearly every *Sepia* examined from off the coast of

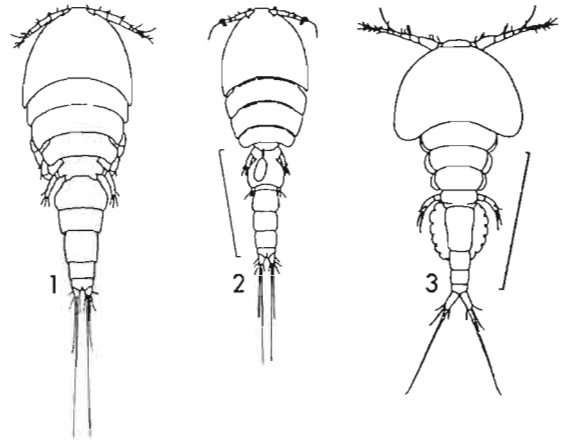


Fig. 1-85: Harpacticoid copepods, Family Tisbidae (Tisbinae), from octopods. 1 and 2: *Octopinella tenacis* from *Benthoctopus profundorum*; (1) female, dorsal view, (2) male, dorsal view. 3: *Yunona marginata* from *Pareledone charcoti*; female, dorsal view. Scales = 0.5 mm. (1 and 2 after Avdeev, 1986; 3 after Avdeev, 1983.)

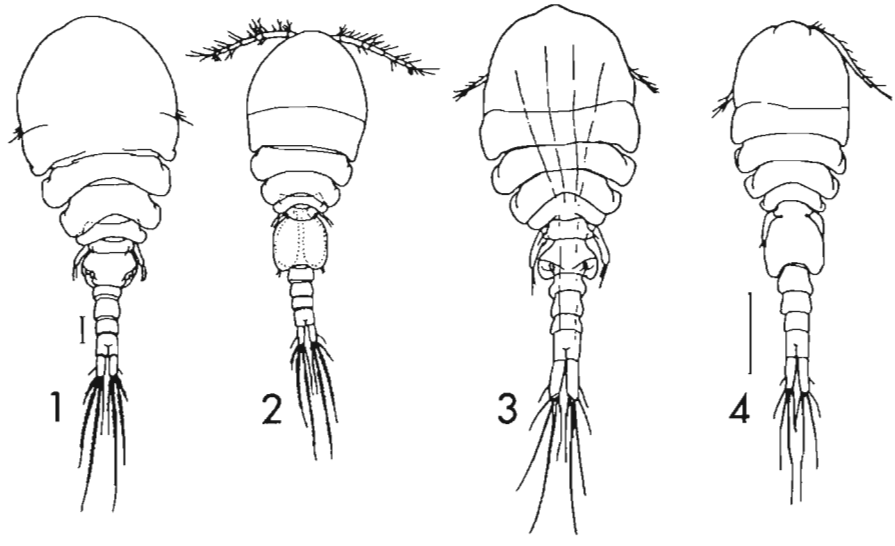


Fig. 1-86: *Doridicola* spp. (Cyclopoida: Lichomolgidae) from cuttlefishes. 1 and 2: *Doridicola longicauda* from *Sepia officinalis*; (1) female, dorsal view; (2) male, dorsal view. 3 and 4: *D. sepiae* from *S. esculenta*; (3) female, dorsal view; (4) male, dorsal view. 1 and 2: Scale = 0.1 mm; 3 and 4 = 0.3 mm. (1 and 2 after Ho, 1983; 3 and 4 after Izawa, 1976.)

the Netherlands. They can easily be seen on the gills of the host due to their contrasting color. Though the body is colorless the intestine, ovaries and ovisacs are bright white and the small eye red. The copepods cling with their second antennae to the tissues of the host. Ovigerous females were abundant in the first weeks of July (Stock, 1956). A second species, *D. sepiae* [= *Lichomolgus sepiae*] (Fig. 1-86, 3 and 4), was reported by Izawa

(1976) from *S. esculenta* in Japan. Stock (1960, 1964) recovered a single specimen of *D.* cf. *agilis* from the gills of *Todarodes sagittatus* at Rosas, Spain.

Members of the genus *Octopicola* live in specific association with octopuses. In the English Channel and in the Mediterranean *Octopus vulgaris* is infected with *O. superbus superbus* (Fig. 1-87, 4 and 5, Fig. 1-88, b and d). In the West Indies, at Barbados and

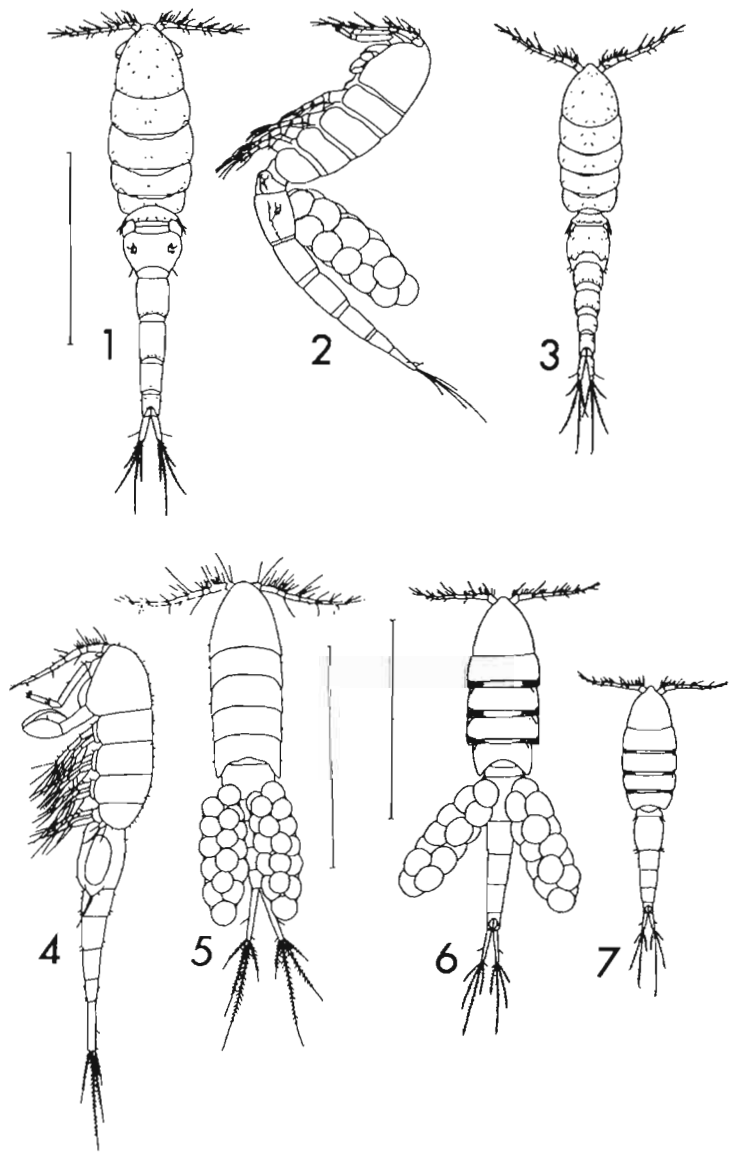


Fig. 1-87: *Octopicola* spp. (Cyclopoida: Lichomolgidae) from octopuses. 1 to 3: *Octopicola regalis* from *Octopus cyanea*; (1) female, dorsal view, without ovisacs; (2) female, lateral view; (3) male, dorsal view. 4 and 5: *O. superbus* from *O. vulgaris*; (4) male, lateral view; (5) female, dorsal view. 6 and 7: *O. stocki* from *O. cyanea*; (6) female, dorsal view; (7) male, dorsal view. 1 to 3: Scale = 0.1 mm; 4 and 5 = 0.5 mm; 6 and 7 = 1.0 mm. (1 to 3 after Humes, 1974; 4 and 5 after Humes, 1957; 6 and 7 after Humes, 1963.)

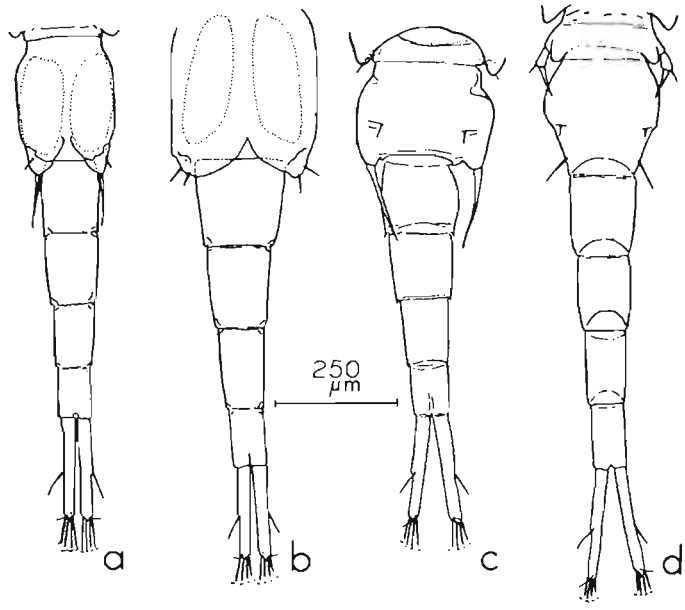


Fig. 1-88: *Octopicola superbus*. Urosomes of subspecies from *Octopus vulgaris*. a and c: *O. s. antillensis* from Curaçao: (a) male; (c) female. b and d: *O. s. superbus* from Roscoff; (b) male; (d) female. (After Stock and co-authors, 1963.)

Curaçao, the same species of host harbors *O. s. antillensis*. The subspecies differ not only morphologically (Fig. 1-88, a and c) but Stock and co-authors (1963) reported that *O. s. antillensis* carries only half as many eggs as the European subspecies, and the individual eggs are larger. Current thinking among cephalopod taxonomists indicates that the common *'O. vulgaris'* may be represented by a complex of species which are distinctly different on either side of the North Atlantic. This would add considerable weight to the morphological differences observed in the copepod parasites. Humes and Stock (1973) also identified *O. s. antillensis* from *Octopus briareus* collected at several sites in Florida (USA). *Octopus cyanea* captured off Madagascar were infested with *O. stocki* (Fig. 1-87, 6 and 7), whereas *O. regalis* (Fig. 1-87, 1 to 3) was present in the same host in the Pacific Ocean at New Caldeonia and Eniwetok Atoll. Additional details on morphology and distributions are included in Delamare Deboutteville and co-authors (1957), Humes (1957, 1963, 1974), Bocquet and Stock (1960), Stock and co-authors (1963), Laubier (1966), Humes and Stock (1972).

These small, cyclopiform copepods normally live in the mantle cavities of their octopod hosts though they also may be found on the body surfaces and amongst the eggs. In the mantle cavity they move about freely over the gills or attach, by means of the second antennae, to the arterial stems beneath the branchial leaflets. No damage to the tissues of the gills or mantle cavities of the hosts has been reported. Delamare Deboutteville and co-authors (1957) noted that the European species, *Octopicola superbus*, inhabits the mantle cavity during the day but becomes more active after dark and moves out on the arms and over the head and mantle. All lichomolgids have a single host life cycle. Delamare Deboutteville and co-authors demonstrated that *O. superbus* exhibits a strong chemotaxis

to the egg masses of the octopus. Even when virtually empty the egg cases are attractive to the copepods. They are probably correct in assuming that autoinfestation regularly takes place. Gotto (1962) suggested that the reproductive rates of lichomolgids (i.e., egg number) reflects the mobility and habits of the host. Members of the genus *Doridicola* infect cuttlefish of the genus *Sepia*, and have a high egg count, whereas species of *Octopicola* occur in association with the more sedentary genus *Octopus* and produce a much smaller number of eggs.

Malacostraca

Of the parasitic malacostracans a few isopods have been discovered on cephalopods. Though rare, they occur principally in the mantle cavity. *Aegathoa loliginea*, originally described by Harger (1878) from *Heterololigo (= Loligo) pealei* is now known to be synonymous with '*Aegathoa oculata*' (Fig. 1-89, 1 and 2) (Richardson, 1905). The genus *Aegathoa* is considered to be a group name which represents a complex of young isopods of

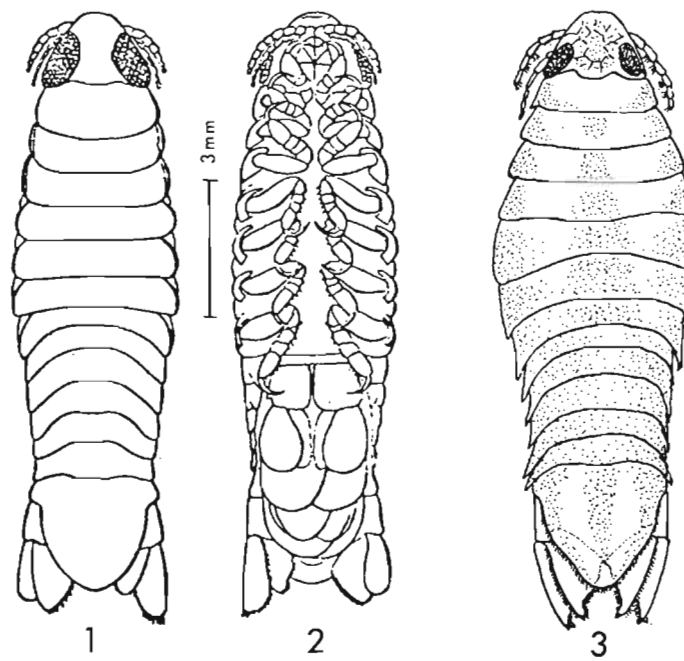


Fig. 1-89: Isopod crustaceans from loliginid squids. 1 and 2: *Aegathoa oculata* from *Heterololigo (= Loligo) pealei*; (1) dorsal view; (2) ventral view. 3: *Nerocila orbignyi* from *Loligo* sp. (1 and 2 after Richardson, 1905; 3 after Szidat, 1955.)

several genera and species. '*A. oculata*' has been reported from a number of fishes found along the Atlantic and Gulf coasts of the United States, Mexico and the West Indies. A second isopod, *Nerocila orbignyi* (Fig. 1-89, 3), was collected by Szidat (1955) from an unidentified species of *Loligo* off the coast of Argentina. A single individual of an undetermined species of *Codonophilus (= Meinertia)* was taken from a specimen of *Sepia elegans* captured at Port-Vendres, France (Dollfus, 1958). A single individual of an unidentified isopod has been recovered from *Abraliopsis felis* in the North Pacific

(Hochberg, unpubl.). Hanlon and Forsythe (unpubl.) noted an unidentified isopod attached toward the distal end of the mouth of a mature male *Sepioteuthis lessoniana*, 175 days old. The isopod remained in the same location for 27 days until the squid was sacrificed for research purposes. The squid did not appear stressed by the isopod's presence. The isopod undoubtedly was introduced into the culture tank along with the many live fishes fed daily to the squids.

All the isopods named above are cymothoids, which as adults typically inhabit the gill chambers, skin and fins of fishes, Narrow host specificity is generally not observed, since these parasites are not permanently attached. Sexual dimorphism is the rule, and the life cycle is protrandric. Males are similar in size and shape to juveniles whereas females are very much larger and their bodies asymmetrically proportioned. Female isopods brood their eggs in a marsupium under the thorax. Following hatching, a free-swimming manca stage is released. During juvenile development, the aegathoid stage attaches to a fish or cephalopod host. After settling on the host, adult male characters are attained with the next molt. The male phase continues through several additional molts until a second individual lands on the host. At this point the larger of the 2 isopods is transformed into a functional female and begins to produce eggs. If the female dies, the remaining male begins to molt and eventually will assume the role of female when another isopod settles on the host. For examples of cymothoid life cycles see Bowman (1960) and Brusca (1978).

Hanlon and Forsythe (unpubl.) observed an unidentified hyperiid amphipod on a laboratory-reared juvenile *Loligo forbesi* (Fig. 1-90). The young squid was observed swimming normally with a barely visible white speck attached to its mantle. Microscopic examination revealed the presence of an amphipod attached by its mouthparts to the squid. SEM of the squid's body clearly showed the site of attachment and attendant skin damage. The laboratory-reared squid were being fed wild-caught live zooplankton, predominantly copepods. Since hyperiid amphipods are similar in size to the desired copepods

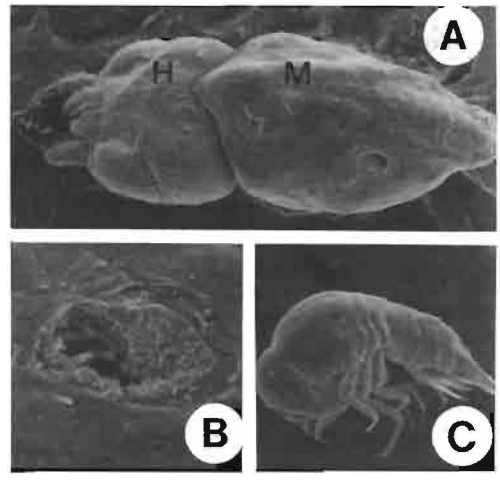


Fig. 1-90: Unidentified hyperiid amphipod found on a 5 day-old *Loligo forbesi* (2.8 mm ML). A: Attachment site on the squid's body revealed by SEM (lower right). B: Details of skin damage; note penetration of body wall. C: Amphipod (TL 1.5 mm). (H) Head; (M) mantle. (Original, provided by Hanlon and Forsythe, unpubl.)

they would not have been excluded. Hanlon and Forsythe suggest that the presence of tiny hyperiid amphipods may have contributed to the high mortality (98 %) seen in this squid species over the first 30 days of culture. Hyperiid amphipods typically attach to and feed on gelatinous zooplankton such as medusae, ctenophores, heteropod mollusks, etc. This is the first report of a hyperiid attached to a cephalopod.

The fact that isopods and amphipods can attach to continuously swimming squids for prolonged periods of time suggests that such relationships may be common in nature.

Brachyuran malacostracans have been reported as commensals in the mantle cavity of several species of squids. Fischer (1943) found specimens of the galatheid *Munida bamffia* in the mantle cavity of *Alloteuthis subulata* dissected by his students in Paris (France). Serene (1961) discovered megalopa larvae of a crab that closely resembles *Monolepis orientalis* in a number of *Loligo* captured of Vietnam. On the surface these would appear to be 'accidental' associations, but Serene indicated that, in all cases, only 1 megalopa was found per host and that, in each case, the coloration blended perfectly with that of the host cephalopod.

1.3 STRUCTURAL ABNORMALITIES AND NEOPLASIA

R. T. HANLON and J. W. FORSYTHE

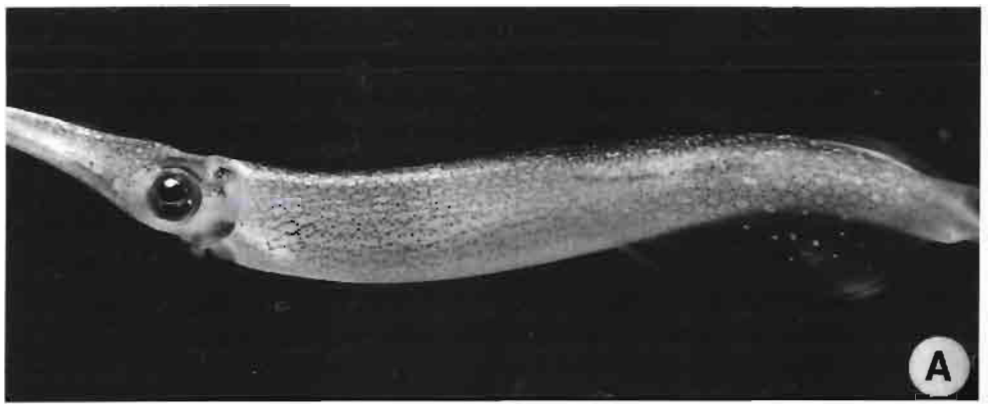
A wide range of structural abnormalities has been reported by a variety of cephalopod systematists during the past 100 years. These include various oddities such as bifurcation of arms, too many arms, and dual hectocotylization in males. However, these have been observed mainly on preserved specimens and nothing is known of the dysfunction they may produce or their implications for disease.

The internal supporting structures of some cephalopods can be altered from repeated physical contact of these animals with structures in their tanks when kept for long periods in captivity. For example, Fig. 1-91, A illustrates how the semi-rigid gladius of *Loligo plei* can bend after the squid bangs into the aquarium wall repeatedly and with considerable velocity; the entire shape of the squid changes, thus affecting swimming and feeding. Cuttlefish *Sepia officinalis* cultured in the laboratory occasionally hit the walls often enough to cause a structural change in the cuttlebone. It is highly unlikely that these abnormalities occur in nature.

Colmers and co-authors (1984) reported a curious abnormality in which hatchling cephalopods lacked one or both statoliths in the statocyst organ for reception of gravity and angular acceleration. This resulted in death because the cephalopods could neither swim properly nor capture food. Eventually it was determined that low levels of the element strontium in the seawater prohibited biomineralization of the statoliths; keeping levels of strontium at 8 mg l^{-1} (normal) negated the problem (Hanlon and co-authors, 1989). It is conceivable that, since cephalopods inhabit nearly all known marine habitats, such variations in seawater could affect embryogenesis in other species worldwide.

In our laboratory we have observed a structural abnormality of the eye of *Octopus maya* (Fig. 1-91, B). Occasionally, hatching octopuses were observed with the orbit of one or both eyes completely exposed. The condition may represent a developmental abnormality or merely a physical/mechanical accident during hatching that pops the eye orbit out of its muscular socket. The affected eye is non-functional and octopuses with bilateral damage are blind; however, the condition is not lethal in laboratory populations.

Neoplasias have not been verified in cephalopods but several 'tumors' have been reported. Jullien and Jullien (1951) found 2 cuttlefish with tumors; one individual had 4, the other 5 tumors on the ventral mantle. These were elevated, hard, whitish nodules measuring several mm in diameter and appeared to be connective tissue that was highly vascularized at the periphery. The connective tissue had lost its normal stratified appearance and was compact and homogeneous within the tumors. There were many compacted fibroblasts adjacent to the tumors and some within the compacted tissue. Epidermis and dermis were gone, thus rendering the white color of the tumors. Nigmatullin (5 Atlant NIRO, Kaliningrad, USSR 236000) provided the following notes on tumors observed in *Octopus vulgaris* in 1971 off the northwest coast of Africa (Cape Blanc area). Approximately 1.6% of 2000 specimens (12 to 18 cm mantle length) had the distal quarter of their mantle enlarged by a factor of 3 to 8×. The enlargements appeared as distinct tumorous



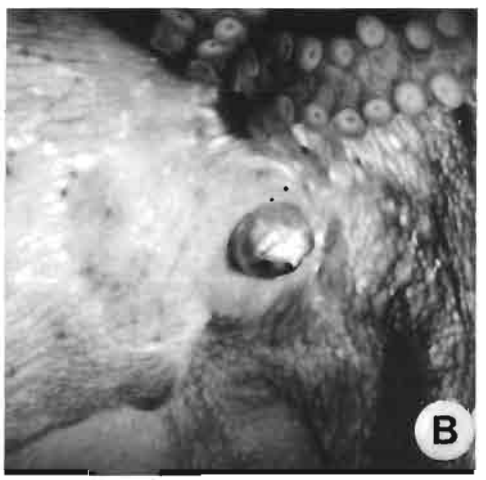


Fig.1-91: Structural abnormalities. A: Structural abnormality in the squid *Loligo plei* in which the gladius is bent in an s-curve due to repeated hitting of the wall (normally the gladius is straight). B: Abnormal eye in *Octopus maya* in which the eyeball has developed outside the eye socket. (Original.)

growths of glassy consistency and gray color, and the adjacent mantle muscle tissue was degenerating. The skin at the edges was stretched. The specimens were preserved in formalin but not analyzed further.

Jullien (1928, 1940) and Jacquemain and co-authors (1947) injected several purported carcinogens into cephalopods to observe their defense responses and to attempt to induce tumors. Some of these carcinogens resulted in 'lesions' or 'tumors' but none were verified to be neoplastic; by the nature of their studies the authors were more interested in defense responses. In summary, there is no hard information on neoplasia in cephalopods.

Perhaps information on the etiology of viral and neoplasic disorders should be accumulated in view of the widespread consumption of cephalopods by humans. Lin and co-authors (1983) presented evidence that squid, octopus and other seafoods contain unusually high levels of various amines that, along with other evidence, indicates dietary factors may play an important role in the etiology of human stomach cancers and other gastrointestinal tumors.

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2. DISEASES OF ANNELIDA

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The phylum Annelida incorporates coelomate metamerically segmented worms. It includes earthworms and leeches, as well as numerous marine and limnetic species. The phylum is divided into 3 large classes: Oligochaeta, Polychaeta and Hirudinea. Most of the living marine annelid species are members of the Polychaeta which are generally considered to display the more primitive features of the phylum. Oligochaeta includes the earthworms as well as freshwater and marine forms, whereas the Hirudinea is typified by aquatic and terrestrial leeches.

Accounts of parasitism, or of commensal relationships, within all 3 annelid classes are common in the literature. However, these reports are often restricted to morphological descriptions and taxonomic classifications of the parasitic species, or they focus on annelids as intermediate hosts for the parasites of commercially important animals. Accurate descriptions of pathological activities in parasitic interactions are rare. This chapter, therefore, includes descriptions of pathological conditions in terrestrial and limnetic annelids, especially where information on marine forms is lacking.

DISEASES CAUSED BY MICROORGANISMS

Agents: Viruses

Devauchelle and Durchon (1973) have identified an iridovirus that infects the spermocytic cells of the marine polychaete *Nereis diversicolor*. Viral particles were observed in the cytoplasm of germinal cells where condensation of viroplasm and envelope formation yielded active virions. The virions were icosohedral in shape and ranged in diameter from 165 to 185 nm.

Viral particles have also been isolated from *Enychytraeus fragmentosus* (Bonami, 1976). However, in this case, particles were recovered from lyzed material so that their capacity to infect cells could not be determined. Indeed, in a recent study of the leech *Piscicola geometra,* Ahne (1985) demonstrated that, whilst this annelid acted as an effective intermediate host for the infection of carp by spring viremia virus (SVCV), it transmitted the virus to fish by mechanical means. Viral proliferation was not detected in the leech, suggesting that cellular infection did not occur.

Agents: Bacteria

Reports of bacterial interactions with annelids are relatively common (for polychaetes consult Cavanaugh, 1985; Dilmore and Hood, 1986; for oligochaetes, Giere, 1981; Richards and co-authors, 1982). However, in marine species the pathological effects of bacterial infection are largely unknown. Indeed, the interaction may often be symbiotic

rather than parasitic. For instance, apparently stable symbioses have been identified between 2 species of Gram negative bacteria and gutless oligochaetes of the genus *Phallodrilus* (Felbeck and co-authors, 1983; Giere and Langheld, 1987). Worm eggs become infected with the bacterium at the time of oviposition. This infective process is facilitated by intrusion of large stores of bacteria from a genital pad abutting the female genital pores. During ontogenesis, bacteria are identifiable in both extra and intra-cellular forms. Rather than being pathological, this interaction is apparently essential to the metabolic requirements of these gutless worms, providing an essential source of carbon fixation.

Bacteria have also been identified in exogenous association with annelids. In both the hydrothermal-vent polychaete *Alvinella pompejana* and the marine oligochaete *Tubifoides benedii,* filamentous epibacteria have been observed attached to the epidermis in the posterior region of the animal (Desbruyeres and co-authors, 1983; Dubilier, 1986) (Fig. 2-1). In neither case was the interaction demonstrably pathogenic.

The only report of morbidity induced by bacterial infection in marine worms comes from Fanthom and Porter (1909). These authors identified *Bacillus arenicolae* in the intestinal epithelium of the polychaete *Arenicola ecaudata.* The bacterium caused swelling and cell damage. According to the authors, the infection hastened the death of infected worms, although experimental confirmation of this effect was not provided.

Indeed, clear evidence for pathological activity by bacteria on annelids has only been obtained for terrestrial oligochaetes. The earthworm *Lumbricus terrestris* has proven to be extremely susceptible to fatal infection by the biological control agent *Bacillus thuringiensis.* Similarly, a blister disease in the earthworm *Eisenia foetida* has been attributed to the pathological effects of *B. thuringiensis.* Worms affected by this disease develop blisters containing large concentrations of bacteria that resemble *B. thuringiensis.* The blisters ulcerate causing mortality amongst the infected subjects (Smirnoff and Heimpel, 1961; Heimpel, 1966). However, the generalized susceptibility of worms to such infection and the correlation of pathological effects with bacterial activity has been questioned (Benz and Altwegg, 1975).

DISEASES CAUSED BY PROTOZOANS

Agents: Flagellata

Most of the reports available on flagellate infection in annelids relate to the role of worms as intermediate hosts. For instance, recent evidence suggests a substantial affinity of trypanosomes for a variety of aquatic leeches, although the physiological effect of trypanosome infection is largely undocumented. Ray and Choudhury (1984) have demonstrated that rhyncobdellid leech *Helobdella novica* is an effective vector for the transmission of *Trypanosoma rotatorium* to a variety of anuran amphibian species. The parasite is held in the crop and intestinal caecae of the leech vector, where it occurs in 4 discrete life cycle stages: epimastigote, spheromastigote, amastigote, and metacyclic forms. The authors suggested that at least partial maturation within the intermediate annelid host is essential to the life cycle of the parasite. Similar observations are available for a number of other vector leech species (Nigrelli, 1944; Barrow, 1953; Diamond, 1958). Woo and Bogart (1986) demonstrated that *T. ogawai* developed to the metatrypanosome stage in

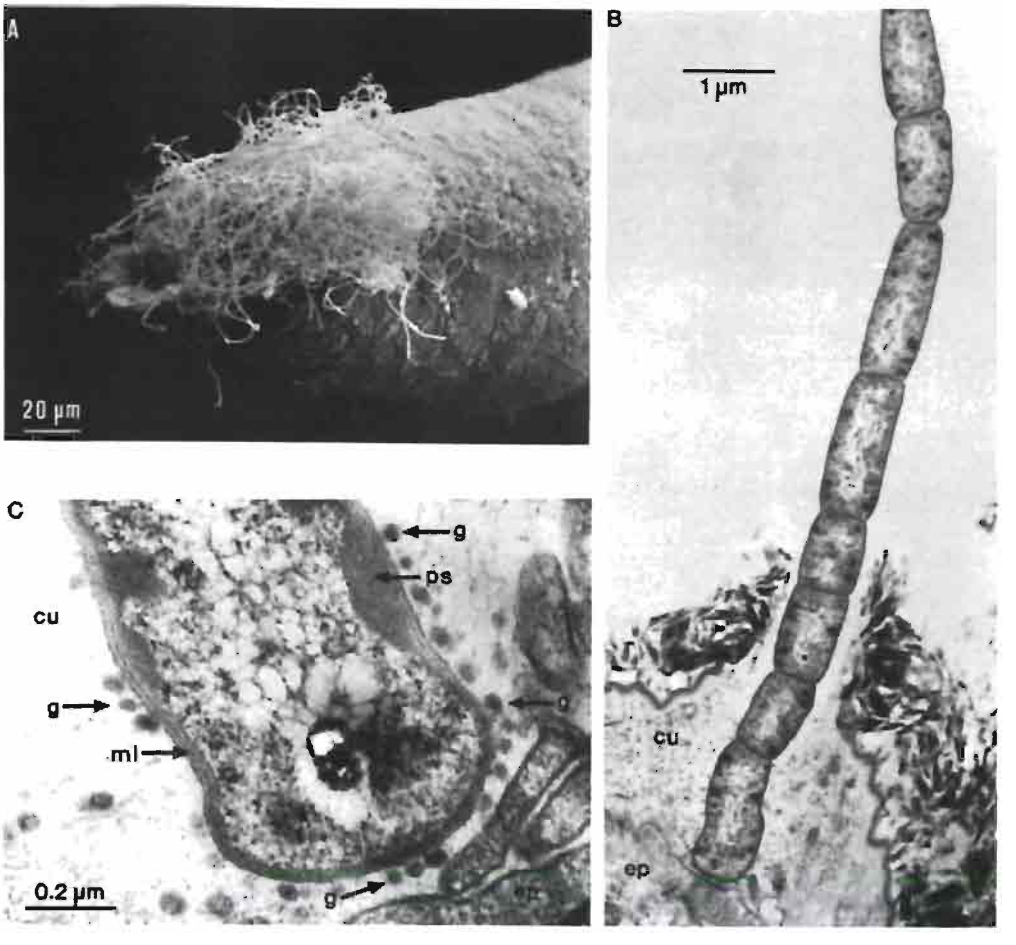

Fig. 2-1: Filamentus epibacteria attached to the epidermis of the marine oligochaete *Tubifoides benedii*. A: Epibacteria attached to the tail of *T. benedii*. B: Bacterial filament embedded in the cuticle. C: Basal cell of bacterial filament showing globules that surround the bacterial cells in the cuticle. (cu) cuticle, (ep) epidermis, (g) globules, (ml) middle layer of cell wall, (ps) periplasmic space. (After Dubilier, 1986.)

the crop of the leech *Bactrobdella picta* within 9 days of infection at 21 °C. Metatrypanosomes from leeches were infective when inoculated onto salamanders 16 days after infection.

This intermediate host activity of leeches is not restricted to trypanosomes. Khan (1984) has shown that the marine leech *Johanssonia arctica* is a natural vector capable of simultaneously transmitting both *Trypanosoma murmanensis* and the piroplasm, *Haemohormidium beckeri*, to American plaice. A similar capacity for simultaneous transmission of trypanosomes and hemogregarians has been reported in fresh-water systems (Lainson, 1981). Neither case, however, described the effect or even the tissue distribution of these pathogens within the host leech species.

The only complete description of the infective mechanisms involved in the infestation of annelids by flagellates comes from the marine polychaete *Axiothella rubrocincta*. This

worm is prone to infection by the haplozoan *Haplozoon axiothellae*. Siebert (1973) identified the gut as the major site of infection. Here, cysts were found to contain the parasite in various stages of maturation. Usually, a single trophocyte is attached to the intestinal epithelium and is surrounded by several mitotic gonocytes and several sporocytes. Cellular attachment to the epithelium is achieved by a highly modified suction disc associated with a number of rigid spines or stylets concentrated around a stylet sac. Apparently, stylets are used to repeatedly pierce the membrane of the host's intestinal epithelium cells. Such cellular disruption provides a flow of host cytoplasm that may be absorbed by the suction disc and distributed, via a series sub-cellular vesicles, throughout the trypanosome. Whilst this method of nutrition is clearly harmful to the host, its precise effect has not been reported.

In contrast, Codreanu and Codreanu (1928) have provided conclusive evidence for mortality arising from euglenid infection in the freshwater oligochaete *Chaetogaster diastrophus*. A considerable percentage of *C. diastrophus* from natural populations were found to be infected with the euglenid *Astasia chaetogastis*. This parasite multiplies rapidly in the body cavity of the host, so that infection is always fatal within 8 to 13 days of the original challenge. The infective euglenid is also capable of surviving outside the host species, although only its parasitic form is capable of replication.

A similar pathogenicity has been reported for the interactions between the flagellate *Hexamitus tubifici* and polychaetes of the genus *Tubifex*. Infection of these worms leads to reduced activity, a loss of pigmentation and eventually death (Ryckeghem, 1928).

Agents: Gregarinia

Infection by gregarian sporozoans appears to be particularly common amongst both polychaetes and oligochaetes (Labbé and Racovitza, 1897; Dogiel, 1909; Reichenow, 1932; Tuzet and Ormières, 1962; Landers and Gunderson, 1986). Moreover, circumstantial evidence supports pathological effects for at least some of these infections. In an ultrastructural description of a new gregarian species *(Cygnicollum lankesteri)* from the polychaetes *Laetmonice hysterix* and *L. producta,* Desportes and Théodoridès (1986) paid particular attention to the attachment mechanism of the parasite. The apparatus consists of a conical structure with a basal rim that is apparently inserted into the epithelial cells of the host's intestine. An annular bundle of 6 to 12 tubes is situated in the basal rim and is associated with a cylinder giving rise to a complex of microtubules radiating to the trunk of the sporozoite. These structures, reminiscent of the feeding mechanism of flagellates (described above), may yield a pathological effect through saprophytic action.

Mechanical disruption of host tissue arising from gregarian infestation is not universal. Identification of free gregarians in the coelom of the maldanid polychaete *Axiothella rubrocincta* suggests that the potential exists for a far more commensal relationship with few deleterious effects (Landers and Gunderson, 1986). A limitation of harmful effects has also been demonstrated by Fowell (1936). This author identified a coccidian sporozoan that exclusively inhabits the nuclei of gut cells in its polychaete host, *Polydora flava*. Although the nuclei are damaged, there appears to be little pathological effect upon the host.

Instances in which sporozoan infection does not induce morbidity may also reflect the efficacy of host defense. Pixell-Goodrich (1916) has demonstrated that gregarian

trophozoites in the polychaete *Glycera siphomostoma* are readily encapsulated by the host's coelomic epithelial tissues. Encapsulation yields cysts that, when dislodged from the epithelium, contained masses of host phagocytes enclosing invading gregarians.

Agents: Microsporozoa

In addition to gregarians, microsporozoans are demonstrably parasitic in annelids. Spelling and Young (1986a) have rediscovered the microsporozoan *Nosema glossiphoniae* in the aquatic leech *Glossiphonia complanata*. Electron microscopical analysis revealed both meronts and mature spores of the protozoan within leech muscle cells (Fig. 2-2).

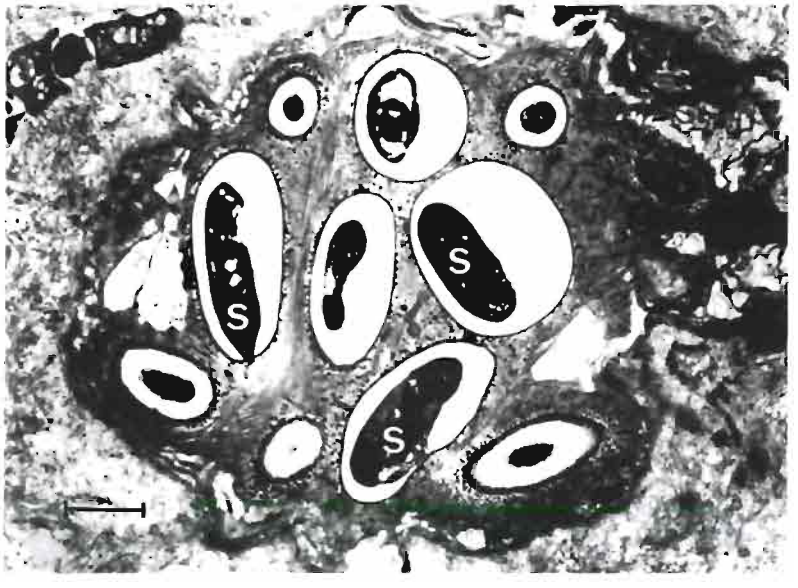

Fig. 2-2: Spores of the microsporozoan *Nosema glossiphoniae* in a muscle cell of the leech *Glossiphonia complanata*. Scale bar = 2 μm. (After Spelling and Young, 1986a.)

When infected, these muscle cells appeared as tubes with invaginated walls. Meronts were recognized in these structures as irregularly shaped cells of ca. 3 × 10 μm and bounded by a single plasma membrane. Spores were elongate (5 × 2 μm) with an internal plasmalemma and 2 outer spore coats. The outermost layer was thrown into long fingerlike projections.

Whilst the morphology of infection by these 2 stages of the sporozoan life cycle is again suggestive of significant host tissue trauma, the precise pathology of the infection was not investigated. Similarly, Mrazek (1899) reported a neuronal hypertrophy in the oligochaete genus *Lophius*, that is apparently derived from infection with the microsporozoan *Glugea lophii*, but the author did not describe the consequences of such infection.

In freshwater oligochaetes, however, pathological activity of microsporidians has been established. Naidu (1959) found that coelomic infection of 4 different oligochaete species with only a few sporocytes from the microsporidian *Mrazekia caudata* is invariably fatal.

Agents: Myxosporozoa

Studies of annelid infestation by myxosporozoans are largely descriptive. Perhaps the most interesting case deals with the taxonomy of the infective organism. In this situation, however, taxonomic considerations are of considerable consequence to the parasitic life cycle (Wolf and Markiw, 1984; Corliss, 1985). The myxosporozoan *Myxosoma cerebralis* has been known for some time to be responsible for 'whirling disease' in European brown trout. However, it has proven impossible to infect fish with its spores. Similarly, the actinosporean *Triactinomyxon gyrosalmo* is commonly found in tubificid oligochaetes, although its capacity to infect worms has never been demonstrated, and initial stages of this sporozoan's life cycle are not evident within annelid hosts. It is now apparent that this confusing situation reflects a taxonomic inconsistency arising from the substantial morphological changes that sporozoans may undergo in different hosts. Recent reports suggest that *M. cerebralis* and *T. gyrosalmo* represent different stages in the life cycle of a single species (Fig. 2-3). Hence, tubificid worms act as intermediate hosts that are infected by *M. cerebralis*. The spores give rise to larvae in the mucosal lining of the gut. These larvae then assume the appearance of triactinomyxids before being passed to the secondary fish host.

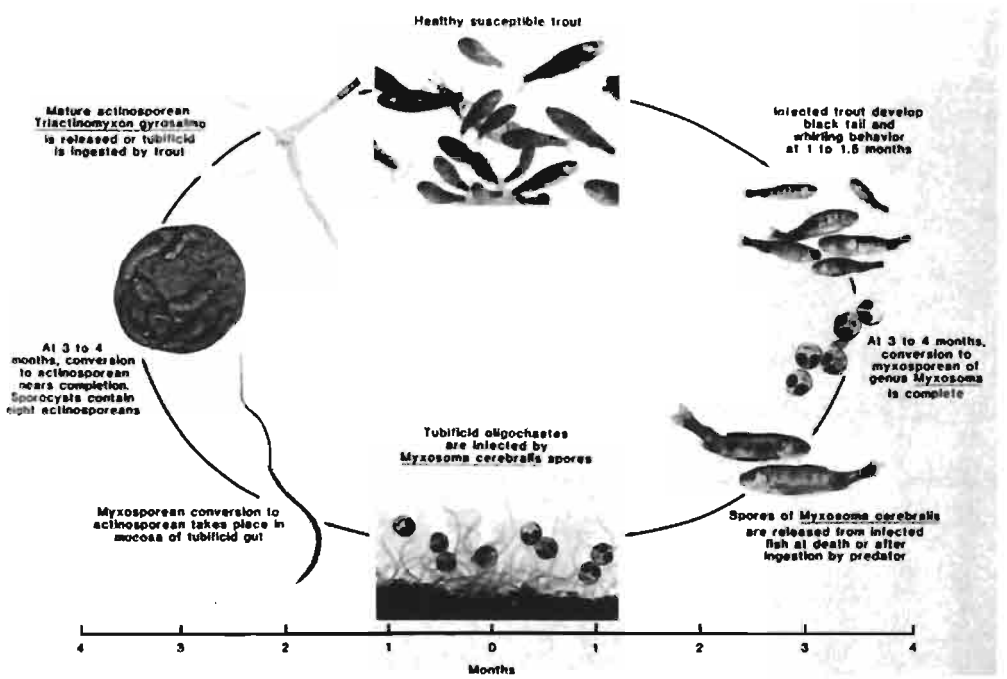

Fig. 2-3: Biphasic life cycle of a myxozoan infecting tubificid oligochaetes and trout at different stages. (After Wolf and Markiw, 1984.)

Despite these details on the life history of *Myxosoma cerebralis*, the effect of the parasite on its intermediate annelid host has not been investigated. Pathological effects have, however, been described in the relationship between the actinomyxid sporozoan *Triactinomyxon naidanum* and the freshwater oligochaete *Nais communis* (Naidu, 1956).

The parasite is apparently host specific and causes some degree of morbidity. It was reported that infested worms are less active than uninfested individuals and lacked the zones of fission that are present in normal individuals.

Agents: Ciliophora

Reports of ciliates infesting annelids are relatively scarce. However, Kozloff (1961, 1965) has identified 3 new species of ciliates from the sabellid polychaetes *Sabellaria cementarium, Eudistylia polymorpha* and *Schizobranchia insignis.* This represented a novel host group for ancistrocomids which had previously been found in gastropods and pelecypods. Ciliate parasites have also been recovered from aquatic oligochaetes. Naidu (1961) demonstrated that nearly all individuals from the freshwater oligochaete *Aelosoma travancorense* are infected in their native environment by the astomatous ciliate *Radiophryoides putyoraci.*

The most comprehensive pathological study of ciliate infestation comes from Stout (1954). He found that enchytraeid worms were susceptible to infestation by the ciliate *Tetrahymena rostrata.* The parasite apparently enters the worms via degenerate setal follicles or through sites of accidental injury. The loss of setae providing such sites for parasitic penetration is apparently a common event. Setal loss leaves only a thin cuticular barrier to penetration that is often weakened by the extrusion of damaged body tissue. Ciliates are actively attracted to this site by histolysis or peptone release.

After penetrating the epidermis of their enchytraeid host, *Tetrahymena rostrata* generally infests body wall tissue. The ciliate does not appear to associate with gut tissue, unlike many other pathogenic microorganisms. In the body wall, ciliates multiply rapidly. At 20 to 25 °C it was determined that cell division occurs every 2 to 3 h. Such division usually takes place in the absence of encystment, although rare exceptions have been found to form small, rapidly dividing cysts. The speed of ciliate proliferation is such that mature worms can eventually enclose more than 200 parasites within a small region. In such cases, the parasite totally destroys the host tissue within 24 to 36 h. After death of the host annelid, the incumbent ciliates generally remain free-swimming until leaving the shell of the host body. The parasite does, however, have the capacity to encyst in the absence of food or in areas of overcrowding.

DISEASES CAUSED BY METAZOANS

Agents: Cestoda

Reports by Calentine (1962, 1965a, b) have detailed the parasitism of cryophyllaeid cestodes on oligochaetes of the genera *Limnodrilus* and *Tubifex. Limnodrilus hoffmeisteri* from the Iowa River, USA, were found to incubate sexually mature specimens of the cestode *Archigetes iowensis.* These annelids are also prone to infestation by another cestode, *Hunterella nodulosa.* Oncospheres of this parasitic species enter the annelid's body via the gut. They penetrate the body cavity through the gut wall and usually migrate to the anterior of the host.

Kashin (1984) has shown that similar penetration by other species of cestode may be facilitated by highly adapted penetration glands. These glands incorporate specialized

ducts that secrete substances with both adhesive and lytic activity. The ducts associate with embryonic hook mechanisms and are presumably essential to the penetration of host tissue during the migration period of larval development. Smaller ducts also secrete substances that are putatively protective against host macrophagial activity.

After penetrating the body cavity of *Limnodrilus hoffmeisteri*, larval *Hunterella nodulosa* develop to the procercoidal stage within 45 days. Procercoidia generally attain only rudimentary gonads and appear to have only a limited effect upon host fitness. Up to 3 fully developed procercoidia have been found to develop within a single host without affecting its capacity to attain sexual maturity.

Pathogenic effects of cestode infestation are far more pronounced in associations with worms of the genus *Tubifex*. In experimental situations, both *Tubifex tubifex* and *T. templetoni* are readily infested by the cestodes *Biacetabulum macrophalum* and *B. frequens*. Infestation occurs after the annelids have ingested cestode eggs. Oncospheres derived from the ingested material rapidly penetrate the gut wall, migrate anteriorly in the body cavity and develop to the procercoidal stage within 50 to 60 days. Young procercoids are generally restricted to seminal vesicles, but more mature individuals may break through the seminal wall into the coelom. In naturally infested hosts, usually only 1 procercoid is present in each worm, although far greater infestations are detected in experimentally infested individuals. This level of infestation leads to mortality amongst experimentally infested hosts within 120 days. However, only immature worms are prone to natural infestation.

A similar pathology is demonstrable for infestations involving the cestode *Glaridacri catostomi*. Tubificid worms experimentally infested with this parasite are short-lived compared to uninfested conspecifics. Procercoidia of *G. catostomi* often damage intersegmental septa during maturation and may also rupture the body wall leading to mortality of the host. Again, juvenile hosts are the target for natural infestations. Infested immature annelids do not become sexually mature.

Agents: Trematoda

Annelids have only infrequently been identified as intermediate hosts for trematode parasites, although a few reports of such infestations are available (Shaw, 1933; Iles, 1960; Oglesby, 1961). In a recent example, Brown and Prezant (1986) recovered a digenetic trematode from the deposit-feeding polychaete *Scoloplos fragilis*. Metacercarian cysts of the trematode were initially identified in live hosts, even though cysts were often mistaken for host ova (Fig. 2-4). Most *S. fragilis* collected over a 2-year period exhibited externally evident infestations, with an average of 68 metacercariae found in each host individual. Metacercarian cysts were most prevalent in the coelom and musculature at the junction of thorax and abdomen.

In experimental infestation trials, trematodes proved to be capable of penetrating *Scoloplos fragilis* within 5 min of contact. All areas of host epidermis were amenable to penetration except the gills. Encystment of cercariae was usually completed within 12 h.

Trematode parasites have also been identified in leeches. Riggs and Ulmer (1983) recovered mature trematodes belonging to 5 species from leeches of the genus *Haemopis*. The trematodes were encapsulated in the intestinal caecae of the hosts, suggesting that active host defense limited their pathogenic effect (Fig. 2-5). Similarly, Spelling and Young

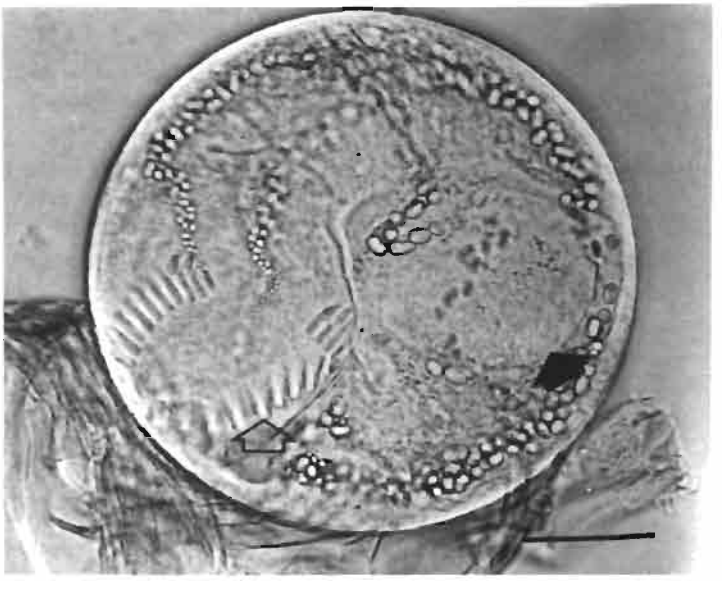

Fig. 2-4: Live encysted metacercaria isolated from the deposit-feeding polychaete *Scoloplos fragilis*. Scale bar = 45 μm. (After Brown and Prezant, 1986.)

(1986b) suggested that the infestation of 3 lake-dwelling leech species by the trematode *Cyathocotyle opaca* did not substantially alter the fitness of host populations. In 2 of the leech species, the frequency of infestation was extremely low, whilst higher rates of infestation in the third host species did not correlate with increased mortality. Fecundity was found to be unaffected by infestation with the trematode. Observations on growth, attainment of sexual maturity, cocoon production and survival revealed little difference between infested and uninfested leeches.

Agents: Nematoda

There are few reports regarding the infestation of polychaete and hirudinean annelids by nematodes. Recently, Poinar (1984) identified a new species of rabditid nematode from the leech *Dina anoculata*. However, the mechanism of infestation, and indeed the localization of parasites within the host, were not described. Similarly, larval nematodes of either the genus *Contracacaecum* or *Ascaris*, have been found in numerous polychaete species, although their pathological effect remains unclear (Norris and Overstreet, 1976).

More detail is available for the relations between nematodes and oligochaetes. Oligochaete worms often act as hosts for mermithidian nematodes. Indeed, Poinar (1976, 1978) suggests that these nematodes can infest a variety of freshwater oligochaetes from at least 3 different families. Most often, an infestive juvenile emerges from an egg fertilized and laid in the external environment by a free-living adult. It is this larval stage that usually penetrates the host annelid, although infestation resulting from the ingestion of fertilized eggs has also been demonstrated (Smith, 1985). Larvae develop continually until their emergence from the infested individual. Emergence of the nematodes is usually fatal to the host oligochaete.

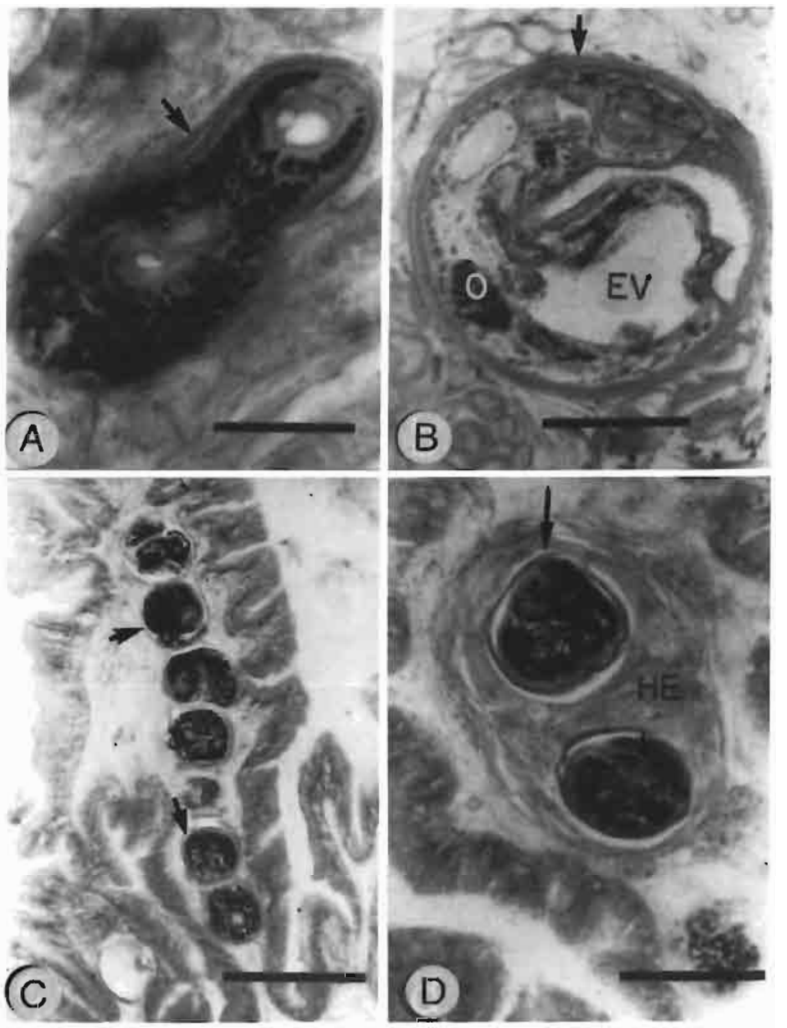

Fig. 2-5: Parenteric helminths of haemopid leeches. A: Unidentified, unencapsulated metacercaria; scale bar = 38 μm. B: Progenetic metacercaria of *Hirudicolotrema richardsoni* in body wall musculature of *Haemopis plumbea;* scale bar = 100 μm. C: Seven unidentified metacercariae arranged along the surface of a rugal blood vessel (not visible in this section); scale bar = 220 μm. D: Host encapsulation response to 2 unidentified metacercariae in a ruga of *H. plumbea;* scale bar = 100 μm. (EV) excretory vesicle; (HE) host encapsulation response; (O) ovary; (unlabeled arrows) metacercarial cyst wall. (After Riggs and Ulmer, 1983.)

This infestive process most likely reflects the utilization of annelids as the sole or paratenic host. However, freshwater oligochaetes have also been identified as intermediate vectors for nematodes that are of serious veterinary or medical import. For instance, members of the genus *Eustrongylides,* which are lethal parasites of piscivorous birds, employ limnodrillid oligochaetes as their primary intermediate hosts (Lichtenfels and Stroup, 1985). Whilst these parasites apparently kill a number of their annelid hosts, their major form of transmission requires consumption of infested Limnodrillids by a freshwater

fish. The fish subsequently acts as a secondary intermediate host prior to infestation of piscivorous birds (Weisberg and co-authors, 1986).

A similar, but perhaps more intriguing, life cycle is that of the giant kidney worm *Dioctophyma renale*. This nematode is, in its adult stage, a serious parasite of mammals including humans. Its larval stages, however, infest branchiobdellid oligochaetes (Woodhead, 1945, 1950). Eggs of *D. renale* are ingested by the branchiobdellid *Cambarincola chirocephala*. *C. chirocephala* is itself an ectoparasite of a freshwater crayfish. Nematode eggs hatch in the gut of *C. chirocephala* and the larvae migrate through the gut wall to the visceral tissue of the worm. The larvae then encyst (up to 6 larvae annelid^{-1}) until their branchiobdellid vector, attached to its own crayfish host, is eaten by a catfish. The catfish then assumes the nematode infection which may in turn be passed on to a tertiary mammalian host. Again, despite this detail regarding life cycle, pathological effects of nematode infestation on annelid hosts that are not consumed by catfish remains unclear.

Agents: Crustacea

A number of taxonomic reports have described crustacean associations with marine annelids. For instance, Ho (1984) has named a new genus and species of copepod that is parasitic on 3 species of polychaete from the genus *Spiophanes*. A variety of similar parasitic associations have been documented by Gotto (1979). Thus, the copepod *Aphanodomus terebellum* has been shown to feed on the hemolymph of the polychaete *Thelepus cincinnatus*. This parasite adheres to the wall of blood vessels surrounding the host's gut. It may make its way to this site via the vascular system after penetrating the epithelium of the gills and entering the branchial circulation.

Another copepod, *Melinnacheres steenstrupi,* also gains nutrition from polychaete blood. In this case though, the crustacean remains ectoparasitic. It attaches directly to the gill lamellae of terebellids using modified mandibles. The parasite then sucks blood from vessels within the gills. An alternative source of nutrition is utilized by *Phylodicola petiti*. This crustacean feeds on the coelomic fluid of phylodocid polychaetes. It gains access to the coelom by inserting 2 long rhizoids associated with the jaws through the host's epidermis and into its body cavity. Food is withdrawn either by diffusion or osmosis.

Clearly, these forms of parasitism would be of considerable detriment to the host, although little information is available regarding their exact effects. However, it is known that, as individuals of *Aphanodomus terebella* grow within the circulatory system of their polychaete host, they displace the host's intestine and severely damage muscle tissue surrounding the gut.

TUMORS AND OTHER ABNORMALITIES

There are a number of studies that indicate the existence of neoplastic growth in annelids. However, most cases relate to tumors in terrestrial species and are insufficiently detailed to determine whether true neoplastic growth is involved. The most notable example of a purportedly neoplastic condition in a marine species has been documented by Thomas (1930a, b, 1931). The author described white globular tumor-like growths in various tissues of the polychaete *Nereis diversicolor*. The 'granulomata' were found to be associated with degenerating host oocytes and setal bristles. Moreover, it was demon-

strated that such tumorous growth could be transferred between worms by inoculation with *Bacillus tumefasciens,* a bacterium associated with granulomatous cells surrounding degenerating oocytes. The inoculated bacteria were apparently transformed after injection, yielding granules that invaded tumor cells and appeared to propagate tumor growth.

The nature of this apparent neoplasia has been further resolved by Dales (1983) who suggested that tumor formation was both a natural and transitory response to unwanted tissue. Dales noted that large numbers of oocytes normally remain unspawned by *Nereis diversicolor,* and so an efficient mechanism of oocyte clearance, possibly involving 'tumor' formation, is required. It was suggested that much of the cellular activity described previously as neoplastic may represent normal physiological alterations in preparation for spawning. *N. diversicolor* undergoes suppressed metamorphosis in which muscle tissue is destroyed at the onset of sexual maturity. The associated infiltration of phagocytes and the transportation of residual granulatoma through the body wall may not be readily distinguishable from true neoplastic growth. The neoplastic basis of tumor growth in *N. diversicolor* is also brought into question by both the transient nature of the condition and the apparent lack of active cell division within tumors. Tumorous worms collected from their native environment underwent rapid regression of the granulomata, so that gross tumor morphology eventually disappeared. It was apparent that such improvement in the condition was due only to time and not to changed milieu.

Indeed, after inspecting Smithsonian collections, Dales (1983) finally concluded that myoblastomas of the terrestrial annelid *Lumbricus terrestris* (Hancock, 1961), represent the only true annelid neoplasm presently known. Neoplasia was induced by painting earthworms with methylchloranthrene-acetone or through X-irradiation. Tumors resulting from irradiation resemble the myoblastomas of vertebrates and usually induce the formation of giant cells with as many as 40 nuclei. Such obvious cellular dysfunction is characteristic of true neoplastic transformation. Accordingly, tissue aggregations associated with tumors were clearly distinct from aggregates of normal amoebocytes.

Descriptions of developmental abnormalities in annelids are also largely restricted to terrestrial species. However, there are intermittent reports of malformed reproductive organs and segmental structures in both polychaetes and leeches (Gibson, 1887; Buchanan, 1893; Green, 1923).

Similarly, identification of degenerative syndromes in aquatic species is rare. Dev (1965) has described a nephridial atrophy in the Indian leech *Hirudinaria granulosa.* Approximately 3 % of a natural population suffered from this condition. Degenerative activity was characterized by a disorganized nephridial structure and a reduction in the size of nephridial cells. Atrophied nephridia also lack the characteristic symbiotic bacterial content typical of normal leeches and exhibit reduced alkaline phosphatase activity. No causative agent has been ascribed to this condition.

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3. DISEASES OF CRUSTACEA

In the marine environment the class Crustacea is represented by a myriad of forms that are extensively distributed and successfully established in a diverse array of habitats. The majority of the 26,000 species of crustaceans (Barnes, 1969) occur within marine ecosystems. Many occupy basic positions in aquatic food chains. Others are familiar, important commercial fishery species, and a few are being extensively aquacultured to meet the ever expanding human demand for high-quality protein from the sea.

3.1 DISEASES CAUSED BY MICROORGANISMS

J. A. Brock and D. V. Lightner

The diseases of marine crustaceans caused by microbial pathogens are an important, although relatively small, area of study within the field of invertebrate pathology. With a very few exceptions, clinical documentation and research into crustacean disease episodes has not received a similar level of time and resource allocation as has been directed toward diseases of terrestrial insects. Nevertheless, over the years a fair body of information has been published. Moreover, greater attention is now being given to this area due to the economic impact of disease in commercial aquaculture. Thus, the next several decades should reveal a tremendous expansion of knowledge on the details of diseases and pathological processes in this important animal group.

Diagnostics for crustacean diseases rely heavily upon morphological pathology. Information on marine crustacean microbial diseases is generally most completely documented for morphologic pathology, both at the light and electron microscopic levels. Excellent books on normal microscopic anatomy for the blue crab (Johnson, 1980) and penaeid shrimp (Bell and Lightner, 1988) are available. In recent years an increasing interest has been shown for the development and application of routine molecular and immunologic diagnostic procedures. This evolution toward use of rapid, precise molecular methods for crustacean microbial pathogen determination will open the way for increased understanding, particularly from an epidemiological perspective, of many crustacean microbial diseases.

Serious infectious disease problems are well documented in captive-wild or aquacultured crustaceans. Viral, rickettsial, bacterial and fungal pathogens cause frequent and notable population diseases. For example, gaffkemia of lobsters or IHHN virus disease of marine shrimp are prime examples where crowding, declining environmental conditions, commonly found in captive rearing situations, and human manipulation ignite the disease process and epidemic animal losses follow.

Interestingly, and as is known for insects (Odier, 1975), infection by multiple pathogens is rather commonly encountered in marine crustaceans. Multiple virus infection of hosts, organs and, in rare cases, within the same cells is reported for marine crabs (Bonami, 1976; Johnson, 1983) and marine shrimp (Nash M. B. and co-authors, 1988;

Lightner and Redman, in press). Multiple infection by viruses with one or more other organisms including rickettsial, bacterial, fungal and protozoan pathogens is also known in cultured marine shrimp (Anderson and co-authors, 1987; Tsing and Bonami, 1987). Although interesting in terms of pathogenesis and crustacean defenses, these multiple infections also complicate precise diagnosis and management of diseases in cultured crustacean stocks.

Pollution is frequently cited as a significant factor predisposing marine crustaceans to intensification of attack from microbial pathogens. Experimentally, exposure to certain anthropogenic and toxic metals has been shown to increase baculovirus activity at the cell and organ levels (Couch, 1974a, 1976; Couch and Courtney, 1977; Owens and Hall-Mendelin, 1988). However, hard evidence is lacking that demonstrates pollutants induce pathogen-caused epidemics in wild crustacean populations under natural conditions.

Sindermann (1979, p. 1) stated: "Epizootics of infectious diseases and resultant mortalities has been a matter of record in natural populations of Crustacea for more than a century". A close examination of the literature reveals only a handful of examples for the marine environment during this period. There is some early literature on mass mortalities of copepods and related forms in the wild caused by fungal attack (Vallin, 1951; Höhnk and Vallin, 1953) and a chlamydia-like pathogen associated with natural mortalities of the dungeness crab (Sparks and co-authors, 1985), but more recent episodes of these occurrences have either not taken place or remain unpublished. This seems odd and leaves the impression that the epidemics reported may have had a more complex etiology than indicated by a simple, single pathogen-host relationship. Moreover, there are no cases that demonstrate microbial diseases, if fact, play a significant role in the long-term population ecology of a marine crustacean host species in the natural environment.

In the natural environment pathologically significant infections of marine crustaceans are documented. An example of a disease in the natural environment that is well described morphologically at the organ and cell level is the 'herpes-like' virus infection of king crabs (Sparks and Morado, 1985, 1986). However, the impact of this disease on natural crab populations and the commercial crab fisheries remains unclear. Black mat disease, a systemic mycosis of tanner crabs (Sparks and Hibbits, 1979; Sparks, 1982a), is well documented to affect large numbers of crabs in certain areas of the commercial fishery in Alaska. Yet, the economic impact of black mat disease on the tanner crab fishery needs to be documented. Thus, examples of catastrophic diseases caused by microbial agents which affect natural populations of marine crustaceans are unusual. And no similar examples in marine decapods are documented that compare to the notable crayfish plague (krebspest), which is a disease of the freshwater crustacean *Astacus astacus* caused by the fungus *Aphanomyces astaci*. It may be very important to the understanding of why this fungus resulted in such a significant disease problem in that the pathogen *A. astaci* was apparently an exotic introduced into Europe.

Concern regarding pathogen transfer with movement of marine invertebrates is not a new topic (Sindermann, 1979, 1986, 1988e; Brock and co-authors, 1983; Lightner, 1983, 1985; Garland, 1988). The record clearly indicates that viral and other obligate pathogens are present in a variety of marine crustaceans. The evidence also shows that movement of crustaceans results in pathogen transfer as well. Notable effort has been put forth to point out this problem and to provide a management strategy for reduction of this risk (Sindermann, 1986, 1988e). However, it remains to be seen if implementation of these

procedures will be undertaken. Increased pressure to move marine crustaceans between widely divergent geographic locations is only going to increase with the growth of crustacean aquaculture enterprises.

Agents: Viruses

Viruses are the most diverse and numerous of the microbial agents described from marine Crustacea. The first viral infection documented from an aquatic invertebrate was reported by Vago (1966) in the marine brachyuran crab *Macropipus depurator*. Indeed, prior to 1966 invertebrate viruses were only known from terrestrial insects and mites (Johnson, 1984a). Presently, over 30 viruses have been reported from marine crustaceans. These viruses are nearly exclusively known from marine crabs and shrimps. The exceptions are 2 viruses described from the marine entoniscid isopod *Portunion conformis*. One of these agents, a picorna-like virus was also found infecting tissues of the isopod's crab host, *Hemigrapsus oregonensis*.

Viruses in the following families have been reported from marine crustaceans: Parvoviridae, Herpesviridae, Baculoviridae, Picornaviridae, Reoviridae, Birnaviridae, Rhabdoviridae and Bunyaviridae. In addition, agents have been described that, based on the available information, cannot be assigned to family. The majority of viruses identified from marine crustaceans have been tentatively placed into recognized virus families by the researchers that discovered the agent. In some cases, agents have been reassigned by a subsequent investigator. For many of the crustacean viruses, family assignment has been based on insufficient basic information about the agent, and while morphological and developmental characteristics are usually documented — agents are most often studied *in situ* by electron microscopy of host tissues — other biophysical data and biochemical features often are undetermined. Modern molecular virological methods have only sporadically been applied to the study of marine crustacean viruses. Reasons for this are many and include the absence (although recent advancements suggest this may change soon — see Chen and co-authors, 1986; Chen and Kou, 1989; Luedeman and Lightner, 1989) of cell culture systems with which to grow agents *in vitro*. Therefore, for the majority of the marine crustacean viruses, family assignments are tentative because fundamental data necessary for classification are lacking. This is reflected in the fact that acceptance of these designations by the International Committee on Taxonomy of Viruses (ICTV) has been accomplished only for a single agent, *Baculovirus penaei* Couch (Matthews, 1982; Brown, 1986). In this review, no attempt is made to reclassify viruses, and family designations for viral agents conform to previous assignments given by the discoverer or those suggested by Johnson (1984a, 1988a, 1988c) and Johnson and Lightner (1988). It is clear, however, that marine crustacean virus taxonomy deserves an intensive research effort.

A number of viruses found in crustacean tissues have not been associated with a disease syndrome. For some of these agents, the host's critical life stages susceptible to disease may not have been examined (Johnson, 1984a). Other viruses may simply be well adapted to their host and the infection does not lead to disease. Depth and scope of information available about virus diseases of marine crustaceans varies considerably; details are often sparse. The most complete information is usually available for organ, cell and ultrastructure pathology and disease signs associated with virus infection. Little is known about the population effects of crustacean viral diseases in nature.

Viral diseases of marine crustaceans are known primarily from observations gathered at the cell, organ and organism level for animals in captivity or confinement rearing. And while several examples of virus infection-associated cell damage have been reported from crustacean hosts examined from the wild (Couch, 1974b; Sparks and Morado, 1986); corroborating data that demonstrate these infections result in morbidity and mortality under natural conditions are lacking. Thus, it is not clear that marine crustacean viruses cause diseases at the population level under natural conditions, and quantified data are lacking that directly implicate viruses as significant biotic agents in the population ecology of marine crustaceans.

On the other hand, for a number of crustacean viruses, disease impacts are well documented for captive crab and cultured marine shrimp. In marine shrimp aquaculture, viruses constitute the economically most significant biotic diseases of these animals. Management of viral diseases is particularly important to the success of semi-to-intensive shrimp farming. The lack of accomplishment in this area has contributed to farm failures. Since shrimp farming currently relies primarily on wild-caught stock, shrimp viral pathogens are repeatedly introduced into culture systems. Additionally, these pathogens continue to be disseminated over wide geographical areas with movement of shrimp stocks for aquaculture purposes (Brock and co-authors, 1983; Lightner and co-authors, 1983d; Colorni and co-authors, 1987; Lightner and Redman, in press). Clearly, viroses are of major importance to the developing shrimp farming industries worldwide.

There is an expanding literature on viruses of Crustacea; excellent general reviews of the subject include: Bonami (1976); Johnson, P. T. (1978, 1983, 1984a); Couch (1981); Sparks (1985); Mari and Bonami (1986). Viroses of marine shrimp have been reviewed specifically by Lightner (1977) his Chapter 3.1.1, 1983, 1985, 1988, his Chapters 3.1.1 to 6) and Bonami (1987). Johnson (1984a, 1988c) and Johnson and Lightner (1988) are recommended for readers interested in the subject of taxonomy of marine crustacean viruses.

The present review of marine crustacean viroses is organizationally based on virus location within the cell (nucleus or cytoplasm), the agent, and the host species. Examined first are the nuclear viruses which include the baculoviruses; other presently unclassified enveloped rod-shaped nuclear viruses; the enveloped icosahedral nuclear viruses; a herpes-like virus; and the nonenveloped isometric parvo-like viruses.

Baculoviruses are double-stranded DNA, rod-shaped, enveloped viruses known widely from certain groups of insects, mites, crabs and penaeid shrimps. The density of insect baculovirus nucleocapsids in CsCl is 1.47 g cm^{-3}, and 1.18 to 1.25 g cm^{-3} for virions (Matthews, 1982). Baculoviruses are ether and heat labile. The CsCl density characteristics for shrimp and crab baculoviruses are undocumented. Couch (1974a) reported the first baculovirus infection of a crustacean, the pink shrimp *Penaeus duorarum*. Historically, 10 baculoviruses have been reported from marine crustacean hosts. Recently, 3 of these agents — infecting the nuclei of hemocytes and hematopoietic tissues of 3 brachyuran portunid crabs *Callinectes sapidus, Carcinus maenas,* and *Carcinus mediterraneus* — were shown to differ from baculoviruses in that the nucleocapsid is not a true cylinder; therefore, they may not belong in the Baculoviridae after all (Johnson, 1988c). The remaining 7 agents, considered baculoviruses, infect entodermally derived cells, mainly hepatopancreas epithelium of their marine crab or shrimp hosts. Two (BP and MBV) of the 3 baculoviruses described from shrimp are occluded forms. The third shrimp

baculovirus (BMNV) and the 4 baculoviruses (Baculo-A, Baculo-PP, Tau and Tau 2) known from marine crabs, are nonoccluded. An additional nonoccluded baculovirus infection of cultured *Penaeus monodon* is also suspected (Fig. 3-3, c). The occluded shrimp baculoviruses are assigned to the Type-A baculoviruses (Couch, 1974b; Lightner and Redman, 1981; Lightner and co-authors, 1983c; Lester and co-authors, 1987); however, results from recent comparative studies (Johnson and Lightner, 1988) on details of morphological and some developmental characteristics of the 7 crustacean baculoviruses (BP, MBV, BMNV, Baculo-A, Baculo-PP, Tau, Tau 2), indicate these baculoviruses show the greatest affinity to the nonoccluded virus of *Oryctes* that is the type for the proposed Subgroup C of the Baculoviridae. Thus, the occluded and nonoccluded crustacean baculoviruses may all belong to the proposed Subgroup C (Johnson and Lightner, 1988). *Baculovirus penaei* Couch, which was extensively characterized morphologically (Couch, 1974b), biophysically and biochemically (Summers, 1977), was the first — and the only to date — crustacean virus accepted by the ICTV (Matthews, 1982).

Baculovirus penaei Couch was first discovered in wild-caught pink shrimp *Penaeus duorarum*, taken originally from Apalachee Bay near Cedar Key, Florida (USA) and experimentally exposed to 3 to 5 $\mu g\ l^{-1}$, of the polychlorinated biphenyl (PCB) Arochlor 1254 (Couch, 1974a). Fresh squash preparations of hepatopancreas from these shrimp revealed conspicuous pyramidal bodies (Fig. 3-1, a), that on electron microscopy examination were associated with a rod-shaped, enveloped nucleocapsids (Couch, 1974a, b). Subsequent studies have indicated *B. penaei* is widespread in American penaeids (Lightner, 1983; Lightner and Redman, in press).

The average size of the *Baculovirus penaei* (BP) nucleocapsid is 270 × 50 nm in length and diameter for BP from Gulf of Mexico and the Eastern Pacific Coast of Central to South America (Couch, 1974a; Johnson and Lightner, 1988; Lightner and Redman, in press). Nucleocapsids from *Penaeus marginatus* in Hawaii are smaller with an average size of 216 × 33 nm (Brock and co-authors, 1986c). The BP envelope is 8.5 nm thick (Couch, 1974a, b; Johnson and Lightner, 1988) and loosely fit to the nucleocapsid except in occluded virions or virions with unilateral envelop expanions. On the average virions are 75 nm in diameter (from Gulf and Pacific Coast penaeids) or 56 nm (from the Hawaiian penaeid). There is no significant difference in size between the occluded and nonoccluded virus (Johnson and Lightner, 1988; Overstreet and co-authors, 1988). *B. penaei* has double-stranded, cyclic DNA with a molecular weight of $75 \times 10^6 +/- 2 \times 10^6$ daltons (Summers, 1977). The virion stability to heat, acid and ether has not been reported. Clerx and Lightner (1985) showed partially purified but badly degraded *B. penaei* nucleocapsids (from *P. marginatus*) banded at 1.17 to 1.18 gm cm^{-3} in a 20 to 50 % sucrose gradient.

Baculovirus penaei (BP) is commonly found to infect the hepatopancreas of juvenile to adult pink and brown shrimp *Penaeus aztecus* in nature (Couch, 1974b). In wild populations of pink shrimp collected from Apalachee Bay, Florida (USA), Couch (1976) reported patent infections (polyhedral occlusion bodies present) averaged 20 % and ranged from 0 to 80 % in samples of over 2,000 shrimp studied over a 4 year period.

In addition to pink and brown shrimp, other penaeids found infected — wild, captive-wild or cultured — include: *Penaeus vannamei, P. stylirostris, P. setiferus* (Lightner, 1985), *P. schmitti, P. penicillatus, P. brasiliensis, P. paulensis* (Lightner and Redman, in press), *P. subtilis* (Bueno and co-authors, 1989; Lightner and Redman, in press), and *P. marginatus* (Brock and co-authors, 1986c).

Available information indicates the host range of BP in decapod crustaceans is limited to species of penaeid shrimp. In the only reported evaluation of alternate host species for *Baculovirus penaei*, grass shrimp *Paleomonetes* spp., blue crabs *Callinectes sapidus*, stone crabs *Menippe mercenaria*, as well as mud crabs *Panopeus* sp. and *Neopanope* sp. — collected from areas within the natural range of *B. penaei* infected penaeids — were examined and did not have patent BP infection (Couch and co-authors, 1975).

Geographically, BP is widespread in cultured and wild penaeids in the Americas. The known distribution of BP is in the 3 separate regions: Hawaii, the Eastern Pacific Coast and the Gulf of Mexico. In Hawaii, BP is known only from wild *Penaeus marginatus* collected from an inshore area off the south coast of Oahu, Hawaii (Brock and co-authors, 1986c). On the Pacific Coast, BP ranges from northern reaches of the Sea of Cortez south along the coast of Central America, Ecuador and Peru. BP occurs throughout the Gulf of Mexico and Caribbean ranging from Florida, along the Gulf Coast, Central America and south to the State of Bahia in central Brazil (Lightner and Redman, in press). BP has not been recorded from wild, cultured or imported penaeids outside of the Americas (Lightner and Redman, in press).

Biotypes of *Baculovirus penaei* may occur within its range and particularly between the geographical areas separated by physical barriers. Brock and co-authors (1986c) reported *B. penaei* nucleocapsids from the Hawaiian penaeid *Penaeus marginatus* to be significantly smaller in length and diameter than BP infecting penaeids in the Gulf of Mexico (Couch, 1974b). Also, BP studied from *P. vannamei* on the Eastern Pacific Coast are somewhat larger in size than BP from the Gulf (Lightner and co-authors, 1985). Further study on this problem is needed. Determination of the antigenic relatedness of BP between and within these areas is suggested.

Baculovirus penaei from infected hepatopancreas tissues of broodstock, juvenile and larval (bioassay trials) *Penaeus vannamei* was experimentally transmitted by oral exposure to third substage protozoea (P_3's), mysis (M) and post larval (PL) stages of *P. vannamei* (Overstreet and co-authors, 1988) and juvenile and subadult shrimp (Leblanc and Overstreet, in press). In studies with shrimp larvae the investigators fed BP infected tissue to rotifers or *Artemia* subsequently preyed upon by shrimp larvae and post larvae. These studies, using specific BP-free test shrimp, confirmed horizontal transmission of the pathogen; further, that as shrimp age administered BP virus has less impact on shrimp survival, and in older shrimp patent infections may not develop. BP mortality in experimental trials with larval *P. vannamei* (Overstreet and co-authors, 1988) reached 100 %, but decreased to sporadic in older, juvenile-to-adult shrimp (Couch, 1976; Overstreet and co-authors, 1988; Leblanc and Overstreet, in press). Usually, in older shrimp (Leblanc and Overstreet, in press), less than 1 % of hepatopancreas cells were patently infected, but exceptions did occur where 80 % or more of the hepatopancreas cells had cytopathologic changes indicating BP infection.

Baculovirus penaei is found as a primary determinant of acute epidemics in hatchery-reared populations of larval and post larval *Penaeus aztecus* (Couch, 1978, 1981), *P. vannamei* (Lightner, 1983, 1988 his Chapter 3.1.2) and *P. stylirostris* (Lightner and Redman, 1989); captive-wild *P. duorarum* (Couch, 1974a, b; Couch and co-authors, 1975) and post larval *P. marginatus* (Brock, unpubl.). However, BP infection of groups of cultured larval-post-larval *P. stylirostris* may occur with negligible mortality (Lightner and Redman, 1989). Thus, presence of virus infection in shrimp populations does not automat-

ically result in an epidemic disease. Juvenile through adult populations of farmed shrimp, if cultured under stressful conditions, are reported to undergo disease with a subacute-to-chronic course (Lightner, 1988 his Chapter 3.1.2). However, BP virus is endemic in many pond-cultured juvenile-to-subadult *P. vannamei* populations with little recognized impact on health and productivity of these shrimp. Thus, BP infection is present, but apparently often remains inapparent and subclinical. Couch (1974a, b, 1978, 1981) reported mortality of captive-wild juvenile-through-adult *P. duorarum* and *P aztecus,* but also noted that a quantitative relationship between hepatopancreas cell pathology and shrimp mortality was often not evident in the material studied. Lightner and Redman (1989) found, by direct microscopic examination, that BP incidence in hatchery-reared groups of post larval *P. stylirostris* was 80 to 100 %, but declined to undetectable in these shrimp as 45 day old juveniles.

Baculovirus penaei appears to be well adapted to juvenile-through-adult life stages of shrimp under natural conditions. BP is not known to be a significant disease determinant in wild shrimp populations. Although *B. penaei* has been recognized in several commercially important species of penaeid shrimp for the past 15 years, a disease syndrome caused by this virus with direct impacts on survival of shrimp in shrimp fisheries is not documented. Couch (1974a, b), Couch and co-authors (1975), and Couch and Courtney (1977) have proposed that BP is potentially a serious health determinant in wild shrimp populations if exposed to pollution. Experimentally, prevalence and intensity of patent BP infection increased dramatically in Arochlor 1254 (PCB) exposed test groups as compared to controls (Couch and Courtney, 1977). Further, results from an earlier study (Couch, 1976) suggested exposure to sublethal levels of another potential pollutant, mirex, also enhanced BP infection. Thus, pollution-induced BP disease in natural shrimp populations, as suggested by (Couch 1974b; Couch and co-authors, 1975), would appear to be a potential concern.

Gross signs of infection of juvenile-to-adult shrimp range from none in wild and wild-captive pink shrimp (Couch, 1974b) to reduced feeding and growth rates and increased surface and gill fouling due to various epibiotic organisms in cultured penaeid species (Lightner, 1988 his Chapter 3.1.2).

Cellular infection by *Baculovirus penaei* is limited to hepatopancreas and anterior mid-gut epithelium (Couch, 1974b; Lightner, 1983; Johnson and Lightner, 1988). At the light microscopy (LM) level patently infected cells have marked cytopathologic changes described by Couch (1974a, b) and Overstreet and co-authors 1988). These changes are nuclear hypertrophy, chromatin diminution and margination, nucleolar degeneration or loss, and formation of intranuclear, tetrahedral inclusion (occlusion) bodies (Fig. 3–1c, d). By phase or bright field microscopy of squash preparations of the hepatopancreas the polyhedral inclusion bodies (PIBs) are tetrahedral or pyramidal in three dimensional form (Fig. 3-1, a, b), range in size from 0.5 to 20 μm (base) and number per nuclei from 1 to 6 (Couch, 1974b; Couch and co-authors, 1975) in natural infections of juvenile-to-adult shrimp, and up to 100 per nuclei in experimentally infected larvae (Overstreet and co-authors, 1988). A systemic or local inflammatory response to BP infection and/or BP induced cytopathology in shrimp has not been reported. However, Overstreet and co-authors (1988) suggested that ingestion by *Penaeus vannamei* larvae of free virions may result in a systemic infection by BP. Systemic BP infection, if this occurs, may be more pathogenic to the crustacean host and be the cause of significant clinical disease. However,

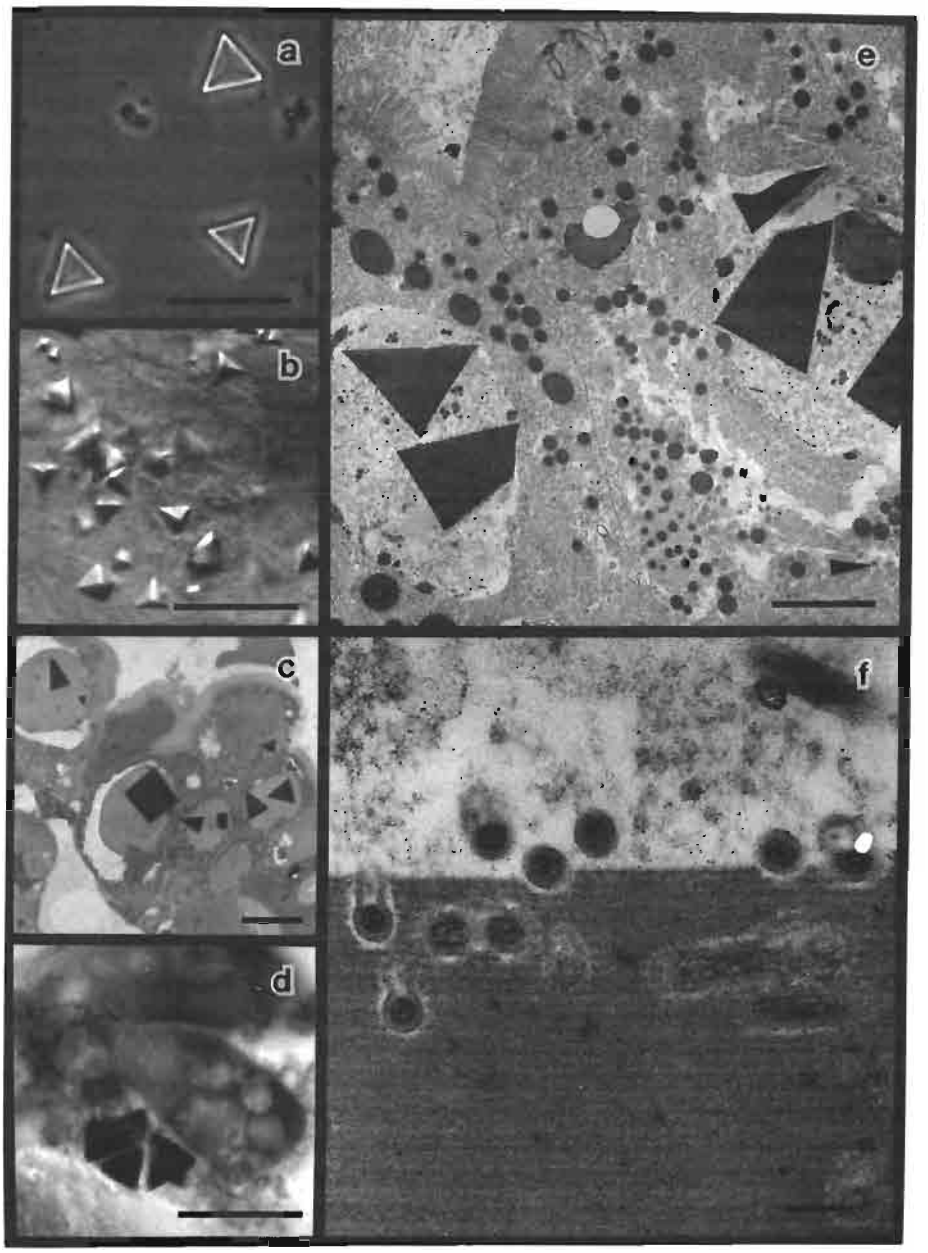


Fig. 3-1: *Baculovirus penaei* (BP) from *Penaeus vannamei* (a, b, and d), *P. stylirostris* (e), and *P. marginatus* (c and f). (a) Tetrahedral occlusion bodies of BP; unstained wet mount; bright field; bar = 25 μm. (b) BP occlusions in feces; Hoffman interference contrast; bar = 25 μm. (c to e) BP occlusion bodies in infected hepatopancreatic epithelial cell nuclei; c: o-toludine blue; bar = 10 μm; d: Brown and Brenn stain; bar = 10 μm; e: TEM showing BP occlusion bodies in the nuclei of adjacent cells; bar = 5 μm. (f) TEM of BP virions in cross and oblique section that are becoming occluded by the developing occlusion body; bar = 100 nm. (Originals.)

investigators working with BP virus have yet to demonstrate BP virions outside of the hepatopancreas or the lumen of the digestive tract. Thus, available data do not support the hypothesis that BP infects shrimp systemically.

The polyhedral inclusion (occlusion) bodies (PIB's, POB's or OB's) are quite distinctive microscopically (Fig. 3-1, a-d) and are the basis for definitive light microscopic diagnosis (fresh wet-mount preparations or histology) of BP infection in penaeid shrimp (Lightner, 1983, 1988 his Chapter 3.1.2). Further, shrimp baculovirus occlusion bodies fluoresce under ultraviolet light following staining with aqueous 0.1 % phloxine (Thurman and co-authors, 1989). This provides a rapid, specific diagnostic tool for identification of baculovirus occlusions. A highly sensitive (10 ng protein per 100 µl) enzyme-linked immunosorbent assay (ELISA) for detection of BP is reported (Lewis, 1986). However, it is not clear if the ELISA antibody is to baculovirus antigens, to PIB polyhedrin, or to both.

Ultrastructure changes found in BP infected hepatopancreas cells, in addition to those recognizable at the LM level, include: nuclear membrane proliferation (membranous labyrinths), myeloid bodies in cytoplasm, increase in free ribosomes, reduction in number of mitochondria, changes in fibrillar and granular stroma of the nucleoplasm and rod-shaped nucleocapids and virions (Couch, 1974a, b). Additionally, Lightner and Redman (1989) reported prominent aggregations of microfilament bundles with *Baculovirus penaei* virions in close association in the nuclei and cytoplasm of infected hepatopancreatocytes of post larval *Penaeus stylirostris*.

Under high magnification the PIB (Fig. 3-1, e, f) has a crystalline structure, a linear lattice of round subunits, each approximately 11 to 20 nm in diameter arranged in rows roughly 5 nm apart (Couch, 1974b). Cytochemical reactions suggest the PIB matrix consists of protein and RNA (Couch, 1976). The major polypeptide of the polyhedra has a molecular weight of approximately 50,000 to 53,000 daltons (Summers, 1977; Clerx and Lightner, 1985). BP polyhedrin is related to, but not the same as, polyhedrins and granulins of insect baculoviruses (Summers, 1977; Clerx and Lightner, 1985; Johnson and Lightner, 1988). The unit structure of the crystal is larger, and shrimp baculovirus polyhedra do not have the polyhedral membrane (Summers, 1977).

Based on morphological criteria, Couch (1974b) categorized several stages of the BP infection cycle in hepatopancreas cells of *Penaeus duorarum*. These stages are eclipse, early, intermediate and advanced infection. Eclipse and early infections are characterized by nuclear enlargement, reduction of heterochromatin and segregation of the nucleoplasm into regions of granular and fibrillar stromata. Virions and virogenic figures are apparent in the early but are lacking in the eclipse phase. Intermediate infections are characterized by greatly hypertrophied nuclei that contain few to numerous virions, aberrant stromatic patterns of the nucleoplasm, degenerate or absent nucleoli, nuclear membrane proliferation (production of membranous labyrinths), an increase in free ribosomes, and decrease in mitochondria and less endoplasmic reticulum in the cytoplasm. Cells in the advanced stage show increases in the above and additionally, the presence of one or more PIBs. Eclipse, early and intermediate may constitute latent stage of BP infection (Couch, 1974b).

There are presently no regional reportings of infectious diseases of shrimp. Thus, a reliable estimate of the economic significance of BP disease to shrimp hatchery and farm productivity in the Americas cannot be given. It is clear, however, the BP disease is one of 2 common virus infections of cultured shrimp in the Americas, the other being IHHN

virus. Further, BP disease is the most frequently encountered cause of virus associated mortalities in *Penaeus vannamei* hatcheries (Akamine and Moores, 1989), which points out the significance of this virosis to *P. vannamei* farming.

Control of BP disease in commercial shrimp hatcheries in regions where the virus is endemic in broodstock varies depending on the husbandry and management of the hatchery system. Data reported on this topic are limited; however, clinical observations suggest that in some facilities BP causes periodic-to-frequent high losses while other hatcheries have managed to reduce, or eliminate, occurrences of the disease by use of one or a combination of the following management techniques: avoidance of BP virus through exclusive use of nauplii spawned from females that are not passing PIB's in their feces or are PIB-negative on hepatopancreas biopsy examination (wet-mount preparations); reduction or elimination of vertical transmission between infected female shrimp and eggs/nauplii apparently accomplished by application of improved hygiene during spawning and hatching; reduction through more rigorous husbandry and sanitation procedures of cross-infection of BP between tank batches of larvae cultured concurrently; elimination of BP transmission from contaminated culture vessels or equipment to newly stocked groups of larvae by disinfection with an alkaline disinfectant (Akamine and Moores, 1989); or culture of larvae in seawater free of pesticides, chlorinated hydrocarbons or heavy metals that may serve to enhance BP disease in exposed groups of shrimp larvae.

Apparently successful strategies for management of BP disease are applied by shrimp hatchery operators. By and large these involve adopting procedures to avoid transmission of the virus from broodstock to offspring and culture conditions which minimize larval stress. According to Matthews (1982), for insect baculoviruses verticle transmission occurs via fecal contamination of eggs. Thus, management steps are taken in shrimp hatcheries to prevent post spawning contact between eggs and broodstock feces that clinically appear to result in reduction of BP disease; this could be explained on the basis of breaking the transmission of BP from broodstock to offspring. Also, proper nutrition and an environment free of dangerous levels of synthetic organic or heavy metal pollutants are of obvious importance. As for other viroses of cultured marine poikilothermic vertebrates and invertebrates, there are no effective chemical treatments known for control of these diseases. Vaccination of shrimp for protection from virus disease has not been reported.

Monodon baculovirus was first discovered during an epidemic disease of laboratory-reared, adult *Penaeus monodon* imported from Taiwan into Mexico and maintained in quarantine (Lightner and Redman, 1981; Lightner and co-authors, 1983c). Subsequent papers (Anderson and co-authors, 1987; Nash, G. and co-authors, 1988; Lightner and Redman, in press) document monodon baculovirus and MBV disease to be widespread in cultured *P. monodon*. There are no published records of MBV infection in wild shrimp populations, although wild shrimp are undoubtedly a primary source for MBV recognized in cultured shrimp.

Monodon Baculovirus is rod-shaped, singly enveloped and replicates within the nucleus (Fig. 3-2, d). The average size of nucleocapsids is reported to be 246 × 42 nm and virions 324 × 75 nm (Lightner and co-authors, 1983c). Slight bending of enveloped virions is occasionally observed (Johnson and Lightner, 1988). MBV described from Australian *Penaeus monodon* has nucleocapsids that are 260 to 300 × 45 to 52 nm in length and diameter, respectively (Doubrovsky and co-authors, 1988). The plebejus baculovirus (PBV), reported as a new baculovirus distinct from MBV (Lester and co-authors, 1987) is

likely an MBV-type baculovirus. The main distinctive feature given was in the capsid envelope, with PBV envelopes having 2 electron-dense zones and MBV having, but a single zone. Slight differences in nucleocapsid and virion dimensions and periodicity of the crystalline lattice of the occlusion body polyhedrin between PBV and MBV were also reported (Lester and co-authors, 1987). However, Lightner and Redman (in press) consider the baculovirus from *P. plebejus* to be an MBV-type agent. The MBV nucleic acid is presumed to be dsDNA, but study results confirming this assumption (Lightner and co-authors, 1983c) have not been published. MBV was successfully cultured *in vitro* in primary cell cultures of the lymphoid (Oka) organ where it caused localized cytopathic effect (CPE) within 2 to 3 days of exposure (Chen and Kou, 1989).

Monodon baculovirus has a diverse host range and wide geographic distribution on the Indo Pacific coasts of Asia, Australia, Africa, the Mediterranean coast of southern Europe, Kuwait and Israel (Lightner and Redman, in press). MBV may be a complex of several related strains of similar virus, but studies on this point are not available. MBV-type baculoviruses have been documented to result in moderate disease of *Penaeus monodon, P. merguiensis, P. penicillatus,* and *P. plebejus* and has been found as a subclinical infection of *P. esculentus, P. semisulcatus* and *P. kerathurus* and *P. vannamei* (Lightner and Redman, in press). MBV is reported from exotic penaeids imported into the Americas for research and aquaculture development (Lightner and co-authors, 1983c) and in an American penaeid, *P. vannamei,* exposed to the virus (Lightner and Redman, in press). MBV was introduced into Mexico, Hawaii, Tahiti, Ecuador, Texas and Brazil (Lightner and co-authors, 1983c; Lightner and co-authors, 1985b; Lightner and Redman, in press) with shipments of live shrimp for aquaculture development.

Monodon baculovirus appears to be common in *Penaeus monodon* culture throughout Southeast Asia. For example, Anderson and co-authors (1987) and Nash, G. and co-authors (1988) reported MBV is endemic to pond cultured populations of *P. monodon* in Malaysia. Additionally, MBV infection was diagnosed in 7 of 8 shrimp farms included in a disease survey of *P. monodon* in Taiwan (Lightner and co-authors, 1987a). Indeed, MBV is probably endemic in most areas where *P. monodon* is cultured.

The natural reservoir for MBV is presumed to be wild *Penaeus monodon* and other species of penaeid shrimp within the geographical range of the virus. Further, although not proven, wild-caught broodstock shrimp spawned to provide nauplii to hatcheries in Taiwan, the Philippines, Malaysia, Australia, etc. are the probable source of MBV in culture facilities. Verticle and horizontal transmission are likely similar for BP and MBV viruses. Presumably MBV is transmitted per os by ingestion of free virus, occlusion bodies and by cannibalism (Johnson and Lightner, 1988). The MBV virus may remain viable for a considerable time within the occlusion body and may serve as a source of infection between successive groups of cultured shrimp stocked into ponds. In hatcheries, MBV is believed to be transmitted from broodstock to offspring (Bonami and co-authors, 1986), but the exact means of vertical transmission has not been determined. Vertical transmission may be by surface contamination of spawned eggs, as is found for some of the insect baculoviruses (Matthews, 1982).

However, unlike BP successful experimental transmission of MBV has not been reported. Lightner and Redman (1981) did not observe MBV infection in juvenile and adult *Penaeus stylirostris* and *P. californiensis* that were held for 60 days in a tank with known MBV-infected adult *P. monodon*. Species and age resistance factors are obvious

reasons why experimental transmission was not observed in this study. Likewise, Bonami and co-authors (1986) were unsuccessful in horizontal transmission of MBV to post larval (PL) *P. monodon* and suggested that vertical may be more important than horizontal transmission in MBV disease. However, again experimental circumstances may have influenced the study results. The enveloped MBV virion is likely inactivated by freezing to 0 °C and slow thawing and this may also be a reason why horizontal transmission was unsuccessful in the Bonami and co-authors (1986) trial, if the source of MBV was frozen tissues.

Published descriptions of MBV disease in *Penaeus monodon* are based exclusively on studies of clinical infections of cultured shrimp. MBV disease is stage/age and stressor-mediated. Early larval stages of *P. monodon* have not been documented with MBV disease, but late larval, post larval and young juvenile shrimp are the most susceptible stage/ages to severe disease. A decreased severity with increased size/age of shrimp is reported (Lightner and co-authors 1983c). While there is limited data on this point, the available clinical information suggests a similarity here between MBV disease in *P. monodon* and BP disease in *P. vannamei*. That is, virus exposure to larval and post larval stages is potentially much more likely to result in mortality than exposure to juvenile-to-adult shrimp stages. Crowding and infection by facultative pathogens appeared to enhance the prevalence and severity of MBV disease in raceway-cultured *P. monodon* (Lightner and co-authors, 1983c). Differences in disease susceptibility based on shrimp species are known (Lightner and Redman, in press). Owens and Hall-Mendelin (1988) reported enhancement of patent MBV infection in *P. plebejus* exposed to 2 ppm nickel. Presumably, exposure to PCBs and mirex would similarly enhance activity of MBV, as it does for BP (Couch, 1976; Couch and Courtney, 1977).

Post larval *Penaeus monodon* with moderate-to-severe MBV infection are reported often to be smaller and darker (bluish grey to dark blue-black) than less or non-affected post larvae (tan colored). Severely affected *P. monodon* are lethargic, do not feed well and are predisposed to microbial fouling and bacterial infections expressed as localized 'shell disease'-type lesions of the gills or general cuticle, or as bacterial septicemias (Lightner, 1988 his Chapter 3.1.3).

Microscopically, MBV pathology is limited to cells of the hepatopancreas and, less often, the anterior mid-gut epithelium in heavy patent infections (Lightner and co-authors, 1983c). MBV has a deleterious effect on the host shrimp by destruction of its hepatopancreatic epithelial cells (Johnson and Lightner, 1988). Three stages of cytopathological development of MBV infection of hepatopancreas epithelium and a histologic severity index grading scale have been described (Lightner and co-authors, 1983c). At the LM and ultrastructure levels MBV pathology in *P. monodon* differs only slightly from that reported (Couch, 1974a, b, 1981) for BP in *P. duorarum* (Lightner and co-authors, 1983c). The principal differentiating feature is the shape of the occlusion body. In MBV the occlusion body is subspherical and not tetrahedral (Fig. 3-2, a-d). Additionally, the membranous labyrinths in MBV appear to arise from the Golgi complex rather than the nuclear membrane as occurs in BP (Lightner and co-authors, 1983c). Prior to the appearance of occlusion bodies early infections may be detected in sections because the nuclei are hypertrophied and the nucleolus and chromatin are marginated or missing (Johnson and Lightner, 1988).

Monodon baculovirus occlusion body morphology and development have been well

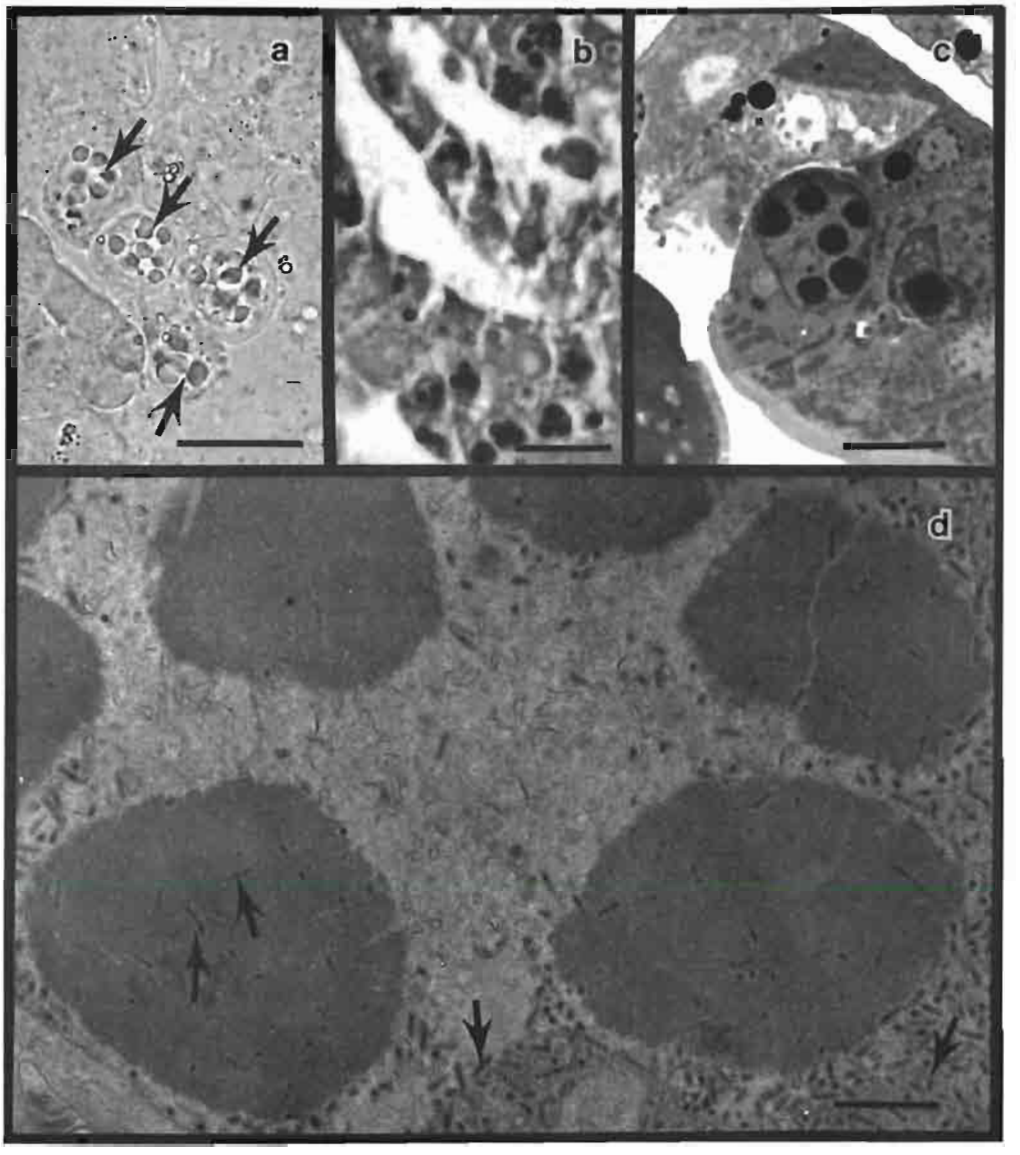


Fig. 3-2: *Penaeus monodon*-type baculovirus (MBV) in hepatopancreatic epithelial cells of *P. monodon*. (a) Multiple occlusion bodies in nuclei (arrows). Tissue squash, 0.05 % malachite green stain; bar = 25 µm. (After Lightner and co-authors, 1983c. Reprinted with the permission of Elsevier Science Publishers.) (b) Multiple MBV infected cells; Brown and Brenn stain; bar = 25 µm. (c) High magnification light photomicrograph of an MBV infected cell; o-toluidine blue; bar = 10 µm. (d) TEM of the nucleus of an MBV infected cell; arrows indicate occluded and free virions; bar = 1 µm. (b to d Originals.)

described (Lightner and Redman, 1981; Lightner and co-authors, 1983c; Johnson and Lightner, 1988). The MBV occlusion body is subspherical, up to 10 µm in diameter and consists of a paracrystalline network of polyhedrin subunits 17.7 ± 2.8 nm in diameter arranged in evenly spaced regular arrays 26 to 27 nm from center to center (Lightner and

co-authors, 1983c). The molecular weight of the MBV polyhedrin is ca 60,000 daltons (Chen and Kou, pers. comm., cited in Thurman and co-authors, 1989). The periodicity of the polyhedrin subunits in MBV from *Penaeus plebejus* is reported to be 20 nm (Lester and co-authors, 1987).

Diagnosis of MBV infection is based on demonstration of the characteristic subspherical occlusions (Fig. 3-2, a-c) in wet-mount preparations or stained sections of hepatopancreata (Lightner and co-authors, 1983a). Staining wet-mount smears with 0.05 % aqueous solution of malachite green with bright field illumination (Lightner, 1983, 1985) or 0.1 % phloxine and use of ultraviolet optics (Thurman and co-authors, 1989) aids in the detection of the spherical occlusion bodies which otherwise appear similar to lipid droplets. In sectioned material stained with hematoxylin and eosin, the occlusions are eosinophilic and very distinctive (Lightner and co-authors, 1983c). Establishing if MBV is the cause of clinical disease in a group of shrimp depends on interpretation of the history and clinical signs and determination of infection severity. Application of the histological infection severity grading criteria provided by Lightner and co-authors (1983c) is useful in this regard. Thus, shrimp dying from MBV disease have great numbers of hepatopancreatocytes undergoing degeneration and cultured shrimp groups suffering population disease have a high prevalence of individuals with histologically heavy MBV infections. There are currently no described procedures to identify different strains of MBV, if indeed these exist, or variations in virus pathogenicity. Perhaps, application of cell-culture methods described by Chen and co-authors (1986) and Chen and Kou (1989) will provide a mechanism to explore these areas.

There is little information reported on prevention and control of MBV disease in cultured shrimp. Avoidance through exclusion of the agent has been suggested (Lightner, 1988 his Chapter 3.1.3). Bonami and co-authors (1986) mention a population of *Penaeus monodon* cultured in Tahiti that are possibly resistant to MBV disease, but data to substantiate the point are lacking and the 'resistance' may as likely be a function of age rather than genetic factors. In terms of verticle transmission, use of procedures for management of BP in commercial hatcheries in the Americas may have application in the control of MBV in Asian shrimp culture.

A third baculovirus disease of culture shrimp is baculoviral mid-gut gland necrosis (BMN). The BMN baculovirus is nonoccluded and tentatively assigned to the Subgroup C of the Baculoviridae (Sano and co-authors, 1981). The BMN virions (Fig. 3-3, d) are 72 nm by 310 nm in average diameter and length, respectively (Sano and co-authors, 1981, Sano and co-authors, 1984). Bending of enveloped virions is observed occasionally (Sano and co-authors, 1984). The biochemical and additional biophysical characteristics of BMNV have not been reported. An indirect immunofluorescence technique has been developed for rapid diagnosis of BMN disease (Sano and co-authors, 1984). Similar to the other shrimp baculoviruses, the BMN virus infects hepatopancreatic epithelium.

Baculoviral mid-gut gland necrosis is known only from hatchery cultured *Penaeus japonicus* in the Kyushu and Chugoki areas of Southern Japan, where it has caused a disease problem every year since 1971 (Sano and co-authors, 1984). Interestingly, even though *P. japonicus* has been extensively translocated to other geographical locations for aquaculture development, BMNV infection and disease is only known from cultured Kuruma shrimp hatcheries in Southern Japan (Lightner and Redman, in press). Baculovirus mid-gut gland necrosis virus has not been reported from other species of

penaeid shrimp. BMN lesions are focal and infrequent in captive-wild adult female and cultured juvenile *P. japonicus* (Momoyama, 1988). The source of BMNV in Kuruma shrimp hatcheries is documented to be wild-caught female spawners (Momoyama, 1988). As with the other penaeid baculoviruses in maturation and hatchery settings, virus particles are thought to be shed with the feces and contaminate the eggs and nauplii, thus spreading the infection from broodstock to offspring. However, Momoyama and Sano (1989) were unsuccessful in experimentally transmitting BMNV to the eggs and naupliar stages of *P. japonicus*; thus the role of fecally shed BMNV in the transmission of this infection has not been demonstrated unequivocally. BMNV is not known to result in a disease in wild shrimp populations.

Experimentally, horizontal transmission of BMNV has been successful through water borne exposure to zoea through 10-day old post larval stages (Momoyama and Sano, 1989), and by feeding infected hepatopancreas tissues to PL_2s (Sano and co-authors, 1981; Sano and co-authors, 1985; Momoyama and Sano, 1988). In some trials, BMNV infection could be detected as early as 18 to 24 h post exposure using an indirect fluorescent antibody staining procedure (Sano and co-authors, 1984; Sano and co-authors, 1985). Patent, moderate to advanced BMNV infection is easily detectable microscopically in squash unstained preparations using dark field illumination and phase contrast microscopy, or in stained squash and H & E stained paraffin preparations by brightfield microscopy (Momoyama, 1983).

Although 75 % of shrimp in experimentally exposed groups of mysis larvae were infected, after 4 days, cumulative mortality was variable and ranged 16 to 44 % in mysis larvae and 83 % in PL_2s; but it was not different from controls for older post larvae shrimp (PL_9) (Sano and co-authors, 1985; Momoyama and Sano, 1988). BMN infected larvae had reduced growth as compared to the non-infected controls, but the growth effects were negligible when older PLs were exposed (Momoyama and Sano, 1989). These data indicate age resistance of *Penaeus japonicus* to BMNV disease, an apparent similarity with BP disease in *P. vannamei* (Overstreet and co-authors, 1988).

In shrimp hatcheries, BMNV disease occurs in mysis through 20-day post larvae and has been reported to reach 98 % mortality (Sano and co-authors, 1981; Sano and Fukuda, 1987). Both in hatchery and experimental infection, BMNV disease is characterized grossly in early post larval stages by a white, turbid hepatopancreas. Severely affected post larvae may float inactively on the water surface (Lightner, 1988 his Chapter 3.1.4).

As with the other penaeid shrimp baculoviruses, BMNV infects hepatopancreatocytes. Polyhedral occlusions are not formed (Sano and co-authors, 1981; Sano and co-authors, 1984). Pathologically, hepatopancreas collapse, marked hypertrophy (Fig. 3-3, a,b) of infected hepatopancreas cells, chromatin margination, diminished nuclear chromatin, nucleolar dissociation, karyorrhexis are reported (Sano and co-authors, 1981; Momoyama, 1983; Sano and co-authors, 1984).

Ultrastructural changes include cytoplasmic collapse, nuclear hypertrophy resulting in karyorrhexis and the presence of numerous rod-shaped baculoviral (Fig. 3-3, d) virions (Sano and co-authors, 1981; Sano and co-authors, 1984).

Momoyama and Sano (1989) report that rinsing eggs and nauplii with clean seawater prior to stocking into previously disinfected rearing tanks prevents BMNV disease. Moreover, since 1985 these steps have been instituted in commercial shrimp hatcheries in Japan and, concurrently, BMNV epidemics have been reduced or climinated in these

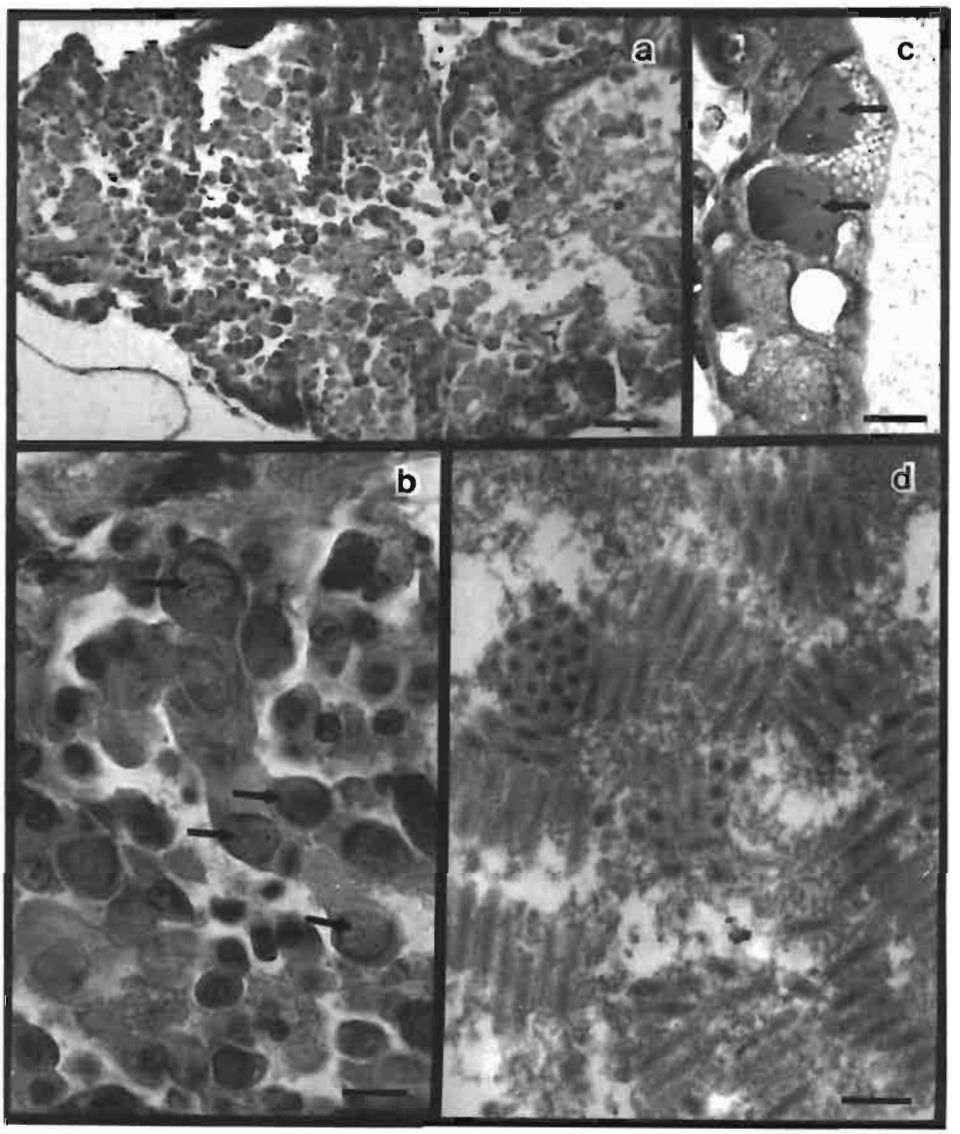


Fig. 3-3: Baculoviral midgut gland necrosis (BMN) and other nonoccluded baculovirus diseases affecting the hepatopancreas (HP) of penaeid shrimp. (a) Section of the HP of a postlarval *Penaeus japonicus* with severe BMN; H&E; bar = 50 μm. (b) Higher magnification of HP cells from Fig. 3-3a showing BMN infected cells with hypertrophied nuclei lacking occlusion bodies (arrows); H&E; bar = 10 μm. (c) Section of the HP from *P. monodon* infected with a nonoccluded baculovirus; several hypertrophied nuclei lacking occlusion bodies are indicated (arrows); H&E; bar = 10 μm. (d) TEM of BMN virions within the nucleus of an HP cell in *P. japonicus;* bar = 250 nm. (After Lightner, 1988. Reprinted with the permission of Elsevier Science Publishers.)

facilities. Lightner (1989) recommends avoidance of BMN virus as the preferred method of control.

Johnson (1976a, 1983, 1984a) reported a nonoccluded baculovirus infection of hepatopancreatocytes of the blue crab *Callinectes sapidus*. The agent is designated the

hepatopancreatic virus of *Callinectes sapidus* or 'Baculo-A', and is provisionally assigned to the Subgroup C of the Baculoviridae (Johnson and Lightner, 1988).

Baculo-A nucleocapsids measure 240 to 254 nm × 43 nm, and virions (Fig. 3-4, c) 260 to 300 nm × 60 to 70 nm (Johnson and Lightner, 1988). The trilaminar envelopes are 8 to 10 nm thick and have subapical, unilateral envelope expansions. Biochemical and further biophysical data for Baculo-A have not been reported.

Baculo-A infected crabs in all stages of the molt cycle were collected in May through October from various locations, in both high and low salinity waters, between Long Island Sound and Chesapeake Bay, USA (Johnson, 1983). Baculo-A infection prevalence averaged 6 % in the 1,500 crabs studied, but varied, usually in the range of 4 to 20 %, between collection times and locations. In one collection from Chincoteague Bay, 18 of 34 (52 %) crabs were infected. Johnson (1983) states Baculo-A is the most ubiquitous virus of the blue crab.

Baculo-A is not recorded to cause overt disease signs in the blue crab life stages examined (Johnson, 1976a, 1983, 1984a; Johnson and Lightner, 1988). Infections are known only through microscopic examination, tend to be focal in distribution and are limited to epithelial cells of the hepatopancreas. Of the hepatopancreas cells, absorptive and reserve cells are most commonly infected and embryonal cells (E or stem cells) are not attacked by Baculo-A (Johnson, 1983).

Microscopically, infected cell nuclei are hypertrophied (Fig. 3-4, a) about twice normal size, some have a faint Feulgen positive center and an intense Feulgen-positive marginal zone along the nuclear membrane (Johnson, 1976a, 1983). Feulgen negative bodies, probably remains of nucleoli, are found in some patently infected nuclei (Johnson, 1983).

Ultrastructurally, nuclei with advanced infection have virions concentrated in somewhat orderly arrays along the nuclear membrane (Fig. 3-4, b). These probably correspond to the intense Feulgen-positive bands seen with light microscopy (Johnson and Lightner, 1988).

There is no known effect by Baculo-A on wild blue crab populations (Johnson, 1983).

Pappalardo and Bonami (1979); Pappalardo (1981, cited in Pappalardo and co-authors, 1986) and Pappalardo and co-authors (1986) reported on a highly pathogenic baculovirus infection of the hepatopancreas of the portunid crab *Carcinus mediterraneus*. The agent is nonoccluded, shares the general characteristics of the Subgroup C baculoviruses, and is named τ (Tau) in reference to the location of its discovery, in the lagoon of Thau, near Montpellier, France (Pappalardo and Bonami, 1979).

Tau virions are 300 to 350 nm × 70 to 80 nm in length and diameter, respectively and are bent or bow-shaped, often the bend is subapical, or straight (Pappalardo and Bonami, 1979; Johnson and Lightner, 1988). In negatively stained preparations of Tau the dimensions of nucleocapsids and virions are 300 to 320 × 60 to 70 nm and 340 to 380 × 80 to 90 nm, respectively (Pappalardo and Bonami, 1979). Nucleocapsid extensions or 'tails' are reported in negatively stained preparations of Tau (Pappalardo and Bonami, 1979; Johnson and Lightner, 1988). Biochemical and additional biophysical characteristics of Tau are not reported.

Both natural and experimental infections of Tau in *Carcinus mediterraneus* are documented. Transmission in the laboratory was achieved by injection of 0.2 ml of a tissue extract and orally by feeding test crabs pieces of hepatopancreas tissues (Pappalardo and

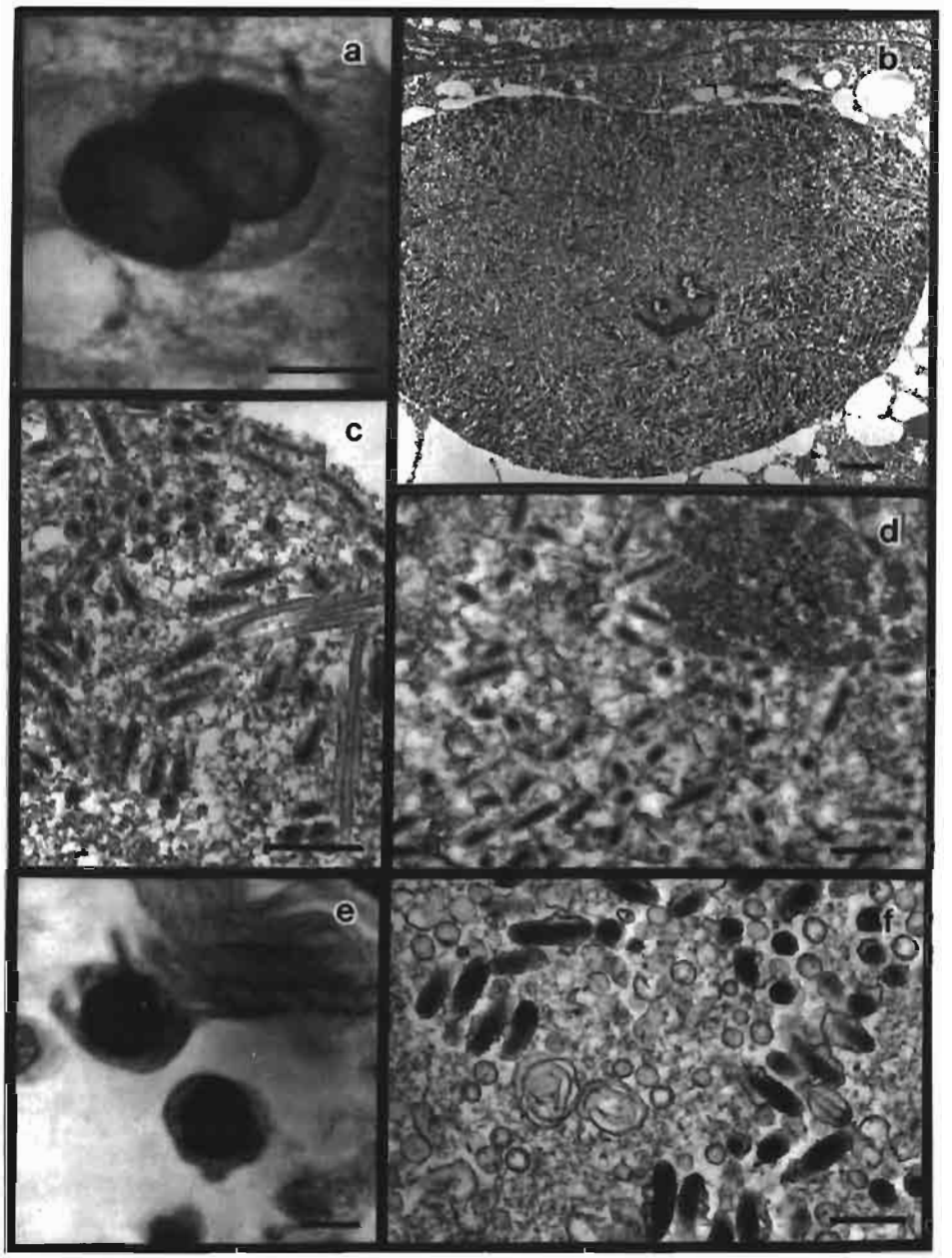


Fig. 3-4: Nonoccluded baculoviruses of crabs and unclassified rod-shaped nuclear viruses of *Callinectes sapidus* and *Paralithodes platypus*. (a) Baculo-A in *C. sapidus;* light micrograph showing infected and hypertrophied nuclei of a binucleate HP cell; Feulgen; bar = 10 μm. (b) TEM of a Baculo-A infected HP cell nucleus; bar = 1 μm. (c) Higher magnification TEM of Baculo-A virions; bar = 50 nm. (d) TEM of Baculo-PP virions in an hepatopancreatocyte nucleus of *Paralithodes platypus;* bar = 25 nm. (e) Baculo-B in hemocytes of *C. sapidus;* light micrograph showing infected hemocytes with hypertrophied nuclei; bar 10 μm. (f) Baculo-B virions in the nucleus of a *C. sapidus* hemocyte; bar = 250 nm. (Courtesy of P. T. Johnson.)

co-authors, 1986). Injected crabs had a cumulative mortality of 100 % after 25 days compared to 20 % mortality in the controls. The onset and progression of the disease was slower in crabs held at 18 °C versus crabs at 23 °C. Virus infection was confirmed by electron microscopic examination of negatively stained homogenates of the hepatopancreas from experimentally exposed crabs. However, positive virus presence was demonstrated in only 55 % of the inoculated individuals that died with disease signs. As suggested by the authors, this low positive confirmation rate likely indicates the limitation of the virus detection method used. *Per os* challenge studies resulted in variable transmission of the disease with an average of 35 % infection after 30 days for both live crabs and those that died during the study period (Pappalardo and co-authors, 1986).

In the injection study (Pappalardo and co-authors, 1986), Tau was introduced into the haemocoel of previously unifected *Carcinus mediterraneus*. Although virus particles were not actually observed passing through the basement membrane of the hepatopancreas, the authors suggest that the positive infections of the hepatopancreas epithelium resulted because the Tau virus crossed from the haemocoel and through the basement membrane of the hepatopancreas tubules.

In the Mediterranean shore crab, Tau virus is reported to cause a disease syndrome characterized by high mortality. Affected crabs display decreased aggressive behavior, increased lethargy, inappetence, eventually followed within a few days by death (Pappalardo and co-authors, 1986).

Gross pathologic changes associated with Tau virus infection have not been reported. Histologically, lesions are limited to the hepatopancreas. All hepatopancreas epithelial cells types, including stem cells, and the mid-gut epithelium, are attacked. Infected nuclei are characterized pathologically by marked nuclear hypertrophy, loss of nucleoli, chromatin margination and karyolysis (Pappalardo and co-authors, 1986). Tau-infected nuclei are strongly Feulgen-positive along the margin of the nuclear membrane with a uniform, Feulgen-positive center. The cytoplasm of infected cells is highly vacuolated and disorganized with enlargement of the perinuclear cisternae. Complete cellular breakdown is followed by discharge of the cell contents into the lumen of the hepatopancreas tubules. Similar pathologic change has been reported for infection of the mid-gut epithelium (Pappalardo and co-authors, 1986).

Ultrastructurally, Tau-infected, hypertrophic nuclei contain rod and bow-shaped particles, some of which are enveloped. Tubular structures of variable length and 50 to 60 nm in diameter are present in parallel arrangement in the nucleoplasm of infected nuclei. Virus particles often occupy peripheral areas of the nucleus, central regions contain a fibrillar stroma with nucleoli and heterochromatin absent or degenerating. Perinuclear cisternae are frequently enlarged and possess proliferating membranes. Cytoplasmic alterations include reduction of numbers of organelles, increased size of mitochondria, presence of vacuoles and vesicles many of which contain virions, and free virions within the cytoplasm (Pappalardo and co-authors, 1986).

The distribution and impact of Tau virus on natural populations of *Carcinus mediterraneus* is undocumented.

During the course of ultrastructure studies, Mari and Bonami (1986) discovered a baculovirus infection of the hepatopancreas of *Carcinus maenas*. The agent appears similar to the Tau baculovirus. This hepatopancreas baculovirus of *C. maenas* has been designated by these authors as Tau 2. We have found no further published information on Tau 2.

A nonoccluded baculovirus, Baculo-PP, infects hepatopancreas epithelium of the blue king crab *Paralithodes platypus* (Johnson and Lightner, 1988). Cell infections are sporatic and, based on available information, Johnson and Lightner, considered the agent to be nonpathogenic.

The Baculo-PP nucleocapsid measures 37 to 40 nm × 190 to 210 nm and has squared ends. The virions (Fig. 3-4, d) are 70 nm at the narrowest diameter by 230 to 265 nm long (Johnson and Lightner, 1988). Envelops are trilaminar and about 10 nm thick. Nucleocapsids have 'tails' extending from one end (Johnson and Lightner, 1988). Biochemical and further biophysical data for Baculo-PP have not been reported.

Baculo-PP was found in 2 populations of blue king crabs from Alaskan waters. Twelve of 30 crabs (40 %) collected in April 1982 at Olga Bay, Kodiak Island and 4 of 20 (20 %) crabs sampled in June 1982 from waters around the Pribilof Islands were infected (LM examination). Baculo-PP was not observed in crabs collected in the summer 1982 through February 1983 from St. Matthews Island, St. Lawrence Island and in an additional collection during that time from the Pribilof Islands (Johnson and Lightner, 1988).

Microscopically, Baculo-PP infected hepatopancreas cell nuclei are moderately hypertrophied, Feulgen positive centrally with strong, non-uniform in width, Feulgen-positive margins. Nucleoli are generally absent from infected nuclei. Baculo-PP infects all cell types of the hepatopancreas epithelium (Johnson and Lightner, 1988).

Baculo-PP is not known to cause disease in natural populations of blue king crabs.

Infections by 4 rod-shaped nuclear viruses of uncertain affinity have been described from marine portunid crabs. Host crab species affected are *Carcinus maenas* from European shores (Bazin and co-authors), 1974) and from North American waters (Johnson, 1988e), as well as *C. mediterraneus* (Mari and Bonami, 1986), and *Callinectes sapidus* (Johnson, 1983). Each of the viruses infect nuclei of hemocytes, hematopoietic or other mesodermal cells. When discovered and up until recently these agents were tentatively designated as nonoccluded baculoviruses (Bazin and co-authors, 1974; Bonami, 1976; Couch, 1981; Johnson, 1983, 1984a; Sparks, 1985). Johnson (1988e) suggests this designation needs reconsideration because these viruses differ from baculoviruses in that the nucleocapsid is not a true cylinder. In this regard, the viruses are similar to the Polydnaviridae and other unclassified rod-shaped nuclear viruses (Johnson, 1988e). None of the agents are known to produce a disease in their crab host.

The first hemocyte-infecting rod-shaped nuclear virus was discovered by Bazin and co-authors (1974) during an ultrastructure study of regenerating limb buds of the European shore crab *Carcinus maenas*. The virus is nonoccluded, enveloped, rod-shaped and was originally designated, and subsequently reported to be, a nonoccluded baculovirus (Bazin and co-authors, 1974; Bonami, 1976; Couch, 1981; Johnson, 1983, 1984a; Sparks, 1985). Mari and Bonami (1986) refer to this virus as B1.

The B1 virus is bacilliform with nucleocapsid and virion dimensions of 230 to 280 nm × 75 to 80 nm and 90 to 100 nm × 300 to 320 nm, respectively (Bazin and co-authors, 1974). Biochemical characteristics and additional biophysical features of B1 are not reported.

The B1 virus of the European shore crab infects hemocytes and connective-tissue cells (Bazin and co-authors, 1974) in regenerating limb buds, and probably elsewhere as well. Ultrastructurally, infected cells have hypertrophied nuclei and chromatin margination. Virions occur in small groups and are associated with vesicles.

An apparently similar hemocytic virus infects the same tissues of the Mediterranean

shore crab *Carcinus mediterraneus* (Mari and Bonami, 1986). These authors named this virus B2. The nucleocapsid is 280 to 320 nm × 70 to 80 nm in length and diameter, respectively. Virions are dispersed in the nucleoplasm and are 340 to 380 × 90 to 110 nm (Mari and Bonami, 1986).

The distribution of hemocytic nuclear viruses B1 and B2 in European and Mediterranean shore crab populations and their pathogenicity toward the crab hosts is undocumented.

A well described infection by a hemocytic nuclear virus is known from the blue crab *Callinectes sapidus*. The agent was named Baculo-B by its discover (Johnson, 1983). As with the other hemocytic nuclear viruses, Baculo-B is rod-shaped, nonoccluded, enveloped and infects hemocytes and hematopoietic cells of its crab host (Johnson, 1983, 1984a). Johnson (1988e) recommends the name Baculo-B be retained until the agent can be studied in sufficient detail to be placed positively to family. Baculo-B virions (Fig. 3-4, f) are ca 100 × 335 nm and often occur in ordered arrays within the nucleoplasm. The development of virions is associated with intranuclear vesicles (Johnson, 1983, 1988c). Biochemical and additional biophysical data on Baculo-B are not reported.

Using histological methods, Baculo-B infections were identified in 19 of 1,500 blue crabs collected from Chesapeake Bay, Maryland and Chincoteague Bay, Virginia, USA (Johnson, 1988e).

By light microscopy, Baculo-B infected cells (Fig. 3-4, e) have pale, hypertrophied (1.6x normal size) nuclei that are homogeneous throughout or have a thin band of chromatin along the nuclear membrane (Johnson, 1983). Hyperchromatic areas may be present in the center of some nuclei. The cytoplasm of infected cells is reduced to a thin band around the nucleus and lacks cytoplasmic granules (Johnson, 1983, 1988e).

Baculo-B is not known to cause disease in blue crabs (Johnson, 1983).

A fourth hemocytic nuclear virus was recently reported from *Carcinus maenas* in North American waters (Johnson, 1988e). The virus is named rod-shaped virus of *C. maenas* (RV-CM) and was found in one of 74 *C. maenas* examined in several collections made during 1982-83 from Woods Hole, Massachusetts, USA. Grossly, the infected crab's hemolymph clotted abnormally and was milky in appearance. The crab was concurrently heavily infected by a rhabdo-like virus similar to EHV of the blue crab (Johnson, 1988e).

The nucleocapsids are straight before becoming surrounded by an envelope and are 190 to 540 nm × 95 to 110 nm in length and diameter, respectively (Johnson, 1988e). Thickness of the envelope is 7 to 9 nm. Once closed, the envelope becomes spherical and this apparently causes the nucleocapsid to bend into a curved or V-shape, to be able to fit into the available space (Johnson, 1988e). Biochemical and additional biophysical data an RV-CM have not been reported.

Under bright field microscopy RV-CM infected cell nuclei in the hematopoietic tissue and hemolymph were enlarged minimally, but were occasionally up to twice normal size. These nuclei stained positively by the Feulgen method and are similar appearing to Baculo-B infected cell nuclei in the blue crab (Johnson, 1988e).

Ultrastructurally, infected nuclei have marginated chromatin, may contain one or more nucleoli with virions and vesicles evenly distributed throughout in heavily infected nuclei (Johnson, 1988e). Johnson (1988e) observed both curved and V-shaped enveloped nucleocapsids and reported the EHV-like virus was often present in the cytoplasm of RV-CM infected cells.

The RV-CM agent has no known impact on *Carcinus maenas* populations.

Three hexagonal nuclear viruses are reported from marine crabs. Each virus was compared by the discoverer to the Herpesviridae. Crab hosts for these viruses are the blue crab *Callinectes sapidus* (Johnson, P. T., 1976b, 1978, 1983, 1984a, 1988a, b); the mud crab *Rhithropanopeus harrisii* (Payen and Bonami, 1979); and the blue king crab *Paralithodes platypus* (Sparks and Morado, 1986). Two of the hexagonal nuclear viruses, those from the blue crab and the blue king crab are highly pathogenic to their respective crab hosts (Johnson, 1976b, 1983; Sparks and Morado, 1986). The herpes-like virus from the mud crab is not known to be pathogenic to its host (Payen and Bonami, 1979).

Johnson (1988a) provides convincing evidence that the hexagonal nuclear viruses infecting the blue crab and the blue king crab differ from members of the Herpesviridae to such an extent that these viruses should not be placed in this family. Johnson's recent study showed that the blue crab agent is an enveloped intranuclear particle. Herpesviruses become enveloped after leaving the nucleus (Gillespie and Timoney, 1981). In addition, the blue crab and the blue king crab hexagonal nuclear viruses are larger than the known herpesviruses. Johnson (1988a) concluded that the blue crab and blue king crab viruses are morphologically and developmentally similar, and therefore related. Sparks and Morado (1986), however, maintain that the blue king crab hexagonal virus is a herpes-like virus. Sparks and Morado (1986) found that the intranuclear particles are surrounded by a bilayered capsid which appears to be the structure that Johnson (1988a) describes as the 'envelope' in the material from the blue crab. Sparks and Morado (1986) acknowledge that Johnson is not in agreement with their descritpion of the intranuclear particles. It is abundantly apparent that classification of these 2 'herpes-like' nuclear viruses must await the application of additional virological methods to define better their biochemical and biophysical features. This situation reflects the obvious limitation and difficulty of attempting virus classification based on criteria established solely by observational methods.

The herpes-like virus reported from the mud crab is similar in size, morphology and development to the only other invertebrate herpes-like virus, described from the eastern oyster *Crassostrea virginica* (Farley and co-authors, 1972, cited in Johnson, 1988a), and the vertebrate herpesviruses (Gillespie and Timoney, 1981). Neither the mud crab or eastern oyster herpes-like viruses are particularly pathogenic to their hosts (Johnson, 1988a).

The Blue crab enveloped nuclear virus was discovered in 1974 in an immature female blue crab *Callinectes sapidus,* collected from Chincoteague Bay, Virginia (Johnson, 1976b). Subsequent studies revealed the virus, named Herpes-Like Virus or HLV, infecting additional blue crabs from Chincoteague Bay and also Assawoman Bay, Delaware (Johnson, P. T., 1978). Johnson (1983) reported an HLV prevalence of 13 % in juvenile wild crabs studied.

In the most recent paper regarding this virus (Johnson, 1988a) has redesignated the agent as Bi-Facies Virus (BFV). Two types of development and 2 final forms, Type A and Type B, are recognized for BFV.

The size of the enveloped Type A particles (face to face) ranges 197 to 233 nm and the Type B particles (face to face) are 174 to 191 nm (Johnson, 1988a). Type A particles have 2 envelopes and Type B particles have 1, which is identical to the inner envelope of the Type A particle. The diameter of the enveloped Type B particle is similar to that of the inner enveloped Type A particle. The envelopes are 7 to 10 nm thick. Type A (Fig. 3-5) and B particles occur in nuclei, the cytoplasm or extracellularly. Each particle is mor-

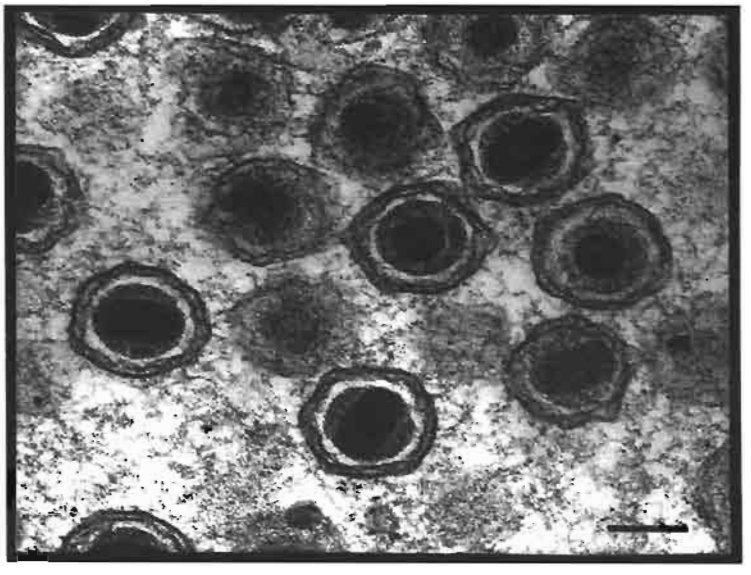


Fig. 3-5: Bi-faces virus of *Callinectes sapidus*. TEM of Type A particles; bar = 100 nm. (After Johnson, 1983. Reprinted with the permission of Academic Press, Inc.)

phologically identical, independent of location in the cell or extracellular space. Developmental stages of the 2 types of particles occur within the same nuclei, usually with one type in much greater abundance than the other. In section, the single envelope of Type B is irregular and appears as an elliptical-to-circular structure. The shape of the outer envelope of the Type A particle is icosahedral (Johnson, 1988a).

Within the envelopes of the completed particles there is a short rod-shaped electron dense core surrounded by and electron-dense sphere (Fig. 3-5). Based on staining characteristics and development, the core is assumed to contain the viral nucleoprotein (Johnson, 1988a). The core size differs slightly between Type A and Type B particles with the Type B core being narrower and slightly longer than that found in the Type A particle. The mean size for Type A and Type B cores are (length and diameter) 138 × 75 nm and 156 × 70 nm. Johnson (1988a) provides a detailed description of the morphogenesis for both the Type A and Type B particles. The biochemical characteristics of the Bi-Facies virus have not been reported.

Bi-Facies Virus causes mortality in blue crabs. Terminally infected crabs are lethargic and anorectic; death follows soon after onset of these disease signs (Johnson, P. T., 1978). The disease course may take up to 60 days in naturally infected crabs, whereas crabs die in 30 to 40 days after experimental challenge (injection and *per os* exposure) to BFV (Johnson, P. T., 1978).

Hemolymph fails to clot and is chalky white in blue crabs with symptomatic BFV infection, but other organ systems appear grossly normal (Johnson, P. T., 1976b, 1978). Hemolymph turbidity is due to tiny refractile bodies (virus particles) and lysed cellular debris (Johnson, P. T., 1978).

The blue crab BFV mainly infects hemocytes and fixed phagocytes in the interstitium of the hepatopancreas (Johnson, 1976b). Hematopoietic cells, epidermis, gill epithelium

and connective tissue cells are also attacked (Johnson, 1976b, 1988a). BFV infected cells have markedly hypertrophic nuclei with Feulgen-positive granules or nuclei stain faintly to deeply Feulgen-positive. Cowdry-Type A inclusions and large Feulgen-negative cytoplasmic inclusions are occasionally observed (Johnson, 1976b, 1983).

BFV infection was not found in the heart, skeletal muscle, gonad, gut epithelium and nervous tissue (Johnson, 1976b). Johnson (1976b, 1983, 1988a) does not mention if BFV infects the antennal gland or the bladder epithelium of blue crabs.

By electron microscopy, cells heavily infected by BFV contain numerous icosahedral virus particles that occur singly or in groups embedded in a granular/fibrillar matrix within hypertrophic nuclei (Johnson, 1988a). The nuclear chromatin is marginated. Multiple crystalline inclusions may occur in infected nuclei. The cytoplasm of infected cells is reduced, contains mitochondria, free ribosomes and vesicles, lacks rough endoplasmic reticulum and Golgi apparatus, and large, finely granular inclusions are often present as are similar appearing inclusions within hypertrophied nuclei (Johnson, 1988a).

In many infected cells, groups of electron dense rods of a size range 60 × 220 nm occur in the cytoplasm (Johnson, 1988a). These rods are also present in the cytoplasm of hemocytes with normal appearing nuclei and are usually embedded in the finely granular cytoplasmic inclusions common to BFV infected cells.

The distribution and impact of BFV in wild-crab populations is unknown.

The hexagonal nuclear virus reported by Sparks and Morado (1986) from the blue king crab *Paralithodes platypus* probably also infects 2 other species of commercially important anomuran lithoidid crabs, the red king crab. *Paralithodes camtschatica* and the golden king crab *Lithodes aequispina* (Sparks and Morado, 1985). Based on characteristic bladder and antennal gland epithelial lesions, the virus is known from natural infections of these crabs over a wide geographic range in Alaskan waters. Infected crabs were collected in all areas sampled from the Cook Islands and Bristol Bay to the Pribilof and the Western Aleutian Islands. The infection is of interest particularly in view of the catastrophic decline in Alaskan red and blue king crab populations in recent years (Sparks, 1985; Sparks and Morado, 1985, 1986).

The outer dimensions of the blue king crab 'herpes-like' virus nucleocapsid (Fig. 3-6, b) is ca 140 × 165 nm in diameter (Sparks and Morado, 1986). The capsid is reported to be composed of 2 electron-dense layers that surround a central, electron-opaque cylinder measuring 55 to 60 nm × 90 to 105 nm. The nucleoid is encompassed by a toroidal structure, 90 to 105 nm that is of intermediate electron density (Sparks and Morado, 1986). Enveloped cytoplasmic particles have not been reported for the blue king crab agent. The biochemical attributes of this virus are unknown.

In a 3 year study period (1982–1984) prevalence of virus infected red king crabs, based on light microscopy findings of the characteristic cytopathology, varied 4.1 % (2/49 crabs examined), 17 % (15/88) and 8.1 % (5/62), respectively (Sparks and Morado, 1985). Infection prevalence in blue king crabs varied from 5.1 % (2/39) to 15.9 % (7/44) in collections made in 1983 from the Bering Sea and St. Matthew Island areas. Eight of 54 (15 %) of golden king crabs examined histologically from various sample locations had the putative virus infection (Sparks and Morado, 1985).

Disease signs and gross lesions have not been reported for this virus infection of king crabs. Microscopically, stained sections of bladder (Fig. 3-6, a) and antennal gland show extensive destruction of these tissues; parenchymal cells have hypertrophied nuclei,

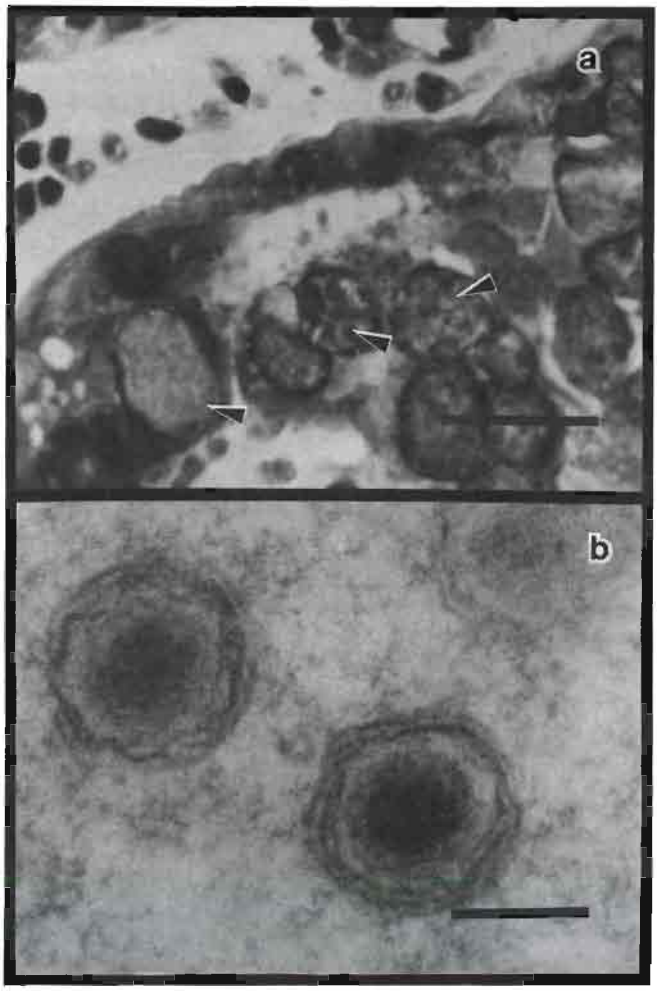


Fig. 3-6: 'Herpes-like' virus of *Paralithodes platypus*. (a) LM of heavily infected bladder epithelium with markedly hypertrophied nuclei and marginated chromatin (arrow heads); H&E; bar = 50 μm. (b) TEM of the 'herpes-like' nucleocapsids; bar = 100 nm. (After Sparks and Morado, 1986. Reprinted with the permission of Inter-Research.)

chromatin margination and possessed unevenly stained, spherical forms of eosinophilic ground substance that sometimes contain variable-shaped, eosinophilic inclusion bodies (Sparks and Morado 1985, 1986). The nuclear ground substance and pleomorphic inclusion bodies stain Feulgen-negative to weakly Feulgen positive. Podocytes are infrequently infected and an inflammatory response is minimal or absent altogether (Sparks and Morado, 1986).

Electron microscopy examination of tissues from infected crabs has only been accomplished from a diseased blue king crab (Sparks and Morado, 1986). Affected bladder and antennal gland nuclei are enlarged, have a thin zone of chromatin encircling the nucleoplasm and contain numerous hexagonal virus particles (Fig. 3-6, b). The ground substance is made-up of finely granular matter, and is the apparent site of viral particle

formation. The intranuclear inclusions have frayed ends when virus particles were numerous in nuclei (Sparks and Morado, 1986).

Sparks (1985) and Sparks and Morado (1985, 1986) present evidence of extensive destruction of antennal gland and bladder tissues associated with the herpes-like virus of king crabs, and suggest that this agent may be an important factor contributing to crab mortality in wild populations.

A herpes-like virus has been reported to infect the male gonad of the mud crab *Rhithropanopeus harrisii* in France (Payen and Bonami, 1979). The virus was observed in mesodermal cells surrounding the primary spermatogonia in the testicular germative zone. Within the nucleus the virus nucleocapsid is 75 to 80 nm with a dense central core 40 to 45 nm in diameter. The virus particles are enveloped in the cytoplasm and are 100 to 110 nm in diameter (Payen and Bonami, 1979). The agent is comparable in size, morphology and development to the majority of vertebrate herpesviruses and the herpes-like virus identified from the oyster *Crassostrea virginica* (Farley and co-authors, 1972, cited in Johnson, 1988a) in the size of the enveloped particle, 100 to 110 nm; it is unenveloped while in the nucleus (Payen and Bonami, 1979; Johnson, 1988a).

The mud crab herpes-like virus is not reported to cause disease signs in its host.

Among invertebrates, parvoviruses are known only from insects (Kelly, 1981) and certain decapod crustaceans (Lightner and Redman, 1985a; Mari and Bonami, 1988). Foster and co-authors (1981) reported 23-nm-diameter virus-like particles in phagosomes of tissue phagocytes in the heart found during electron microscopy examination of tissues from a solitary brown shrimp *Penaeus aztecus*. These authors as well as Lightner and co-authors (1983b) suggest the agent is probably a parvovirus. No further information has been forthcoming in the literature, and this virus, its nucleic acid type and site of replication are undetermined. Based on size and isometric morphology, the agent could be tentatively classified as a parvo-like or picorna-like virus.

Lightner and Redman (1985a) report infection and disease by a parvo-like virus in certain species of cultured penaeid shrimp. The infection is designated hepatopancreatic parvo-like virus (HPV) disease and was found more or less simultaneously in cultured populations of 4 species of penaeid shrimp in 4 distinctly separate culture facilities in Asia (Lightner and Redman, 1985a).

Hepatopancreatic parvovirus is an intranuclear, 22 to 24 nm-diameter (Fig. 3-7, d, e), isometric agent (Lightner and Redman, 1985a). Based on its intranuclear location and histochemical reaction, the HPV nucleic acid type is suggested to be DNA (Lightner and Redman, 1985a). HPV occurs within an intranuclear inclusion body composed of an electron dense, fine granular virogenic stroma. Occasionally, HPV particles are arranged in geometric patterns suggestive of an array formation (Lightner and Redman, 1985a). HPV has not been described in locations outside the nucleus of hepatopancreas epithelium. Biochemical and additional biophysical features of HPV are unstudied.

Currently, Lightner and Redman (in press) have documented HPV infection from 9 penaeid species, including *Penaeus monodon, P. esculentus, P. semisulcatus, P. merguiensis, P. indicus, P. chinesis, P. penicillatus* and *P. vannamei*. Geographically, HPV has a wide distribution and is known to occur in areas of Asia, Australia (Paynter and co-authors, 1985) and Africa. Furthermore, the HPV agent has been imported into Israel from Kenya (Colorni and co-authors, 1987) and into the Americas with *P. penicillatus* introduced into Brazil in 1987 (Lightner and Redman, in press) and with *P. chinesis*

imported into Hawaii. The Hawaiian introductions of *P. chinesis* were destroyed and HPV was eradicated (Brock, unpubl.). Recently, HPV lesions were found in cultured *P. vannamei* in Ecuador (Lightner and Redman, in press).

The mode of transmission for HPV is unclear. There are no literature reports on the experimental transmission of HPV and knowledge of this aspect of the epidemiology of HPV disease is based on clinical reports, all of which lack control data for comparison. According to Paynter and co-authors (1985) wild-caught *Penaeus esculentus* showed only low infection prevalence after 40 days of confined enhancement-rearing, possibly indicating a prolonged incubation period or a lack of horizontal transmission of HPV in this shrimp species. Chong and Loh (1984) reported high HPV infection prevalence in 2 shrimp farms (> 50 %) using hatchery derived seed as opposed to 2 fattening farms (< 15 %) that grew-out wild-caught seed. Lightner and Redman (in press) suggest this may indicate that HPV is transmitted either vertically from parent broodstock or horizontally with efficiency only during the larval stages. Studies to clarify the means of HPV transmission and mode of infection are clearly needed.

Population and individual shrimp signs of HPV disease are reported, but are not specific for this disease (Lightner and Redman, 1985a). The signs are reduced growth, mortality, inappetence, reduced preening as evidence by increased surface epicommensal fouling and greater susceptibility to secondary bacterial and fungal infection (Lightner and Redman, 1985a; Lightner, 1988 his Chapter 3.1.5). Paynter and co-authors (1985) found no abnormal signs associated with a single, severely infected, wild-caught *Penaeus esculentus* that was enhanced for disease development prior to sacrifice for histopathology examination.

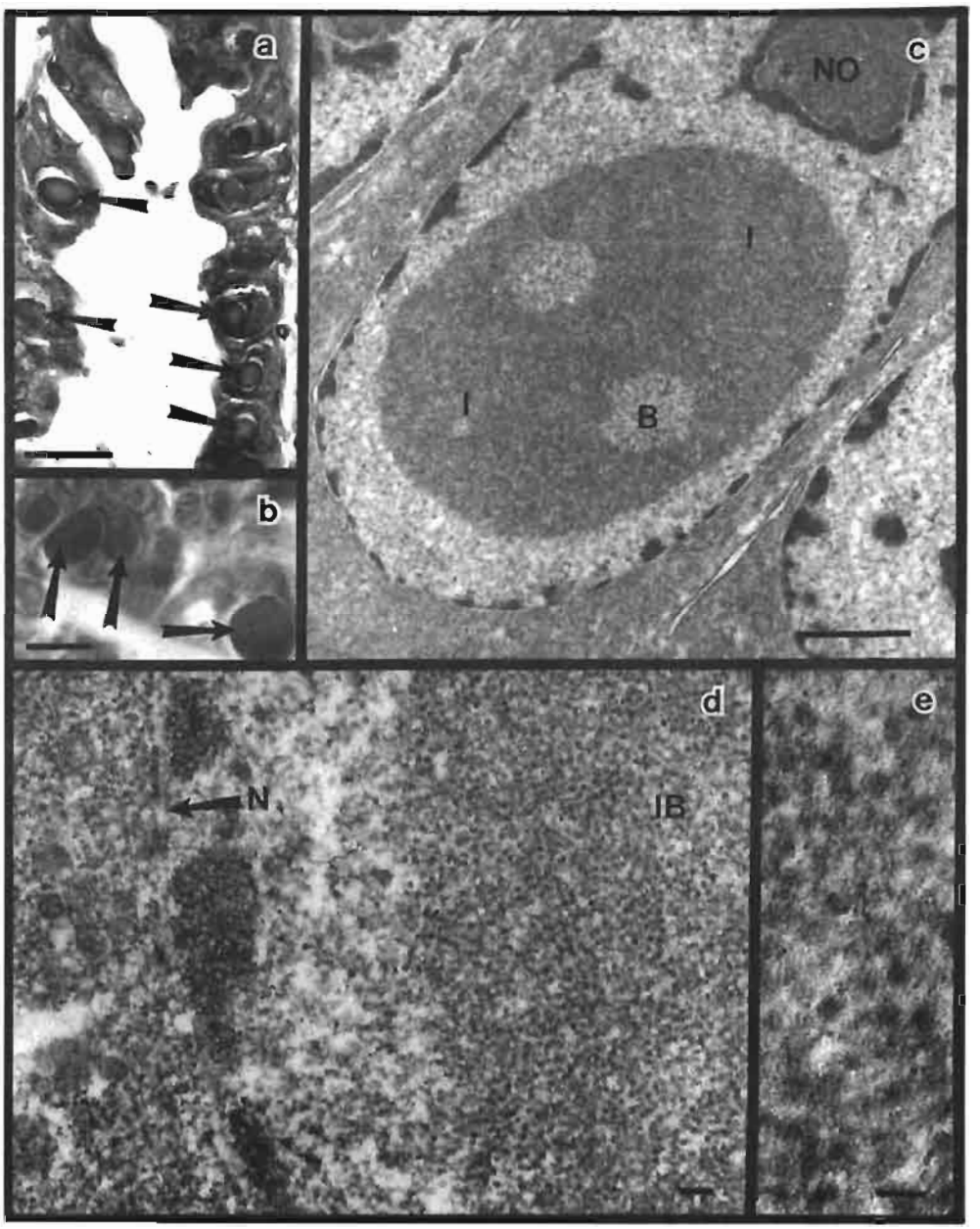
Gross lesions associated with HPV infection are hepatopancreas atrophy and abdominal muscle opacity (Lightner and Redman, 1985a). Cumulative HPV-associated mortality is reported to be 50 to 100 % after 4 to 8 weeks in susceptible juvenile stages of cultured *Penaeus merguiensis* (Lightner and Redman, 1985a).

HPV infects hepatopancreatic epithelium and less often epithelium of the anterior mid-gut and dorsal epigastric caecum (Lightner and Redman, 1985a). In the hepatopancreas, cells near the distal aspect of the hepatopancreatic tubules, the E-cells (stem cells), are most often infected. HPV infection results in formation of prominent intranuclear inclusion bodies (Fig. 3-7, a, b) that when fully developed are basophilic (H & E), PAS-negative and strongly Feulgen-positive (Lightner and Redman, 1985a). In early cell infection inclusions are small spherical-shaped, eosinophilic intranuclear bodies, that as the infection progresses, develop into large, usually single, dense basophilic inclusions that fill and greatly distent the nucleus. Microscopically, Lightner and Redman (1985a) found atrophy of the hepatopancreas in HPV infected shrimp. The parenchymal cells of the organ are reduced in size and have markedly fewer lipid droplets and/or secretory vacuoles. Apparently, however, infected hepatopancreata lack cell necrosis and host inflammation (Lightner and Redman, 1985a).

Ultrastructurally, developing HPV inclusions (Fig. 3-7, c, d) are composed of fine granular, electron-dense material, the virogenic stroma, and virus particles (Lightner and Redman, 1985a). HPV particles (Fig. 3-7, e) are usually spherical and some maybe in close association with microtubules or microfibrils (Lightner and Redman, 1985a). Roubal and co-authors (1989) found the pathological aspects of HPV in *Penaeus merguiensis* from Australia identical to the descriptions reported by Lightner and Redman (1985a).

Studies concerning control of HPV disease in penaeid shrimp are not reported. Avoidance of this virus infection in cultured shrimp is suggested (Lightner, 1988 his Chapter 3.1.5).

Another putative parvovirus is reported from the Mediterranean shore crab *Carcinus mediterraneus*. The virus was first discovered in crabs caught in Prevost Lagoon near Montpellier, France (Mari and Bonami, 1986). The agent is a small, 23 to 27 nm virus,



designated PC84, and is provisionally assigned to the Parvoviridae (Mari and Bonami, 1988). The virus causes a lethal disease in its host (Mari and Bonami, 1986).

The PC84 virus is an icosahedral, unenveloped agent (Mari and Bonami, 1986, 1988). Isopycnic centrifugation in CsCl gradient produced 2 bands of virus particles designated L and H particles with buoyant densities of 1.25 and 1.34 g cm^{-3}, respectively. By negative staining these particles are 29 to 31 nm in diameter, and in TEM preparations they are 23 to 27 nm in diameter. Preliminary results (Mari and Bonami, 1988) indicate the virus nucleic acid to be double stranded DNA. The molecular weight of the L particle 1.4×10^6 daltons.

The disease signs, nonspecific for this virus infection, include weakness, lethargy and anorexia (Mari and Bonami, 1988). Experimentally, infected crabs die between 10 and 25 days after inoculation with 0.2 ml of a cell-free suspension of purified virions from a diseased crab (Mari and Bonami, 1988).

PC84 infects connective tissue cells systemically resulting in necrosis and defense reactions characterized by degenerating cells and nodule formation (Mari and Bonami, 1988). Nuclei are not hypertrophied and are uniformly Feulgen-positive. Epithelial cells of the gills, mid-gut and hepatopancreas, all organs with heavy virus infection, are reported not to be infected.

Ultrastructurally, PC84 virions are abundant in fibroblastic and myoepithelial cells (Mari and Bonami, 1988). Nuclei of these cells lack chromatin which is replaced by an amorphous, electron-dense material with numerous viral particles in clearer areas. Affected cells have enlarged perinuclear cisternae. Numerous icosahedral virus particles are present in the cytoplasm of these cells and paracrystalline arrays of virions are found in the intercellular spaces (Mari and Bonami, 1986, 1988).

The distribution and impact of PC84 in wild populations of the mediterranean shore crab has not been reported.

Vago (1966) was the first to discover a virus infection in a crustacean. The virus, named the paralysis (P) virus, was found in the tissues of the portunid crab *Macropipus depurator*. Infected crabs were collected from Sète on the Mediterranean Coast of France. The agent localizes in the cytoplasm of connective tissue cells and probably hemocytes (Bonami, 1973; Bonami and co-authors, 1976). Infection by the P virus results in slowly developing paralysis in the crab host.

The paralysis virus is nonenveloped, icosahedral RNA virus with cytoplasmic development (Bonami, 1973, 1976; Bonami and co-authors, 1976). By negative staining

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Fig. 3-7: Hepatopancreatic parvovirus (HPV) of penaeid shrimp. (a and b) Light micrographs of HPV-infected HP cells of *Penaeus merguiensis;* dense basophilic intranuclear inclusion bodies (arrows) are present in markedly hypertrophied HP cell nuclei; both H&E; bars = 25 μm (a) and 10 μm (b). (Originals.) (c) TEM of an HPV-infected HP cell from *P. orientalis* that contains an HPV intranuclear inclusion body (I); embedded within the inclusion are 2 intranuclear bodies (B), structures seen in parvovirus-infected nuclei of other individuals. Host cell nucleolus (No) displaced by inclusion body; bar = 1 μm. (d) Higher magnification TEM of an HPV-infected HP cell that shows unorganized masses of 22 nm diameter virus particles developing within the virogenic stroma of the inclusion body (IB), which is within the nucleus (N = nuclear membrane); bar = 0.1 μm. (e) Higher magnification TEM of 22 nm diameter HPV virions from Fig. 3-7d showing angular profiles; bar = 50 nm. (c to e after Lightner and Redman, 1985a. Reprinted with the permission of Academic Press, Inc.)

technique the P virus is 58 to 65 nm in diameter (Vago, 1966), with an average size of 61 nm (Bonami, 1973). The icosahedral virions have a central electron dense zone of 30 nm and a bilayered 15 nm-thick capsid with subunits in the outer layer. Capsomeres are 8 to 9 nm in diameter. Membrane structures are associated with viral areas in the cytoplasm (Bonami, 1973; Bonami and co-authors), 1976). The P virus is a putative reovirus (Bonami, 1973, 1976; Bonami and co-authors, 1976).

Experimental inoculation of purified P virus into crabs results in trembling of the legs in 6 days in more than 60 % of exposed individuals, and increase in severity of the signs by the 9th day following injection. As the disease progresses the paralysis becomes generalized and mortality in infected groups in repeatedly 70 to 85 % (Bonami, 1973).

P virus infection of *Macropipus depurator* causes a disease characterized clinically by slowly developing paralysis, slight darkening of the exoskeleton and death (Vago, 1966; Bonami, 1973; Bonami and co-authors, 1976).

The P virus attacks connective tissues and probably hemocytes with heavy involvement of the gill and interstitial tissues of the hepatopancreas and mid-gut. Paracrystalline arrays of virus particles that are 5 to 10 μm in size occur in the cytoplasm of infected connective tissue cells (Bonami and co-authors, 1976). The hepatopancreas or mid-gut epithelium is not infected. Severe destruction of gill tissues has been reported (Bonami and co-authors, 1976). Johnson (1983) pointed out that the presence of the paralysis virus in nervous tissues is unknown. Nevertheless, the signs associated with the disease implicate infection of the nervous system.

The P virus occurs as a dual infection with the S virus of *Macropipus depurator* (Bonami, 1973, 1976; Bonami and co-authors, 1976). While the 2 viruses are present in the same organs, each infects different cell types. Simultaneous inoculation of the paralysis virus and the S virus, or inoculation of either of these viruses into crab hosts previously infected with the other agent results in disease signs and high mortality (Bonami, 1973).

The impact of the paralysis virus in wild populations of *Macropipus depurator* is not known.

Johnson and Bodammer (1975) initially reported a cytoplasmic reo-like virus (RLV) disease of the blue crab *Callinectes sapidus*. RLV is known from laboratory-reared juvenile-to-adult, male and female crabs collected from Chincoteaque Bay, Virginia (USA), a high salinity area; and the Tred Avon River, a low salinity tributary of Chesapeake Bay, Maryland (USA). However, the prevalence of the disease in wild blue crab populations in these areas is undetermined (Johnson, 1983).

Blue crab RLV is a nonenveloped, icosahedral 55 to 60 nm particle (Johnson and Bodammer, 1975; Johnson, 1983). The agent has not been studied in purified preparations. An electron-dense nucleoid-like structure is present in the central area of RLV virions. The capsid is composed of 7 nm-sized circular subunits (Johnson and Bodammer, 1975; Johnson, 1983). RLV forms cytoplasmic paracrystalline arrays (Fig. 3-8 a-d) that stain Feulgen-negative (Johnson and Bodammer, 1975). These features and the virion size are the basis for tentative assignment RLV to the reovirus group. The biochemical and further biophysical features of RLV are not reported.

Blue crabs with RLV disease were concurrently infected by RLV and one or more other pathogens including a rhabdo-like virus (RhVA); an enveloped helical virus (EHV); the baculovirus, Baculo-A; the nuclear virus, Baculo-B; and an unidentified microsporidian (Johnson, 1977a; Johnson and Farley, 1980). Johnson (1983, 1984a, 1988d) concluded

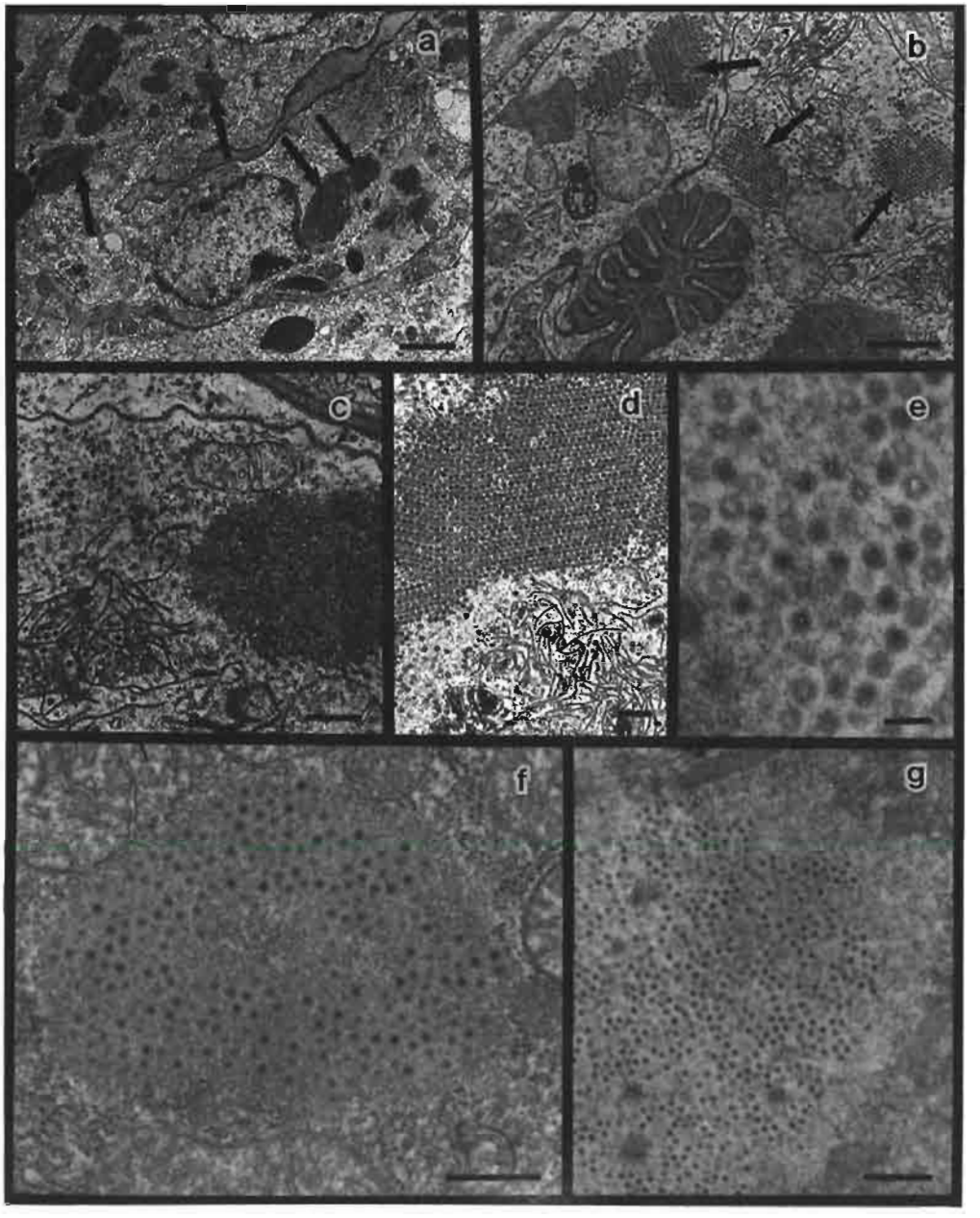

Fig. 3-8: Reo-like virus infections in crabs (RLV) and shrimp (REO). (a and b) Low magnification TEMs of RLV (arrows) in the cytoplasm of cells making up the wall of a blood vessel in *Callinectes sapidus;* bars = 0.5 μm (a) and 250 nm (b). (c) TEM of sinuous strands associated with development of RLV in *C. sapidus;* bar = 100 nm. (d) TEM of a paracrystalline array of RLV and an adjacent cluster of sinuous strands in *C. sapidus;* bar = 100 nm. (a to d by courtesy of P. T. Johnson.) (e) High magnification TEM of a reo-like (REO) virus in the HP of *Penaeus japonicus;* bar = 100 nm. (f to g) Low magnification views of REO in the cytoplasm of HP cells of *P. japonicus* cultured in France (f) and in Hawaii (g); bars = 0.5 μm. (e to g Originals.)

that RLV and RhVA act synergistically to result in the observed clinical signs of RLV disease. Crabs experimentally infected by inoculation of hemolymph containing RLV and RhVA died as early as 3 days post injection; while crabs fed infected tissues died after 12 to 32 days (Johnson, P. T., 1978, 1983). Groups of RLV-RhVA infected crabs held in captivity showed a continuous mortality over a 1 to 2 month period. Disease signs first started after 9 days in captivity and include trembling of appendages, disorientation, paralysis, sluggishness, inappetence and disruption of the normal molt cycle (Johnson and Bodammer, 1975; Johnson, 1977a, 1988d). Gross lesions include discolored exoskeleton; red-to-brown colored gills fouled by epizoic organisms; and pale, small hepatopancreata. Reduced clotting to the hemolymph with normal hemocyte aggregation also characterizes the disease (Johnson, 1988d).

On light microscopy examination of stained tissue sections, hemocytes and hematopoietic tissues are universally affected (Johnson, 1983). The hematopoietic tissues are hypertrophied and necrosis of cells is found, as are abnormal mitotic figures and multinucleate hyaline hemocytes (Johnson and Bodammer, 1975; Johnson, 1983, 1988d). Focal areas of tissue damage (necrosis, hemocyte invasion) are present in the gill epithelium and epidermis, and extensive damage to the brain, various ganglia, associated nerves and nerves within the heart are reported (Johnson, 1977a, 1983). Marked invasion of nervous tissue including the eyestalk nerves by hyaline and granulocytic hemocytes is commonly found, as is extensive disruption, lysis and necrosis of glial elements and ganglia. While the hepatopancreas epithelium is unaffected the interstitial tissues are hypertrophied and cells are vacuolated and necrotic. Also not directed affected are the mid-gut epithelium, epithelium of the caeca and the gonad and Y-organ tissues (Johnson, 1977a, 1983). With H & E staining, the cytoplasm of heavily infected cells is basophilic and homogeneously-opaque. Basophilic, angular cytoplasmic inclusions may be present. These inclusions are Feulgen and Periodic Acid-Schiff (PAS) negative and are not alcianophilic (Johnson, 1977a).

Ultrastructurally, the cytoplasmic inclusions are made up of paracrystalline arrays (Fig. 3-8, a, b, d) of RLV virions (Johnson, 1977a). The virions are often associated with microtubules and microfilaments (Fig. 3-8,c,d); (Johnson and Bodammer, 1975).

The impact of RLV disease in natural blue crab populations is unstudied (Johnson, 1977a).

A putative reovirus infection of gill epithelium of *Carcinus mediterraneus* was reported by Bonami (1976). The agent is nonenveloped, paraspherical virus approximately 55 nm in diameter that localized within the cytoplasm of gill epithelium. Infected gill epithelial cells are destroyed (Bonami, 1976). The putative reo-like virus associates in groups of 6 and forms characteristic rosettes (Mari and Bonami, 1986). Inoculation of purified virus into crabs or exposure of susceptible crabs to infected tissues results in death in 8 days (Bonami, 1976). This infection of gill epithelium has not been encountered in more recent studies of *C. mediterraneus* (Mari and Bonami, 1986). The distribution of the putative reo-like virus in wild crab populations and the impact of the virus on natural crab populations have not been reported.

A second reo-like virus was reported from *Carcinus mediterraneus*. The agent has been designated virus W2 (Mari and Bonami, 1986) and infection of interstitial cells of hepatopancreas, mid-gut, gills and the hemocytes has been found. W2 localizes within the cytoplasm and is 55 to 60 nm in diameter in tissue section, and 65 to 70 nm in negative

contrast preparations (Mari and Bonami, 1986). W2 also forms rosettes in groupings of 6 particles.

The ability of W2 virus to cause disease and the distribution of this virus in natural populations of *Carcinus mediterraneus* have not been reported.

A similar cytoplasmic reo-like virus, designated W virus, apparently infects the hepatopancreas epithelium of *Carcinus maenas* (Mari and Bonami, 1986). W virus, like W2, also arrays in rosettes. Additional information on W virus has not been published.

A third reo-like virus, designated RC84, infects the cytoplasm of hepatopancreas epithelium of *Carcinus mediterraneus* (Mari and Bonami, 1986). RC84 localizes principally in the B-cells, but the virus has been observed in the R-cells as well (Mari and Bonami, 1986). Electron microscopy reveals viral areas up to 8 μm in length with the virus arrangement in not any particular way although paracrystalline arrays are at times present. RC84 is 70 to 75 nm in diameter with a 15 nm outer layer and 50 nm dense core (Mari and Bonami, 1986). The effect of RC84 on the crab host and the distribution of the virus in crab populations has not been studied.

Penaeid shrimp reo-like virus (REO) is a nonenveloped, icosahedral cytoplasmic virus with an average size of 61 nm in purified preparations (Tsing and Bonami, 1987) to 50 to 70 nm in tissue sections (Tsing and Bonami, 1987; Lightner, 1988 his Chapter 3.1.6; Nash, M. B. and co-authors, 1988). While the nuclei acid type was given as ds-RNA by Lightner (1988), data to confirm this have not been published. REO is believed to be an RNA virus because of its cytoplasmic location and the Feulgen-negative staining characteristics of the presumed REO cytoplasmic inclusion bodies reported by Lightner (1988). In the only report found on the subject, attempts were unsuccessful at demonstration of viral RNA from REO infected tissues (Clerx and Lightner, 1985). The REO capsid appears to form 2 shells (Tsing and Bonami, 1987). Biochemical and additional biophysical properties of shrimp REO virus have not been reported.

The penaeid shrimp reo-like virus is known from juvenile through adult *Penaeus japonicus* cultured in France and Hawaii, and from subadult *P. monodon* cultured in Malaysia (Tsing and Bonami, 1986, 1987; Lightner, 1988 his Chapter 3.1.6; Nash, M. B. and co-authors, 1988). REO infection of penaeid larvae has not been reported as has been the occurrence of the virus in wild shrimp populations.

Shrimp REO virus was transmitted experimentally by injection of purified virus and oral feeding of crushed hepatopancreas tissues to unspecified-sized *Penaeus japonicus* (Tsing and Bonami, 1986, 1987). Mortality and diagnosis of REO infection occurred in all trials after 5 to 6 weeks from initial exposure to REO infected tissues. There was no mention of control groups for these trials. The investigators state that the slow development of the disease is an indication of the limited pathogenicity of REO virus in *P. japonicus*.

The significance of REO infection for health in juvenile-to-adult *Penaeus japonicus* and *P. monodon* is not clear. Tsing and Bonami (1987) observed disease signs and mortality associated with REO infection of *P. japonicus*, but these investigators state that the virus may have limited pathogenicity and acts primarily as a stress factor that predisposes the host shrimp to *Fusarium* disease. Further evidence of low virulence presented by Tsing and Bonami (1987) include finding REO virus in tissues of clinically normal individuals; a prolonged incubation period in experimental infection trials and the absence of a disease problem at the fish farm where they obtained the REO-infected *P.*

japonicus used in their laboratory studies. From the data presented, it is not clear that these investigators had REO-free shrimp for use as controls in their studies. This further complicates an interpretation of the results presented.

An uncertain relationship between REO virus and disease in the shrimp host is also reported for *Penaeus monodon* (Nash, M. B. and co-authors, 1988). Reovirus infection was found concurrently with infection of *P. monodon* by other pathogens including MBV, a rickettsia and Gram-negative bacteria. The discovery of the reovirus was an unexpected finding in the course of the disease study (Nash, M. B. and co-authors, 1988), and the role of the REO virus as a primary pathogen of *P. monodon* remains unclear.

Shrimp reovirus has been suggested (Tsing and co-authors, 1985; Lightner, 1988 his Chapter 3.1.6) to be an etiologic factor in the idiopathic disease 'gut and nerve syndrome' (GNS) described by Lightner and co-authors (1984) in *Penaeus japonicus* cultured in Hawaii. At present, this hypothesis is supported by association only. That is, GNS and REO virus infection were found concurrently in populations of cultured *P. japonicus*. However, studies have not been conducted which demonstrate REO exposure results in the GNS cytopathology. Moreover, this situation is complicated as REO-free *P. japonicus* were apparently not available for use as controls in infection trials that have been carried-out to date. Thus, the relationship between REO and GNS will remain equivocal until infection trials can be undertaken with REO-free *P. japonicus*.

Tsing and Bonami (1986, 1987) report the signs and gross lesions of REO virus infection of *Penaeus japonicus* as reduction in sand burying behavior and increased reddish color of telson, uropods and hepatopancreas; while Lightner (1988 his Chapter 3.1.6) found inappetence, lethargy, reduced preening (evidenced by increased epicommensal fouling), and opacity of the abdominal muscle as the signs and lesions of REO infection.

Shrimp reovirus localizes in the cytoplasm of the F and R cells of the hepatopancreas (Tsing and Bonami, 1987; Lightner, 1988 his Chapter 3.1.6; Nash, M. B. and co-authors, 1988). By light microscopy reovirus infection is easily overlooked because the eosinophilic to magenta-staining cytoplasmic inclusion in atrophied hepatopancreas cells are a variable finding (Lightner, 1988 his Chapter 3.1.6). Nash, M. B. and co-authors (1988) report cytoplasmic degeneration, focal rounding-up and necrosis of hepatopancreatic cells with luminal sloughing of these cells, but the role of REO in these changes is not clear because of other pathogens, including numerous intra-luminal bacteria, were occasionally observed invading hepatopancreas cells and concurrent MBV infection of the hepatopancreata of these shrimp.

By electron microscopy, Tsing and Bonami (1987) report large REO viral areas (Fig. 3-8, e-g) within the cytoplasm of R and F cells without any crystalline arrangement, the virus particles being spread among an electron dense material. Viral arrays, at times in lattice-like arrangements, were reported by Nash, M. B. and co-authors (1988). These researchers noted a ca 40 nm dense nucleoid (core) in the virus particle.

At present, diagnosis of reovirus infection must be based on demonstration of the virus by transmission electron microscopy (Lightner, 1988 his Chapter 3.1.6; Lightner and Redman in press). The magenta to eosinophilic cytoplasmic inclusions in hepatopancreatocytes are an uncommon finding and cannot, therefore, be depended upon as a sensitive indicator of the presence of shrimp REO infection. Immunologic methods for detection of this agent are not developed. Such sensitive, specific probes would aid greatly the recognition of REO-infected, as well as REO-free groups, of *Penaeus japonicus* and *P.*

monodon, and would also be helpful in detection of REO presence in other penaeid species and distribution in wild-populations of shrimp.

Lightner (1988 his Chapter 3.1.6) recommends avoidance as the means of control for REO in *Penaeus japonicus*. However, the location or distribution of REO-free stocks of *P. japonicus* is unknown. Nash, M. B. and co-authors (1988) suggest future studies on penaeid shrimp REO virus should include pathogenicity trials and attempts to isolate and culture the agent.

Kuris and co-authors (1979) described dual infection by a putative picornavirus and a larger, reo-like virus (Johnson, 1983, 1984a) in cells of the entoniscid isopod *Portunion conformis*, a parasitic castrator. The diameter of the un-enveloped, icosahedral reo-like virus was 58 nm in sectioned material and 61 nm in negatively stained extracts (Kuris and co-authors, 1979). These authors were uncertain if the reo-like virus was pathogenic to the isopod host.

Viral agents resembling birnaviruses are reported to be isolated from 2 marine decapods. The Birnaviridae look like the Reoviridae morphologically but differ in that birnaviruses are somewhat smaller, have a different capsid morphology and are biophysically and biochemically quite distinct from the Reoviridae (Johnson, 1984a). Viruses in these 2 families cannot be reliably differentiated by electron microscopy examination of thin tissue sections (Johnson, 1984a).

A well known birnavirus is the infectious pancreatic necrosis virus (IPN) of salmonid and other fishes. IPN has been recovered from marine molluscs, and Hill (1976, cited in Johnson, 1984a) reported the isolation in fish cell cultures of a birnavirus from tissues of the European shore crab *Carcinus maenas*. Additionally, Bovo and co-authors (1984, cited in Bonami, 1987) reported the isolation in fish cells of an IPN-like virus from an adult Kuruma shrimp *Penaeus japonicus*. No further information on birnaviruses from marine decapods is apparently available and the impact of birnavirus infections on decapod health is unknown. In view of the similar morphological appearance of the reoviruses and birnaviruses, it seems prudent to carry-out standard cell culture virus recovery tests on marine decapod tissues found with putative reovirus infection.

Johnson, P. T. (1978) reported infection by a cytoplasmic virus associated with a lethal disease in a captive group of about 200 juvenile blue crabs *Callinectes sapidus*, collected during summer from Tangier Sound in the Chesapeake Bay (USA). Crabs in this group were used in nutritional studies and over a 2 month period most of the crabs died from the virus infection. The virus is designated Chesapeake Bay virus (CBV) or blue crab picorna-like virus (Johnson, P. T., 1978, 1983).

The Chesapeake Bay virus (Fig. 3-9g) is a non-enveloped icosahedron ca 30 nm in diameter (Johnson, P. T., 1978, 1983). CBV replicates within the cytoplasm where large inclusion bodies composed entirely of virus particles are formed. Paracrystalline arrays of virus are often observed in infected cells (Johnson, 1983). The aggregations of virus particles are Feulgen-negative. This and site of replication is presumptive evidence that the virus nucleic acid is RNA (Johnson, 1983). The core of the particle may be either electron dense or lucent. The core is sourrounded by a layer of dark granules or short rods that appear to be connected to spikes that project from the surface of the virus (Johnson, 1983). CBV has not been isolated in purified preparations. The biochemical and additional biophysical features of this virus are unknown.

The CBV has been reported from blue crabs captured from Tangier Sound. In the

captive group, tissues from 3 of 5 crabs studied with the electron microscope also had concurrent infection by one or more viruses including a rhabdo-like virus (RhVA), the enveloped helical virus (EHV) and RLV (Johnson, 1983).

Diseased crabs exhibit abnormal behavioral signs distinctive for CBV disease. Disoriented swimming, erratic movements, head-down position at rest are reported (Johnson, P. T., 1978, 1983). Furthermore, the molt pattern is disrupted and often CBV infected crabs are blind. The course of the disease in individual crabs is variable and may be quite leisurely, taking a month or more after onset of abnormal behavioral signs before death occurs (Johnson, P. T., 1978).

The CBV virus primarily infects tissues of ectodermal origin (neurons, retina, gill and general epidermis, stomach and hindgut epidermis, antennal gland and bladder epithelium), less frequently tissues of mesodermal origin (hemocytes and hematopoietic) Nervous-system connective tissues, muscle and midgut epithelium were not infected (Johnson, 1984a).

Microscopic pathology found in CBV disease include hypertrophy of infected cells that contain cytoplasm filled with a homogeneous, Feulgen-negative material (Fig. 3-9f). CBV lesions are often focal, but may be diffuse in certain organs, and massive destruction of the epithelium of the gill or the entire retina is common (Johnson, 1984a). Host response to CBV infection appears limited. Podocytes of the gill and antennal gland contain dense Feulgen-negative inclusions. However, these inclusions lack intact virus and are of uncertain origin and significance (Johnson, 1984a).

Ultrastructurally, infected cells have cytoplasm engorged with CBV particles. Large, Feulgen-negative inclusions are formed and paracrystalline arrays of virus particles occur (Johnson, 1983).

A presumptive diagnosis of CBV disease of blue crabs can be made based on the clinical signs and light microscopy findings of hypertrophied cells with dense Feulgen-negative cytoplasm. Confirmation requires demonstration of the virus by EM (Johnson, 1984a).

The impact of CBV in natural blue crab populations has not been investigated.

A putative picornavirus infection of the parasitic entoniscid isopod *Portunion conformis,* a parasitic castrator, and the grapsid shore crab *Hemigrapsus oregonensis* is reported (Kuris and co-authors, 1979). This was the first report of virus infection in a parasitic crustacean, as well as in the order Peracarida, and the second published documentation of a picornavirus infection in Crustacea.

Hemigrapsus oregonensis were collected in January and October, 1977 from San Francisco Bay. Captured crabs were grossly normal except for internal isopod infection. Three virus-infected *Portunion conformis* were examined by electron microscopy, each had dual infection by 2 spherical viruses located within the cytoplasm of cells. Biophysical attributes of the viruses were studied after extraction of hepatopancreas tissues from 21 crabs hosts (pooled) and from 42 pooled parasites. Recovery of the viruses was by differential centrifugation.

Kuris and co-authors (1979) found cellular infection in *Portunion conformis* by the 2 viruses to be widespread. The viruses were especially abundant in the epidermal layer and the hepatopancreas, but particular cell-types infected were not reported (Johnson, 1983). Virus particles nearly completely filled the cytoplasm of infected cells and subcellular organelles were lacking. The smaller particle was 25 nm in sectioned material, more

abundant that the larger particle and often occurred in (conspicuous), irregular shaped crystalline arrays. This virus measured 29 nm in negative stained extracts, appeared identical in crab host and parasite extracts, and had 3 major capsid proteins with a sedimentation coefficient of 160 to 165S (Kuris and co-authors, 1979).

Dual infection by both types of viruses occurred in the same cells of *Portunion conformis*. The larger virus was not recovered from the tissues of the crab host (Kuris and co-authors, 1979).

The impact of the putative picorna-like and reo-like viruses on the isopod and/or the crab host is unknown. Infected parasites and crabs appeared healthy at the time of dissection. However, Kuris and co-authors (1979) indicated that dead isopod parasites had been recognized in different crab populations along the Pacific coast and suggested that isopod mortality should be studied in relation to virus infection.

The infection by the picornavirus in 2 unrelated crustacean hosts in unique (Johnson, 1983) as is the mode of transmission for both viruses. Kuris and co-authors (1979) provided hypothetical explanations concerning the transmission and suggest that further studies on the host-agent relationships are warranted.

An acute, highly contagious lethal disease of the Pacific blue shrimp *Penaeus stylirostris* was first recognized in 1980 in the University of Arizona's experimental shrimp culture facility on Oahu, Hawaii. Epidemiological and pathological studies undertaken by D. Lightner and coinvestigators soon revealed the viral etiology, appropriately designated infectious hypodermal and hematopoietic necrosis (IHHN) for the primary lesions encountered on light microscopy examination of shrimp dying from the disease (Lightner and co-authors, 1983b).

IHHN virus is a un-enveloped icosahedral agent (Fig. 3-9d, e) that measures 17 to 27 nm in tissue sections and 20 to 22 nm in purified preparations (Lightner and co-authors, 1983b; Lightner and Redman, in press). Replication occurs within the cytoplasm where the virus is usually found in small aggregates (27 nm-diameter particles) and rarely in membrane-bound inclusions with paracrystalline arrays (17 nm-diameter particles) (Lightner and co-authors, 1983b). IHHN virus appears to have a single stranded polyadenylated RNA genome, 7–8 kilobases in length (Clerx and Lightner, 1985). Lu and co-authors (1989) confirm the nucleic acid type as RNA by colorimetric analysis (orcinol test) and found the RNA content is 4.7 % by weight of the virus particle. The buoyant density of IHHNV in CsCl is reported to be 1.33 g cm^3 (Lu and co-authors, 1989). IHHNV can be frozen and thawed and retain its infectivity and the virus can survive for more than 5 years when stored frozen at −20 or −70 °C (Lightner and Redman, in press). Also, IHHNV retains its infectivity in tissues stored in 50 % glycerol for at least 14 days (Lightner and co-authors, 1987b). This provides a convenient method for transport of IHHNV-infected tissues to the laboratory. Other biophysical and biochemical features of IHHN virus have not been reported.

IHHN virus is infectious for all species of penaeid shrimp tested thus far. However, various shrimp species have dissimiliar susceptibility to disease from this agent (Lightner and co-authors, 1983a,b,d; Brock and co-authors, 1983; Lightner, 1985; Lightner and Redman, in press). *Penaeus stylirostris* is highly susceptible to disease from IHHNV (Lightner and co-authors, 1983a,b); *P. monodon* is less so, and *P. vannamei, P. setiferus, P. aztecus* and *P. duorarum* are regarded as least prone to disease from this virus (Lightner and co-authors, 1983d; Lightner and Redman, in press). It is not known if IHHN is infectious for other invertebrate or non-invertebrate hosts.

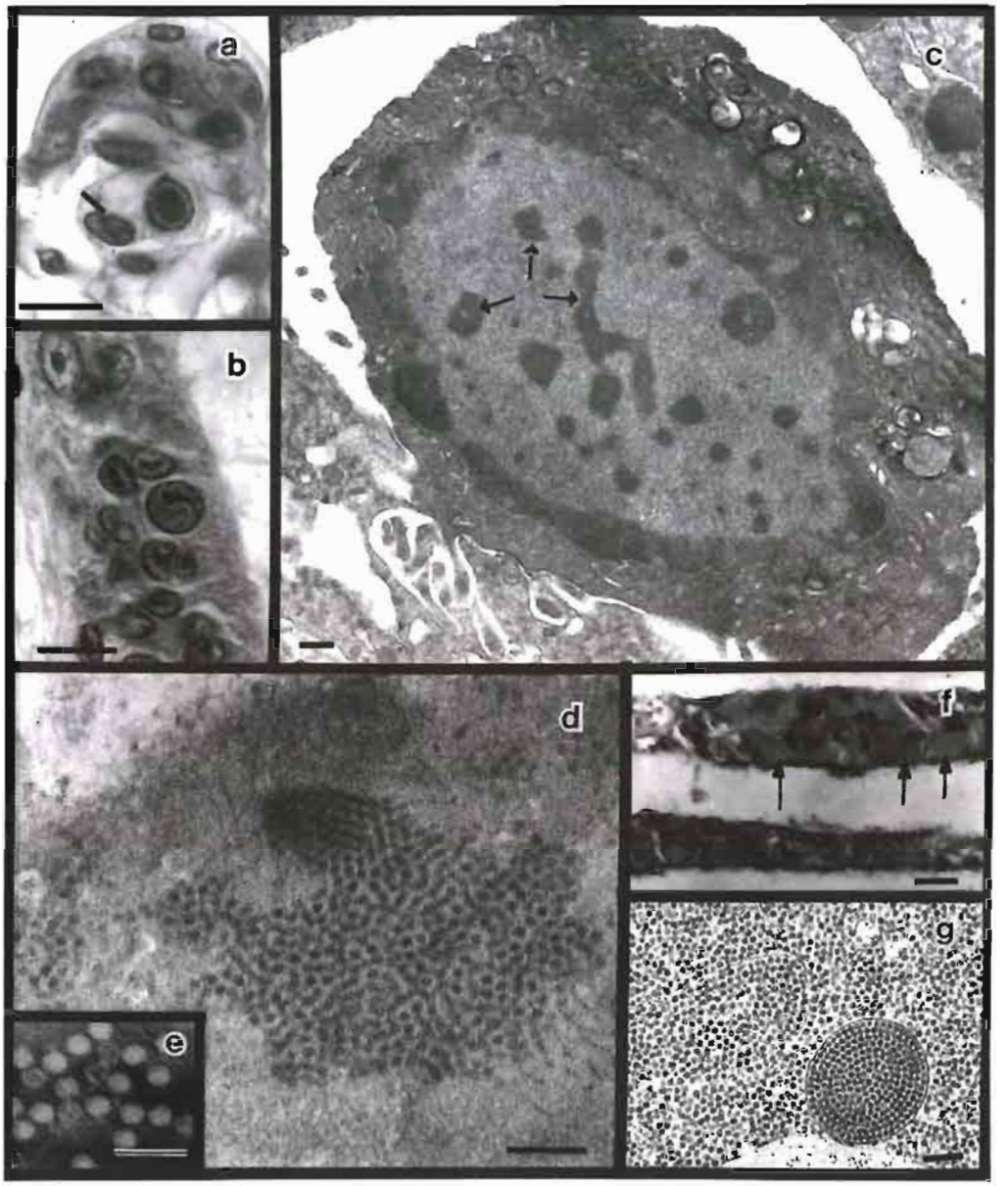

Fig. 3-9: IHHN and CBV viruses. (a and b) Light micrographs of IHHN pathognomonic Cowdry type A eosinophilic intranuclear inclusion bodies in gill and foregut cuticular hypodermis of *Penaeus stylirostris;* H&E; bars = 25 μm (a) and 10 μm (b). (After Lightner, 1988. Reprinted with the permission of Elsevier Science Publishers.) (c) TEM of an IHHN intranuclear inclusion body in a hemocyte of *P. stylirostris;* nuclear chromatin displaced against the nuclear membrane, while the center of the nucleus has become filled with a proteinaceous granular matrix that contains electron-dense spheres and strands (arrows); bar = 0.1 μm. (d) TEM of a paracrystalline array of ca 20 nm IHHNV particles in the cytoplasm of a hemocyte from *P. stylirostris;* bar = 50 nm. (c and d Originals.) (e) TEM of purified IHHN virions from cesium chloride density gradient centrifugation; 2% PTA; bar = 50 nm. (f) Light micrograph of gills from the *Callinectes sapidus* infected with CBV, a picorna-like virus; large cytoplasmic inclusions are evident in several cells (arrows); alcian blue and nuclear fast red; bar = 20 μm. (e and f by courtesy of P. T. Johnson) (g) TEM of CBV virions from the bladder epithelium of *C. sapidus;* bar = 155 nm. (Original.)

IHHN virus is carried latently in IHHN-disease resistant penaeids as well as early life stages prior to the onset of disease signs. IHHNV has been widely disseminated geographically through transfers of latent to patently infected post larval and other life stages of shrimp (Brock and co-authors, 1983; Lightner and co-authors, 1983b,d; Bell and Lightner, 1987b). Presently, IHHNV is distributed worldwide in penaeid culture; interestingly, its natural reservoir host(s) and range in wild penaeid populations is undocumented (Lightner, 1985; Lightner and Redman, in press). These authors present evidence that suggests *P. monodon* may be a natural reservoir host for IHHNV.

The IHHN virus is transmitted horizontally by exposure to infected water, *per os* ingestion of infected shrimp and by direct injection of the virus (Bell and Lightner, 1984). The virus is also thought to be transmitted vertically from infected broodstock shrimp to their offspring, but this has not been proven nor has the means of transmission been established by controlled study data.

The reported signs of IHHN disease are based principally on observations made with juvenile cultured *Penaeus stylirostris*. Clinically affected juvenile *P. stylirostris* display weak, erratic, inverted, surface swimming; eventually they stop movement and slowly sink to the tank bottom with a slowed righting response. Other signs are nonspecific and include anorexia, lethargy and weakness. Mortality may exceed 90 % (Fig. 3-10) within several weeks of onset of mortality in 0.05 to 2.0 g *P. stylirostris* (Bell and Lightner, 1987b). Gross lesions of IHHN infection are white to buff, mottling the cuticle; further

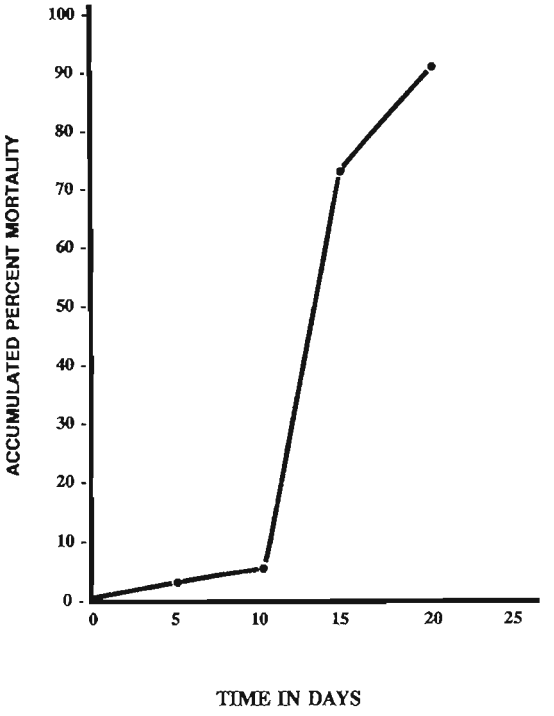

Fig. 3-10: IHHNV epidemic in intensive cultured juvenile *Penaeus stylirostris*. Accumulated percent mortality over a 20 day period. (After Lightner and co-authors, 1983b; redrawn. Reprinted with the permission of Academic Press, Inc.)

observed were generalized opacity of striated muscles, soft cuticle and — in chronic infections — multifocal, tiny melanized foci within the cuticle hypodermis (Bell and Lightner, 1987b).

The microscopic pathology of IHHN is characterized by multifocal areas of cellular necrosis, nuclear hypertrophy, pyknosis, karyorrhexis and Cowdry-Type-A intranuclear inclusions (Fig. 3-9, a-c) within cells of ectodermal (neurons, fore and hindgut, antennal gland, gills and general cuticle epidermis) or mesodermal (hematopoietic tissue and hemocytes, striated muscle, heart, lymphoid organ and connective tissues) origin (Lightner and co-authors, 1983b). Infection of midgut epithelium occurs, but is an uncommon finding (Lightner and co-authors, 1983b; Bell and Lightner, 1987b).

Diagnosis of IHHN disease is based on histopathological demonstration of Cowdry Type A inclusions in tissues of ectodermal or mesodermal origin. For Cowdry A inclusions to be visible (ie., take up eosin stain shrimp must be in an acid fixative (Lightner and co-authors, 1983a) such as Davidson AFA (Humason, 1979). These inclusions and associated cytopathologic changes are widespread in shrimp showing clinical signs of IHHN disease. However, most shrimp species carry IHHN virus as a latent infection. Therefore, determination of infection in subclinically affected shrimp or groups of shrimp requires use of enhancement procedures and/or indicator shrimp bioassay (Lightner and co-authors, 1983a,b,d; Lightner and Redman, in press). Bell and co-authors (in press) indicate that histological examination of peripheral trunk nerves in a pereiopod appendage can be used to successfully identify IHHNV infection in carrier adult *Penaeus vannamei*. The procedure has the advantage that sacrifice of the shrimp is not necessary and sensitivity of the test is similar to the more time consuming and expensive multiorgan histological examination. However, indicator bioassay is presently the most sensitive method available and involves *per os* or inoculation transmission of IHHNV to the sensitive shrimp host species, *P. stylirostris*. Post exposure incubation ranges from 14 to 30 days when test and the control indicator shrimp are sacrificed and examined histologically for Cowdry A inclusions. Tissue culture or specific immunological methods have not yet been developed for IHHN virus diagnosis. Such procedures are needed so that rapid, specific diagnosis of this virus infection can be made (Brock and co-authors, 1983; Lightner and co-authors, 1983d; Lightner, 1985; Lightner and Redman, in press).

Published information is limited on management and control procedures for IHHN disease in shrimp culture. Avoidance of the virus through quarantine and inspection of shrimp stocks is recommended (Brock and co-authors, 1983; Lightner and co-authors, 1983b; Lightner, 1985, 1988 his Chapter 3.1.1), but success rates for this strategy have not been documented. Management procedures such as culture of IHHNV-disease resistant species or strains of shrimp, lowered stocking density, ideal environmental conditions and use of nutritionally balanced feeds are options that, if available, may be implemented to reduce the disease impacts from IHHN virus. Specific treatments or vaccination protocols for IHHN disease of marine shrimp are unknown.

Other possible picorna-like viruses of marine crustaceans are reported. Bonami (1976) found in a moribund *Macropipus depurator* 2 types of un-enveloped paraspherical virus-like particles, 24 and 31 nm in diameter. Experimental inoculation of semi-purified virus preparations into normal crabs resulted in the diseases signs. The disease is termed V31-24 complex disease of *M. depurator* (Bonami, 1976; Johnson, 1983). No further information on this disease is apparently available. Tsing and Bonami (1987) report other

small viral particles 25 to 30 nm in diameter during the course of purification of the reo-like virus of *Penaeus japonicus*. According to these authors further work on the nature of these viral particles is in progress.

Four rhabdo-like viruses are reported from decapod crustaceans, all are known from marine crabs. The decapod rhabdo-like viruses include 3 agents identified from tissues of the blue crab *Callinectes sapidus* — rhabdo-like virus A-RhVA (EGV-2) (Jahromi, 1977; Yudin and Clark, 1978, 1979a, b; Johnson, 1983), rhabdo-like virus B-RhVB (EGV-1) (Yudin and Clark, 1979b) and the enveloped helical virus (EHV) (Johnson and Farley, 1980; Johnson, 1983, 1984a), and the Y-organ virus of *Carcinus maenas* (Chassard-Bouchard and Hubert, 1975; Chassard-Bouchard and co-authors, 1976). Only one of the agents, the RhVA (EGV-2), and apparently only in association with RLV, is implicated as a cause of disease signs in the host (Johnson, 1983, 1984a, 1988d). The other agents are of no known significance as pathogens to their crab hosts.

The blue crab rhabdo-like viruses A and B (RhVA & RhVB) were so named by Johnson (1983). RhVA was originally reported by Jahromi (1977), and later rediscovered by Yudin and Clark (1978, 1979a, b) along with RhVB, during an ultrastructure study of the mandibular gland, which they mistakenly identified as the ecdysial gland (Johnson, 1983). Yudin and Clark (1978, 1979a, b) named the 2 rhabdo-like viruses Ecdysial gland virus 1 (EGV-1) and ecdysial gland virus 2 (EGV-2). However, as the viruses were not found in the ecdysial gland cells, Johnson (1983) renamed the agents rhabdo-like virus A (RhVA) and rhabdo-like virus B (RHVB) for EGV-2 and EGV-1, respectively. Blue crab RhVA was discovered by Jahromi (1977) during the course of an electron microscopical study of neuromuscular junctions of the gastric mill muscles of blue crabs. Yudin and Clark (1978, 1979a, b) also found the same virus during an electron microscopical study of the mandibular organ. Johnson (1983) reported RhVA in other organ systems as well.

Rhabdo-like virus A (Fig. 3-11, a, b, d) develops within the cytoplasm and is bacilliform, 25 to 30 nm by 100 to 150 nm with 2 rounded ends (Yudin and Clark, 1978, 1979a, b). Johnson (1983) reported the virus can be long, up to 600 nm, and flexuous. The core of RhVA is generally electron lucent (Yudin and Clark, 1979a, b), but at times electron dense areas are observed (Johnson, 1983). An electron-dense ring about 4 nm in thickness surrounds the core, and a granular layer is outside the ring (Johnson, 1983). The morphogenesis and structure of RhVA suggest it is a Rhabdovirus, but the small diameter is not characteristic of Rhabdoviridae (Johnson, 1983). The virus has not been isolated in purified preparations and the biochemical and biophysical features of RhVA are unreported.

RhVA apparently has a wide distribution in blue crabs as infected crabs have originated from Chesapeake Bay, Maryland and Chincoteague Bay, Virginia (Johnson, 1988d); probably other areas along the Atlantic coast (Johnson, 1983); and the East Lagoon region of Galveston, Texas (Yudin and Clark, 1978, 1979a, b).

Manifestation of RhVA infection, that is, the apparent proliferation of the agent in host cells, is considered to be stress-mediated by the investigators that have studied this virus infection (Jahromi, 1977; Yudin and Clark, 1978, 1979a, b; Johnson, 1983, 1984a, 1988d). Stressors that are implicated in activation of the virus include transport and holding in artificial seawater (Jahromi, 1977); bilateral eyestalk ablation or infection by a second Rhabdo-like virus (RhVB) (Yudin and Clark, 1978, 1979a, b); or concurrent infection by other viral agents including reo-like virus (RLV), an enveloped helical virus

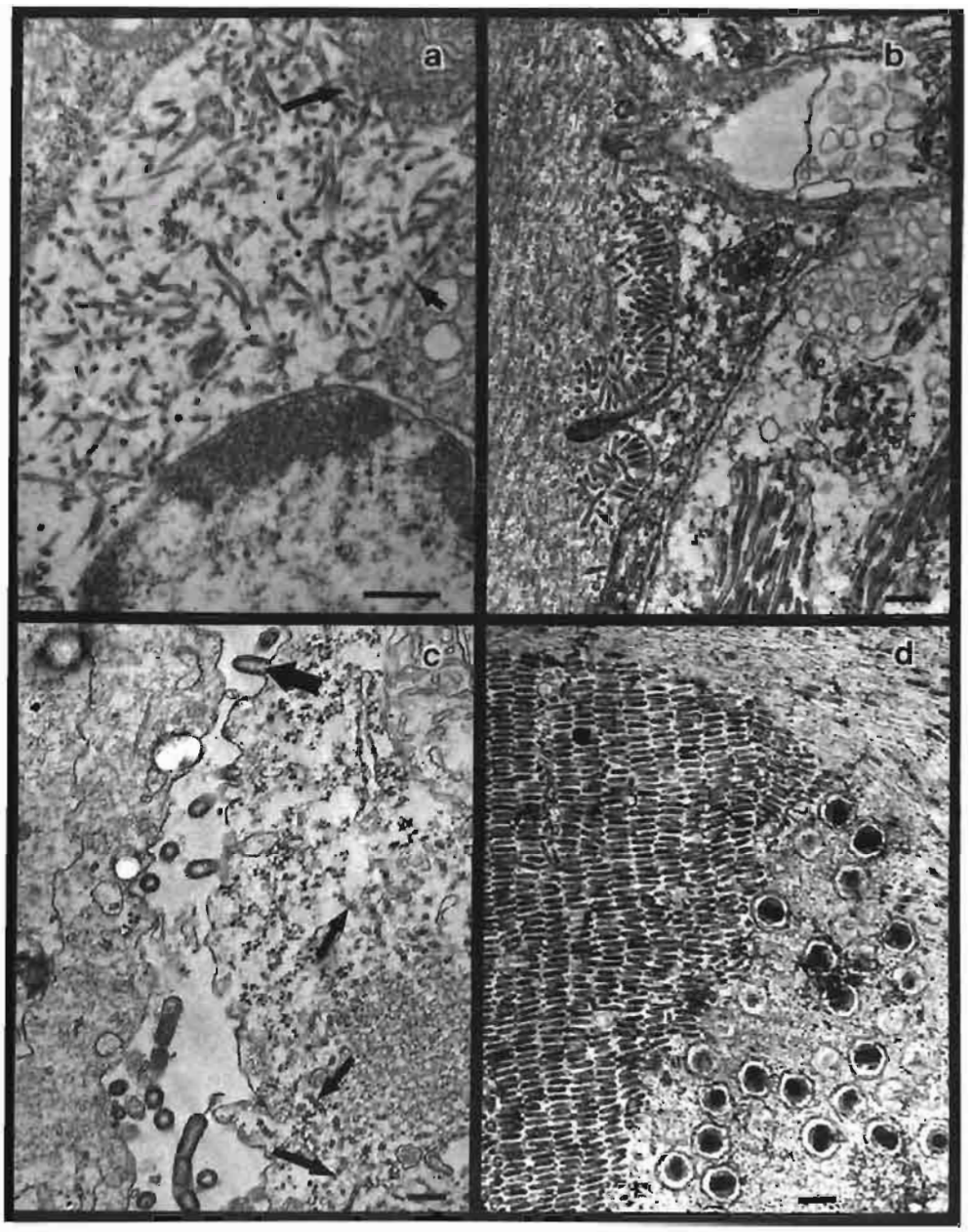

Fig. 3-11: Rhabdo-like virus infection of crabs. (a) Rhabdo-like virus (RhVA) in the nuclear cisterna and endoplasmic reticulum of a *Callinectes sapidus* hemocyte; virions shown arising from the endoplasmic reticulum (arrows); TEM; bar = 200 nm. (b) TEM showing accumulations of RhVA in close association with host cell organelle membranes; bar = 200 nm. (a and b by courtesy of P. T. Johnson.) (c) Enveloped helical virus (EHV) in a *C. sapidus* hemocyte; virions are budding through the plasma membrane (thick arrows), and nucleocapsids are present in the cytoplasm (thin arrow); bar = 200 nm. (After Johnson and Farley, 1980. Reprinted with the permission of Academic Press, Inc.) (d) TEM of a dual infection of a *C. sapidus* hemocyte by RhVA (left) and by the bi-faces virus, bar = 200 nm. (Courtesy of P. T. Johnson.)

(EHV), picorna-like virus (CBV), the Bi-Facies virus (Fig. 3-11, d), and the rod-shaped nuclear virus, Baculo-B (Johnson, 1983, 1984a).

Yudin and Clark (1979a) found the activity of RhVA in ablated blue crabs was transitory. Virus was observed in mandibular gland tissues between Days 3 and 5 post ablation, with no infected glands encountered on Days 1, 2 and 6 of the study.

A disease syndrome attributable to sole infection by RhVA is not recognized. The virus infected crabs studied by Jahromi (1977) and Yudin and Clark (1978, 1979a, b) were behaviorally and physically considered to be normal by these investigators. However, Johnson (1983, 1984a, 1988d) concluded that paralysis of blue crabs infected by RLV maybe in part due to concurrent infection by RhVA.

Rhabdo-like virus A infection of the mandibular organ does not affect the size of the organ (Yudin and Clark, 1979a, b); however, heavily RhVA infected mandibular organ cells and hemocytes can be distinguished by the light microscope because of large accumulations of virus particles within these cells (Yudin and Clark, 1979a, b; Couch, 1981). Other light microscopy changes associated with RhVA infection have not been reported.

Rhabdo-like virus A infects Schwann (glial) cells, endothelial cells, probable fibroblasts and hemocytes associated with the gastric mill muscles (Jahromi, 1977); mandibular organ cells (Yudin and Clark, 1978, 1979a, b); and hematopoietic tissue cells, hemocytes, connective tissue cells, reserve cells associated with connective tissues, epidermis, bladder epithelium, epicardial tissue cells and glial of peripheral nerves and the ventral nerve cord (Johnson, 1983).

Ultrastructurally, RhVA proliferates within the nuclear envelope, the tubular endoplasmic reticulum (TER) and the plasma membrane (Yudin and Clark, 1979a, b). RhVA particles are commonly encountered in a longitudinal or parallel fashion between, but not attached to, the inner and outer membranes of the nuclear envelop. However, it is the TER, which originates from the nuclear envelope, that is the main site of RhVA multiplication. As virus proliferates the TER swells and eventually RhVA-filled vacuoles are formed. These vacuoles, engorged with virus, fuse with the plasma membrane and appear to be a mechanism for release of the virus into the extracellular space. Apparently, RhVA does not proliferate within the smooth endoplasmic reticulum (Yudin and Clark, 1979a, b). Jahromi (1977) described a similar pattern of proliferation within Schwann cells and reported RhVA to be less abundant in other cell types where the virus was observed.

Rhabdo-like virus A particles apparently mature along the outer membrane of mandibular gland cells and extend into the hemal channels. Occasionally, hemal channels were observed filled with free and paracrystalline arrays of particles, but only randomly distributed particles were found in the larger hemal sinuses (Yudin and Clark, 1979a, b). RhVA attached to the plasma membrane of hemocytes and necrotic hemocytes engorged with RhVA particles were occasionally present.

In conjunction with RLV, RhVA appears to be pathogenic to blue crabs held in captivity. However, the impact of RhVA on wild blue crab populations is unknown.

Rhabdo-like virus B was discovered by Yudin and Clark (1978) during an electron microscopical study of mandibular organ. Rhabdo-like Virus B virions are 50 to 70 nm × 100 to 170 nm, bacilliform shaped particles. In cross section the center of the virus is a hollow core surrounded by a nucleocapsid. The envelope has surface projections (Yudin and Clark, 1978, 1979b). The site of and description of RhVB development and the biophysical and biochemical characteristics of RhVB have not been reported.

In Yudin and Clark's material, RhVB was found in 2 of 60 (3 %) of the control, non-ablated crabs; one of these crabs also had a concurrent infection by RhVA (EGV-2) (Yudin and Clark, 1979b). No pathologic alterations to the gland cells' internal ultrastructure was reported from RhVB infection. RhVB was associated with the calyx material that coats the hemolymph sinuses of the mandibular organ or within pockets in the extracellular space positioned between the calyx coat and the plasma membrane. Virus particles were not observed within the cytoplasm of parenchymal cells or found budding through the plasma membrane (Yudin and Clark, 1978, 1979b). These authors conclude that RhVB (EGV-1) probably proliferated and matured in some other tissue and accumulated in the Y-organ via the hemolymph.

The distribution of RhVB seems to be limited as the agent is known only from crabs collected from Galveston, Texas (Yudin and Clark, 1978, 1979b).

Johnson and Farley (1980) reported an enveloped helical virus (EHV) in tissues of blue crabs collected from the Tred Avon River, a tributary of Chesapeake Bay, Maryland and Tangier Sound, Virginia. The agent was always observed in crabs infected by one or more other viruses, i.e., CBV, RLV, RhVA or Baculo-B.

The enveloped helical virus virions (Fig. 3-11, c) are membrane bound and variable in shape ranging from ovoid (105 × 194 nm) to rod-like (105 × 300 nm). Indistinct spikes project from the enveloping membrane (Johnson, 1983). Virions are enveloped by budding through the plasma membrane. Helical shaped flexous strands about 23 nm in diameter are found within granular areas, probable sites of nucleocapsid formation, that occur in the cytoplasm of infected cells (Johnson, 1983).

The enveloped helical virus was noted in extracellular locations – between the plasma membrane of adjacent hematopoietic cells and hemocytes or underneath or within the basal lamina surrounding lobes of the hematopoietic tissues (Johnson and Farley, 1980). EHV was also found within the basal lamina of mesodermal cells lining the antennal gland labyrinth, but not elsewhere. EHV is never abundant and disease signs observed in some of the examined crabs were probably due to the presence of other viruses (Johnson and Farley, 1980; Johnson, 1983, 1984a).

Johnson (1984a) assigned EHV to the Rhabdoviridae, although earlier reports (Johnson and Farley, 1980; Johnson, 1983) were uncertain of the classification of EHV. The virus has developmental and morphological characteristics similar to that of the Paramyxoviridae but is smaller (Johnson, 1983). Johnson and Farley (1980), Johnson (1983, 1984a) stated EHV is closely related to the Y-organ virus of *Carcinus maenas*.

The occurrence of EHV in natural crab populations outside of Cheaspeake Bay and Tangier Sound is unknown.

A rhabdo-like virus, named Y-organ virus, is reported from *Carcinus maenas* (Chassard-Bouchaud and Hubert, 1975; Chassard-Bouchaud and co-authors, 1976). Infected crabs were collected from Roscoff, France. The putative rhabdovirus was discovered during electron microscopic study of the Y-organ where groups of virus particles were observed beneath and within the basal lamina.

Y-organ virus of the European shore crab is an enveloped, variable-shaped (ovoid to elongate) 70 to 90 nm × 150 to 170 nm particle (Chassard-Bouchaud and Hubert, 1975; Chassard-Bouchaud and co-authors, 1976). The envelope is 9 nm thick with external spicules ca 7 to 10 nm long. Viral development occurs in the cytoplasm in association with numerous ribosomes and electron dense filaments that are nucleocapsids (Chassard-

Bouchard and co-authors, 1976). The Y-organ virus buds through the plasma membrane (Chassard-Bouchard and co-authors, 1976) and this feature distinguishes it from the S virus of *Macropipus depurator,* that buds into intracytoplasmic vesicles, with which the Y-organ virus is morphologically similar (Chassard-Bouchard and co-authors, 1976; Johnson, 1983). The biochemical and additional biophysical characteristics of the Y-organ virus of *Carcinus maenas* have not been documented.

Infection of other tissues in *Carcinus maenas* by the Y-organ virus has not been investigated. There are no disease effects known for this virus infection of *C. maenas.*

In marine Crustacea, 3 putative bunya-like viruses have been reported from crabs. These agents are the crab hemocytopenic virus from *Carcinus maenas,* the S virus from *Macropipus depurator* and *C. mediterraneus,* and a bunya-like virus infection noted during electron microscopy study of Y-organ tissues from *C. mediterraneus.* The first 2 agents are reported to cause diseases in their crab hosts.

The crab hemocytopenic virus (CHV) was discovered by Bang (1971) in one of 700 *Carcinus maenas* collected from waters near Roscoff, France. Hemolymph from this crab inoculated into other crabs transmitted the infection within 2 to 19 days post injection. By electron microscopy, a cytoplasmic viral agent was found (Bang, 1971). The virus, later named crab hemocytopenic virus (CHV) of *C. maenas* is tentatively assigned to the Bunyaviridae (Johnson, 1984a).

The crab hemocytopenic virus is a spherical 55 to 80 nm-diameter particle that occurs throughout the intracytoplasmic membrane system of infected hemocytes (Hoover, 1977). CHV apparently buds into intracytoplasmic vesicles and often is associated with the Golgi complex. Enveloped particles have not been demonstrated (Hoover, 1977). The virus has not been studied in purified preparations and the biochemical and biophysical features of CHV are undertermined.

Experimental infection of *Carcinus maenas* by CHV results in a transient impairment of *in vitro* clotting and marked reduction in circulating hemocytes (Bang, 1971, 1974; Hoover, 1977). In the original work (Bang, 1971) the infection was apparently discovered when withdrawn hemolymph from a crab failed to clot normally. Serial passage transmitted this clotting defect to other apparently healthy crabs. When crabs were injected with 0.05 ml of undiluted blood, clotting failure became apparent between 2 to 19 days following exposure (Bang, 1971). Of 73 crabs inoculated with known positive material, 74 % developed clotting failure within 10 days. Additionally, the time in days to onset of clotting failure was correlated with dilution of the inoculum (Bang, 1971).

Hemocyte count decreased as early as 2 days prior to the clotting defect being detectable. A series of studies (Bang, 1971) showed the infection causes abnormal cellular clotting, decrease in peripheral amebocyte count, clumping of amebocytes in peripheral tissues and blood and abnormal amebocyte behavior on glass. In his classical studies, Bang (1971, p. 617) stated "the failure of blood to clot on glass at 20 °C was considered as evidence of infection" and primarily used this clinical tool to track the behavior of CHV infection in *Carcinus maenas* under laboratory conditions.

During peak periods of infection, CHV occurred in large amounts in hemolymph, serum and hemocytes (amebocytes). The virus remained infectious during 10 months when frozen at −70 °C. Male and female crabs are equally susceptible to CHV infection (Bang, 1971).

Not all crabs experimentally exposed develop the disease. Moreover, about two-thirds

of experimentally infected crabs recover (i.e., *in vitro* clotting of hemolymph returns), and of those approximately 50 % regain clotting in 4 to 6 days from onset of the defect (Bang, 1971). However, CHV virus was detectable as late as 40 days after recovery. Crabs that did not recover died, presumably due to clotting failure (Bang, 1974).

Bang (1974) noted other signs beside clotting defect that accompanied CHV infection including general weakness, and in about 10 % of the *Carcinus maenas* stiffness of all the appendages (a spastic inhibition rather than due to paralysis *per se*). Crab mortality ranged from 18 to 32 % in the test groups within 1 month from inoculation and was about double that observed in the controls. However, Bang (1974) cautions that these results could be misleading because of the highly artificial conditions these crabs were held (no feed and repeatedly bled).

Previously infected *Carcinus maenas* that had recovered were not resistant (as compared to controls) when reinoculated with CHV-infected hemolymph (Bang, 1974). Bang also observed autointerference, i.e., hemolymph withdrawn from inoculated crabs after the second day of patent infection (hemolymph clotting defect detectable) and serum withdrawn from crabs from Days 1 through 8 of patent infection resulted in reversal of the dilution effect: crabs inoculated with highly dilute serum either developed more positives or a shorter incubation period before onset of the clotting defect. Bang (1974) speculated this interference could be due to the virus itself, presence of another virus or of an interferon-like substance.

Hoover and Bang (1976) and Hoover (1977) studied in detail the histopathology of CHV infection in *Carcinus maenas*. Her work indicates that CHV infection results in significantly increased numbers of aggregated, mostly intact hemocytes, within connective tissues of the hepatopancreas, gills, heart and ovary. Hemocyte aggregation accounts for the decrease in circulating hemocytes and the delayed clotting of hemolymph. Infiltration of the ovary by hemocytes with focal to extensive replacement of ovarian tissues was also observed. Focally degenerating hemocytes, hemocytic nodules and encapsulations and damaged striated muscle adjacent to the hepatopancreas and the ovary were also associated with experimental CHV infection of *C. maenas* (Hoover, 1977).

Studies to determine the impact of CHV on health of wild *Carcinus maenas* populations have not been undertaken. The impact of CHV on natural crab populations is unknown.

The S virus was first recognized in *Macropipus depurator* collected near the city of Sète on the French Mediterranean coast. The agent is enveloped and pleomorphic with cytoplasmic development (Bonami and Vago, 1971; Bonami and co-authors, 1975; Bergoin and co-authors, 1982). Usually ovoid in shape, spherical and elongate forms of the S virus also occur. Ovoid forms measure 80 to 130 nm × 190 to 230 nm; spherical forms are 80 to 150 nm, and elongate forms are 50 to 70 nm × 240 to 230 nm (Bonami and co-authors, 1975). The envelope is bilayered and approximately 22 nm thick. Projections are found on the outer layer and the subunits of the envelope form a helical structure. Flexous elements with perpendicular striations occur in the electron dense interior. These may represent nucleocapsids (Bonami and co-authors, 1975). The virions bud through membranes into intracytoplasmic vesicles and are released from these vesicles into the extracellular space (Bonami and co-authors, 1975). The nuclei acid type is reported to be ss-RNA (Bonami and co-authors, 1975; Bergoin et al. and co-authors, 1982).

The classification of the S virus is uncertain. Bonami and co-authors (1971) concluded

the S virus shares similarities with the Rhabdoviridae, but did not assign the virus to a family. Later, Bonami and co-authors (1975) indicated the S virus also has characteristics of both the Paramyxoviridae and the Bunyaviridae. Johnson (1984a) has tentatively placed the virus in the bunyavirus group.

Bonami and Vago (1971) reported the S virus disease is common in *Macropipus depurator* populations on the Mediterranean coast with the S virus being identified from sick wild-caught crabs on several occasions.

Inoculation of infected hemolymph, tissues or purified S virus results in 70 to 80 % mortality in 15 to 20 days. Disease signs commence on about Day 10 following experimental exposure (Bonami and Vago, 1971; Bonami and co-authors, 1971). Crabs infected by the S virus show progressive weakness, reduction of activity and slight darkening of the exoskeleton (Bonami, 1976).

The S virus infects the cytoplasm of cardiac cells and endothelial cells of the blood vessels associated with the hepatopancreas (Bonami and Vago, 1971; Bonami and co-authors, 1971; Bonami and co-authors, 1975).

Although the S virus is reported as a cause of disease in *Macropipus depurator* in the natural environment (Bonami and Vago, 1971; Bonami and co-authors, 1971, 1975; Bergoin and co-authors, 1982); data on this point are sparse. The impact of the S virus on wild crab populations is unclear.

In both normal and *Sacculina*-parasitized *Carcinus mediterraneus* collected from the wild, electron microscopy findings indicated a bunya-like virus infection of the Y-organ cells (Zerbib and co-authors, 1975). The agent, an oval virus, measured 60 × 180 nm and developed within cytoplasmic vesicles (Zerbib and co-authors, 1975; Bonami, 1976). Although the agent is smaller, its finding could well represent another documentation of the S-virus (Zerbib and co-authors, 1975). No further information on this virus has apparently been published (Johnson, 1984a).

Agents: Rickettsia and Chlamydia

Four rickettsia-like and 1 chlamydia-like pathogens are reported from marine Crustacea. The recognized agents are described from infections of crabs or penaeid shrimp. The chlamydia-like agent of the dungeness crab *Cancer magister* was associated with a mortality syndrome in natural crab populations in Washington State coastal waters (USA). The taxonomic relationships are poorly understood between the marine crustacean rickettsia-like and chlamydia-like agents to established species known from vertebrate and other invertebrate hosts.

Members of the order Rickettsiales are small procaryotic, rod-shaped, coccoid, often pleomorphic, intracellular Gram-negative microorganisms. All recognized members of this order divide by binary fission (Moulder, 1974). Rickettsias are known from a wide range of vertebrate and invertebrate hosts. Within marine invertebrates, rickettsia-like organisms have been described only from molluscs and decapod crustaceans including 2 marine crabs and several species of penaeid shrimp. The crustacean rickettsias are ovoid-to-rod-shaped, develop within intracytoplasmic vacuoles and stain Gram-negative and weakly Feulgen-positive. Only a superficial understanding exists on the taxonomic relationships of the marine crustacean rickettsias to others in the order Rickettsiales. Johnson (1984b) and Brock and co-authors (1986a) suggested the organisms they observed infecting

Paralithodes playtpus and *Penaeus marginatus*, respectively, belong to the genus *Rickettsiella* (Weiss, 1974).

The first rickettsial organism from a marine decapod crustacean was reported infecting *Carcinus mediterraneus* (Bonami and Pappalardo, 1980; Pappalardo and Bonami, 1980. The organism attacks connective tissues and was discovered during the course of an ecopathological study of *C. mediterraneus* collected from the Sète region on the Mediterranean Coast of France.

The rickettsia-like organism measures 2 × 0.7 μm, has a plasma membrane and cell wall, contains ribosomes, fibrils and nuclear material. Transverse constriction of the rod-shaped forms was observed suggestive of binary fission (Bonami and Pappalardo, 1980). The agent is known only from *C. mediterraneus*. Experimental infection by inoculation of 0.2 ml of infected hepatopancreas tissue extract resulted in crab deaths within 15 days of infection (Bonami and Pappalardo, 1980). The rickettsia infects connective tissue cells of the hepatopancreas, gut, gonad and gills where it forms 10 to 20 μm diameter microcolonies. The rickettsial microcolonies stain Feulgen-positive and form within intracytoplasmic vacuoles (Bonami and Pappalardo, 1980). Cell rupture releases the agent to infect other cells.

The impact and distribution of the rickettsia-like agent on wild populations of *C. mediterraneus* has not been documented.

Johnson (1984b) reported a rickettsial infection of the hepatopancreas epithelium of a wild-caught, immature female *Paralithodes playtpus*. The specimen was the only infected individual in a sample of 106 crabs, that were collected from various areas in the Bering Sea and waters around Kodiak Island, Alaska.

Johnson (1984b) described the agent as a Feulgen-positive rickettsia that measures 0.3 × 0.6 to 1.0 μm. The rickettsia forms round, 10 to 40 μm cytoplasmic inclusions that are lightly Feulgen-positive. Dividing forms were not observed. The cytoplasmic inclusions are membrane-bound vesicles filled with 0.3 × 0.5 μm organisms.

The *Paralithodes platypus* rickettsia infects hepatopancreas epithelium. In the specimen studied, infection was massive. Occasional necrotic, encapsulated hepatopancreatic tubules were present, ovarian development was arrested and aspects of the molt cycle did not appear to be synchronized normally. Johnson (1984b) concluded the infection would have terminated in death of the host.

The distribution and impact of the rickettsia on *Paralithodes platypus* populations is unknown. Johnson (1984b) surmised that early infections could have been overlooked by light microscopy because the agent is only weakly Feulgen-positive.

Chong and Loh (1984) reported dual infection of the hepatopancreas of *Penaeus merguiensis* by a 'chlamydial' agent and hepatopancreas parvovirus (HPV). Lightner and co-authors (1985b) examined the material and suggested the prawns were infected by a rickettsialike agent rather than a chlamydia.

Chong and Loh found heavy rickettsial infection of hepatopancreas epithelium (Fig. 3-12, a) in cultured prawns examined from 1 of 4 farms evaluated in the Singapore area. Prawns at this location were derived as hatchery seed and grown in ponds. Gross signs or lesions were not associated with the rickettsial infection. Reduced feed consumption and moderate mortalities were reported from infected ponds when the prawns were about 1 g in average weight (Chong and Loh, 1984). Electron microscopic description of the organism was not given.

Brock and co-authors (1986a) reported a second case of hepatopancreatic rickettsia-

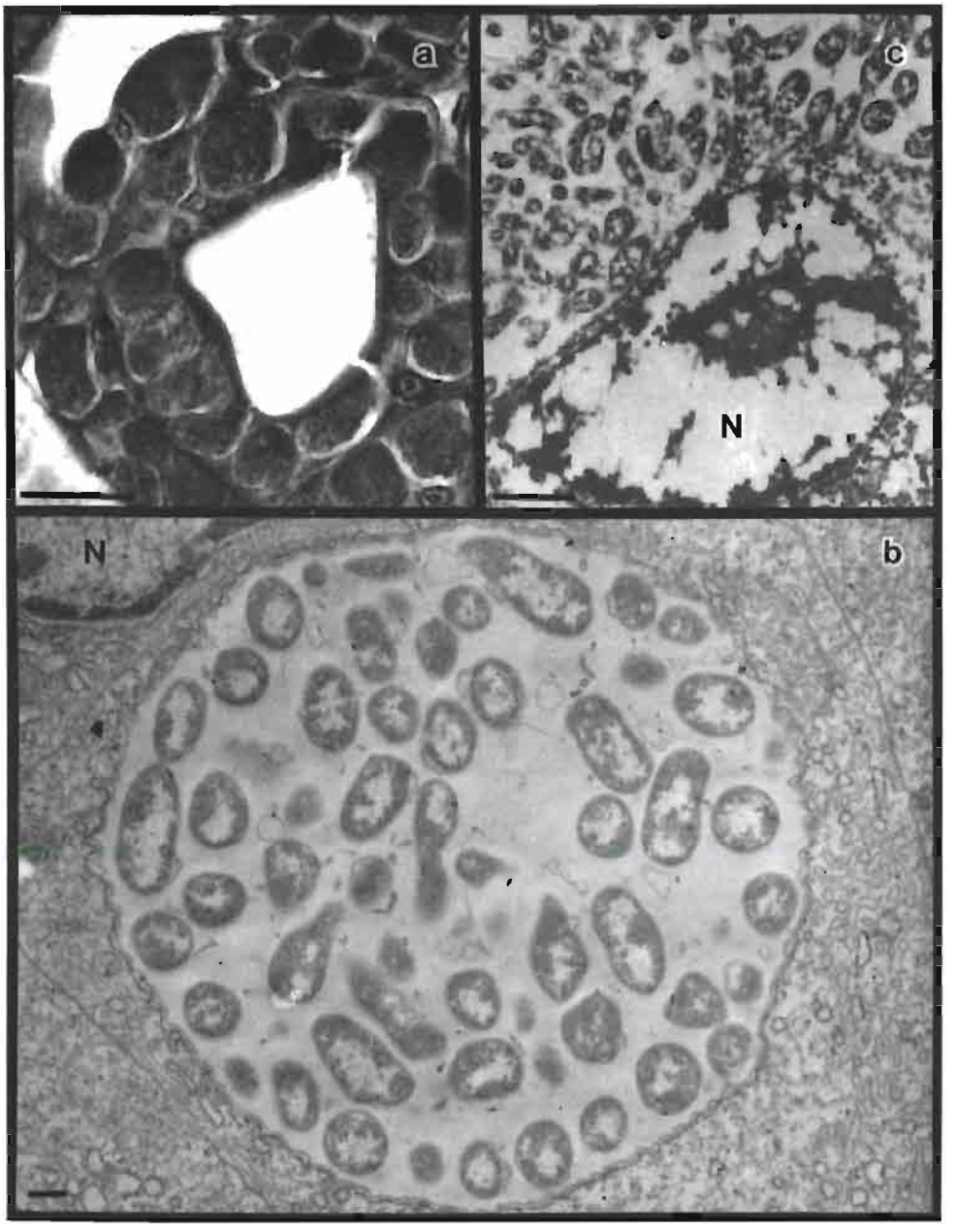

Fig. 3-12: Rickettsia in penaeid shrimp. (a) Histological section of the HP of a juvenile *Penaeus merguiensis;* cytoplasm of every HP tubule epithelial cell nearly filled with masses of rickettsia, bar = 25 μm. (b) TEM of a rickettsial inclusion adjacent to the nucleus (N) but within the cytoplasm of an HP cell from *P. marginatus;* bar = 1 μm. (c) TEM of rickettsial organisms within the cytoplasm of an infected fixed phagocyte of *P. monodon;* nucleus (N) not infected: bar = 1 μm. (Originals.)

like infection in wild-caught, captive-held *Penaeus marginatus*. Rickettsia infection was detected by light microscopy after 30 days of captive rearing, and only moderately active infections were found.

The hepatopancreatic rickettsia of *Penaeus marginatus* measured 0.2 to 0.7 × 0.9 to 1.6 μm. The agent (Fig. 3-12, b) forms large aggregations within variable-sized (7 to 46 μm) intracytoplasmic vacuoles. Dividing forms were not observed.

Experimentally, *per os* exposure of infected hepatopancreas tissues to juvenile *Penaeus stylirostris* successfully transmitted the rickettsia and resulted in a disease syndrome characterized by lethargy, inappetence, delayed escape response and white colored hepatopancreata (Brock and co-authors, 1986a). Captive, infected *P. marginatus* showed no outward signs of disease.

The rickettsial infection of *Penaeus marginatus* and *P. stylirostris* was limited to hepatopancreatic epithelium. Infected cells were often hypertrophied, the cytoplasm largely replaced by a developing rickettsial microcolony. A host inflammatory response to the infection was present in areas where marked destruction of the hepatopancreas had occurred, but largely absent elsewhere, even though many hepatopancreatocytes contained rickettsial microcolonies.

The rickettsia-like organism appeared to be only mildly pathogenic to *Penaeus marginatus* but a potentially serious pathogen to juvenile *P. stylirostris*. The distribution and impact of the rickettsia in wild populations of *P. marginatus* is unknown.

Anderson and co-authors (1987) first reported on a systemic rickettsial infection in tiger shrimp *Penaeus monodon*, pond cultured in the State of Johore, Malaysia. Some rickettsial-infected shrimp also had concurrent infections by monodon baculovirus, a cytoplasmic reo-like virus and Gram-negative bacteria. (Anderson and co-authors, 1987).

The rickettsia (Fig. 3-12, c) most often occurred within large intracytoplasmic vacuoles where it formed microcolonies 19 to 33 μm in diameter (Anderson and co-authors, 1987). In heavy infections the organism was widespread in mesodermal and ectodermal tissues, but did not infect endodermally derived cells (Anderson and co-authors, 1987; Brock, 1988). This feature is a distinct contrast to the penaeid shrimp rickettsias that infect hepatopancreatic epithelium. The systemic rickettsial infection is known only from cultured *P. monodon* in Malaysia. Experimental transmission of the agent has not been reported.

The disease signs associated with systemic rickettsial infection are difficult to attribute to the rickettsial infection alone because other pathogens were present in the tissues of shrimp sampled from ponds. Nevertheless, the signs associated with the disease were first noted about 7 to 9 weeks after stocking: reduced feed consumption, lethargic-to-erratic swimming behavior, weak shrimp aggregating along the edge of the pond; within a few days, mortalities started and continued for several weeks (Anderson and co-authors, 1987). Moribund and dead shrimp were small for their age, had darkened, fouled gill filaments, empty digestive tracts, opaque abdominal muscles and whitish nodules on the mig-gut wall (Anderson and co-authors, 1987).

Histopathologically, rickettsial microcolonies, hemocyte aggregation and encapsulation were associated with areas of degeneration and necrosis with the most severe changes in the lymphoid organ. However, connective tissues in the gills, hepatopancreas, antennal gland, ventral nerve cord, striated and cardiac muscle were also affected (Anderson and co-authors, 1987; Brock, 1988).

Occurring concurrently in *Penaeus monodon* infected by systemic rickettsia were other biotic agents including Gram-negative bacteria (such as septicemia); bacterial, algal and protozoan epizoic gill fouling; and hepatopancreatic viruses — monodon baculovirus and reo-like virus (Anderson and co-authors, 1987; Nash, M. B. and co-authors, 1988). Thus the etiology of the observed disease is complex and the exact role attributable to the systemic rickettsia unclear. However, major pathologic tissue changes reported by Anderson and co-authors (1987) and Brock (1988) were mainly associated with rickettsial microcolonies suggesting an important role of the agent in the disease processed observed.

The distribution and impact of systemic rickettsial infection in wild populations of *Penaeus monodon* is unknown. As suggested by Anderson and co-authors, 1987), *P. monodon* may represent an atypical host for the rickettsial agent. Diagnosis of rickettsial involvement in a disease occurrence in cultured shrimp can be established by demonstration, using histological methods, of the prominent microcolonies in stained tissue sections.

In the case report given by Anderson and co-authors (1987) mortality control was achieved when the farm switched species of shrimp raised from *P. monodon* to *P. merguiensis*. Antibiotic therapy has been suggested (Anderson and co-authors, 1987; Brock, 1988) as a potential means of control, but data on treatment trials is unavailable.

Present information indicates that several rickettsial agents infect marine crabs and shrimps. The number of different agents is unclear, but at least 2 different organisms are involved. One attacks endodermal cells, another infects mesodermal and ectodermal tissues. Analysis of the relations between crustacean rickettsial agents and comparison to rickettsias known from vertebrates and other invertebrates would be a productive area for future study.

Chlamydias are small, obligate intracellular microorganisms known from vertebrate and invertebrate hosts. Chlamydial multiplication is characteristic for members in this group; it includes small, rigid-walled 'elementary bodies', the infectious forms, that change into larger, thin-walled 'initial bodies' dividing by fission and resulting in the formation of daughter cells which eventually reorganize and condense (intermediate bodies) to become elementary bodies (Page, 1974). Chlamydia are Gram-negative, contain DNA and RNA, but are not able to generate adenosine triphosphate and are thus referred to as 'energy parasites' (Storz and Page, 1971). Chlamydias cause the economically significant diseases, psittacosis (*Chlamydia psittaci*) and trachomoniasis (*C. trachomatis*). The chlamydia affecting the scorpion *Buthus occitanus* and the agent known from *Cancer magister* also result in severe disease for their respective host species (Sparks and co-authors, 1985). Within marine Crustacea, chlamydia-like agents are known only from the well documented disease in *C. magister* (Sparks and co-authors, 1985) and a single mention of infection by a chlamydia-like organism of the hepatopancreas of the Kuruma shrimp *Penaeus japonicus* (Lightner and co-authors, 1985b).

High mortalities of economically important *Cancer magister* were reported in crab pots as well as holding facilities in Willapa Bay, Washington, USA in February, 1979 (Sparks and co-authors, 1985). In a study of the epidemic, Stevens and Armstrong (1981, cited in Sparks and co-authors, 1985) estimated 6,461 crab mortalities along a 7.9 km transect of the southwest Washington coast. Apparently, the disease affects *C. magister*, populations each winter and spring in northern Puget Sound, although mortalities from this disease are unknown in the summer and fall (Sparks and co-authors, 1985).

The agent is partially characterized based on morphological criteria. Cytoplasmic

infections were found and the reticulate (initial), intermediate and ellipsoidal bodies identified. Sparks and co-authors (1985) mention that 2 reviewers of the material did not believe the agent to be a chlamydia-like microbe, but offered no other suggestions for its identity. Progress on the *in vitro* cultivation, biochemical characterization, or isolation of the agent from tissues in purified preparations are not reported.

The chlamydia-like agent is known from dungeness crabs *Cancer magister* in the Puget Sound area of Washington, USA. Its occurrence elsewhere within the range of the dungeness crab has apparently not been studied. Crabs were collected monthly from the wild from January 1978 through February 1982, and examined histologically. The disease has only occurred in crabs sampled between December and March, and the incidence during these periods was 6 % (18/295), with the highest attack rate in 1979 (13 % 8/64), the period when crab mortalities were reported (Sparks and co-authors, 1985). These investigators related the seasonal occurrence of the disease to low water temperature. The disease has not been reproduced experimentally.

In the histological survey, only crabs with heavy chlamydia infections were found. Sparks and co-authors (1985) reasoned this indicated a rapid, progressive course after infection. Disease crabs were lethargic. Withdrawn hemolymph had the normal collection of hemocytes, but contained abundant tiny refractile bodies, whose exact structure has not been described.

Histologically, diseased crabs exhibit massive systemic infections. The chlamydial agent infects cells of mesodermal and ectodermal origin. Heavy, intracellular proliferation of the organism causes extreme hypertrophy of infected cells resulting in an apparent confluent field of microcolonies (Fig. 3-13, a) as adjacent cells are infected (Sparks and co-authors, 1985). Cell necrosis and moderate-to-dense accumulations of hemocytes accompany the chlamydial infection in some tissues. Fixed tissue phagocytes are universally and markedly infected (Sparks and co-authors, 1985), but encapsulation by hemocytes has not been reported.

Ultrastructurally, microcolonies are membrane-bound and completely, or nearly so, displace cytoplasmic organelles. Cells that contain the putative condensing forms (Fig. 3-13, c) and elementary bodies are usually devoid of organelles except the nucleus which is emarginated and the chromatin condensed along the borders (Fig. 3-13, b). Cells with reticulate (initial), intermediate and ellipsoidal bodies are less densely packed and, hence, contain more cytoplasmic organelles (Sparks and co-authors, 1985).

Dungeness crab chlamydial disease is caused by a highly pathogenic chlamydia-like microorganism most closely allied to the Chlamydiales. Sparks and co-authors (1985) suggested that the agent is a significant factor in the health of wild-populations of dungeness crabs in the Puget Sound area of Washington, USA. The chlamydial disease of *Cancer magister* is perhaps one of the better documented examples available of a biotic agent with potential impact at the population level on a marine crustacean in its native environment.

Agents: Bacteria

Marine bacteria play a significant role in causing diseases of captive-wild and aquacultured marine crustaceans. However, that these organisms are the cause of diseases in wild-populations is unclear and, as Sparks (1985, p. 181) writes, is "difficult to establish". For example, the isolation of Gram-negative bacteria including known poten-

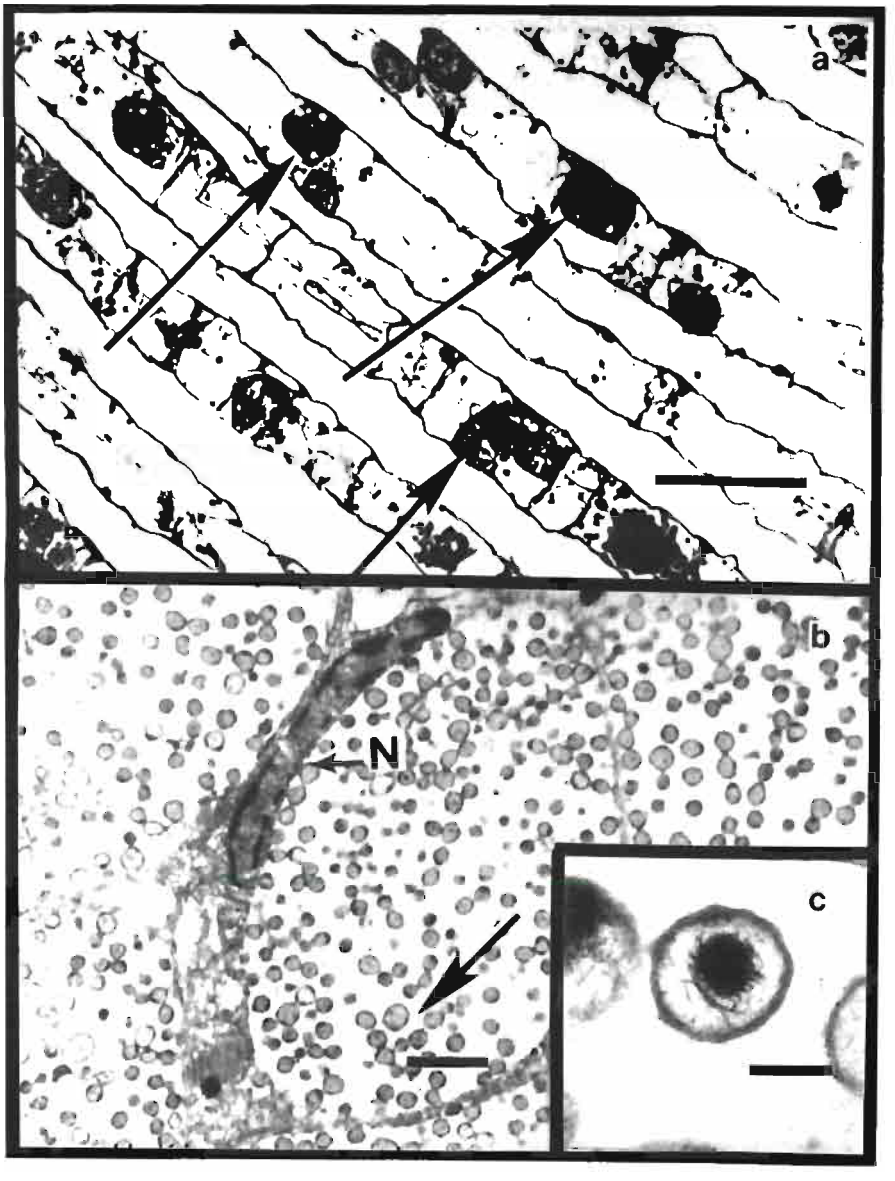

Fig. 3-13: Chlamydia-like infection of *Cancer magister*. (a) LM of colonies of Chlamydia-like agents (arrows) infecting connective tissue stroma of gill lamellae; bar = 150 µm. (b) TEM of 'condensing forms' infecting gill stem cells; cells are swollen and nuclei (N) marginated; bar = 2 µm. (c) TEM of 'condensing form' from gill stem tissue; bar = 250 nm. (a to c after Sparks and co-authors, 1985. Reprinted with the permission of Academic Press, Inc.)

tial pathogens — such as *Vibrio* sp., *Pseudomonas* sp. and *Aeromonas* sp. — has been reported from apparently healthy, recently collected *Callinectes sapidus* (Colwell and co-authors, 1975; Sizemore and co-authors, 1975), but that this is 'normal' for the crab or, more specifically: it is disputed that recently collected live crabs are the same as wild crabs untouched in their natural habitat. Colwell and co-authors (1975) concluded their results

indicate that the hemolymph of normal, healthy blue crabs is non-sterile. On the other hand, Bang (1970) and Johnson (1983) maintain that bacteria are not naturally present in crustacean hemolymph. However, through wounding, during ecdysis or by other means bacteria enter the hemolymph of a 'stressed' host, and bacteremia rapidly develops. Under appropriate conditions (host, agent, environment) and for individual crabs, bacteremia may rapidly cascade into fulminating bacterial disease.

A similar situation exists for the microbial epibionts. In the wild, bacterial epibionts are normally present on the cuticle surfaces of marine crustaceans (Baross and co-authors, 1978). Usually, infection levels are low and the crabs are not functionally compromised. However, in captive rearing and under circumstances related to environmental quality, the agents proliferate and can result in, or contribute to, disease syndromes. Epibiont disease occurs when the host's functional capacity is impaired; not because the microbes are present *per se,* but because pathologically high densities of microbial colonization has taken place.

Fundamental determinants of bacterial disease expression in marine crustaceans encompass much more than the microorganism itself. Circumstances are necessary that provide or promote exposure and allow entry of the pathogen, enhance activity of the microbe, depress defenses of the crustacean host or a combination of these factors in order for the disease to occur. Unfortunately, the current level of understanding, in a quantitative way, of the relations between bacteria, crustacean host and environmental factors is, for most bacteria-caused diseases, superficial. The exception to this generalization is gaffkemia of lobsters. For the other bacterial diseases, these relations are lumped into descriptive categories such as 'stressed environments' or 'stressed hosts'. That such conditions or states exist is obvious, but what they are comprised of, is not. Studies on the cause-and-effect relations between host, bacterial agent and environmental parameters are scarce and few quantitative data are available. Thus, our understanding is poor in this area, emphasizing the difficulty in establishing a complete understanding of the etiology for some bacterial diseases of marine crustaceans. Clearly, physiological, nutritional or behavioral stresses or enhanced potentials for opportunistic pathogen multiplication and/or transmission due to crowding and degradation of water quality are common determinants in bacteria-mediated diseases in captive crustacean populations. Understanding the interactions of these determinants may be highly important for effective prevention or control of these diseases in captive populations. This area deserves greater emphasis in future studies; the work on gaffkemia of lobsters can serve as a model.

Although Gram-negative bacteria are predominant in marine environments (Brisou and co-authors, 1965) and usually constitute the major intestinal flora of crustaceans, Gram-positives are important for marine crustaceans as well. Yasuda and Kitao (1980) studied the gut flora of captive and wild larval and adult stages of *Penaeus japonicus. Vibrio* was the dominant agent genera, and *Pseudomonas, Aeromonas* and other Gram-negative bacteria were also found. However, Vanderzant and co-authors (1970a); Vanderzant and co-authors (1971); Christopher and co-authors (1978) recovered Gram-positive coryneform bacteria from pond-reared shrimp, as well as species of *Vibrio, Moraxella, Pseudomonas,* etc. Also, Lee and Pfeifer (1975) reported isolation of *Bacillus* sp. and *Staphylococcus* sp. from dungeness crab meat. The findings of these studies indicate that both Gram-negative and Gram-positive bacteria comprise the natural flora associated with marine crustaceans.

To date, Gram-negative bacteria are the predominant forms associated with crustacean diseases. The Gram-negative disease agents are opportunistic pathogens and their role is etiologically complex. Not uncommonly, these diseases are considered 'secondary infections' (Lightner and Lewis, 1975; Delves-Broughton and Poupard, 1976). While the direct cause of the disease are microbial agents, contributing etiologic determinants (physical, chemical, biochemical) are important for disease expression. Furthermore, mixed infections involving agents from several taxa are not uncommon. This is particularly true for diseases involving the crustacean cuticle or cuticular surfaces.

Epizoic bacterial colonization, by both filamentous and non-filamentous agents, on cuticular surfaces of the crustacean host has been abundantly documented in marine and freshwater crustaceans. In nature, bacterial communities on cuticular surfaces are usually limited by one or more factors before reaching densities that can cause disease in the host. However, microbial epibionts continue to cause problems in cultured crustaceans (Fisher, 1977a, b, c; Lightner, 1977 his Chapter 3.1.4; Lightner, 1988 his Chapter 3.1.10).

All species of life stages of marine Crustacea are susceptible to epibiotic microbial infestation. According to Gharagozlou-van Ginneken and Bouligand (1975), bacterial cuticle colonization is a normal condition in 2 species of harpacticoid copepods (*Porcellidium* spp.) collected from the Mediterranean Sea and the English Channel. Johnson and co-authors (1971) observed filaments of *Leucothrix mucor* on the cuticle of the copepod *Acartia clausi* maintained in an aquarium, and found *L. mucor* on a variety of benthic marine decapods *(Pagurus longicarpus, Carcinus maenas, Cancer irroratus, Lithodes maia, Palaemonetes pugio, Crangon septemspinosa)* collected from the wild. Eggs and larvae of *Cancer magister, Homarus americanus* and *H. gammarus, Pandalus platyceros* (Fisher and co-authors, 1975; Fisher, 1977a, b, c, 1988a, c), eggs of cultured *Palaemon serratus* (Delves-Broughton and Poupard, 1976); and larvae through adults of *Penaeus* spp. (Lightner 1977 his Chapter 3.1.4, 1983, 1985, 1988 his Chapter 3.1.10; Johnson, 1978; Baticados, 1988) suffer from epibiotic infestations, particularly those caused by filamentous *L. mucor.* Solangi and co-authors (1979) reported epibiotic fouling disease caused by *L. mucor* in cultured adult *Artemia.*

The most important filamentous epibiont disease agent of marine crustaceans is *Leucothrix mucor.* It is best known as an epiphyte of macroscopic algae (Lewin, 1959; Brock, 1966; Kelly and Brock, 1969; Johnson and co-authors, 1971; Bland and Brock, 1973, and others cited in Bland and Brock, 1973); it is also the periphyte most often documented from benthic marine Crustacea in nature and cultures (Johnson and co-authors, 1971; Overstreet, 1973; Fisher and co-authors, 1975; Shelton and co-authors, 1975; Delves-Broughton and Poupard, 1976; Fisher, 1977a, b, 1983a; Lightner, 1977 his Chapter 3.1.4, 1983, 1985, 1988 his Chapter 3.1.10; Johnson, S. K., 1978; Baticados, 1988). In culture, other filamentous and chain-forming bacteria reported as agents in filamentous gill and surface fouling of penaeid shrimp are *Thiothrix* sp., *Flexibacter* sp., *Cytophaga* sp., *Flavobacterium* sp. (Lightner, 1988 his Chapter 3.1.10), and a filamentous bacterium tentatively identified as *Bacillus cereus* var. *mycoides* (Barkate and co-authors, 1974).

A variety of free-living non-filamentous bacteria are important causes of larval mortality in dungeness crabs (Fisher, 1977a, c), and of 'aggregation' fouling disease in penaeid shrimp larvae (Lewis and co-authors, 1982). Interestingly, only infrequently have identifications been reported for the bacterial flora of these non-filamentous forms. Lewis and co-authors (1982) reproduced 'aggregation' symptoms experimentally with

Pseudomonas piscicida, Aeromonas formicans and *Flavobacterium* sp. isolated from clinical cases of fouling disease in larval shrimp.

Strains of *Leucothrix mucor* — which is found only in marine environments (Bland and Brock, 1973) — did not grow when the salinity decreased below about 7 ppt (Kelly and Brock, 1969). *L. mucor* is an obligately aerobic, Gram-negative chemo-organothroph with temperature, salinity and pH optima of 28 °C, 31 ppt and 7 to 8, respectively (Kelly and Brock, 1969). The genus *Leucothrix* has 1 species with strains that have 48 to 51 % G + C. However, Steenbergen (1979) found the G + C % to be 59.5 % for *L. mucor* isolated from *P. stylirostris* cultured in Mexico. The Mexican isolate also lacked antigens cross reacting with other *L. mucor* strains, and Steenbergen (1979) concluded that the Mexican organism is a different species or belongs to another genus.

Leucothrix mucor has a developmental cycle with 2 forms: multicellular filaments and gonidia. The filaments are 3 to 5 μm wide at the base and vary in length, often being 100 to 500 μm, and attach by a holdfast to the host. Under unfavorable conditions single cells of the filament round-up, detach to form gonidia which can move by gliding and attach via a holdfast. Once attached, gonidia divide and filaments form (Bland and Brock, 1973).

Gonidia and filaments are easily identified by direct microscopic examination. *Leucothrix mucor* can be cultured routinely in Provasoli-enriched sea-water (PES) medium, supplemented with 0.1 % monosodium glutamate (MSG) (Brock, 1966 cited in Bland and Brock, 1973). Tubes are incubated for 10 weeks at a temperature similar to that from which the samples were collected. *L. mucor* can be subcultured from primary isolation tubes onto 1 % agar plates made with PES and 0.1 % MSG (McKee and Lightner, 1982). The bacterium apparently will settle on a non-living substrate; however, under conditions of low carbon content in sea-water, extensive growth of *L. mucor* is not supported (Bland and Brock, 1973). These authors conclude that in nature an obligate relation exists between *L. mucor* and the host organism. Under culture conditions with high available carbon contents in the water, heterotrophic biomass flourishes, and *L. mucor* colonizes non-living substrates (Lightner, 1983). Experimentally, Fisher (1983a) demonstrated substantial increase in bacterial populations on incubating eggs of *Palaemon macrodactylus* with addition of nutrient broth to sea-water, but bacteria present were not identified.

Slight infestation by bacterial epibionts occurs naturally in crustaceans, and low levels of epibiotic microbial fouling are normal on marine crustaceans in the wild. Under conditions of low nutrient availability in the water and through preening activity of the crustacean host, colonization levels, in terms of host disease, remain insignificant. If conditions are appropriate, i.e., confinement rearing and increasing microbial colonization levels, the bacterial mat can impair normal physiological functions of the host. Then, disease symptoms and animal mortality may occur. Life stage of the host, quantity of microorganisms per unit of surface area and location on the host's body are relevant features to expression of the disease. Eggs and larval stages are more apt to suffer significant disease from epibionts than are juvenile-through-adult stages. Fouling of gill lamellar surfaces (Fig. 3-14, a, b), chemoreceptor sites, etc. can be more significant than fouling that occurs on cuticular surfaces not directly involved with gas exchange or other vital functions. Appendage fouling of larvae may result in impairments to swimming and feeding abilities.

Fisher and Wickham (1976, 1977) suggested that microbial fouling of eggs and larvae of *Cancer magister* could explain the decline in dungeness crab catches recorded from the

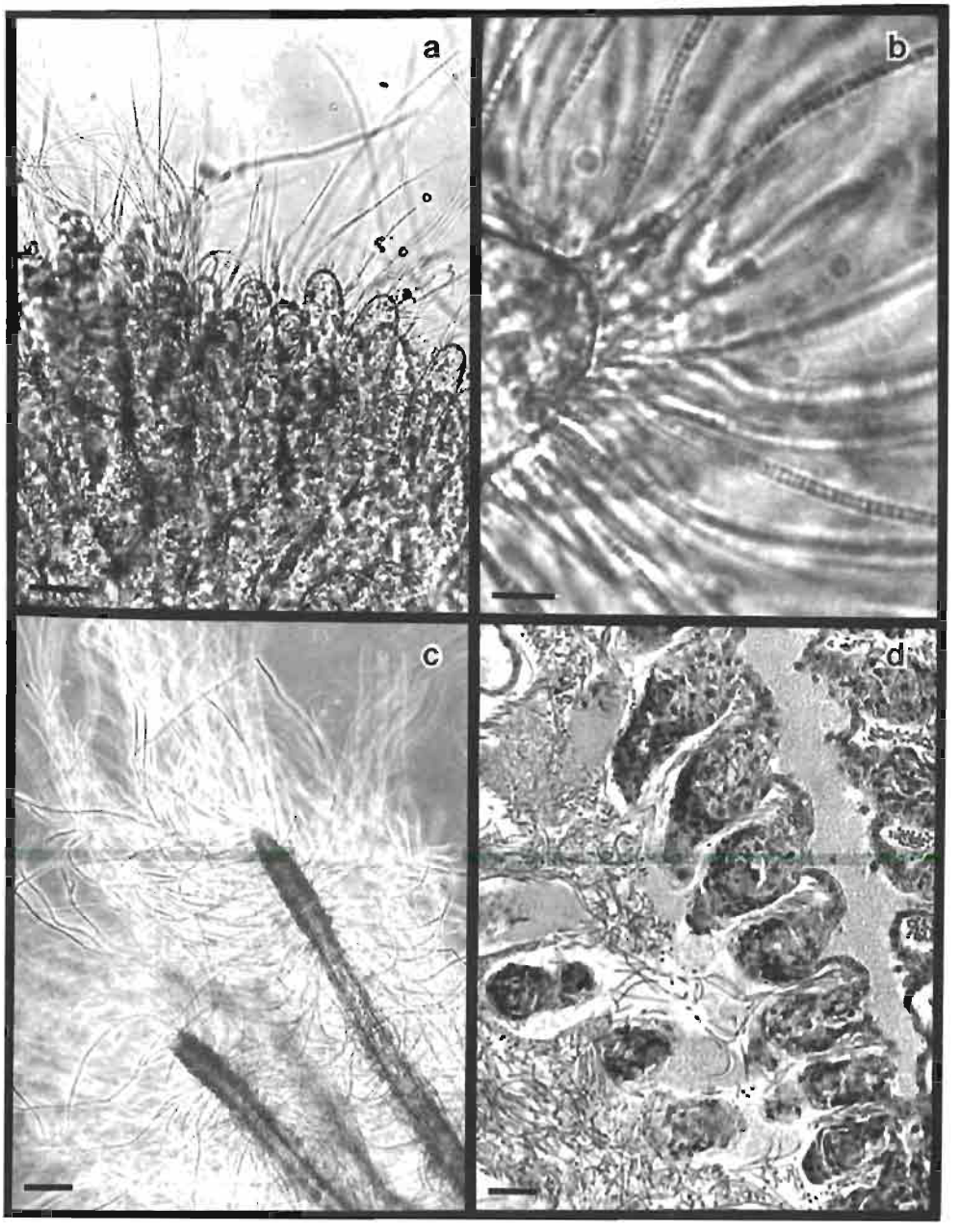

Fig. 3-14: Gill and surface fouling of penaeid shrimp. (a) Severe fouling of the gills of a juvenile *Penaeus californiensis* by the filamentous bacterium *Leucothrix mucor;* no stain; bar = 50 μm. (Original.) (b) Higher magnification view of *L. mucor* filaments fouling the surface of a gill lamellus; no stain; bar = 5 μm. (Reprinted with the permission from Lightner, D. V. 1983. © CRC Press.) (c) Severe fouling of a gill mastigobranchia of a juvenile *P. californiensis;* phase contrast; bar = 50 μm. (After Lightner, 1988. Reprinted with the permission of Elsevier Science Publishers.) (d) Histological section of the gills of a juvenile *P. californiensis* fouled by filamentous blue-green algae *Spirulina subsala;* H&E; bar = 25 μm. (Original.)

San Francisco Bay region. They found higher levels of microbial fouling associated with increased egg mortality in the later stages of egg development for crabs collected from locations receiving effluent from San Francisco Bay. Eggs examined from crabs collected from areas further north along the California coastline showed lower levels of microbial fouling. The internal pathology of crabs was not examined. Therefore, the possible role of a systemic biotic disease, i.e., chlamydial disease of dungeness crabs (Sparks and Morado, 1985), as a potential factor in the decline of crab abundance is unknown.

Microbial epibionts are a continual problem to contend with in cultured marine crustaceans (Barkate and co-authors, 1974; Lightner, 1975, 1977 his Chapter 3.1.4, 1983, 1985, 1988 his Chapter 3.1.10; Nilson and co-authors, 1975; Delves-Broughton and Poupard, 1976; Fisher and co-authors, 1976a; Aquacop, 1977; Fisher, 1977a, b, c, 1988a, c; Fisher and Nelson, 1977; Johnson, S. K., 1978; Solangi and co-authors, 1979; Lewis and co-authors, 1982; Johnson, 1983; Bell and Lightner, 1987a; Baticados, 1988). Morphologically, the disease manifests itself via the presence of microbial epibionts on cuticular surfaces. In heavy infestations of gills, discoloration is apparent, and color changes range from yellow-brown to brown-black. Mixed infections with bacteria, algae (Fig. 3-14, d) and protozoans are commonly encountered (Lightner, 1977 his Chapter 3.1.4, 1988 his Chapter 3.1.10).

Infestation by filamentous forms (Fig. 3-14, a-c) can be detected under a dissecting microscope with transmitted light or under a compound microscope (100× or higher). Non-filamentous bacteria require evaluation with a compound microscope under oil immersion (Fisher, 1977a, b, c, 1988a, c). According to Lightner (1977 his Chapter 3.1.4, 1988 his Chapter 3.1.10), in larger shrimp filamentous growth can be found especially on the setae of uropods, pleopods, pereiopods, antennal scales and other mouth appendages, gill lamellae and tips of gill mastigobranchiae (Fig. 3-14, c). Lightner (1983) provided a wet-mount microscopic grading scale assessing the severity of epibiotic infestation by *Leucothrix mucor* of gills and appendages of cultured penaeid shrimp. The grading of disease incidence and severity guides culturists in management or treatments for reducing fouling organisms by direct observational data on abundance of fouling organisms on critical gill surfaces.

Bacterial epibiont infestation results in none-to-minimal structural changes to the cuticle; internal pathological responses to infestation are not known. However, Solangi and co-authors (1979) noted in their study of *Leucothrix mucor* infection of *Artemia* that the cuticle at the site of attachment was rougher than adjacent cuticle; no underlying cellular damage was found, in spite of slight penetration at the site of the holdfast.

Reports supported by measurement data have not been published on functional internal alterations (i.e., reduced hemolymph O_2, decreased pH, etc.) which would explain how microbial epibionts cause mortality. However, interference with O_2 uptake and resultant tissue hypoxia are the stated reasons for morbidity and death from microbial epibiont fouling disease (Fisher, 1977c, 1988c; Lightner, 1977 his Chapter 3.1.4, 1983, 1988 his Chapter 3.1.10). Reduced gas exchange (because bacterial colonies on critical gill lamellar or egg surfaces act as a physical barrier restricting water flow) and the formation of a 'mucoid layer' by non-filamentous bacteria (that impedes the rate of gas exchange, or competition for available O_2 in water passed over the gills by the aerobic microflora) have been suggested as potential means by which the microorganisms reduce O_2 available to the host animal (Fisher, 1977c, 1988c).

Management of microbial epibiont infestation in cultured crustaceans should take into consideration: Nutrient loading in the water (incoming and rearing), host species and stage of host development (susceptibility to infestation and preening), and use of chemical compounds. Of particular importance is good water quality (Barkate and co-authors, 1974; Nilson and co-authors, 1975; Delves-Broughton and Poupard, 1976; Aquacop, 1977; Fisher and Nelson, 1977, 1978; Lightner, 1977 his Chapter 3.1.4, 1983, 1988 his Chapter 3.1.10; Sunaryanto, 1986). Primarily, this reflects rearing conditions with seawater low enough in soluble nutrients to be restrictive to growth of heterotrophic bacteria.

According to Fisher (1977c, 1988c), inline use of activated charcoal, microfiltration and ultraviolet irradiation of incoming water can help to limit the introduction of soluble and particulate organics. While useful for incubating eggs, this may do little for larvae or juvenile/adults, since addition of feeds add large amounts of nutrients to the water. Pretreatment with antibiotics of live food (e.g. *Artemia* sp.) introduced into larval cultures was not effective in disease prevention (Fisher, 1988c). Addition of microalgae (e.g. *Phaeodactylum tricornutum*) supports the survival of larval dungeness crabs in the light but is detrimental in the dark (Fisher and Nelson, 1977). Physical characteristics of rearing container and diet have been suggested to be important factors (Fisher, 1988a, c). Rate of water exchange, water distribution and flow patterns that prevent the accumulation of detritus, uneaten feed and soluble organics in the container are of obvious relevance to control heterotrophic bacteria. Feeds that breakdown rapidly in water release high levels of soluble and insoluble inorganics. Obviously, crowding is an important consideration in the control of epibiotic microbial fouling. High stocking densities lead to increased nutrient loading of the water and increased abundance of fouling organisms, but quantitative relations of nutrient levels and bacterial response to these for particular cultured marine crustaceans have not been investigated. This is an important area for future studies.

Ecdysis results in shedding of epibionts with the exuviae. Also, preening has been shown to be effective in reducing epibiont infestations. Bauer (1979) and Fisher (1983a) showed that ablation of cleaning chelipeds of the caridean shrimps *Heptacarpus pictus* and *Palaemon macrodactylus* resulted in severe gill and egg fouling in brooding females. Duration of the egg incubation period is also important. For example, *Cancer magister* broods eggs for 3 months, *Homarus americanus* for 9 months. Preening of eggs by the adult female must be important for epibiont control in these species.

There are life-stage differences in susceptibility to epibiotic microbial fouling. Eggs and larval stages appear to be more prone to fouling than juveniles and adults (Fisher and Nelson, 1977), possibly because the former life stages lack the ability to preen. Thus, younger life-stages may require increased management efforts to control fouling disease.

Chemical control of epibiont microbial diseases in cultured crustaceans has received considerable attention. Principal compounds useful for control are malachite green, various antibiotics and chelated copper compounds.

Fisher (1988a, c) recommends malachite green for treating eggs of *Cancer magister* and eggs and larvae of *Homarus* spp. For *C. magister* eggs, 1 ppm (mg l^{-1}) malachite green is administered to the water for 30 min 3 times per week. Malachite green is toxic to larval crabs and should not be used for these stages. Lobster eggs tolerate well a bath treatment of 5 ppm solution of malachite green in seawater for 10 min, and lobster larvae are treated similarly every other day for 2 min. Malachite green is not effective in treating bacterial

epibiont infestations of larval penaeid shrimp, nor is this compound approved for fishery use in the United States.

Fisher (1977a, b, c, 1988a, c) and Fisher and Nelson (1977, 1978) summarize the results of treatment trials for microbial epibionts in cultures of *Cancer magister* larvae. In zoea larvae, antibiotics administered to the water 3 times weekly improved survival, as compared to non-treated controls. Chloramphenicol (1–10 ppm) was particularly effective and gave similar results as 100 ppm each of penicillin and streptomycin. Lower dosages of penicillin and streptomycin improved survival over non-treated controls, but were less satisfactory from the standpoint of survival than the chloramphenicol or high penicillin/streptomycin treatments. One part per million each of penicillin and streptomycin increased larval survival without a noticeable reduction of filamentous epibionts. The investigators remarked that this may indicate the non-filamentous forms are more important as a cause of mortality than the filamentous bacteria. Fisher and Nelson (1977, 1978) concluded that periodic treatment with antibiotics is necessary for high survival of cultured *C. magister* larvae.

According to Lewin (1959) filamentous *Leucothrix mucor* are inhibited in culture by addition of 25 ppm streptomycin. Solangi and co-authors (1979) found a 100 ppm oxytetracycline bath for 48 h an effective treatment for *L. mucor* infestation of cultured *Artemia* sp. Salinity reduction to 10 ppt provided similar improvement in survival (Solangi and co-authors, 1979).

Antibiotic treatments are, in many cases, standard procedure for larviculture of penaeid shrimp. These treatments presumably control microbial epibionts and other Gram-negative bacteria causing shell disease and septicemia. Some antibiotics commonly used in shrimp hatcheries are chloramphenicol 0.5–10 ppm, nitrofurazone 1–5 ppm, oxytetracycline 100 ppm, neomycin 10 ppm, streptomycin 1–4 ppm (Lightner, 1983, 1988 his Chapter 3.1.10). These compounds are administered as water treatment for general control of bacteria, of which epibiotic fouling is one manifestation. However, after the hatchery period of cultured penaeid shrimp, antibiotic treatment is not a standard practice except, possibly, for very high-density cultures. For post-hatchery and older shrimp, antibiotics are administered in the feed (Bell and Lightner, 1987a).

Filamentous bacterial gill fouling, usually involving *Leucothrix mucor* as the dominant agent, can be a significant problem in intensive grow-out (juvenile-subadult) shrimp culture (Lightner, 1975, 1983, 1988 his Chapter 3.1.10). A chelated copper compound, Aquatrine, that has US Environmental Protection Agency approval for use in shrimp culture, and is soluble in seawater, is effective in the control of filamentous bacterial gill fouling, especially due to *L. mucor* (Lightner and Supplee, 1976). Copper dosages recommended for control of filamentous gill fouling of raceway-cultured shrimp are 0.1 ppm copper added for 24 h as a drip with continuous water flow-through, or 0.2 to 0.5 ppm copper in 4 to 6 h static bath treatment (Lightner, 1983, 1988 his Chapter 3.1.10). Delves-Broughton and Poupard (1976) reported the successful treatment of *L. mucor* infected eggs of *Palaemon serratus* with a 1 min dip in a 1:2,000 (500 mg l^{-1}) solution of copper sulfate.

Bacterial shell disease (brown or black spot disease) is an ulcerative condition of the external crustacean integument (Rosen, 1970). Ulcerative lesions may be focal to multifocal, small to large, static to slowly spreading, but are usually confined to the chitinous exoskeleton (Fig. 3-15, a, b). The etiology is complex, but eventually involves apparently

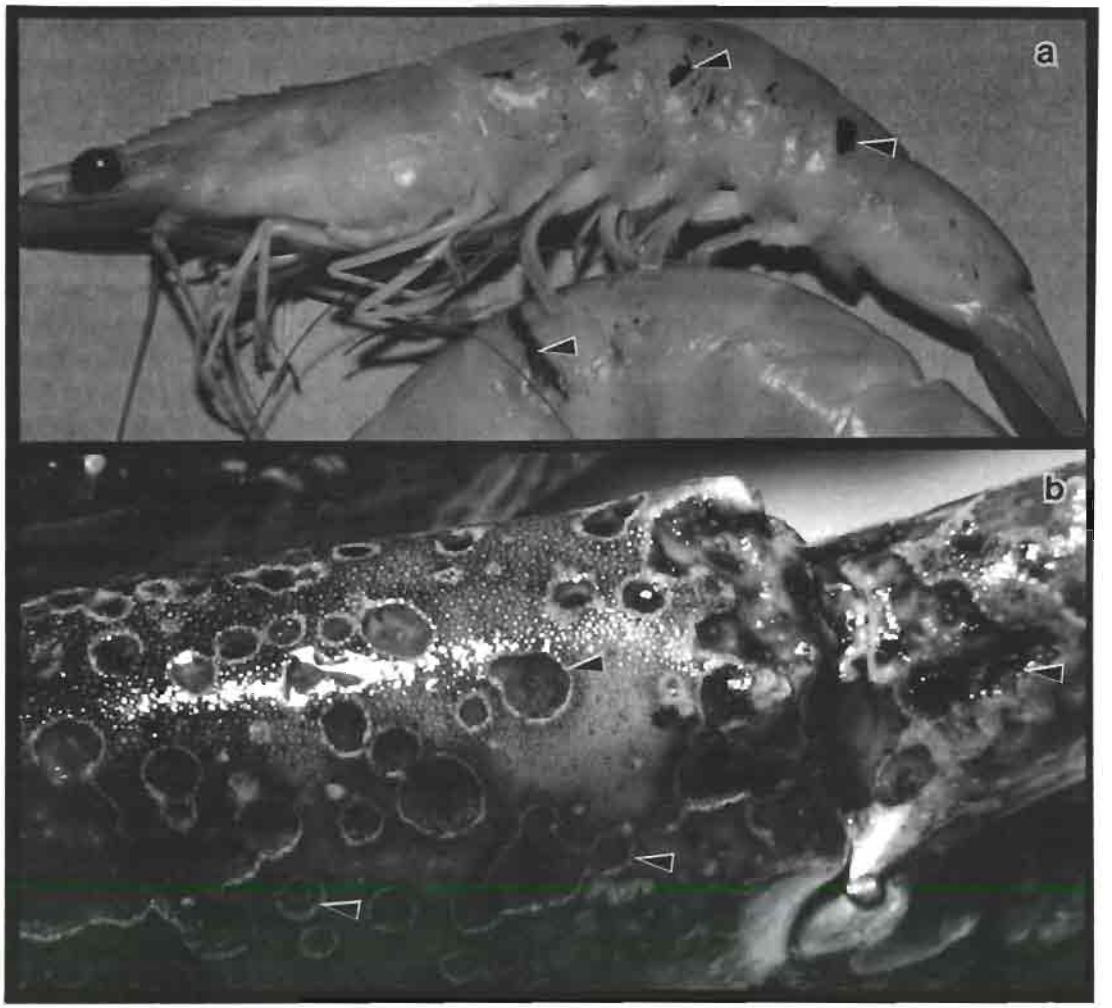

Fig. 3-15: Shell disease. (a) Multifocal, melanized 'black spot' cuticular lesions (arrow heads) of a subadult *Penaeus vannamei*. (b) Multiple 'pitted' exoskeleton lesions (arrow heads) on a chela of an adult female *Scylla serrata*. (Originals.)

low-virulence, Gram-negative, chitinoclastic bacteria, and/or fungi (see p. 339). Lesions are usually melanized indicating an active host inflammatory response to the breach in the cuticle and degradation of chitin in the integument. An initiating determinant such as physical (wounding) trauma (Delves-Broughton and Poupard, 1976) or chemical (pollution) injury (Gopalan and Young, 1975), that provides an opening through the outer, protective epicuticle, seems to be a prerequisite for development of shell disease lesions.

Shell disease is described from a variety of freshwater and marine Crustacea. Excellent reviews are those by Rosen (1970), Johnson (1983) and Getchell (1989). The disease has been documented from a wide range of natural environments (Rosen, 1970) as well as from captive, cultured and impounded crustacean stocks (Johnson, 1983). Bogdanova (1957) and Mann and Pieplow (1938 cited in Rosen, 1970) noted shell disease in

several species of freshwater mysids, amphipods and decapods. In marine Crustacea, shell disease is reported from decapods, although undoubtedly its occurrence is not limited to this order. Hess (1937) first described the disease in *Homarus americanus;* they coined the descriptive name 'shell disease'. Within the marine macrura, shell disease of bacterial etiology is also recorded from *H. gammarus* (Roald and co-authors, 1981; Fisher, 1988b), *Panulirus argus* (Sindermann and Rosenfield, 1967) and *P. guttatus* (Iversen and Beardsley, 1976). Shell disease has further been documented from the anomurans *Paralithodes camtschatica* and *P. platypus* (Bright and co-authors, 1960 cited in Sindermann and Rosenfield, 1967). Shell disease has been reported from 8 brachyurans, the majority of which are commercially harvested: *Callinectes sapidus* (Rosen, 1967; Sindermann and Rosenfield, 1967; Cook and Lofton, 1973; Sandifer and Eldridge, 1974 cited in Getchell, 1989), *Cancer magister* (Sindermann, 1977a; Baross and co-authors, 1978), *C. irroratus* (Young and Pearce, 1975), *Chionoecetes tanneri* (Baross and co-authors, 1978), and *Menippe mercenaria, Panopeus herbstii, Carpilius corallinus* and the land crab *Cardisoma guanhumi* (Iversen and Beardsley, 1976). Shell disease is also known from *Palaemon serratus* (Forster and Wickens, 1972; Delves-Broughton and Poupard, 1976), and *P. platyceros* (Forster and Wickens, 1972), *Crangon septemspinosa* (Gopalan and Young, 1975) and *Pandalus borealis* (Rinaldo and Yevich, 1974); *Penaeus duorarum, P. setiferus* and *P. aztecus* (Cook and Lofton, 1973), *P. occidentalis* (Iversen and Beardsley, 1976) and *Penaeus* spp. (Lightner, 1977 his Chapter 3.1.3, 1988 his Chapter 3.1.9; Johnson, S. K., 1978; Baticados, 1988).

Since the earliest report (Hess, 1937), American and Canadian investigators have emphasized a bacterial etiology for shell disease (Currier and Duemmling, 1949; Sawyer and Taylor, 1949, Bright and co-authors, 1960 cited in Sindermann and Rosenfield, 1967; Rosen, 1967, 1970; Cook and Lofton, 1973; Fisher and co-authors, 1976a; Lightner, 1977 his Chapter 3.1.3, 1983, 1985, 1988 his Chapter 3.1.9; Sindermann, 1977a, 1988a; Baross and co-authors, 1978; Fisher and co-authors, 1978; Malloy, 1978; Cipriani and co-authors, 1980). Hess (1937) and Rosen (1967) recovered chitin-digesting bacteria from shell lesions of *Homarus americanus* and *Callinectes sapidus.* Attempts by Hess and Rosen to infect healthy lobsters and blue crabs with the cultured isolates were unsuccessful. Cook and Lofton (1973) recovered chitinoclasts from lesions in 3 species of penaeid shrimp and in the blue crab, and classified the bacteria as members of the genera *Vibrio, Pseudomonas* and *Beneckea**. Cook and Lofton (1973) found that the predominant bacteria isolated from lesions, designated *Beneckea* type I, caused necrosis of cuticle in blue crabs, but only in areas of the cuticle that were first damaged. The investigators concluded the bacterium functions as an opportunist causing disease if the epicuticle layer is first disrupted.

Other researchers report similar findings. Malloy (1978) studied shell disease in *Homarus americanus,* collected in waters of Yarmouth and Cape Sable, Nova Scotia. *Vibrio* spp. and *Pseudomonas* spp. were identified from shell lesions, and a *Vibrio* sp. caused similar lesions in experiments when the cuticle had been damaged prior to inoculation (Malloy, 1978). In addition, Cipriani and co-authors (1980) found 10 distinct phenotypes of bacteria among isolates from 20 lesions. The bacteria were placed into 4 genera: *Vibrio, Altermonas, Spirillum* and *Flavobacterium.* In experiments, 4 isolates were

* *Beneckea* is listed as a Genus *Incertae sedis* (Shewan and Véron, 1974) and a number of species formerly placed in *Beneckea* are now included in *Vibrio* (Lightner, 1988 his Chapter 3.1.9).

able to initiate lesion formation in abraded cuticle of otherwise healthy penaeid shrimp. Laboratory-induced infections did not result in death. The bacteria that caused lesions were 2 *Vibrio* spp., 1 *Alteromonas* sp. and 1 *Spirillum* sp. Cipriani and co-authors (1980) concluded that shell disease is an injury-related, low-virulence condition in which several heterotrophic bacterial taxa that elaborate lytic exoenzymes (lipases, chitinases, proteases) are involved. Delves-Broughton and Poupard (1976) isolated myxobacteria and *Vibrio* spp. from shell lesions of cultured *Palaemon serratus*. These researches also concluded that shell disease occurred as a "secondary infection following mechanical injury" (p. 203).

Baross and co-authors (1978) isolated chitin-digesting bacteria from all shell lesions studied in tanner and dungeness crabs *Chionoecetes tanneri* and *Cancer magister*. Sixty bacterial isolates were examined in the study. The tanner crab chitinolytic isolates were phenotypically heterogeneous with 3 distinct groups recovered. The dominant group, 28 isolates, was luminescent and identified as *Photobacterium* sp., 8 isolates resembled *Moraxella* spp. and the others were similar to *Vibrio anguillarum*. The isolates from dungeness crab lesions were phenotypically similar to *V. anguillarum* (Baross and co-authors, 1978).

A variety of abiotic determinants have been reported that initiate, or contribute to, shell disease in Crustacea. Of importance are circumstances that cause trauma or wounding of the integument and thus a break in the epicuticle; chronic contact exposure to high levels of heterotrophic bacteria (i.e., increased water temperature and/or high nutrient loading); or pollutants which degrade the epicuticle causing lack of proper formation or maintenance of the epicuticle and lack of molting of the host (Rosen, 1970; Cook and Lofton, 1973; Gopalan and Young, 1975; Young and Pearce, 1975; Fisher and co-authors, 1976b; Baross and co-authors, 1978; Malloy, 1978; Cipriani and co-authors, 1980; Estrella, 1984). For example, crowding of lobsters or crabs in impoundments or shedding tanks during periods of elevated temperature and poor water circulation is likely to result in increased incidence of shell disease, and almost certainly, gaffkemia as well. Getchell (1989) notes that shell disease is not a major problem for lobster dealers in Massachusetts (USA) where coastal impoundments are uncommon and lobsters are rapidly moved through the marketplace. However, the disease continues to cause problems where lobsters are held in coastal pounds. Experimental exposure to high levels of bacteria and 'pollution' resulted in development of shell disease. According to Young and Pearce (1975), healthy crabs held in aquaria with sewer sludge developed shell lesions while controls remained free of the disease. Similarly, Gopalan and Young (1975) recorded a 50 % incidence of shell disease in *Crangon septemspinosa* raised in natural seawater; controls in artificial seawater fortified with antibiotics remained uninfected. Dietary deficiency that causes a defect in the epicuticle has been suggested as an abiotic determinant for shell disease in lobsters (Fisher and co-authors, 1976b; Fisher, 1988b). In summary, as Johnson (1983) concludes, many factors may contribute to the occurrence of shell disease in Crustacea.

Shell disease has been reported by several authors to be 'definitely contagious' (Rosen, 1970; Johnson, 1983; Sindermann, 1977a, 1988a). Taylor (1948 cited in Rosen, 1970) observed a rise from 0 to 72 % in shell disease prevalence in impounded lobsters over a 4 month period. Experimentally, shell disease has been successfully induced when the epicuticle was removed prior to bacterial inoculation (Bright and co-authors, 1960 cited in Sindermann and Rosenfield, 1967; Cook and Lofton, 1973; Malloy, 1978; Cipriani and co-

authors, 1980); however, induction attempts were unsuccessful if the epicuticle was left intact (Hess, 1937; Sawyer and Taylor, 1949; Rosen, 1967). This suggests that additional factors are involved in shell disease transmission. Clearly, wounding is an important physical determinant. However, a complete understanding of the factor(s) necessary for shell disease to be contagious has not been defined under controlled conditions. Getchell (1989) surmised that shell disease is contagious only under conditions of poor environment or crowding.

Incidences of shell disease in wild populations of marine crustaceans vary. Bright and co-authors (1960 cited in Sindermann and Rosenfield, 1967) found the natural incidence of shell disease in king crabs to be 11 % or less. Hess (1937) and Malloy (1978) indicated shell disease is rare in wild lobster populations, but prevalence of affected individuals can be high in lobster floats. Fifty percent or more wild-caught blue crabs may have shell disease (Sindermann, 1977a, 1988a). Gopalan and Young (1975) reported 30 % incidence of shell disease in *Crangon septemspinosa* in certain locations in the New York Bight (USA). Seventy six percent of adult females, 29 % of adult males and 0 % of juveniles examined had shell disease in natural populations of tanner crabs (Baross and co-authors, 1978). The latter point out that adult females in the family Majidae do not molt after the puberty molt, and that lesions were vastly more common and severe in this non-molting subpopulation. According to Getchell (1989), the impact of shell disease-induced mortality in natural crustacean populations has not been determined.

Incidence of shell disease is also variable in captive-wild or cultured marine crustaceans. In shedding floats in Maryland (USA) about 3 % of blue crabs were affected (Rosen, 1967), and on some occasions 100 % of tank-reared penaeid shrimp may be affected (Lightner, 1988 his Chapter 3.1.9).

Investigators differ on their findings for shell disease-related (caused) mortality. Taylor (1948 cited in Johnson, 1983) observed a mortality rate of 71 % in lobsters with shell disease, and of 6 % in the unaffected lobsters. According to Rosen (1970) shell disease is not rapidly fatal, but many affected individuals do die. Aquacop (1977) noted high mortality in hatchery-reared larval penaeid shrimp and in *Macrobrachium rosenbergii* affected by 'bacterial necrosis', a form of shell disease. Cipriani and co-authors (1980) observed no mortality of penaeid shrimp with experimentally induced shell lesions following bacterial inoculation. These publications emphasize the variation in the expression of shell disease related to host susceptibility, pathogen and environmental factors; the occurrence of relatively high incidence of shell disease in marine crustaceans — natural, captive or cultured — may be a clear indication that the population is acutely-to-chronically exposed to a deteriorated environment or to nutritional conditions that are unsuitable for supporting the natural integrity of the integument. Pathologically, shell lesions are usually limited to the integument (Fig. 3-16, c). Exceptions to this are reported for crustaceans with a thin cuticle (i.e., shrimp and larval stages) where the lesion may extend into the epidermis or subepidermal tissues (Gopalan and Young, 1975; Fisher, 1977d, 1988b). Lesions extending into deeper layers may result in death at the time of ecdysis because of failure of separation of the old and new exoskeleton at sites of shell lesions (Fisher and co-authors, 1976b), or provide a portal of entry for internal bacterial infection (Lightner, 1983).

The pathology of shell disease is similar for all crustaceans. Any cuticular surface may be affected. Sites and distribution of lesions undoubtedly reflect the direct and contributo-

ry etiologies involved in the disease process for a given situation. Affected areas are soft, sometimes friable, and cratered (Fig. 3-15, b). Shell erosions are frequently melanized (Fig. 3-15, a); lesions may progressively enlarge and eventually coalesce covering large areas of the integument (Getchell, 1989). Distal segments of affected appendages may be missing in shrimp (Gopalan and Young, 1975) and larval stages. Shell lesions are shed when the crustacean molts, but missing tips of appendages may not regenerate (Gopalan and Young, 1975). Borders of shell lesions may seal by melanization. Presumably, this 'healing' follows elimination of chitinoclastic bacteria in the lesion.

Monitoring shell disease occurrence is easily done by gross examination and may have a place in population surveys and health assessment of impounded and cultured stocks. Getchell (1989) suggests rapid diagnosis of chitinoclasts by use of a new filter paper spot test (O'Brien and Colwell, 1987 cited in Getchell, 1989) that assays for chitobiase.

Management of shell disease is practical in captive and cultured populations. Rosen (1967) suggests reduced crowding and shortened holding time for blue crabs in shedding operations. For lobsters, shell disease problems may be reduced by wound avoidance, and proper husbandry and system hygiene (Stewart, 1980, 1984). Fisher and co-authors (1978) and Fisher (1988b) suggest animal isolation for wound prevention, system sterilization, disinfection of incoming water, waste removal, selective culling of affected individuals and proper nutrition to provide for a healthy epicuticle. Stewart (1980) and Johnson (1983) confirm the importance of reduced crowding, keeping organics at low levels, maintenance of low water temperature and prompt removal of exuviae from holding chambers as hygienic measures, and environmental manipulations that reduce bacterial biomass, curb microbial multiplication, animal aggression and occurrence of shell disease.

An 8 min dip of lobster larvae in 20 ppm malachite green has been effective as treatment for shell disease (Fisher and co-authors, 1978). Antibiotic treatment is indicated (Lightner, 1988 his Chapter 3.1.9) for control of shell disease in cultured shrimp and other crustaceans as well (Austin and Alderman, 1987). However, the treatments suggested should be accompanied by identification and correction of factors predisposing for shell disease.

Gram-negative bacteria also invade the internal tissues of marine crustaceans. Of particular importance in captive rearing and aquaculture are systemic infections where bacteria are distributed and multiplying, often virtually unchecked, in the hemolymph channels (Fig. 3-16, a) and, in some cases, the other tissues (Fig. 3-16, b, d) as well. Because *Vibrio* spp. are most frequently encountered in septic bacterial diseases of marine crustaceans (see also below), the disease is referred to as vibriosis (Lightner, 1977 his Chapter 3.1.2, 1988 his Chapter 3.1.8).

Gram-negative bacterial septicemic disease (vibriosis) is commonly encountered in marine crustaceans recently captured and in live-holding, or in acute-to-chronically stressed cultured populations. Circumstances under which vibriosis is likely to occur are as follows: For *Callinectes sapidus* recently captured and transferred into shedding or holding tanks, especially during periods of warm weather and high water temperature (Krantz and co-authors, 1969; Johnson, 1976c, 1977b, 1988f). For cultured and captive-held feral penaeid shrimp, all species and life stages, but particularly younger shrimp (larval-to-post-larval-stages) where crowding, high water temperature, poor water quality, low water exchange and low dissolved oxygen are present (Barkate, 1972; Vanderzant and co-authors, 1970b; Lewis, 1973; Lightner and Lewis, 1975; Lightner, 1977 his Chapter 3.1.2,

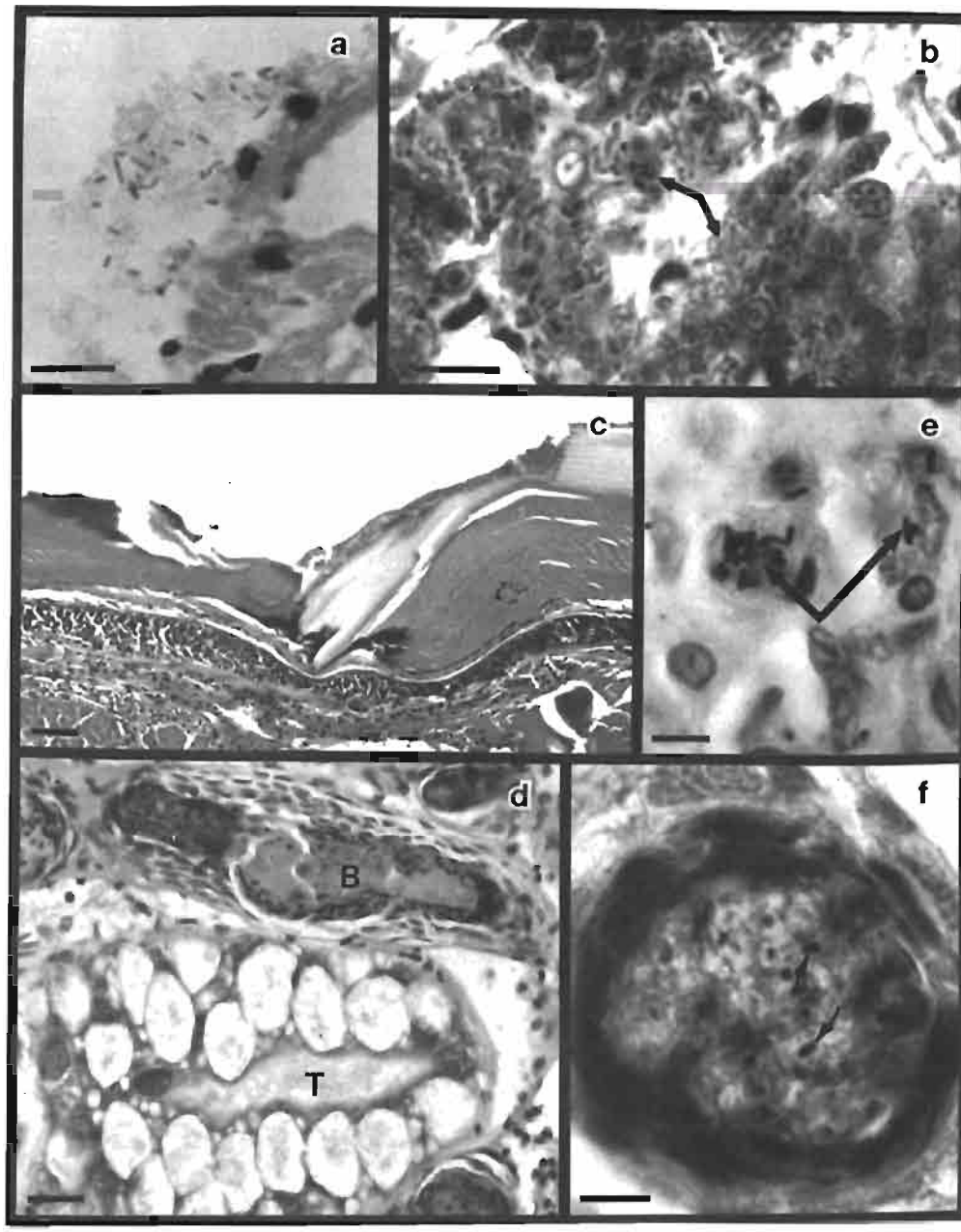

Fig. 3-16: Bacterial infections. (a) Gram-negative rods (*Vibrio* sp.) in hemolymph in the heart lumen of a juvenile *Penaeus stylirostris;* Brown & Brenn; bar = 10 μm. (After Lightner, 1977. Reprinted with the permission of Elsevier Science Publishers.) (b) Masses of bacterial rods (arrows) in the cytoplasm of sheath cells of the lymphoid organ of a juvenile *P. stylirostris;* H&E; bar = 10 μm. (c) Bacterial shell disease lesion in the cuticle of a *P. vannamei;* H&E; bar = 50 μm. (d) Light micrograph of the HP of a *P. stylirostris* showing a normal tubule (T) and a necrotic, inflamed, and septic tubule with masses of bacteria (B) within the lumen of the hemocyte encapsulated tubule; H&E; bar = 25 μm. (e and f) Acid fast bacteria (arrows) of the genus *Mycobacterium* in granulomatous lesions of the lymphoid organ of *P. vannamei;* (e) Ziehl-Neelsen, (f) Brown & Brenn; bar = 5 μm. (b to f Originals.)

1983, 1985, 1988 his Chapter 3.1.8; Leong and Fontaine, 1979; Tareen, 1982; Takahashi and co-authors, 1985b; Sunaryanto, 1986; Baticados, 1988).

Other marine decapods for which Gram-negative bacterial septic disease has been reported are *Carcinus maenas* and *Uca pugilator* (Spindler-Barth, 1976), *Homarus americanus* (Bowser and co-authors, 1981; Rosemark and Fisher, 1988), an unidentified lobster species (Brinkley and co-authors, 1976), and *Chionoecetes bairdi* (Follett and Grischkowsky, 1980; Grischkowsky and Follett, 1982). Gram-negative bacterial sepsis has also been described in natural and experimental infections of the sand flea *Talorchestia longiconus* (Inman, 1927).

The bacterial species that cause Gram-negative septicemias in marine crustaceans are predominantly members of the Vibrionaceae, mainly the genus *Vibrio* (Krantz and co-authors, 1969; Tubiash and Krantz, 1970; Vanderzant and co-authors, 1970b; Lewis, 1973; Lightner and Lewis, 1975; Brinkley and co-authors, 1976; Corliss and co-authors, 1977; Lightner, 1977 his Chapter 3.1.2, 1983, 1985, 1988 his Chapter 3.1.8; Johnson, S. K., 1978; Overstreet, 1978; Leong and Fontaine, 1979; Lewis, 1979; Tareen, 1982; Takahashi and co-authors, 1985b; Guoxing, 1986; Baticados, 1988). Another oxidase-positive Vibrionaceae implicated in septic bacterial disease of shrimp is *Aeromonas* sp. (Lightner and Lewis, 1975; Lightner, 1977 his Chapter 3.1.2, 1983, 1985, 1988 his Chapter 3.1.8). Additionally, Gram-negative *Pseudomonas* sp. have been found to be pathogenic to certain penaeid shrimp and *Chionoecetes bairdi* (Barkate, 1972; Follett and Grischkowsky, 1980; Lightner, 1983, 1985). Of interest is that *Pseudomonas* spp. predominate in septic infections of freshwater crayfish (McKay and Jenkin, 1969; Boemare and Vey, 1977 cited in Johnson, 1983). *V. parahaemolyticus* is most often reported as the offending pathogen in septic bacterial disease of marine crustaceans (Krantz and co-authors, 1969; Tubiash and Krantz, 1970; Vanderzant and co-authors, 1970b; Brinkley and co-authors, 1976; Lightner, 1977 his Chapter 3.1.2, 1983, 1985, 1988 his Chapter 3.1.8; Overstreet, 1978; Leong and Fontaine, 1979). Other vibrios implicated include *V. anguillarum* (Lewis, 1973; Lightner and Lewis, 1975; Bowser and co-authors, 1981; Rosemark and Fisher, 1988), *V. alginolyticus* (Lightner and Lewis, 1975; Brinkley and co-authors, 1976; Lightner, 1977 his Chapter 3.1.2, 1983, 1985, 1988 his Chapter 3.1.8) and *Vibrio* spp. including *V. algosus* (Leong and Fontaine, 1979), *V. harveyi* and *V. splendidus,* luminous species (Baticados, 1988), *V. panulirus* (Kusada and Watada, 1969 cited in Lightner, 1988 his Chapter 3.1.8), *V. cholerae* (non 0-1) (Guoxing, 1986), and *Vibrio* sp. (Lightner and Lewis, 1975; Brinkley and co-authors, 1976; Bowser and co-authors, 1981; Tareen, 1982; Takahashi and co-authors, 1985b; Rosemark and Fisher, 1988).

The epidemiology of septic vibriosis in marine decapods has not been studied in sufficient detail to be precisely understood. Generally, investigators reporting on Gram-negative septic disease and reviewers of decapod bacterial diseases conclude that the organisms involved are facultative pathogens (Lightner and Lewis, 1975; Brinkley and co-authors, 1976; Johnson, 1976c, 1983; Lightner, 1977 his Chapter 3.1.2, 1983, 1985, 1988 his Chapter 3.1.8; Couch, 1978; Overstreet, 1978; Leong and Fontaine, 1979; Bowser and co-authors, 1981; Sparks, 1985; Rosemark and Fisher, 1988). Such opportunistic pathogens are saprophytic heterotrophs that are widespread and constitute a significant portion of the indigenous microflora of marine water, sediments, fish and shellfish. Epidemiologically, the disease, vibriosis, occurs when the marine decapod host is compromised and natural defense mechanisms are unable to either prevent entry of bacteria into the haemocoel, or halt bacterial multiplication once the organisms have invaded.

However, Lewis (1979) has proposed an alternate hypothesis on this point. He suggests that episodes of septic disease in marine shrimp are not precipitated by opportunistic saprophytes, but rather by specific pathogenic strains that reservoir in decapod populations in chronically infected individuals. Carrier hosts may have low tolerance to adverse environmental conditions or other stressors. Once stressed, these reservoir hosts would rapidly incur acute septic disease shedding sizable numbers of pathogens that could initiate epidemic disease. Lewis presents serologic data to support his hypothesis, that is, isolates of *V. anguillarium* most often recovered from diseased shrimp were typed into 2 serogroups. Although these serogroups were widespread geographically, they usually differed from isolates of *V. anguillarum* which Lewis recovered from marine environmental samples.

It is well established that Gram-negative pathogens can be obtained from apparently healthy marine crabs and shrimps (Vanderzant and co-authors, 1971; Lee and Pfeifer, 1975; Sizemore and co-authors, 1975; Follett and Grischkowsky, 1980; Yasuda and Kitao, 1980). Furthermore, a hemolymph survey study conducted on wild-caught adult *Callinectes sapidus,* collected from Chesapeake Bay (USA) under a variety of conditions, commonly demonstrated recovery of Gram-negative taxa in the hemolymph sampled from apparently healthy animals (Colwell and co-authors, 1975). These investigators state (p. 29): "*Vibrio* spp., including *Vibrio parahaemolyticus*, were the major taxonomic groups found in the crab hemolymph"; and concluded: "Clearly the hemolymph of most healthy blue crabs is not sterile." Bang (1970), Lee and Pfeifer (1975) and Johnson (1976c, 1983) do not concur that the hemolymph of normal marine crustaceans contains bacteria. Lee and Pfeifer (1975) cultured hemolymph specimens sampled from 121 wild-caught *Cancer magister* with negative results. According to Sindermann (1971) bacteremias can occur in lobsters immediately after capture. Brinkley and co-authors (1976) recovered low numbers of bacteria (non-*Vibrio* taxa) from the hemolymph of apparently healthy, captive lobsters. These authors concluded the organisms were probably contaminants. Gram-negative bacteria have been isolated from overtly normal cultured penaeid shrimp (Lightner, 1977 his Chapter 3.1.2, 1988 his Chapter 3.1.8). Lightner's interpretation is that small numbers of Gram-negative bacteria, including *Vibrio* spp., can occur in the hemolymph of apparently healthy shrimp. Shrimp become bacteremic through wounding or when subjected to stress due to crowding, handling, molting or capture. The defense mechanisms of the shrimp are capable of controlling low numbers of bacteria, but septic disease ensues if host defenses fail to restrict bacterial entry or restrict bacterial multiplication. If successful, control of stressor-mediated bacterial invasion takes place when the organisms are ousted by the activities of the hosts' defenses (phagocytosis, encapsulation, bacterial killing within the host, elimination by diapedesis).

Lightner and Lewis (1975), Johnson (1976c, 1983), Lightner (1977 his Chapter 3.1.2, 1983, 1988 his Chapter 3.1.8) and Sparks (1985) point out the importance of trauma and wounding as a portal of bacterial entry into internal tissues of the host. Sparks (1985, p. 198) concludes, for penaeid bacterial septicemia in wild-caught shrimp that were traumatized during capture and transfer, or following handling of cultured shrimp: "It is virtually certain that *Vibrio* infections develop by invasion of cuticular wounds." Wounding allows a portal of entry for potential bacterial pathogens which are present as normal microflora on the cuticle of the crustacean host. Johnson (1976c) presents data that support this position. During a 12 day period following collection, 80% (152/190) of

laboratory-maintained, trapped *Callinectes sapidus,* died, as compared to 23 % (28/122) of carefully handled trawled crabs. Mortalities from the disease virtually ceased by Day 9. Bacterial sepsis was diagnosed in 85 % (115/135) of the trapped crabs and in 45 % of the trawled individuals. According to Johnson (1976c), less than 1 % (1/150) of carefully handled crabs died from bacterial infection. Johnson commented that many of the trapped crabs suffered wounding and/or autotomized appendages during capture and handling. These data point-out the tremendous impact that handling may have on occurrence of septic bacterial disease in recently captured blue crabs.

Lightner and Lewis (1975), Johnson (1976c, 1983), Lightner (1977 his Chapter 3.1.2, 1983, 1988 his Chapter 3.1.8) and Sparks (1985) do not exclude the possibility of other portals besides wounding for bacterial entry into the internal tissues of stressed marine crustaceans. However, studies and data that specifically identify and confirm other entry routes, i.e., via the gut or during ecdysis, have not been published. Additional information on bacterial entry routes for Gram-negative septic disease is needed. According to Lasso De La Vega and Brady (1989), adult freshwater prawns *Macrobrachium rosenbergii,* incubated in water with 10^5 to 10^6 bacteria ml^{-1}, became bacteremic (recovered from 10 to $> 10^3$ bacteria ml^{-1} of hemolymph), whereas bacteria were infrequently recovered or not recovered at all from hemolymph sampled from the low exposure group (10^2 to 10^3 bacterial ml^{-1}) or the control group, respectively. The investigators surmise that high bacterial concentration in the water influences bacteria presence in the hemolymph. But precisely how the bacteria enter the prawns is unknown.

Gram-negative bacterial septicemias and localized, internal bacterial infections (Fig. 3-16, b, d) of marine crustaceans follow, as secondary problems, earlier infection and disease by other pathogens or disease processes due to abiotic factors. Some examples to illustrate this point follow: Localized, bacteria-infected abscess-like lesions in the hepatopancreas and bacterial shell disease lesions were documented in a solitary, captive-wild ghost shrimp *Callianassa affinis* that refused feed for 2 months and finally died (Smith and Taylor, 1968). The authors concluded that prolonged malnutrition predisposed the shrimp to bacterial attack. Lightner (1978) reported localized bacterial infections with the formation of large abscess-like hepatopancreas lesions (Fig. 3-16, d) and/or fulminating bacterial sepsis, primarily due to *Vibrio alginolyticus,* as a secondary condition in *Penaeus stylirostris* affected by the necrotizing enteric disease, hemocytic enteritis. Hemocytic enteritis is caused by ingestion of blue green algal toxins (Lightner, 1978). Anderson and co-authors (1987) and Nash, G. and co-authors (1988) found terminal bacterial sepsis in *Penaeus monodon* associated with concurrent monodon baculovirus, reo-like virus and systemic rickettsial infections. In all these cases, based on histopathological interpretation, bacterial involvement appeared to have occurred late in the course of the other disease(s) and, thus, represented a secondary disease condition.

A number of laboratory exposure studies have been carried-out to demonstrate virulence of Gram-negative bacteria isolated from septic bouts in marine crustaceans. Detailed information on these trials can be found for the following marine crustaceans and the horseshoe crab: *Talorchestia longicornus* (Giard and Billet, 1889 cited in Inman, 1927; Inman, 1927); *Limulus polyphemus* (Bang, 1956); *Callinectes sapidus* (Tubiash and Krantz, 1970; Johnson, 1976c); *Carcinus maenas* and *Uca pugilator* (Spindler-Barth, 1976); *Homarus americanus* (Bowser and co-authors, 1981); *Chionoecetes bairdi* (Grischkowsky and Follett, 1982); and various *Penaeus* spp. (Vanderzant and co-authors, 1970b;

Barkate, 1972; Lewis, 1973; Lightner and Lewis, 1975; Leong and Fontaine, 1979; Takahashi and co-authors, 1985b).

The following conclusions are supported, at least in part, by results presented in the above-listed studies. In most cases, route of exposure (injection versus water borne), species of bacteria used, and exposure dosage (# bacteria injected indiv.$^{-1}$) were important determinants. Experimentally, water-borne exposure did not successfully transmit the disease (Barkate, 1972; Lewis, 1973; Lightner and Lewis, 1975; Johnson, 1976c; Leong and Fontaine, 1979; Bowser and co-authors, 1981) and the agents must be injected at high dosages to insure onset of the disease. However, Guoxing (1986) indicated that immersion exposure to *Vibrio cholerae* (non-01) successfully transmitted the disease. Regarding injection exposure, blue crabs and penaeid shrimp are quickly killed when inoculated with *Vibrio* spp. *Vibrio parahaemolyticus* injected into the haemocoel rapidly kills a high percentage of blue crabs (Tubiash and Krantz, 1970) as does injection of bacterially infected hemolymph from septic crabs (Johnson, 1976c). Intramuscular injection of 10^4 to 10^8 bacteria indiv.$^{-1}$ was rapidly fatal to *Penaeus setiferus* (Leong and Fontaine, 1979). *V. parahaemolyticus* was the most virulent species, followed by *V. anguillarum, V. algosus,* and *V. alginolyticus.*

High dosages of microbial agents must be injected for disease signs to develop. Infection dosage of 1×10^5 or less resulted in no mortality of experimentals for *Vibrio anguillarum, V. algosus* and *V. alginolyticus* (Leong and Fontaine, 1979). Lightner and Lewis (1975) demonstrated *Aeromonas* sp. to be pathogenic to shrimp when injected at levels of about 10^5 bacteria indiv.$^{-1}$. Spindler-Barth (1976), working with crabs, reported clear differences in disease development related to exposure dosage; 100% of the crabs died within 2 days after injection (more than 4×10^5 bacteria crab^{-1}), but a dosage of 3×10^5 resulted in disease signs in about one-third of the injected crabs with an onset on Days 14 to 24 post injection. However, if the exposure dosage was reduced to 2×10^5 bacteria crab^{-1}, no disease signs were observed. Experimental injection of *Pseudomonas* sp. into *Chionoecetes bairdi* at dosages of 10^8 to 10^9 bacteria crab^{-1} resulted in significantly higher mortality than in controls (Grischkowsky and Follett, 1982). Bowser and co-authors (1981) reported high mortality of juveniles injected with 10^5 bacteria lobster^{-1} for *Vibrio* sp. and *V. anguillarum.*

Inoculation into marine crustaceans of certain types of Gram-negative bacteria has not resulted in signs of bacterial septicemia. Bowser and co-authors (1981) injected juvenile lobsters with *Enterobacter aerogenes* at a dosage of 10^5 bacteria indiv.$^{-1}$, but disease signs were not observed. Tubiash and Krantz (1970) indicated that blue crabs were killed by injection challenge with *Vibrio parahaemolyticus* and other *Vibrio* spp. but that the crabs were refractory to similar exposure to bacterial pathogens of vertebrate fishes. *Micrococcus conglomeratus,* a Gram-positive coccus, caused no mortalities in *Penaeus setiferus* following injection exposure of 1.9×10^7 bacteria indiv.$^{-1}$ (Leong and Fontaine, 1979). These data point out the role of virulence factors for the species of bacteria and also that high numbers of agents must be injected before fulminating bacterial sepsis is reproduced in crustacean hosts.

Bacterial toxins have been implicated by several investigators in the pathogenesis of bacterial septicemia in marine crustaceans (Vanderzant and co-authors, 1970b; Johnson, 1976c; Leong and Fontaine, 1979; Bowser and co-authors, 1981). Vanderzant and co-authors (1970b) suggested an exotoxin might be involved in the pathogenesis of *Vibrio*

parahaemolyticus infection of shrimp because of the peracute onset (0.5 to 3 h) and rapidly fatal course of the disease. Leong and Fontaine (1979) also cite the inoculation-dependent, swift course following inoculation as suggestive of a toxin-mediated disease process. Additionally, these investigators note that a toxic factor may explain why the agent was not always recovered from moribund test shrimp. Bowser and co-authors (1981) injected sterile wash supernatants from broth cultures of *Vibrio* sp., *V. anguillarum* and heat-treated filtrate of the *Vibrio* sp. Heat treated and unheated sterile filtrates from the high concentration (10^7 bacteria ml^{-1}) cultures resulted in 80 to 100% mortality of juvenile lobsters at 4 h post injection. Bowser and co-authors (1981) suggest the toxic factor is bacterial endotoxin. This interpretation is supported by work of others. Endotoxin can cause formation of *in situ* cellular clots in the horseshoe crab *Limulus polyphemus* (Bang, 1956) and similar clinical signs as observed in blue crabs dying from bacterial disease (Johnson, 1976c).

The signs and gross pathology of bacterial sepsis are not diagnostic for the disease in marine crustaceans (Lightner and Lewis, 1975; Johnson, 1976c; Leong and Fontaine, 1979). These signs and lesions include lethargy and weakness (Krantz and co-authors, 1969; Lewis, 1973; Lightner and Lewis, 1975; Johnson, 1976c, 1977b, 1988f; Leong and Fontaine, 1979; Bowser and co-authors, 1981), although hyperactive behavior preceding inactivity has been noted (Vanderzant and co-authors, 1970b; Lewis, 1973); disorientation including swimming on one side (Lewis, 1973; Lightner and Lewis, 1975); prostration in ventral, dorsal or lateral recumbency (Lewis, 1973; Lightner and Lewis, 1975; Leong and Fontaine, 1979); dorsal flexion of the tail or a head down position (Lewis, 1973; Lightner and Lewis, 1975); continuous slow movement of pleopods and pereiopods (Lewis, 1973; Lightner and Lewis, 1975; Leong and Fontaine, 1979); focal-to-diffuse opacity of the striated musculature (Lewis, 1973; Lightner and Lewis, 1975; Leong and Fontaine, 1979; Tareen, 1982; Takahashi and co-authors, 1985b); expansion of cuticular melanophores on the dorsal surfaces, resulting in darkening of these areas (Lightner and Lewis, 1975); expansion of the cuticular erythrophores of the pereiopods and pleopods, resulting in a reddening of these appendages (Lightner and Lewis, 1975; Leong and Fontaine, 1979; Tareen, 1982); irregular-sized white, cloudy areas to melanized foci in the gill lamellae (Lightner and Lewis, 1975; Johnson, 1976c; Overstreet, 1978; Takahashi and co-authors, 1985b), ventro-lateral edge of the branchiostegites (Lightner and Lewis, 1975), antennal gland and Y-organ (Johnson, 1976c), lymphoid organ (Takahashi and co-authors, 1985b) and the posterior pair of legs (swimming paddles) of *Callinectes sapidus* (Johnson, 1976c); variable-sized antemortem, acellular jelly-like clots in hemal sinuses (Johnson, 1976c) and increased turbidity and reduced clotting of withdrawn hemolymph (Lightner and Lewis, 1975; Johnson, 1976c; Spindler-Barth, 1976). Death from bacterial sepsis is often quite rapid in experimentally infected penaeid shrimp and occurs within 2 to 4 h following onset of clinical signs (Lightner and Lewis, 1975). However, in her experiments Spindler-Barth (1976) reported a subacute course in that bacterial infected crabs died within a few days after the external signs of infection were apparent.

Two reports provide data on an effect on molting of bacterial sepsis. Spindler-Barth (1976) observed that infected *Carcinus maenas* remained about 3 times longer in intermolt stages C_3 and C_4 than did non-infected crabs. Leong and Fontaine (1979) noted an opposite effect in experimentally infected *Penaeus setiferus*. Test groups of shrimp injected with 1.5×10^6 and 1.5×10^5 *Vibrio alginolyticus* indiv.$^{-1}$ molted at a rate of 5.6 and 25%,

respectively; whereas no molting occurred in the saline-injected and non-injected control groups. These investigators speculate that injection of the bacteria had a slight effect on inducing molting by altering the balance of the molting hormones.

Distinctive clinical pathology is present in decapods and *Limulus polyphemus,* dying from bacterial sepsis. Workers who investigated the hemolymph have found a marked reduction in number of circulating hemocytes (Cantacuzène, 1925 cited in Johnson, 1976c; Lightner and Lewis, 1975; Johnson, 1976c) that coincided with the loss of hemolymph clotting. Failure for hemolymph to clot occurs because of hemocytopenia due to sequestering in organs as hemocyte aggregations (Bang, 1956; Johnson, 1976c).

The hemolymph of septic decapods contains numerous Gram-negative rods; diagnosis of bacteriemia is made by direct microscopic examination of stained (Fig. 3-16, a) or unstained preparations, or by recovery of microorganisms on standard bacteriologic media. The Gram-negative bacteria that cause septic disease in marine crustaceans are not fastidious. These organisms readily grow on Tryptic Soy Agar (TSA) (Lightner, 1977 his Chapter 3.1.2, 1983), Brain Heart Infusion Agar (BHI) (Vanderzant and co-authors, 1970b; Bowser and co-authors, 1981), Marine Agar (Lightner, 1977 his Chapter 3.1.2, 1983) and the selective media Thiosulfate Citrate Bile Salts Agar (Lightner, 1977 his Chapter 3.1.2, 1983). Marine vibrios are typically halophilic, and addition of 1.5 to 3% NaCl to TSA and BHI is necessary for bacterial recovery from clinical samples (Lightner and Lewis, 1975; Lightner, 1977 his Chapter 3.1.2, 1983).

Spindler-Barth (1976) provides the only information on alteration of serum and tissue chemical constituents in decapods affected by bacterial septicemia. This investigator reported an increase in glucose in the hemolymph of infected versus healthy *Uca pugilator,* and a decrease in hemolymph total protein, copper and total lipids. Spindler-Barth attributed the elevated glucose to a stress reaction. Also found was a reduction in the glycogen content in tissues of the muscle, hepatopancreas, gills and heart in the infected group.

Lewis (1973) reported results of immunoelectrophoresis of hemolymph from *Penaeus aztecus,* infected with live *Vibrio anguillarum.* At 48 to 72 h post-inoculation a component electrophoretically similar to vertebrate beta globulin was found in the serum of challenged shrimp. This component was not detected in the serum of control shrimp. Lewis (1973) suggests the component was an inducible substance that developed as a result of exposure to the infectious agent. However, Huang and co-authors (1981), working with the freshwater prawn *Macrobrachium rosenbergii,* demonstrated the presence of natural agglutinins within the serum of healthy prawns, but observed no increase in titer following vaccination with formalin-killed *V. anguillarum* cells. Huang and co-authors (1981) noted the agglutinins of *M. rosenbergii* were dissimilar from vertebrate antibodies in structural and functional attributes. Differences between the 2 studies are in the species of host animal used and live vs. formalin-killed bacterial inoculant.

Several published studies are available on the microscopic pathology associated with bacterial septicemia in marine Crustacea. In addition, Smith and Ratcliffe (1980) report the sequential micropathological response of *Carcinus maenas* to injection with killed Gram-negative bacteria. Johnson (1976c), in a very thorough study of the morphological pathology in *Callinectes sapidus* found, in addition to the changes already listed (p. 315), progressive formation of hemocyte aggregations with necrotic centers in the cardiovasculature system. By the third day, numerous hemocyte nodules, often centrally rich in

bacteria, occurred in the gills, antennal gland and the Y-organ. Large antemortem plasma clots and focal, massive areas of ischemic tissue necrosis were observed along with marked hepatopancreas atrophy, amitosis in the apical acinar epithelia and massive sloughing and necrosis of acinar epithelium in some tubules. The fixed phagocytes in the hepatopancreas, and in other locations as well, were swollen, necrotic and contained bacteria. The microscopic pathology observed was attributed to direct effect of the bacteria and endotoxins on the host tissues (Johnson, 1976c).

Smith and Ratcliffe (1980) obtained results similar to those reported by Johnson (1976c). Injected, heat-killed bacteria were sequestered in gills, heart and hepatopancreas in association with clumps of hemocytes that later organized into nodules, some of which developed necrotic centers. By 7 days post injection many of the nodules had cleared from the gills. Smith and Ratcliffe noted that hemocyte clumping probably follows recognition of the bacteria as foreign and subsequent attachment of the microorganisms to the surfaces of 'sticky' hemocytes, which eventually adhere to each other and form nodules. Foreign particle adhesion to hemocytes turned out to be very effective in rapidly immobilizing bacteria and containment of bacterial infection in the shore crab.

Tissue pathology associated with experimental bacteria infection of *Homarus americanus* has been reported by Bowser and co-authors (1981). Their observations are limited to the hepatopancreas of lobsters injected with *Vibrio* sp., toxin and heated toxin preparations. The tissue changes were similar for all 3 exposures, but more pronounced in lobsters injected with toxin as opposed to bacteria. Tissue changes consisted of necrosis of tubule cells, intertubular edema and B-cells that had extreme vacuolation. These changes are quite different than those reported for the blue crab, but possibly represent a peracute course of the disease, or the difference between naturally acquired infection (blue crabs) with mixed-bacterial involvement versus rapid injection exposure to a high bacterial/toxin titer for the lobster.

Using an indirect fluorescent antibody technique, Lewis (1973) reported *Vibrio anguillarum* administered to *Penaeus setiferus* to accumulate primarily in the hepatopancreas. This observation is consistent with the findings of Johnson (1976c) and Bowser and co-authors (1981) regarding the localization of large numbers of bacteria within this organ.

Diagnosis of Gram-negative bacterial septic disease in marine Crustacea is based on history, signs and gross pathology, and clinical pathology/microbiology findings. Gram-negative sepsis is suspected in recently captured or transferred crustaceans, or those exposed to environmental stressors, that have an acute onset of disease with high mortality. Focal-to-diffuse opacity of striated muscle, patchy white areas in the gill lamellae or other subcuticular sites, marked loss of clotting of the hemolymph, hemocytopenia and acellular hemolymph clotting are highly suggestive of the disease. Demonstration of Gram-negative bacteria in direct microscopic examination of stained smears of the hemolymph or recovery of Gram-negative bacteria by hemolymph culture is diagnostic for the disease. However, because Gram-negative bacterial sepsis (vibriosis) is stressor-mediated (Lightner and Lewis, 1975; Brinkley and co-authors, 1976; Johnson, 1976c; Lightner, 1977 his Chapter 3.1.2, 1983, 1985, 1988 his Chapter 3.1.8; Bowser and co-authors, 1981) and may be secondary to other disease processes (Lightner, 1978; Anderson and co-authors, 1987; Nash, G. and co-authors, 1988), it is imprudent to limit the examination to the above listed steps. Other biotic and abiotic diseases must be ruled-out

by the appropriate diagnostic tests. Failure to carry-out an in-depth examination may result in an unsatisfactory outcome when control procedures are implemented.

Management strategies for prevention and control of Gram-negative septic bacterial disease in cultured marine crustaceans have been discussed by several investigators (Corliss and co-authors, 1977; Lightner, 1977 his Chapter 3.1.2, 1983, 1985, 1988 his Chapter 3.1.8; Tareen, 1982; Lewis and Lawrence, 1983; Takahashi and co-authors, 1985a; Sunaryanto, 1986; Baticados, 1988; Rosemark and Fisher, 1988). Preventive measures for cultured penaeid shrimp include maintenance of suitable water quality with low bacterial biomass through use of disinfection or filtration of incoming or recirculated water; avoidance of temperature extremes or rapid variation; use of nutritionally balanced diets and reduced handling, crowding and exposure to water hypoxia. Avoidance of environmental and nutritional stress are suggested as preventative steps for cultured lobsters (Rosemark and Fisher, 1988). Preventative measures given for wild-caught blue crabs (Johnson, 1977b, 1988f) include careful handling of crabs during capture and transport, routine cleaning of crab traps and holding tanks, and water filtration and sterilization.

Immunoprophylaxis or vaccination of penaeid shrimp is reported to provide some protection against vibriosis in field trials with pond reared shrimp (Lewis and Lawrence, 1983). Huang and co-authors (1981) reported vaccination of *Macrobrachium rosenbergii* did not result in increase agglutinin titers nor protection from injection challenge to *Vibrio anguillarum* Thus, the status of vaccination for protection of crustaceans against vibriosis remains an area for more study before a procedure can be recommended for disease control.

Chemical treatment is often used prophylactically and therapeutically for management of vibriosis in cultured marine shrimp. Treatments are more often recommended for use in the hatchery phase than in nursery or grow-out periods of culture reflecting the markedly greater significance of vibriosis to larviculture. Chemicals applied are both antibiotic and non-antibiotic substances. Their use is accompanied with variable results. A few examples of commonly listed substances for treatment of penaeid larvae are ethylenediaminetetraacetic acid (EDTA at 10 to 50 ppm), malachite green (5 to 10 ppb), chloramphenicol (1 to 10 ppm), furazolidone (1 to 5 ppm) and others (Lightner, 1977 his Chapter 3.1.2, 1988 his Chapter 3.1.8; Sunaryanto, 1986; Baticados, 1988). In juvenile to adult penaeids antibiotics (oxytetracycline, furacin) are administered in the feed (Corliss and co-authors, 1977; Lightner, 1977 his Chapter 3.1.2, 1988 his Chapter 3.1.8; Takahashi and co-authors, 1985a). Use of medication in food animals such as shrimp must comply with government regulations for these substances.

Roper (1979) reported a unique condition of wild spider crabs *Leptomithrax longipes* in New Zealand waters. About 1% of the crabs collected and examined were sexually indeterminant; a study indicated these were male crabs undergoing feminization. A rod-shaped bacterium was found in tissue smears of blood and unspecified tissues, and the author suggested these bacteria were the cause for the feminization of male crabs. No further information on this possible unique outcome of bacterial infection in a decapod host was found. With the present information, however, no conclusion can be drawn regarding a cause-and-effect relation between bacteria and feminization.

During histopathologic examination Johnson (1983) noted as rare the findings of localized bacterial infection of mid-gut and hepatopancreas of wild-caught *Callinectes*

sapidus. Bacterial colonies were found within inflammatory lesions. The agent was not identified and its significance as a cause of diseaes in the blue crab suggested to be minimal (Johnson, 1983). Also reported in wild-caught blue crabs is an occasional infection by a filamentous Gram-negative bacterium within the lumen of the hepatopancreas. The agent attaches to the brush border of hepatopancreas epithelial cells. Microvilli are denuded at attachment sites of the bacteria and individual cell necrosis was noted. However, Johnson (1983) concludes the agent is minimally pathogenic to the crab host.

An infection of *Artemia* sp., by a spirochete bacteria has been described by Tyson (1970, 1974a, b) and reviewed by Sparks (1985). Spirochetes were first observed during an electron microscopic study of the antennal (maxillary) gland of adult brine shrimp and later studied using dark field and electron microscopy from a total of 7 brine shrimp found to be massively infected. Incidence of spirochete infection was 1 % (3/261) in specimens of *Artemia* hemolymph studied using dark-field microscopy. In all, tissues from 7 spirochete-infected brine shrimp have been examined at the ultrastructure level. Sparks (1985) terms the infection 'brine shrimp spirochetosis'.

Infected brine shrimp with spirochetes have appeared grossly and behaviorally normal (Tyson 1970, 1974a, b). To date, infections have only been demonstrated from brine shrimp that originated as cysts from the San Francisco Bay area (Tyson, 1974a). In *Artemia* hemolymph under dark-field microscopy the agents are 6 to 13 μm long, 0.3 to 0.4 μm wide. Coiling of the spirochete is variable and irregular. Classification of the agent to genus and species has not been undertaken, but based on size, appearance and host, the *Artemia* spirochete may be a member of the genus *Borrelia,* known to infect other arthropods (ticks and lice) (Smibert, 1974).

Electron microscopic examination revealed spirochetes in the cytoplasm of cells in all regions of the antennal gland, as well as in the cytoplasm of hypodermal cells, muscle fibers and hemocytes, and extracellularly in the hemocoelic spaces (Tyson, 1970, 1974a, b). Spirochete-infected cells were not undergoing necrosis. However, Tyson (1974a) observed spirochetes within 'cave-like' indentations at the junction of the basal lamina and the efferent tubule epithelium. In some sections the basal lamina appeared discontinuous.

Nothing is known about transmission, course, or impact of this spirochete infection on the *Artemia* host (Tyson, 1974b). *Artemia* sp. is extremely important as a live food for cultured larval and postlarval fish and crustaceans. Sparks (1985) suggests, and we concur, further inquiry into the nature of the infection is needed.

Gram-positive bacteria cause diseases in marine crustaceans. By far the best known of these diseases, as well as the most thoroughly researched microbial disease of non-insect invertebrates, is gaffkemia of lobsters caused by *Aerococcus viridans* var. *homari*. Other Gram-positive bacteria associated with diseases of Crustacea are a streptococcus of *Carcinus mediterraneus* (Pappalardo and Boemare, 1982), a Gram-positive coccus identified with 'red disease' in *Penaeus monodon* (Lightner and Redman, 1985b), and an acid-fast bacterium (probably *Mycobacterium* sp.) of *P. vannamei* (Lightner and Redman, 1986; Krol and co-authors, 1989) and possibly other penaeid species (Overstreet, 1978).

Gaffkemia is an acute-to-chronic, almost invariably fatal disease of impounded American and European lobsters, *Homarus americanus* and *H. gammarus*. The time course of gaffkemia in lobsters is strongly temperature dependent. The disease and its bacterial nature in the American lobster were initially reported by Snieszko and Taylor

(1947). Further cultural and biochemical characterization were provided, and the name of the causative bacterium, *Gaffkya homari,* suggested by Hitchner and Snieszko (1947). Roskam (1957 cited in Stewart, 1975) assigned the name 'gaffkemia' to the disease in lobsters based on the etiologic agent and septicemic nature of the condition. The bacterium was later renamed *Pediococcus homari* based on the work of Deibel and Niven (1960 cited in Fisher and co-authors, 1978), but in time was shown to be identical to the ubiquitous *Aerococcus viridans* (Kelly and Evans, 1974). Stewart and Zwicker (1974b) suggested the name *Aerococcus viridans* variety *homari,* the name presently in use. Excellent reviews on lobster gaffkemia are available in: Sindermann and Rosenfield (1967); Stewart and Rabin (1970); Stewart (1975, 1980, 1984); Sindermann (1977c, 1988c); Fisher and co-authors (1978); Johnson (1983) and Sparks (1985).

Aerococcus viridans var. *homari* is a Gram-positive, non-motile, catalase negative, beta-hemolytic, facultative anaerobic, tetrad-forming (0.8 to 1.1 μm) coccus that grows on a variety of media in the temperature range 6 to 44 °C with an optimum of 30 °C (Hitchner and Snieszko, 1947). On media, the coccus is non-encapsulated. Colonies are circular, raised 1 to 2 mm in diameter and gray-white (Hitchner and Snieszko, 1947). The agent lacks exoenzymes (Hitchner and Snieszko, 1947; Snieszko and Taylor, 1947; Stewart and co-authors, 1969d). Lobster hemolymph added to artificial media promotes the growth of the agent (Rabin, 1965; Stewart and co-authors, 1969d). Not all strains of *A. v.* (var.) *homari* are lethal to lobsters, and repeated *in vitro* culture leads to a decline of virulence.

Aerococcus v. (var.) *homari* naturally reservoirs in lobster populations along the North American and European Atlantic coastlines, other decapod crustaceans and, possibly, marine sediments (Wood, 1965; Stewart and co-authors, 1966; Vachon and co-authors, 1981). Wood (1965) cultured *A. v.* (var.) *homari* from hemolymph of 2 in 123 (1.6 %) *Homarus gammarus* sampled from Harwich, England, but not from lobsters tested from other locations during a survey conducted in 1962. Stewart and co-authors (1966) noted a 5 % (96/2,035) infection rate of lobsters individually tested from 5 areas off the Canadian Atlantic coastline. *A. v.* (var.) *homari* incidence ranged ca 2 to 40 % in the different sample periods and survey locations. Steenbergen and Schapiro (1974) found a 10 to 50 % incidence of *A. v.* (var.) *homari* in the hemolymph of *H. americanus* entering their laboratory in California that were purchased from a San Diego wholesaler (USA). The bacterium has also been reported as the cause of a low-incidence infection of other decapods in nature, including *Libinia emarginata, Carcinus maenas, Cancer borealis* (Rabin and Hughes, 1968; Gallagher and co-authors, 1979); *Penaeus aztecus* (Luizzo and co-authors, 1965 cited in Stewart and Rabin, 1970). *A. v.* (var.) *homari* survives well in seawater and can be recovered from marine sediments (Goggins and Hurst, 1960 cited in Stewart and Rabin, 1970; Kellog and co-authors, 1974) and surfaces of lobster tanks (Wood, 1965). Schapiro and co-authors (1974) recovered *A. v.* (var.) *homari* from the surface of *H. americanus* shipped into California (USA), but the bacterium is apparently not normally a part of the epiflora of lobsters (Stewart, 1980).

In trials, injection of virulent *Aerococcus v.* (var.) *homari* into lobsters held at 15 °C was nearly always fatal to experimentals within 14 days. Other decapods have also been infected by inoculation, but suffered only mild or no disease; where death occurred it was after a prolonged incubation period. *Panulirus interruptus, Pandalus platyceros,* and *Cancer irroratus, C. borealis, C. magister, Libinia emarginata, Geryon quiquedens, Chionoectes opilio* and *Callinectes sapidus* have all been infected in the laboratory

(Cornick and Stewart, 1968b; Rabin and Hughes, 1968; Tubiash and Krantz, 1970; Schapiro and co-authors, 1974; Cornick and Stewart, 1975).

Gaffkemia is recognized as an epidemic disease of wild-caught, impounded lobsters throughout the natural range of *Homarus americanus* and *H. gammarus*. In years past, the disease has caused substantial economic loss to the lobster trade in the United States, Canada, Holland, France, Ireland and England (Snieszko and Taylor, 1947; Stewart and MacDonald, 1962; Wood, 1965; Adouin and Leglise, 1971 cited in Johnson, 1983). However, incidence of gaffkemia in cultured lobsters in California (USA) has been low, perhaps a result of optimal husbandry practices in these operations (Fisher and co-authors, 1978). The impact of *Aerococcus v.* (var.) *homari* on wild lobster populations is undefined (Stewart, 1980).

Infection in lobsters occurs through breaks in the cuticle. *Aerococcus v.* (var.) *homari* is not transmitted by ingestion as the acidity of gastric fluid destroys the bacterium (Wilder and McLeese, 1961 cited in Stewart and Rabin, 1970; Wood, 1965; Rabin and Hughes, 1968; Stewart and co-authors, 1969c). Thus, the practice of 'pegging' the large claw provides a portal of entry for the bacterium (Stewart and Rabin, 1970). Introduction of as few as 5 *A. v.* (var.) *homari* into the haemocoel of the host results in fatal disease in the lobster, and the course of the disease is unrelated to the initial inoculation dose (Cornick and Stewart, 1968a).

Temperature plays a key role in the time-course of gaffkemia in lobsters. Stewart and co-authors (1969b) found the mean time-to-death of *Homarus americanus* infected by *Aerococcus v.* (var.) *homari* to be 2 days at 20 °C, 12 days at 15 °C, 28 days at 10 °C, 84 days at 5 °C, and 172 days at 3 °C; no deaths were attributable to *A. v.* (var.) *homari* infection over 250 days at 1 °C. At the lowest temperature tested, the pathogen was not eliminated from the experimentals but remained at low levels with unchanged virulence. Therefore, besides the presence of the bacterium the other important determinants for gaffkemia epidemics in lobsters are wounds or breaks in the cuticle and water temperature suitable for disease development.

Lobsters dying from gaffkemia are extremely weak and die in a 'spread-eagle' position (Sindermann and Rosenfield, 1967) or in lateral recumbency (Fisher and co-authors, 1978). Advanced gaffkemic lobsters may have pink discoloration of the ventral abdomen and their hemolymph is thin and pinkish (Snieszko and Taylor, 1947). Small, black specks due to hemocyte aggregations may be noticeable in the gills and other tissue locations in late infections, and macroscopic white-to-discolored, irregular lesions may be visible on dissection in the antennal gland (Johnson and co-authors, 1981).

Lobsters late in the course of gaffkemia have a drastic reduction in number of circulating hemocytes and, as a result, the hemolymph fails to clot (Fig. 3-17) (Snieszko and Taylor, 1947; Stewart and co-authors, 1969a). The hemolymph is heavily infected ($1 \times 10^{8-10}$ bacteria ml^{-1}), and oil-immersion microscopic examination of hemolymph wet-mounts reveals abundant tetrad-forming cocci (Fisher and co-authors, 1978). Concomitant with proliferation of *Aerococcus v.* (var.) *homari* in the hemolymph is a marked decline in hemolymph glucose (Fig. 3-18), a less dramatic reduction of hemolymph non-protein nitrogen and lactic acid (Stewart and co-authors, 1969a; Stewart and Cornick, 1972: Stewart and Arie, 1973a) and significant reduction in glycogen and adenosine triphosphate (ATP) in the hepatopancreas (Figs 3-19, 3-20), heart and abdominal muscle (Stewart and Arie, 1973b). Hemolymph protein level does not decline significantly (Stewart and co-

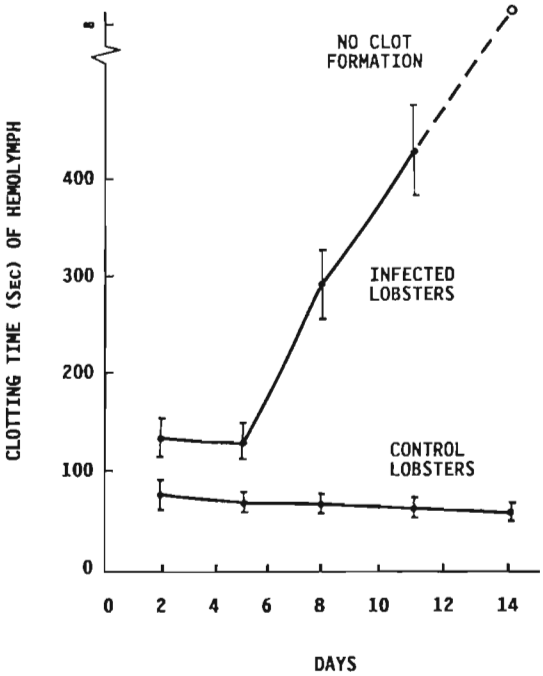

Fig. 3-17: Lobster gaffkemia. Comparison of hemolymph clotting times for gaffkemic versus control *Homarus americanus*. (After Stewart and co-authors, 1969a; redrawn. Reprinted with the permission of the National Research Council of Canada.)

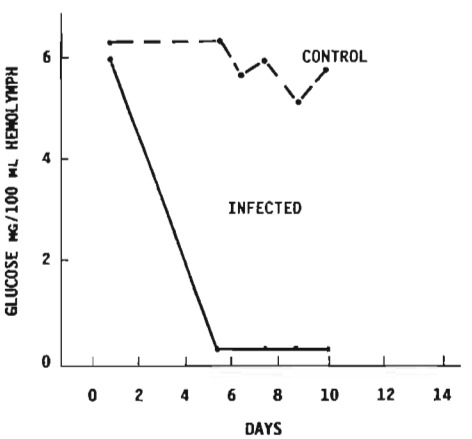

Fig. 3-18: Lobster gaffkemia. Plot of glucose levels in the hemolymph of gaffkemic versus control lobsters. (After Stewart and Arie, 1973a; redrawn. Reprinted with the permission from *Comp. Biochem. Physiol.*, 45 A. © Pergamon Press.)

authors, 1969a; Stewart and Rabin, 1970) nor is the hemocyanin level much altered (Rittenburg and co-authors, 1979). However, a 50% reduction in the oxygen carrying capacity of the hemocyanin in gaffkemic lobsters has been reported (Rittenburg and co-authors, 1979).

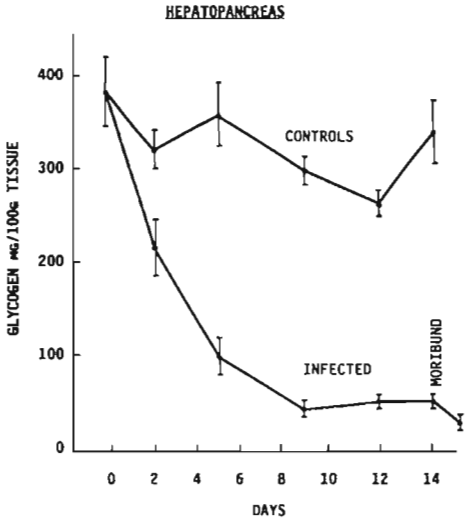

Fig. 3-19: Lobster gaffkemia. Levels of glycogen in the hepatopancreas of gaffkemic versus control *Homarus americanus*. (After Stewart and Arie, 1973b; redrawn. Reprinted with the permission of the National Research Council of Canada.)

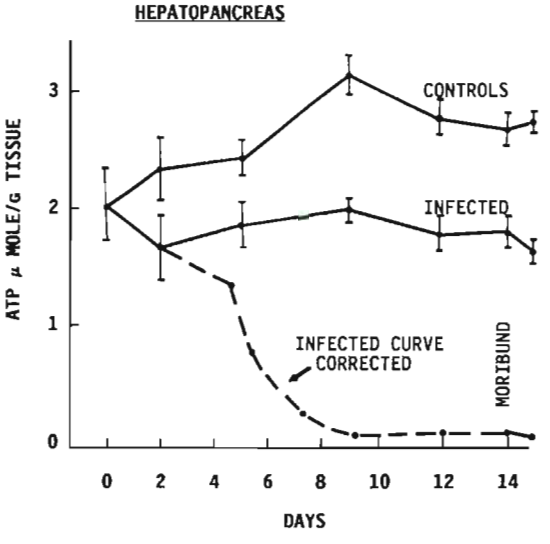

Fig. 3-20: Lobster gaffkemia. Measured levels of ATP in the hepatopancreas tissues of gaffkemic versus control lobsters. Solid lines: actual measurements; dashed line: values corrected for bacterial ATP. (After Stewart and Arie, 1973b; redrawn. Reprinted with the permission of the National Research Council of Canada.)

The progression of histopathologic changes in *Homarus americanus* experimentally infected by *Aerococcus v.* (var.) *homari* was studied by Johnson and co-authors (1981). The main features noted were hemocyte aggregations that increased in number and size over time within hemal sinuses in all tissues; early phagocytosis of the bacteria by the

system of fixed phagocytes; premature release of differentiating hemocytes into the circulatory system; and damage to the antennal gland, an apparent consequence of hemal stasis from occlusion of hemal sinuses by hemocyte aggregations. Other tissues and organ systems were unremarkable microscopically, except for glycogen depletion. According to Johnson and co-authors the microscopic pathologic changes found were indicative of ". . . a nontoxic, non-invasive bacteremia" (p. 127).

The cause of death of gaffkemic lobsters has not been resolved entirely. The apparent absence of exoenzymes produced by *Aerococcus v.* (var.) *homari,* the constant course of experimental infection independent of inoculation dosage but dependent on environmental temperature, the lack of mortality in lobsters injected with culture filtrate (Rabin and Hughes, 1968), the distribution of bacteria limited to hemal sinuses within hemocyte aggregations or fixed phagocytes, and the absence of specific cytopathology suggestive of a toxic effect on lobster tissue (Johnson and co-authors, 1981) implicate death is not due to production of bacterial exotoxin or organ tissue destruction by bacterial invasion, but by another mechanism. Goggins and Hurst (1960 cited in Stewart and Rabin, 1970) noted that wounded gaffkemic lobsters may bleed to death due to deficiency of hemocytes and greatly prolonged clotting time. Stewart and Arie (1973b) and Stewart (1975) suggest that the massive drain on carbohydrate reserves by the bacteria prevents sufficient ATP production for the lobster to carry-out vital metabolic functions so that death soon follows. Additionally, Rittenburg and co-authors (1979) contend that since the oxygen binding capacity of hemocyanin is reduced by 50% in gaffkemic lobsters, hypoxia arising from "bacterial impairment of the lobster's respiratory exchange system" (p. 172) may be the cause of death.

The prime metabolic function of molecular oxygen is to serve as an electron acceptor for ATP production (oxidative phosphorylation). Without adequate ATP, cell and vital organ functions cannot be sustained, and it appears that gaffkemic lobsters die because of an energy (ATP) deficit; the apparent cause being lack of O_2 and nutrient depletion. Also, as clotting ability of the hemolymph is lost, individuals that suffer wounding undoubtedly bleed to death.

There appears to be a lack of internal host defenses against virulent strains of *Aerococcus v.* (var.) *homari* by *Homarus* spp. (Stewart and Rabin, 1970). Although *H. americanus* has non-specific agglutinins (Cornick and Stewart, 1968b), bactericidins (Stewart and Zwicker, 1972) and phagocytosis (Johnson and co-authors, 1981) that provide very effective protection from most microorganisms, lethal strains of *A. v.* (var.) *homari* overcome these defenses.

Attempts at vaccination have met with limited success in *Homarus americanus.* Killed vaccines have been ineffective in improving survival of vaccinates, but did increase the time-to-death upon challenge (Stewart and Zwicker, 1974a). Use of attenuated live *Aerococcus v.* (var.) *homari* was more promising (Stewart and Zwicker, 1974b). Modified-live preparations provide good protection from experimental challenge in *Panulirus interruptus* (Schapiro and Steenbergen, 1974). Fisher and co-authors (1978) conclude that vaccination could have a use for protection of valuable broodstock, but techniques for application to other life stages remain undeveloped.

Aerococcus v. (var.) *homari* shows little resistance to commonly available antibiotics such as tetracycline, penicillin, erythromycin, novobiocin, vancomycin (Aaronson, 1956; Stewart and Cornick, 1967). However, the bacterium is resistant to sulfa compounds

(Stewart and Cornick, 1967). Interestingly, treatment with 25 mg kg^{-1} vancomycin prior to infection provides a high degree of protection from otherwise lethal challenge when administered up to 50 days post-vancomycin treatment (Stewart and Arie, 1974). Vancomycin treatment at 1 mg kg^{-1} administered prior to infection gives complete protection to lethal challenge if given within the 15 days of treatment (Stewart and Arie, 1974). Fisher and co-authors (1978) stated that holding lobsters at low temperature (5 to 10°C) and injection of 20,000 units of penicillin G completely eliminates the bacteria from lobsters. Oxytetracycline has recently been given approval by the United States Food and Drug Agency for use in lobsters.

Management of gaffkemia disease of lobsters is primarily a matter of good husbandry. The devastating outbreaks of the past should be largely preventable using the information now available on the disease. Wounding and crowding of lobsters puts the animals at high risk of infection and should be avoided. Sanitation of holding facilities, prompt removal of moribund or dying lobsters, maintenance of low water temperatures and routine disinfection of water used in recirculation systems are all obvious steps (Snieszko and Taylor, 1947; Fisher and co-authors, 1978). Rearing in individual containers, water-quality control and antibiotic treatment of selected gaffkemic broodstock incoming to lobster culture operations are suggested (Fisher and co-authors, 1978). These authors report that incidence of gaffkemia has been incidental in lobster mariculture in California (USA).

A slowly developing disease in *Carcinus mediterraneus,* caused by the Gram-positive encapsulated diplococci *Streptococcus faecalis liquefaciens,* has been reported by Pappalardo and Boemare (1982). Designated mediterranean crab streptococcosis (Sparks, 1985), the disease is known only from Pappalardo and Boemare (1982). Infected *C. mediterraneus* were collected from apparently polluted and low-saline waters of Languedoc on the French Mediterranean coast. The description of the disease is based principally on experimentally infected wild-caught crabs.

The agent is Gram-positive, facultative anaerobic, oxidase and catalase negative, resistant to crystal violet, 40% bile, sodium chloride, sensitive to tetracyclines and sulfa compounds and liquefies gelatin. The capsule is lost, colony appearance changes (from small, transparent to larger and mucoid) and catalase reaction turns positive after several passages on artificial media (Pappalardo and Boemare, 1982).

The disease is transferred by inoculation and feeding of hepatopancreas tissues from infected crabs. Time to death following inoculation exposure depended on dosage, and for ca 600 bacteria per 25 g crab was delayed to 40 days. Injection of culture ultra-filtrate failed to result in signs of the disease (Pappalardo and Boemare, 1982).

Infected crabs are inappetent, weak, but lack gross lesions. Gram-positive cocci, usually in pairs, are observed in smears of the hepatopancreas. Histologically, the bacterium is found in the cytoplasm of cells within the interstitial loose connective tissues of the hepatopancreas. The bacterium does not attack the hepatopancreatic epithelium, but infects connective tissue cells of digestive tract, gills and gonads. Heavy infection of the connective tissue and generalized bacteremia are noted in the terminal stages of the disease (Pappalardo and Boemare, 1982).

The impact and distribution of the infection in wild crab populations is unknown. Pappalardo and Boemare (1982) indicate that sewage pollution of the immediate environment and osmotic stress may have enabled the development of a variant of *Streptococcus faecalis* pathogenic to *Carcinus mediterraneus.* The uncommon *S. faecalis* is a fish patho-

gen, particularly of salmonids. Outbreaks are thought to occur because of contamination either during fish handling or in the water supply (Richards and Roberts, 1978).

An unidentified Gram-positive coccus was infecting hepatopancreas epithelium of *Penaeus monodon* with red disease, submitted from a shrimp farm in the Philippines (Lightner and Redman, 1985b). The significance and role of the bacterium as an etiologic agent of red disease are unknown. Red disease is thought to have an abiotic etiology, possibly toxic, although Gram-negative bacterial infection of necrotic hepatopancreatic tubules was also noted in the examined shrimp (Lightner and Redman, 1985b). The Gram-positive coccus observed in *P. monodon* differs from the *Streptococcus faecalis* of *C. mediterraneus* in size: 1.0 to 1.5 μm versus 0.5 to 1.0 μm for *S. faecalis;* and in cellular location: hepatopancreas epithelium versus connective tissue cells.

Pathogenic acid-fast bacteria have rarely been noted to infect crustaceans. Shrimp along with other marine animals can harbor *Mycobacterium marinum* (Overstreet, 1978). Infections by acid fast bacteria of 1 adult and 2 juvenile *Penaeus vannamei,* 2 adult *Macrobrachium rosenbergii,* and 1 *Austropotamobius pallipes* were reported by Alderman and co-authors (1986), Brock and co-authors (1986b), Lightner and Redman (1986) and Krol and co-authors (1989). Lightner and Redman documented a probable *Mycobacterium* sp. tissue infection of an adult, wild-caught female *P. vannamei* collected by a shrimp trawler off the Pacific coast of Panama. The shrimp was shipped to a commercial shrimp hatchery in Florida, killed within a few days, and its tissues preserved for histological screening of the hepatopancreas for *Baculovirus penaei* infection. The shrimp's behavior and appearance were normal prior to sacrifice. Only the hepatopancreas and surrounding tissues were collected and preserved for examination. Multiple, melanized hemocyte nodules were observed in the hepatopancreas tubules, intertubular connective tissues, haemocoel, hepatopancreas capsule, adjacent lymphoid organ and ovary. The nodules and adjoining tissues contained numerous, rod-shaped (1×0.5 μm), Gram-positive, acid-fast bacteria (Lightner and Redman, 1986). Lightner (1985) suggests that acid-fast bacterial infection may be more common in marine shrimp, and Krol and co-authors (1989) reported acid-fast bacterial infection of 2 cultured juvenile *P. vannamei.* The latter authors describe in detail the histopathology and ultrastructure of the hemocytic response to the acid-fast bacterial infection. Both affected shrimp exhibited abnormally dark pigmentation in areas that microscopically had multifocal hemocytic nodules composed of a centrally melanized core of intact bacteria and necrotic cellular debris, encompassed by a capsule of multiple layers of small and large granule hemocytes. One individual was moribund, the other behaviorally normal, when removed from the culture tanks. Krol and co-authors (1989) concluded that while the host appeared to contain the acid-fast bacteria, cellular defenses failed to kill and degrade the organism.

Agents: Fungi

Fungi and yeasts play an important ecological role as saprophytes in the marine environment. A few species are recognized as pathogens of fish, molluscs and crustaceans. The majority of fungal pathogens of marine Crustacea are Phycomycetes, but an Ascomycete species *(Trichomaris invadens)* and the Deuteromycete genus *Fusarium* are also represented. Several of the fungal pathogens cause significant diseases of cultured marine crustaceans. Also, in natural environments, epidemic losses have been attributed to

mycotic agents and one fungal pathogen is associated with a chronic disease syndrome of its host.

Previous articles that review fungal diseases of crustaceans include Unestam (1973), Alderman (1976, 1982), Lightner (1981), Johnson (1983) and Sparks (1985). The majority of these reviews address the broader topic of fungal diseases of aquatic animals or aquatic invertebrates. However, the diseases of marine Crustacea are generally considered thoroughly, and the reader is referred to these earlier articles, or the citations in the present work, for additional information. Johnson and Sparrow (1961) consider the fungal groups and species described from marine environments and, where appropriate, relations of the pathogens with marine crustaceans.

Yeasts of the genus *Metschnikowia* affect freshwater cladocerans, *Artemia* sp., and several marine copepods. *Metschnikowia* infections of brine shrimp are recorded from populations in hypersaline land-locked ponds (Spencer and co-authors, 1964; Codreanu and Codreanu-Balcescu, 1981) and, on this basis, are not discussed in this review. For further information on yeast infections of freshwater crustaceans and *Artemia* sp. see Johnson (1983) and Sparks (1985).

Metschnikowia sp. infection of the marine copepod *Calamus plumchrus* and of other copepods during winter and spring in the Strait of Georgia, Nanaimo (Canada) have been reported by Seki and Fulton (1969). They found yeasts either attached to the integument surface, within the digestive tract, or invading tissues. These yeasts had vegetative cells and asci that held a needle-shaped ascospore. The dominant yeast species was *M. krissii* (Seki and Fulton, 1969). Advanced yeast infection of copepods was associated with weakness and secondary invasion by bacteria and protozoans. Seki and Fulton studied the physiological characteristic of *M. krissii*. Bottom sediments from the Strait of Georgia contained many exoskeletons of *C. plumchrus* that Seki and Fulton suggested resulted from yeast-induced copepod mortalities.

Few Myxomycota (Thraustochytriales) are represented as pathogens of marine Crustacea. Artemchuk and Zelezinskaya (1969 cited in Unestam, 1973) reported epidemics in 1963 and 1964 of the planktonic cladoceran *Penilia avirostris* in the Black Sea that were caused by *Hyphochytrium peniliae*. Infected *P. avirostris* were filled with fungal hyphae and the cladoceran populations were reduced by 98%.

Mottling disease (Herrick, 1891, 1895, 1909 cited in Unestam, 1973 and Alderman, 1976, 1982) of *Homarus americanus* is thought to be caused by a chytrid-like fungus (Sindermann and Rosenfield, 1967; Sindermann, 1970). The disease condition, occasionally found in lobsters caught off the coast of Maine and originally thought to be a color variation, was called 'leopard lobster'. Mottling disease is characterized by large, yellowish, irregular patches on the normally green cuticle of the lobsters. The discoloration is due to a slowly developing necrosis of the underlying tissues; if the infection is advanced, the exoskeleton may be blistered (Sindermann and Rosenfield, 1967). Histologically, PAS-positive, thick-walled 30 to 60 μm diameter spherical bodies were seen in the affected tissues (Sindermann, 1970). The impact of the infection on wild-lobsters is unknown. The disease has not been reported from cultured lobster stocks.

A gill infection of the shrimp *Dichelopandalus leptocerus* has been described by Uzmann and Haynes (1968), who believe it to be due to infection by a chytrid-like fungus. However, the identity of the agent is questioned. Johnson (1983) cited Rinaldo and Yevich (1974) as indicating the agent associated with black gill syndrome of an unspecified species

of northern shrimp may be a dinoflagellate. Rinaldo and Yevich (1974) report on a similar black gill syndrome in *Pandalus borealis,* but suggest infecting agents were not found and the etiology of this disease was not determined. The disease was recognized in *Dichelopandalus leptocerus* populations collected during 3 sampling periods: June 1963, January 1964 and October 1966, from sites ranging from southern Nova Scotia (Canada) to the continental slope off southeastern Long Island, New York (USA). Lesion prevalence in 1,341 shrimp examined in the 3 collections varied from 52 to 96% (Uzmann and Haynes, 1968).

Development of the cytrid-like fungus is limited to the gill lamellae. The disease results in a blackened, mottled appearance of gill filaments. In advanced cases the whole gill tissue is darkly colored. Histologically, infection sites are rimmed by melanized, necrotic tissue (Uzmann and Haynes, 1968). The authors suggest the infection may be an important cause of natural mortality of *Dichelopandalus leptocerus,* and possibly other crustacean hosts as well. However, corroborating data have not been forthcoming and the impact of the disease on wild populations of the northern shrimp remains unclear.

The Eumycota, Mastigomycotina and Oomycetes (Phycomycetes) are represented as parasites of marine Crustacea by 3 orders: Lagenidiales, Saprolegniales and Peronosporales. These mycelial fungi have non-septate or sparingly septate hyphae and motile zoospores. While several species are frequent and important pathogens of crustaceans, only agents which cause diseases in marine Crustacea are considered here. The taxonomic placement and ordinal relationships of some of the Oomycete fungi are unresolved (Dick, 1973; Sparrow, 1976). The taxonomic assignments used in the present review are thus provisional and generally follow those provided by Alderman (1982).

The genus *Lagenidium* is the only member of the Lagenidiaceae (Lagenidiales) with species reported as pathogens of marine crustaceans. These species are *L. callinectes, L. chthamalophilium* and *L. scyllae.* Members of *Lagenidium* have endobiotic, holocarpic thalli and biflagellate zoospores. In terms of host, geographic distribution and economic significance the most important phycomycete pathogen of marine Crustacea is *Lagenidium callinectes.* The fungus, initially recognized in 1941 by C.L. Newcombe, was observed infecting ova of *Callinectes sapidus.* Specimens were forwarded to J.N. Couch who described and named the fungus *Lagenidium callinectes* Couch (Couch, 1942). Since then, *L. callinectes* has been recorded to infect eggs and/or larvae of the majority of cultured marine decapods (crabs, lobsters, shrimp) in the Americas. A similar fungus, named *L. scyllae,* has been reported from cultured eggs and larvae of wild-caught mud crabs *Scylla serrata* in the Philippines (Bian and co-authors, 1979). It is not altogether clear that *L. scyllae* is a species distinct from *L. callinectes,* and future work that critically compares isolates of both these fungi is recommended. In terms of infection and disease, these fungi are essentially the same, and where appropriate in this review, *L. scyllae* is included with the discussion on *L. callinectes.*

Lagenidium callinectes is an extensively branched, sparing septate, mycelial fungus with intramatrical, 5 to 14 μm diameter hyphae filled with globules. Extramatrical hyphae are similar sized, non-septate and generally unbranched. Bland and Amerson (1973a) have shown that conversion of the thallus from vegetative mycelium to zoosporangia is induced in pure culture by nutrient depletion. Zoosporangia form as 14 to 25 μm expansion of tips of extramatrical hyphae. Planonts (zoospores) are pyriform, 3 to 9 × 5 to 12 μm, laterally biflagellate (Couch, 1942; Fuller and co-authors, 1964; Bland and Amerson, 1973a, b;

Lightner and Fontaine, 1973; Gotelli, 1974a,b; Armstrong and co-authors, 1976; Fisher and co-authors, 1978). Zoospores are rapidly formed and released. Planonts appear within 8 h after transfer from liquid media to sterile seawater (Fuller and co-authors, 1964). The motile zoospores are positively attracted to crab eggs (Bland and Amerson, 1973a).

Lagenidium scyllae was identified by Bian and co-authors (1979) from infected ova and larvae of *Scylla serrata* in culture facilities in the Philippines. The fungus which Bian and co-authors (1979) describe has holocarpic, sparingly septate, branched hyphae 7.5 to 17 μm in diameter. The discharge tubes vary in length from 37 to 500 μm, and the terminal vesicles from 25 to 72.5 μm in diameter. The zoospores are monoplanetic, laterally biflagellate and irregular in shape and size (average 12.5 to 10 μm). Baticados and co-authors (1977) and Lio-Po and co-authors (1982) report a *Lagenidium* sp. from eggs and/or larvae of *P. monodon* and *Scylla serrata* in the Philippines, and the relationship of these isolates to reported species of *L. scyllae* and *L. callinectes* has not been established.

Lagenidium callinectes grows readily on a variety of standard mycological media (Fuller and co-authors, 1964; Bland and Amerson, 1973a; Lightner and Fontaine, 1973; Nilson and co-authors, 1976; Baticados and co-authors, 1977; Lightner, 1977 his Chapter 3.1.5, 1988 his Chapter 3.1.11; Bahnweg and Gotelli, 1980; Lio-Po and co-authors, 1982). Lightner and Fontaine (1973) obtained primary isolation of *L. callinectes* on thioglycolate media with penicillin (500 units ml^{-1}) and streptomycin (500 μg ml^{-1}) incubated at room temperature (27 to 30 °C) and maintained the fungus on Sabouraud Dextrose agar (Difco) supplemented with 2 % NaCl and 5 % shrimp homogenate. Nilson and co-authors (1976) used seawater corn meal extract agar (CMA) with streptomycin sulfate and penicillin G added at 50 μg ml^{-1} for primary isolation of *L. callinectes.* Bian and co-authors (1979) reported *L. scyllae* is easily cultured on PGY-seawater medium.

Lagenidium callinectes grows in salinities from 5 to 30 ppt (Rogers-Talbert, 1948). The isolate studied by Bahnweg and Gotelli (1980) was obligately marine, and sodium was essential for growth. On the other hand, Armstrong and co-authors (1976) noted that fungal growth and sporulation occurred at 0, 16 and 32 ppt, but the time needed differed considerably depending on salinity. Hyphal growth and appearance of sporangia took 32, 50 and 60 h at 16, 32 and 0 ppt, respectively. *L. scyllae* grows on PGY medium without supplemental NaCl (Bian and co-authors, 1979). Bahnweg and Gotelli (1980) demonstrated good growth between 20 and 27 °C for *L. callinectes* previously adapted to 25 °C. Rogers-Talbert (1948) noted that low temperatures (15 °C) retarded development of the fungus. Interestingly, the number of nitrogen and carbon substrates *L. callinectes* can use was rather limited for a saprophytic oomycete (Bahnweg and Gotelli, 1980). For detailed discussion of the physiological and nutritional requirements of *L. callinectes* readers are referred to Rogers-Talbert (1948), Gotelli (1969), Bland and Amerson (1973a) and Bahnweg and Gotelli (1980).

Lagenidium callinectes or *Lagenidium* sp. have been reported from eggs and/or larval stages of *Callinectes sapidus* (Sandoz and co-authors, 1944; Rogers-Talbert, 1948); *Panopeus herbstii* and *Libinia dubia* (Bland and Amerson, 1974); *Cancer magister* (Armstrong and co-authors, 1976); *Scylla serrata* (Lio-Po and co-authors, 1982); cultured *Penaeus* spp. (Cook, 1971; Lightner and Fontaine, 1973; Bland and co-authors, 1976; Aquacop, 1977; Baticados and co-authors, 1977; Nurdjana and co-authors, 1977; Tareen, 1982; Lightner, 1983, 1985, 1988 his Chapter 3.1.11; Baticados, 1988); *Homarus americanus* (Nilson and co-authors, 1976; Nilson and Fisher, 1977; Fisher and co-authors,

1978; Fisher, 1988d); *Pandalus platyceros* and *Palaemon macrodactylus* (Fisher, 1983b), and the barnacles *Chthamalus fragilis* and *Chelonibia patula* (Johnson, 1958; Johnson and Bonner, 1960). Fuller and co-authors (1964) isolated *L. callinectes* from algal surfaces. It is clear that *L. callinectes* has a wide geographic and host range.

Lagenidium callinectes is readily transmitted experimentally. Rogers-Talbert (1948) observed the fungus spread very rapidly between *Callinectes sapidus;* infections were obtained in eggs of the oyster crab *Pinnotheres ostreum* and the mud crab *Neopanope texiana.* Johnson and Bonner (1960) successfully cross-inoculated *L. callinectes* from *Chelonibia patula* lamellae to ova of *C. sapidus.* Preliminary experiments conducted by Bland and Amerson (1974) indicate that strains of *L. callinectes* collected from ova of *C. sapidus, Panopaeus herbstii* and *Libinia dubia* were equally infective for ova from all 3 crabs. According to Lightner (1981), *L. callinectes* was infectious for *Artemia* sp. Fisher (1983b) routinely infected, with a range of *L. callinectes* isolates, detached eggs of *Palaemon macrodactylus* or eggs on females that had excised first pereiopods (cleaning appendages).

Natural infections by *Lagenidium callinectes* occur in ova of marine Crustacea (Sandoz and co-authors, 1944; Rogers-Talbert, 1948; Johnson and Bonner, 1960; Bland and Amerson, 1974). Infection of larval stages is only known from cultured populations (Rogers-Talbert, 1948; Cook, 1971; Lightner and Fontaine, 1973; Armstrong and co-authors, 1976; Bland and co-authors, 1976; Nilson and co-authors, 1976; Aquacop, 1977; Baticados and co-authors, 1977; Nilson and Fisher, 1977; Fisher and co-authors, 1978; Tareen, 1982; Lightner, 1983, 1985, 1988 his Chapter 3.1.11; Baticados, 1988; Fisher, 1988d). In wild and cultured populations, *L. callinectes* is not known to cause disease in juvenile(beyond post larvae)-to-adult marine Crustacea.

Infection of individual crustacean eggs occurs rapidly, and invariably results in death of the ova (Sandoz and co-authors, 1944; Rogers-Talbert, 1948; Johnson and Bonner, 1960). Although, Sandoz and co-authors (1944) noted that the fungus appears evenly distributed throughout the egg masses, later work by Rogers-Talbert (1948), on blue crabs from Chesapeake Bay (USA), clearly indicate that only eggs on the periphery of the sponge were attacked by *Lagenidium callinectes.* Further, Rogers-Talbert (1948) observed that, even in heavy infections, 75 % of the eggs on a sponge were not infected and hatched normally. Very heavy infections were found in slightly less than 25 % of the crab sponges studied from the natural environment. Rogers-Talbert (1948) concluded that infection by *L. callinectes* is not a significant mortality factor affecting blue crab populations. Bland and Amerson (1974) studied natural infection of blue crab eggs in certain North Carolina (USA) waters; their observations differed significantly from those of Rogers-Talbert. Severe fungal infection by *L. callinectes* was found in 6 of 2,000 ovigerous blue crabs collected from June 1 through August 1, 1971. Concurrent infection by *Thraustochytrium* sp. and the filamentous bacterium *Leucothrix mucor* was also noted. In a similar time period in 1972, 95 % of 174 crab sponges were found infected in early June samples. Both orange and mature brown sponges were infected but involvement was more severe in the older sponges. Often, 30 to 50 % of the eggs were destroyed in a given sponge. By August 1972, 676 sponges were evaluated and 30 % found infected. Infections declined as the summer progressed, and no infected crab sponges were found after late July. The reason for the decline in infection was not determined. These results suggest *L. callinectes* can cause significant egg losses in natural infections. More recent data on this point were not

found in the references considered in this review. Of interest is that *L. callinectes* was not discovered infecting eggs from wild populations of *Cancer magister* collected in waters off California (USA) where egg mortalities in these crabs were studied (Fisher and Wickham, 1976). These data suggest that the eggs of the dungeness crab are resistant to attack under natural conditions, or that *L. callinectes* is not normally present in this area. Armstrong and co-authors (1976) reported infection in cultured dungeness crab larvae and postulated that the fungus inoculation entered with untreated bay water. However, with the information available, the impact of *L. callinectes* on recruitment of blue crab and other marine crustaceans remains unclear.

In culture, *Lagenidium callinectes* is clearly a formidable pathogen to ova and larval stages of many marine crustaceans. Infection is rapid and brought about by the zoospores which settle on eggs or larvae and then encyst (Rogers-Talbert, 1948). The spore germinates by penetration, with a thin tube, of egg shell and membrane or larval cuticle. Growth of the fungus is very rapid once entry has been made, and infected eggs are totally replaced by intramatrical hyphae within 48 h of infection. Multiple spores can infect a single egg or larvae, and the mycelium of 1 spore may produce several sporangia. Extramatrical hyphae and sporangia rapidly form once the nutrients are depleted from the egg or larvae. Sporangia release multiple zoospores within 20 to 50 min (Fuller and co-authors, 1964; Bland and Amerson, 1973a; Lightner and Fontaine, 1973). Thus, infections proceed very rapidly.

Heavily infected crab eggs (Rogers-Talbert, 1948), and crab and shrimp larvae (Armstrong and co-authors, 1976; Lightner and Fontaine, 1973, respectively) appear opaque white. Diseased crab eggs are reduced in size by ca 20% and contain abundant fungal mycelia (Rogers-Talbert, 1948). Shrimp, crab and lobster larvae dying from *Lagenidium callinectes* infection swim or move weakly, or lay on the bottom with feeble movements until death (Lightner and Fontaine, 1973; Armstrong and co-authors, 1976; Nilson and co-authors, 1976). Microscopically, in terminally infected larvae (Fig. 3-21, a), fungal hyphae almost completely fill the body. The response to invasion by *L. callinectes* appears to be only minimal inflammation (Lightner and Fontaine, 1973), perhaps because of the rapid course of the disease. Early infections of larval shrimp are localized, usually in an appendage. These infections rapidly progress to the terminal stage as the fungus grows virtually unchecked by host defenses (Lightner, 1977 his Chapter 3.1.5, 1988 his Chapter 3.1.11).

Epidemics characterized by extremely high population mortality rates result from *Lagenidium callinectes* infection of cultured crustacean larvae. The intensity of *Lagenidium* disease of early shrimp larval (nauplii-protozoea) stages can be extensive, often 100% (Cook, 1971; Lightner and Fontaine, 1973; Baticados and co-authors, 1977; Lightner, 1977 his Chapter 3.1.5, 1983, 1985, 1988 his Chapter 3.1.11). Armstrong and co-authors (1976) observed 40% loss of second-stage dungeness crab zoea. When eggs or first and second stages of lobster larvae are infected, 90% mortality can occur (Nilson and co-authors, 1976). Clearly, *Lagenidium* disease has the potential to cause economically significant epidemics in commercially cultured decapod larvae.

However, as larval shrimp, crabs or lobsters progressively grow and mature, susceptibility to infection by *Lagenidium callinectes* diminishes (Lightner and Fontaine, 1973; Armstrong and co-authors, 1976; Nilson and co-authors, 1976). And while infection of post larval lobsters has been reported (Nilson and co-authors, 1976), disease consequences

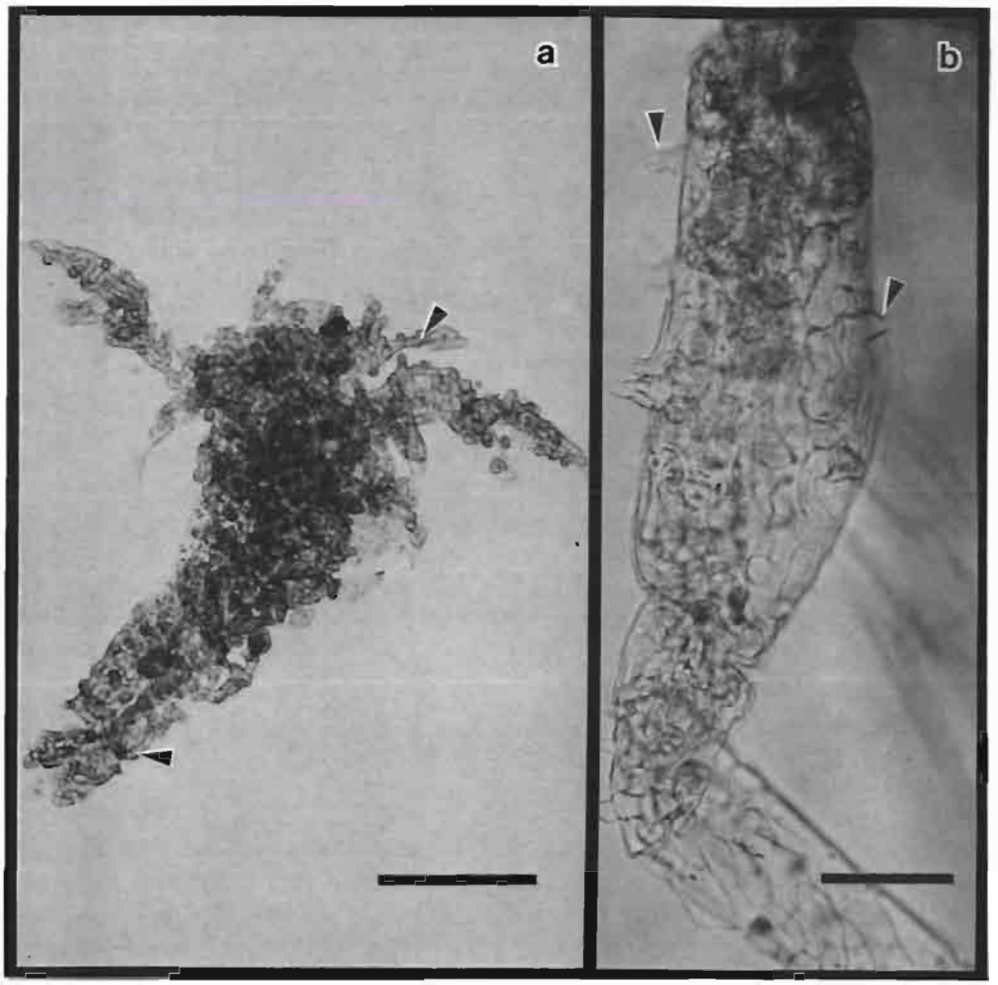

Fig. 3-21: Larval mycosis. (a) *Lagenidium* sp. infection of an *Artemia* sp. nauplius; fungal hyphae (arrow heads) fill the entire body cavity of the host; no stain; bar = 200 μm. (b) *Sirolpidium* sp. infection in a pereiopod of a post larval *Penaeus vannamei;* hyphae have replaced host tissues in the appendage; several discharge tubes are indicated by arrow heads; no stain; bar = 50 μm. (Originals.)

are greatly reduced. In penaeid shrimp culture some confusion may occur because of another fungal infection, due to *Sirolpidium* sp., that is a more common problem in mysis-through-early-post-larval stages. Beyond the post larvae, *L. callinectes* infection of juvenile-through-adult-stages of shrimp, crabs and lobsters has not been demonstrated. Thus, *L. callinectes* is principally a pathogen for decapod eggs and early larval stages. Older decapods and cultured fish are apparently refractory to disease caused by this fungal agent.

The suggested routes of entry into crustacean hatcheries for *Lagenidium callinectes* is via the water supply (Armstrong and co-authors, 1976; Fisher, 1988d) and with transferred animals (Lightner, 1977 his Chapter 3.1.5, 1988 his Chapter 3.1.11; Nilson and Fisher, 1977; Fisher, 1988d). However, besides demonstrating that *L. callinectes* infects lobster

eggs, information indicating that the fungus enters via incoming water or introduced crabs or shrimps has not been published. Thus, the reservoir for *L. callinectes* introduction into culture settings is poorly defined.

Largely because of the potentially tremendous negative economic consequences of larval mycosis in commercial decapod culture, a number of investigators have studied various treatments for prophylaxis and control of the disease (Armstrong and co-authors, 1976; Bland and co-authors, 1976; Nilson and co-authors, 1976; Aquacop, 1977; Fisher and co-authors, 1978; Lio-Po and co-authors, 1982; Lio-Po and Sanvictores, 1986). Lightner (1977 his Chapter 3.1.5, 1983, 1985, 1988 his Chapter 3.1.11), Nilson and Fisher (1977), Fisher (1988d), Armstrong and Fisher (1977, 1988) and Sindermann (1977b, 1988b) reviewed the chemical treatments for shrimp larvae; as well as lobster, dungeness crab and blue crab eggs and larvae. Once the fungus invades eggs or larvae, the infection probably nearly always results in death of the host. Thus, chemical control methods are aimed principally at destruction of the zoospores. Prophylactic treatment of shrimp larval stages with trifuralin (treflan), a herbicide, at 0.01 to 0.2 mg l^{-1} continuous bath has been widely used (Lightner, 1988 his Chapter 3.1.11) following positive *in vitro* results (Bland and co-authors, 1976; Lio-Po and co-authors, 1982). Malachite green was also found effective *in vitro* at dosages of 0.1 mg l^{-1} (Lio-Po and co-authors, 1982), but the compound is less widely used than treflan in shrimp hatcheries, possibly because of mixed results in terms of efficacy (Lightner, 1988 his Chapter 3.1.11). For treatment of larvae of dungeness crab and American lobster, Armstrong and Fisher (1977, 1988) and Fisher (1988d) list treflan at 0.005 mg l^{-1} or captan at 0.06 mg l^{-1} as effective. Nilson and Fisher (1977) and Fisher (1988d) suggest dipping lobster eggs in a 5 mg l^{-1} solution of malachite green in seawater for 10 min and larvae every other day at the same dosage for 2 min.

Also for prevention and control, strict sanitation procedures are important. Lio-Po and co-authors (1982) evaluated a variety of disinfectants and recommend application of benzalkonium chloride (50 mg l^{-1}), Tide detergent (50 mg l^{-1}), formalin (50 mg l^{-1}), malachite green (1 mg l^{-1}) or potassium permanganate (100 mg l^{-1}) mycocidal for *Lagenidium* spp. (24 h exposure). Interestingly, these investigators found the mycocidal dosage of calcium hypochlorite needed to be 500 mg l^{-1} (24 h exposure). Lightner (1988 his Chapter 3.1.11) commented that this finding could explain the variable results for eradication of *Lagedinium* sp. experienced by shrimp culturists for chlorine disinfection at levels of 50 to 200 mg l^{-1}.

Ultra-violet treatment of water used for rearing has also been suggested for control of *Lagenidium* (Armstrong and co-authors, 1976). Rapid, strong disinfection of rearing systems containing heavily infected groups of eggs/larvae is recommended to control the disease (Armstrong and Fisher, 1977; Nilson and Fisher, 1977; Fisher, 1988d).

Johnson (1958) discovered and described *Lagenidium chthamalophilum* parasitic in ova of the barnacle *Chthamalus fragilis denticulata* in Beaufort Inlet, North Carolina (USA). Inoculation experiments indicated that the fungus was specific for ova of *C. fragilis denticulata*. Its irregularly branched hyphae are 10 to 18 μm in diameter. Sporangia of *L. chthamalophilum* almost exclusively form on intramatrical hyphae. Each sporangium has a single emergent discharge tube which expands apically, forming a spherical vesicle. Zoospores are reniform, biflagellate and 9×7 μm in size. Reduction of populations of *C. fragilis denticulata* in certain areas were attributed to the fungal infection (Johnson, 1958).

Sirolpidium sp. (Sirolpidiaceae, Lagenidiales) has been reported as a serious patho-

gen of cultured larval and post larval penaeid shrimp and, apparently, larval lobsters (Lightner, 1977 his Chapter 3.1.5, 1981, 1983, 1985, 1988 his Chapter 3.1.11). *Sirolpidium* sp. infecting penaeid shrimp seemingly have a worldwide distribution (Lightner, 1983). However, information on isolation, *in vitro* culture and formal description of the fungus supporting assignment to the genus *Sirolpidium* has not been published. As a consequence, the present identification is debatable. Also, due to the broad host and geographic range for the disease (Lightner, 1981, 1983, 1985, 1988 his Chapter 3.1.11) it is unclear if one or more fungi species are involved. Adequate study and description is needed of the *Sirolpidium* sp. that causes larval mycosis in penaeid shrimp hatcheries. Moreover, it would be interesting to critically compare the *Sirolpidium* sp. with type material for *Haliphthoros milfordensis*.

The pathogenesis of *Sirolpidium* sp. infection in larval shrimp and lobsters is similar to that of *Lagenidium callinectes* (Lightner, 1981). The 2 fungi are distinguishable by zoospore formation and discharge, and by hyphae width. *Sirolpidium* sp. expels motile zoospores through a terminal discharge tube (Fig. 3-21, b); a terminal sporangium is lacking. The width of *Sirolpidium* sp. hyphae is variable, ranging from 7 to 40 μm. Also, *Sirolpidium* sp. tends to cause a clinical problem in late larval and post larval stages. The infection may result in high mortality. Treatment of *Sirolpidium* sp. infection of penaeid shrimp is similar to that discussed for *Lagenidium callinectes*.

With members reported as pathogens of marine crustaceans, the Saprolegniales are represented by 4 families: Saprolegniaceae, Leptolegniellaceae, Leptolegniaceae and Haliphthoraceae.

Fungi in the family Saprolegniaceae are apparently rare pathogens of marine Crustacea. Only one reference to such infection was found, and there is doubt regarding the identity of the fungus reported because documentation concerning the identifications are sparse. *Saprolegnia parasitica, Achlya racemosa* and *Achlya* sp. were recorded from 'burn spot' (shell disease) lesions of the tiger prawn *Penaeus monodon, Gammarus* sp. and tanaeidaceans *Apseudes* spp. (Gopalan and co-authors, 1980). The crustaceans were collected from backwaters in Cochin, India. Burn spot lesions were noted on 6 of 155 (4 %) juvenile *Penaeus monodon* evaluated in the study. The fungus species were identified from direct microscopic examination of infected tissues.

The genus *Aphanomyces* (Saprolegniales, Saprolegniaceae) contains a species, *A. astaci*, that is a very important pathogen of freshwater crayfish, and is the agent responsible for catastrophic mortalities of wild crayfish populations in Europe (Alderman, 1982). For comprehensive discussion of crayfish plague ('Krebspest'), the reader is referred to Johnson (1983) and Sparks (1985).

The Leptolegniellaceae (Saprolegniales) are represented by 2 species known as pathogens of marine Crustacea.

Leptolegnia marina was originally assigned by Atkins (1929, 1954b) to *Leptolegnia*. Johnson and Sparrow (1961) questioned this identification, and Dick (1971) placed the fungus into the newly erected family and genus Leptolegniellaceae and *Leptolegniella*. However, elsewhere (Unestam, 1973; Alderman, 1976; Sparks, 1985) the fungus is referred to as *L. marina*.

Atkins (1929, 1954b) described a fungal infection of the pea crab *Pinnotheres pisum*, itself a parasite of *Mytilus edulis*. The fungus, *Leptolegniella marina*, invades body, eggs and embryos of *P. pisum*. Fungal attack invariably caused death of the crab. Other

invertebrates were reported by Atkins (1954b) to be susceptible to infection by *L. marina*. Atkins encountered infection in a single *P. pinnotheres* and in 2 lamellibranchs, *Barnea candida* and *Cardium echinatum*. Johnson and Pinschmidt (1963 cited in Unestam, 1973) observed the fungus in blue crab ova. Geographically, *L. marina* has been recorded from waters on both sides of the Atlantic. Gopalan and co-authors (1980) reported *L. marina* from shell disease lesions of shrimp and amphipods in India. However, there is some question regarding the identity of the fungus.

The mycelium of *Leptolegniella marina* is almost exclusively intramatrical and highly branched; it averages 7.5 to 20 μm, occasionally up to 40 μm, in width (Atkins, 1929, 1954b; Alderman, 1976). Sporangia are formed in unchanged hyphae; they release biflagellate zoospores through short hyphal branches that penetrate the crab's cuticle ('discharge papilla'; Atkins, 1929). Zoospores are pyriform and measure 8 to 14 μm (Atkins, 1929; Alderman, 1976).

Infection of the pea-crab manifests itself by the presence of irregular, variable sized, opaque patches under the cuticle of the body or abdomen, or by diffuse white discoloration of gill lamellae. The white patches most often become evident in the posterior region of the gill covering (Atkins, 1929). The time to death in laboratory held crabs ranged from 8 to 57 days at 8 to 17 °C (Atkins, 1954b). Disease of *Pinnotheres pisum* caused by *Leptolegniella marina* is known only from crabs maintained in the laboratory. The outcome of crab infections in nature and the impact of *L. marina* on wild pea-crab and other invertebrate populations has not been studied.

Episodes of mass mortalities of the planktonic copepod *Eurytemora hirundoides* in the Baltic Sea have been reported by Vallin (1951). The epidemics were confined to *E. hirundoides* and occurred during July and August 1951 and 1952. The saprolegnid *Leptolegnia baltica* (Leptolegniaceae, Saprolegniales) caused the mortalities (Vallin, 1951; Höhnk and Vallin, 1953). Masses of dead copepods clogged fishing nets, and wide discolored areas in the water, apparently comprised of dead copepods, were noted. Decaying *E. hirundoides* were heavily infected with *L. baltica*. Branched, aseptate, 15 to 20 μm intramatrical hyphae with oogonia and antheridia filled the copepods. Extramatrical hyphae were rarely branched. Sporangia appeared similar to other hyphae; zoospores, aligned in a single row, were oval to elongate and 9 to 16 μm in size (Vallin, 1951; Höhnk and Vallin, 1953; Alderman, 1976). The disease did not affect other copepod species. Vallin (1951) indicates that the plankton mortality could potentially impact the herring fishery by temporary decreased yields, a consequence of reduced food supply. However, since the early 1950's subsequent disease outbreaks have not been recorded (Sparks, 1985). Today, the role of *L. baltica* as a marine pathogen is vague.

Haliphthoros and *Atkinsiella* (Haliphthoraceae, Saprolegniales), represented by several species (*Haliphthoros milfordensis, H. philippinensis, Atkinsiella dubia* and *A. hamanaensis*), have been reported as pathogens of marine Crustacea. Of these, *H. milfordensis* is the best known; it has been the subject of a number of studies since its discovery in 1958 (Vishniac, 1958). *H. philippinensis* was isolated from cultured larval *Penaeus monodon* in the Philippines during a disease epidemic (Hatai and co-authors, 1980). These authors described the isolate as a species distinct from *H. milfordensis* based on earlier spore formation, distinctive morphology of the sporangia, means of zoospore discharge, and the occurrence of polyplanetic and polymorphic zoospores. However, recognition of the Philippine isolate as a new species warrants critical evaluation, and,

indeed, is in doubt (Alderman, 1982). Tharp and Bland (1977) describe the zoospores of the strain of *H. milfordensis* in their study as polymorphic and conclude "there is great variation in zoospore shape among the various strains of *H. milfordensis* investigated to date" (p. 2942). The photomicrographs of gemmae presented by Hatai and co-authors (1980) appear similar to that depicted in Tharp and Bland (1977) for *H. milfordensis*. Tharp and Bland (1977) noted that morphology and development of *H. milfordensis* differ on various growth substrates. The taxonomic standing of *Haliphthoros* has also been disputed by Tharp and Bland (1977). Vishniac (1958) and Dick (1973) place *H. milfordensis* in the order Saprolegniales (Haliphthoraceae). Sparrow (1973b) assigned the fungus to the order Lagenidiales, and more recently (Sparrow, 1976) to the family Sirolpidiaceae. Comparative studies of *H. milfordensis*, *H. philippinensis* and *Sirolpidium* sp. recovered from outbreaks of larval mycosis of penaeid shrimp and other marine invertebrates could provide valuable insight into the taxonomic relations of these important pathogens.

Haliphthoros milfordensis has branched, non-septate, irregular hyphae 7 to 40 μm in diameter. Fragmentation of the thallus into subthalli occurs early in culture (Tharp and Bland, 1977). The fungus is holocarpic and gemmae form in the vegetative hyphae. Planonts are biflagellate and polymorphic averaging 6 × 8 μm (Tharp and Bland, 1977). The fungus is obligately marine (Vishniac, 1958) and can be readily isolated from clinical material using Cantino's PYG (peptone-yeast extract-glucose) agar (Difco) made with seawater (PYGS) of about 20 to 30 ppt (Tharp and Bland, 1977; Hatai and co-authors, 1980). Addition of antibiotics, such as penicillin and streptomycin sulfate, are indicated for primary isolation.

Haliphthoros milfordensis was initially recovered from ova of the oyster drill *Urosalpinx cinerea* (Vishniac, 1958). The fungus has also been isolated from adult *Penaeus setiferus* collected from the vicinity of Beaufort, North Carolina, USA (Tharp and Bland, 1977), juvenile *Homarus americanus* (Fisher and co-authors, 1975; Fisher and Nilson, 1977), and the alga *Enteromorpha* sp. (Fuller and co-authors, 1964). Experimental infections by *H. milfordensis* have been reported for eggs and larvae of the oyster drill (Ganaros, 1957), eggs of *Pinnotheres* sp. (Vishniac, 1958), *H. gammarus* (Fisher and co-authors, 1975), adult *Penaeus duorarum*, eggs of *Callinectes sapidus* and eggs, nauplii and adult stages of *Artemia* sp. (Tharp and Bland, 1977; Overton and Bland, 1981). *H. philippinensis* was isolated from cultured larval *Penaeus monodon* during a hatchery disease outbreak (Hatai and co-authors, 1980).

In experimental infection trials with crustacean hosts, *Haliphthoros milfordensis* caused epidemic disease. In *Artemia* nauplii, *H. milfordensis* zoospores encysted on the exoskeleton, and infection occurred with spore germination and penetration of the exoskeleton by vegetative hyphae (Overton and Bland, 1981). Experimental infections of blue crab eggs, eggs through adult stages of brine shrimp, and adult pink shrimp resulted in total host mortality (Tharp and Bland, 1977). In young cultured post larval lobsters the fungus caused 46% mortality within 22 days (Fisher and co-authors, 1975). The impact of *H. milfordensis* on natural marine crustacean populations is unknown.

Pathologically, *Haliphthoros* infections of eggs and planktonic larvae are characterized by extensive replacement of host tissues by the invading fungal mycelia. The fungus destroyed first the fat and striated muscle and eventually the gut in experimentally infected brine shrimp larvae. Host responses are often non-existent, but occasionally melanization is observed (Tharp and Bland, 1977). Once the host tissues are decimated, hyphae

penetrate the cuticle and sporulation is initiated. Identical to *Lagenidium*-caused larval mycosis, infection is rapidly transmitted by large numbers of motile zoospores discharged from infected hosts.

Haliphthoros milfordensis infection is generally localized in young lobsters, and mortality is restricted to small juveniles between 5 and 27 mm carapace length (Fisher and co-authors, 1975). Larger juveniles often develop exoskeleton lesions but shed these at the time of molting. Spontaneously infected lobsters (culture tanks) usually display a few large exoskeleton lesions while experimentally exposed small juveniles have numerous small, melanized areas on the appendage joints and ventral abdominal segments (Fisher and co-authors, 1975). However, melanization fails to develop around mycelia that invaded the gill filaments, and heavily infected gills lack host cellular material. In exceptional cases the hepatopancreas may be invaded by the fungus (Fisher and Nilson, 1977). Diseased young juveniles become increasingly lethargic and may have equilibrium deficits. Further, juvenile lobsters with melanized foci move with obvious stiffness and carry their chela directly in front, and on occasion crossed, rather than wide apart as normal lobsters do (Fisher and co-authors, 1975; Fisher and co-authors, 1978). Laboratory infections of adult pink shrimp occurred initially via growth of the fungus into the gill lamellae. Rarely the fungi invaded eyes, eye stalks and exoskeleton (Tharp and Bland, 1977). Behavioral signs in infected shrimp were not reported.

Haliphthoros milfordensis invades its hosts presumably through wounds (Fisher and co-authors, 1975; Johnson, 1983), or possibly via mild chitinolytic activity (Fisher and co-authors, 1975; Fisher and co-authors, 1978). However, Bahnweg and Bland (1980) in a study including 6 strains of *H. milfordensis*, 3 of which were isolated from lobsters, found none of the isolates were capable of chitin hydrolysis. Thus, the chitinolytic activity of the fungus is unconfirmed.

In lobster aquaculture facilities, *Haliphthoros milfordensis* mycosis is controlled primarily through hygiene (Fisher and co-authors, 1975; Fisher and Nilson, 1977; Fisher and co-authors, 1978; Fisher, 1988e). Abrahams and Brown (1977) evaluated 22 chemicals for controlling *H. milfordensis* in American and European lobsters. Fungal growth was inhibited *in vitro* by malachite green at 0.25 mg l^{-1} and Furanace at 2.5 ppm. These chemicals were well tolerated by juvenile lobsters in dip treatments. Lio-Po and co-authors (1985) found *in vitro* inhibition of zoospore production in *H. philippinensis* for malachite green (0.3 mg l^{-1}), Furanace (0.2 to 0.7 mg l^{-1}), formalin (6 to 14 mg l^{-1}) and potassium permanganate (10 mg l^{-1}). Effective dosages of Treflan were considerably higher than reported for inhibition of *Lagedinium* sp. Lio-Po and co-authors recommend an *in vitro* mycocidal dosage to *H. philippinensis* of 20 mg l^{-1}, 100 mg l^{-1} and 200 mg l^{-1} for potassium parmanganate, detergent (Tide) and calcium hypochlorite, respectively. Other chemicals tested were less effective. Lio-Po and co-authors advised caution with use of any chemical treatments for sensitive larval stages of crustaceans. Care to avoid any possible exposure from these chemicals to the natural environment is also important.

Atkinsiella dubia was originally discovered and described by Atkins (1954a) infecting laboratory-held eggs of *Pinnotheres pisum* and *Gonoplax rhonboides*. Atkins demonstrated *A. dubia* was infectious, under artificial circumstances, to eggs of *Trypton spongicola*, *Crangon vulgaris*, *Palaemon serratus*, *Macropodia* sp. and *Portunus depurator*. The fungus was first named *Plectospira dubia* (Atkins, 1954a), but was subsequently redesignated *Atkinsiella dubia* and comprised the type species for the newly erected genus

Atkinsiella and family Haliphthoraceae (Vishniac, 1958). In the Pacific Northwest, Sparrow (1973a) recovered *A. dubia* from laboratory-held eggs of crabs belonging to the genera *Hyas, Oregonea, Pugettia, Chorilia, Skyra, Chianectes* and *Cancer.* Infection of crabs or crab eggs in the natural environment has not been reported (Lightner, 1981), and the importance of *A. dubia* as a pathogen of marine crustaceans appears to be minimal. However, Lightner (1981) isolated *A. dubia* from localized cuticle lesions of an adult, laboratory-held *Penaeus aztecus.* The lesions noted were yellowish-white, friable and limited to the cuticle.

Atkinsiella dubia forms irregularly branched, variable sized (27 to 50 μm) hyphae with bulbous sporangia up to 100 μm in width. The pyriform, biflagellated zoospores are diphasic and form within sporangia or the efferent hyphae that develop as discharge tubes on the sporangia. These hyphae are broad and irregular. Evidence of sexual reproduction has not been observed (Atkins, 1954a; Sparrow, 1973a; Alderman, 1976).

Atkinsiella hamanaensis was described as parasitic on ova of *Scylla serrata* cultured in Japan (Bian and Egusa, 1980). In experiments, *A. hamanaensis* was pathogenic to crab eggs and brine shrimp. The fungus was grown in PYGS broth and is holocarpic with stout, branched, sparingly septate hyphae 12 to 40 μm in diameter. At times, the hyphae tips were expanded into spherical-to-irregular shaped protuberances. Sporangia, formed within thalli, vary in shape from filamentous to saccate lobed, and several terminal or lateral discharge tubes form for each sporangium. Primary and secondary zoospores are pyriform with 2 lateral flagella and an average size of 6 × 5 μm. Zoospores encyst in the sporangium or discharge tubes. Secondary zoospores are morphologically not distinct from primary zoospores. Physiological studies indicated the fungus is euryhaline with reasonable growth over a temperature range of 15 to 32 °C (Bian and Egusa, 1980).

Important as parasites of plants or saprophytes, the Peronosporales are represented by 1 genus, *Pythium,* as pathogens of animals (Alderman, 1982). Two species *Pythium* are known from infections of marine crustaceans. Atkins (1955) described the fungus and the infection of laboratory-held eggs of marine Crustacea (similar host range as reported for *Atkinsiella dubia*) by *Pythium thalassium.* The role of *P. thalassium* as a pathogen is suggested to be minimal (Johnson and Sparrow, 1961). Additionally, Johnson (1970) doubted the taxonomic position of the fungus. Atkins (1955) reported the fungus has highly branched, 5 to 20 μm diameter, predominantly intramatrical hyphae. The extramatrical sporangia are filamentous, single or branched, with thin vesicles, 70 to 90 μm in diameter. Zoospores are biflagellate and 15 μm long. Zoospores encyst and are 10 to 12 μm in diameter. Asexual resting bodies are occasionally found that are spherical, 25 to 50 μm in diameter, and germinate by hyphae (Atkins, 1955).

Infection of *Palaemon serratus* by *Pythium* sp. has been described by Anderson and Conroy (1968) and Delves-Broughton and Poupard (1976). As reproductive stages were not observed, Delves-Broughton and Poupard suggest it to be *Pythium afertile.* The disease occurred in laboratory cultured batches of shrimp and resulted in epidemic losses. The infection started in breaks in the cuticle, but rapidly spread to internal tissues with muscles being heavily invaded by the fungus (Anderson and Conroy, 1968; Delves-Broughton and Poupard, 1976). *Pythium* sp. was isolated on Sabouraud's Dextrose agar from connective tissue and muscle (Anderson and Conroy, 1968; Delves-Broughton and Poupard, 1976). Experimental infection by inoculation and *per os* were reported by these investigators to result in 50% of test shrimp developing symptoms of the disease within

5 to 17 days of exposure. Additionally, injection of the fungus into the haemocoel of *Astacus astacus* resulted in death of exposed individuals (Unestam, 1973).

Of the Deuteromycotina, imperfect fungi, the hyphomycete genera *Fusarium* has 2 species recorded as pathogens of Crustacea. Only infections of decapod crustaceans are known, and for cultured decapods *F. solani* is recognized as important pathogen.

Early reports of imperfect fungi recovered from diseased decapods included 3 species: *Ramularia astaci* isolated from *Astacus astacus* and a crab (Mann and Pieplow, 1938 cited in Unestam, 1973) and *R. branchialis* and *Didmyaria palinuri* infecting *Palinuris vulgaris* and *Homarus gammarus* (Sordi, 1958 cited in Lightner, 1981). These genera are given as synonyms of *Fusarium* (Booth, 1971 cited in Lightner, 1981 and Alderman, 1982). Hence, *Fusarium* spp. is apparently the only member of this group recognized as a pathogen of Crustacea (Alderman, 1981, 1982; Lightner, 1981).

In the recent literature, 2 species of *Fusarium* have been reported as etiologic agents of decapods. Unequivocal identification of the isolated fungus for fusariumosis (Sparks, 1985) of marine decapods revealed *Fusarium solani* (Egusa and Ueda, 1972; Fisher and co-authors, 1978; Alderman, 1981; Lightner, 1981). However, in the freshwater white-footed, native English crayfish *Austropotamobius pallipes, Fusarium tabacinum* infected a solitary, adult, captive-reared individual (Alderman and Polglase, 1985).

Fusarium solani grows readily on most standard artificial media for fungal culture. Sabouraud Dextrose agar, corn meal agar, potato dextrose agar are generally suitable for primary isolation from clinical material. Salt (1 to 3%), antibiotics (penicillin and streptomycin) and occasionally shrimp extract (Lightner and Fontaine, 1975) may be added to improve fungal growth. Antibiotics can be excluded once the fungus is isolated in pure culture.

Pathogenic strains of *Fusarium solani* often produce a brown to purplish-brown diffusible pigment on artificial media (Egusa and Ueda, 1972; Lightner and Fontaine, 1975), but an unpigmented pathogenic isolate has been reported (Alderman, 1981). The *F. solani* mycelium is composed of usually straight (with some branching) septate, and 2.5 to 5.0 μm diameter hyphae. Micro- and macroconidia are formed on thin, short, occasionally branched conidiophores. Microconidia are ovoid to curved, 1- or rarely 2-celled, and 8 to 15 μm in length. Macroconidia are distinctive canoe-shaped 4- to 6- celled and 30 to 40 μm in length (Egusa and Ueda, 1972; Lightner and Fontaine, 1975).

Diseases caused by *Fusarium* species are known as 'burn spot disease' and 'black gill disease' for the principal lesions recorded or as *Fusarium* disease (Lightner, 1977 his Chapter 3.1.6) in recognition of the etiological agent. The first documentation of 'black gill disease' was observed by Ishikawa (1968) in *Penaeus japonicus*. The disease in *P. japonicus* and its etiologic agent, *F. solani*, have been extensively studied (Ishikawa, 1968; Egusa and Ueda, 1972; Fukuyo, 1974; Hatai and co-authors, 1974; Hatai and Egusa, 1978; Hatai and co-authors, 1978; Bian and Egusa, 1981). *Fusarium* disease is also known from other marine decapods including: *P. duorarum* (Johnson, 1974), *P. setiferus* and *P. aztecus* (Solangi and Lightner, 1976), *P. vannamei* (Laramore and co-authors, 1977; Lightner and co-authors, 1979), *P. stylirostris* and *P. californiensis* (Lightner, 1977 his Chapter 3.1.6; Lightner and co-authors, 1979), *Homarus americanus* (Lightner and Fontaine, 1975; Fisher and co-authors, 1978) and *H. gammarus* (Alderman, 1981). Hose and co-authors (1984) concluded for the decapod crustaceans: "The disease and its etiologic agent appear to be truly ubiquitous" (p. 292).

It is apparent from the published information that *Fusarium* is a disease of captive-reared and cultured crustaceans. At the population level, *F. solani* can cause an economically significant disease characterized by endemic but high cumulative mortality (Fig. 3-22) in older, intensively cultured, susceptible host species of marine shrimp (Ishikawa, 1968; Lightner, 1976, 1977 his Chapter 3.1.6, 1981, 1988 his Chapter 3.1.12; Solangi and

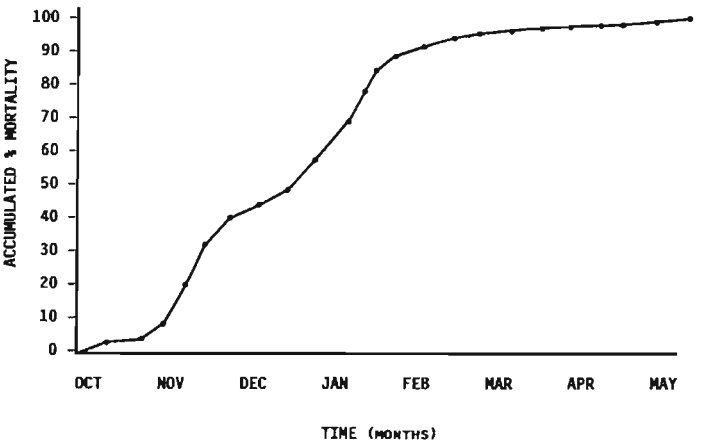

Fig. 3-22: Accumulated percent mortality of a raceway-reared population of *Penaeus californiensis* caused by *Fusarium solani*. (After Lightner and co-authors, 1979; modified.)

Lightner, 1976) and *H. americanus* (Lightner and Fontaine, 1975). However, disease in cultured American lobster populations caused by *F. solani* is only known from a single case report (Lightner and Fontaine, 1975) over a 15 year span. This suggests the disease is of minimal clinical importance to commercial aquaculture of this decapod. The fungus and the disease are not known from decapods in the marine environment, nor is the disease known from eggs, larvae or juvenile life stages of marine crustaceans.

Among the marine decapods, certain penaeid shrimp are highly susceptible to *Fusarium solani*. These shrimp include *Penaeus japonicus* (Egusa and Ueda, 1972; Lightner, 1977 his Chapter 3.1.6) and *P. californiensis* (Lightner, 1976; Lightner and co-authors, 1979; Lightner, 1981; Hose and co-authors, 1984). Lightner (1976) noted that in one case 90 % of cultured *P. californiensis* succumbed while a similar aged and maintained group of *P. stylirostris* suffered only 10 % losses from *Fusarium* disease. Field and laboratory findings (Solangi and Lightner, 1976; Lightner, 1976, 1977 his Chapter 3.1.6, 1988 his Chapter 3.1.12; Lightner and co-authors, 1979; Hose and co-authors, 1984) indicate highly susceptible penaeid shrimp are *P. californiensis* and *P. japonicus; P. stylirostris* is moderately sensitive and *P. vannamei* and *P. monodon* are relatively resistant. The disease is virtually unknown in resistant species cultured at modest densities in tanks and ponds. However, *Fusarium* disease may occasionally be the cause of individual shrimp deaths of resistant species under maturation broodstock captive-rearing conditions.

Fusarium solani is a saprophyte in soil and water. In the only published study (Lightner and co-authors, 1979) of its distribution around a shrimp-rearing facility, *F. solani* was isolated on culture media from 3 species of shrimp with active cuticular lesions;

water supporting shrimp with endemic *Fusarium* disease; rearing tank water of outwardly unaffected shrimp; deep, subsurface well water; and from the air under covered raceways which housed infected shrimp populations. *F. solani* was not found in shrimp feed, beach sand and soil, nearshore surface seawater or from wild-caught shrimp from a nearby fishery. The saprophytic nature of *F. solani* and its distribution in the well water suggest the impracticality of pathogen exclusion as means of control for *Fusarium* disease in shrimp rearing systems.

In experiments, *Fusarium solani* has infected dead tissue and open wounds (Lightner, 1976). Experimental exposure of unwounded and wounded shrimp resulted in disease in the wounded group only (Solangi and Lightner, 1976; Hose and co-authors, 1984). Subadult *Penaeus californiensis* were wounded, and *F. solani* culture material immediately swabbed directly onto the cut surfaces. This resulted in 100% infection of treated shrimp within 14 days (Hose and co-authors, 1984). However, when shrimp were wounded and held in seawater containing spores or if low numbers of spores ($< 10^3$ shrimp^{-1}) were injected into *P. aztecus* and *P. setiferus*, infection did not develop. In contrast, injection of high numbers of spores ($> 10^5$ shrimp^{-1}) resulted in acute-to-subacute death (Solangi and Lightner, 1976) and, in *P. japonicus*, typical clinical signs and lesions of 'black death disease' (Egusa and Ueda, 1972). High acute mortality was also observed in *Cancer pagurus* injected with 4×10^3 spores crab^{-1} or higher doses (Alderman, 1981). In aquaculture settings, *Fusarium* disease apparently arises when conditions support prolific growth of the fungus, susceptible aged host species are present, and integument wounds provide a portal of entry for the fungus.

Marine shrimp susceptibility and resistance to *Fusarium* disease has been discussed by Solangi and Lightner (1976). Based on their observations, hemocyte activity in terms of encapsulation and melanization is important in the apparent resistance of *Penaeus setiferus* and *P. aztecus* to experimental *Fusarium solani* infection which proved fatal to *P. californiensis*. Therefore, shrimp hosts that are most resistant to *Fusarium* disease may have quantitative and qualitative differences in hemocyte activity toward the fungus. Additional hypotheses suggest that subclinical reo-like virus infection (Tsing and Bonami, 1987; Lightner, 1988 his Chapter 3.1.6), shell disease or gut and nerve syndrome (GNS) of *P. japonicus* (Lightner, 1988 his Chapter 3.1.12) may increase host susceptibility to fatal infection by *F. solani*. However, the mechanism(s) for such interactions are unknown. Further, these diseases of *P. japonicus* do not directly apply to the increased susceptibility observed in *P. californiensis*.

Behavioral signs have not been reported for decapods with *Fusarium* disease. Pathologically, *Fusarium* disease appears to have similar presentation and tissue pathogenesis in all decapod crustaceans studied thus far (Lightner, 1981). Infection of cuticular wounds is considered the main portal of entry (Lightner, 1981; Alderman, 1981). Lesions are visible as single to multiple, small to large, slowly developing, ulcerated to raised areas of the gills (Fig. 3-23, a), appendages or general body integument (Egusa and Ueda, 1972; Lightner and Fontaine, 1975; Alderman, 1981). Occasionally, *Fusarium* lesions of shrimp were first recognized as white patches on the eyes (Laramore and co-authors, 1977), and early lesions in lobsters were 'white spots on the exoskeleton' (Lightner and Fontaine, 1975). In terminally infected individuals, wet-mounts of gill lamellae often reveal prolific, occasionally branched, septate hyphae and diagnostic boat-shaped macroconidia (Fig. 3-23,b) (Egusa and Ueda, 1972; Lightner and Fontaine, 1975).

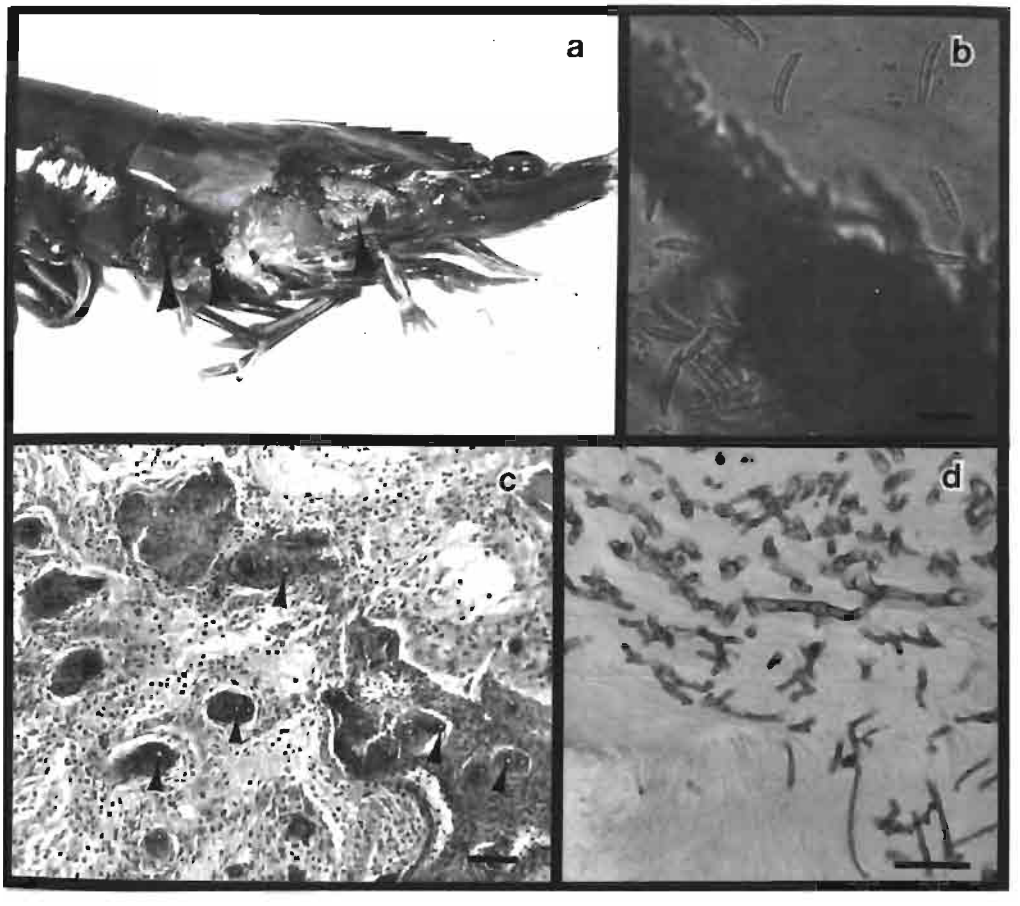

Fig. 3-23: *Fusarium solani* infections of penaeid shrimp. (a) *Fusarium solani* lesions (arrows) in gills and carapace of a subadult *Penaeus californiensis*. (b) Wet-mount of *F. solani* conidiospores prepared from a scraping of the surface of a lesion; no stain; bar = 20 μm. (c) Histological section of a lesion in a uropod of a *P. californiensis;* masses of hemocytes encapsulate *Fusarium* hyphae (arrows) in the lesion; H&E; bar = 50 μm. (d) PAS-stained section of a *Fusarium* lesion showing the abundance of hyphae; bar = 25 μm. (Originals.)

Histologically, melanized cuticular lesions are characterized as pronounced hemocytic responses that appear granulomatous with multiple layers of hemocytes encapsulating hyphal elements (Fig. 3-23, c, d) and distinct melanization of the encapsulated hyphae (Lightner and Fontaine, 1975; Alderman, 1981; Bian and Egusa, 1981). Lesions may extend deep into underlying tissues but invasion occurs by extension rather than systemically via the hemolymph. Infection of internal organ systems (hepatopancreas, antennal gland, lymphoid organ, etc.) has not been reported. Furthermore, spores experimentally injected invariably localized in the distal ends of the gill lamellae (Solangi and Lightner, 1976). Hyphae within the host's exoskeleton are encased by a thin melanized zone lacking hemocytes (Lightner and Fontaine, 1975). Although, focal-to-multiple encapsulation and melanization of hyphae and conidiospores have been observed in the gills, in late stages of the disease hyphal growth may be extensive with only moderate host response to impede

the advancing mycelia. The mitotic activity of the hematopoietic tissue of shrimp infected experimentally with *Fusarium solani* was observed to increase greatly and to account for a hemocytosis found in early infected shrimp (Solangi and Lightner, 1976). Eventually, the hemocytes decrease resulting in hemocytopenia and, in terminal stages, failure of the hemolymph to clot. Solangi and Lightner (1976) recorded average hemocyte counts of 20,000 cells mm^{-3} in early infections that decreased to an average of 3,325 cells mm^{-3} in moribund shrimp.

Changes in hemolymph chemistry for advanced *Fusarium* diseased *Penaeus californiensis* were found by Hose and co-authors who observed significant alterations in serum glucose and total protein (decreased), and alkaline phosphatase and serum glutamic oxaloacetic transaminase (increased) in infected, compared to control shrimp. However, besides the association of these chemistry changes with advanced *Fusarium* disease, no explanation of the role of these changes in the pathogenesis of the disease was given.

Experimental infection of penaeid shrimp, whereby *Fusarium solani* propagules were inoculated into fresh cuticle wounds, resulted in 100% success rate of infection within 2 weeks, and in 50% mortality from the disease by Day 24 (Hose and co-authors, 1984). These and other infection trial results (Egusa and Ueda, 1972; Solangi and Lightner, 1976) show that even localized infection of the cuticle by *F. solani* can result in mortality. The virulence of *F. solani* is suggested to be enhanced through production of potent mycotoxins that may be involved in eventual host death (Lightner, 1976, 1977 his Chapter 3.1.6, 1981, 1988 his Chapter 3.1.12; Hose and co-authors, 1984). Claydon and co-authors (1977) demonstrated production of insecticidal secondary metabolites from a pigmented strain of *F. solani* isolated from lobsters. Additionally, secondary bacterial infection of a host with exhausted defenses may also account for mortality in *Fusarium* disease (Lightner, 1977 his Chapter 3.1.6, 1981, 1988 his Chapter 3.1.12).

Diagnosis of *Fusarium* disease is based on demonstration of the characteristic canoe-shaped macroconidia in wet-mount smears of exoskeleton or gill lesions. A clinical diagnosis can be confirmed by culture, isolation and identification of the fungus on mycological media (Lightner, 1977 his Chapter 3.1.6, 1988 his Chapter 3.1.12; Sindermann, 1977d, 1988d).

Control of *Fusarium* disease in cultured shrimp populations is problematic. Practical methods of chemotherapy for the disease are lacking. Hatai and co-authors (1974) and Lightner and co-authors (1979) tested an extensive array (61 in total) of chemicals none of which were found to be effective *in vivo*, and methods for chemotherapy of *Fusarium* disease in marine crustaceans remain to be developed. Bell and co-authors (1987) noted a statistically significant reduction in the number of *Fusarium* lesions in chronically infected *Penaeus stylirostris* exposed to the commercial microbicide 5-Chloro-2-methyl-4-isothiazolin-3-one and 2-methyl-4-isothiazolin-3-one. Survivals in treated and control groups were not significantly different over the 22 day experiment. Nevertheless, the investigators suggested a longer study period would probably show an increased survival in shrimp receiving the treatment.

Elimination of inoculation of *Fusarium solani* conidiospores into the rearing tank water by filtration or sterilization of water supplies and improved sanitation and hygiene of rearing containers have been suggested as means of control (Lightner and co-authors, 1979). Also, recommended is culture of *Fusarium*-resistant species or development through selective breeding of increased resistance to the disease (Lightner, 1976, 1977 his

Chapter 3.1.6, 1981, 1988 his Chapter 3.1.12; Lightner and co-authors, 1979). For small groups of captive decapods, culling of infected individuals and increased attention to sanitation by frequent removal of detritus and uneaten feed from the container may reduce levels of infective fungus and, in that way, help to reduce disease incidence.

A case of shell disease in *Palinurus elephas,* caused by an unidentified fungus, was noted by Alderman (1973). In other cases of shell disease in *P. elephas* studied by Alderman, bacteria were the probable cause of shell lesions. Thus, the case documented represents an unique occurrence in this lobster of fungal shell disease. Multiple exoskeleton lesions were ulcerated, necrotic, melanized and infected by a mycelial fungus with branching, septate, 1 to 2 μm hyphae. Spores or reproductive structures were not found and only preserved tissues were available for examination; the agent could not be cultured and its identification was not possible. The lobster was caught off Cornwall (England) in spring and held in a tank during summer. When first noticed, shell lesions were reported as severe and, in some areas, penetrated through the exoskeleton so that the underlying muscle was exposed (Alderman, 1973). The lobster eventually died and post mortem examination indicated the cause of death was due to widespread bacteremia. The most likely route for entry of bacteria were the shell lesions.

As pathogens of marine Crustacea, the Ascomycetes are represented by a single species, *Trichomaris invadens.* In Alaskan waters, the occurrence of an encrusting fungus on carapace and appendages of *Chionoecetes bairdi* was long recognized by commercial fisherman and fishery biologists who referred to the conditon as 'black mat syndrome' or 'black mat disease' because of the external gross appearance of the fungal infection, which is "a mat of black encrusting nodules or pustules forming a dense, hard, almost tar-like covering over parts of the exoskeleton" (van Hyning and Scarborough, 1973, p. 1738).

Black mat syndrome was initially described to be a non-invasive fungus encrustation caused by the coelomycete fungus *Phoma fimeti.* Although recognized as a nuisance because pieces of fungus contaminated the crab meat during processing, the surface infestation appeared to have no obvious detrimental impact on the health of tanner crabs, with the possible exception of very heavily infested individuals (van Hyning and Scarborough, 1973). These investigators also noted that the fungus infection was species and possibly area specific, as dungeness and king crabs coinhabitating with black mat diseased tanner crabs were never affected. Black mat syndrome is limited to tanner crabs in the Alaskan fishery and unknown from other Pacific and Canadian Atlantic fishery areas (Sparks, 1985).

Sparks and Hibbits (1979) necropsied 11 tanner crabs with black mat syndrome and 9 unaffected individuals and found microscopically, with use of special staining (Grocott's method for fungi – GMS) that the fungus invades, sometimes extensively and massively, the internal tissues of the crab. Sparks and Hibbits proved the fungus is an invasive pathogen of the tanner crab. Further, they suggested, based on their observations, that the infecting fungus was not *Phoma fimeti,* and subsequently (Hibbits and co-authors, 1981) described and named a new genus and species of marine pyrenomycetes, *Trichomaris invadens,* an apparent obligate fungal parasite in members of the marine crustacean genus *Chionoecetes.*

Trichomaris invadens has sparingly septate, branched black extramatrical hyphae, 5.0 to 5.5 μm in diameter that form an encrusting subiculum on the host's carapace. Intramatrical hyphae are non-pigmented, infrequently septate, branched and 2.0 to 2.5 μm in

diameter, growing tortuously often in tight bundles in internal crab tissues. Perithecia are external and thick walled. Asci are elongate clavate, unitunicate, thin walled and 85.0 to 155.0 × 12.0 to 17.0 μm in size. Ascospores are hyaline, oblong to ellipsoidal 15.0 to 22.0 × 4.6 to 5.0 μm with 1 to 3 septa (usually one) and have a hair-like appendage at either end, 50.0 to 360.0 μm in length (Hibbits and co-authors, 1981). *T. invadens* is a fastidious fungus and has not been cultured successfully on artificial media (Hibbits and co-authors, 1981).

Trichomaris invadens was recorded from 3 species of tanner crabs: *Chionoecetes bairdi, C. opilio* and *C. tanneri*, but extensive infections are known only from *C. bairdi* (Hibbits and co-authors, 1981). The fungus is apparently host specific for the genus *Chionoecetes* (van Hyning and Scarborough, 1973; Hibbits and co-authors, 1981).

According to Brown (1971 cited in van Hyning and Scarborough, 1973) prevalence of the disease in 1,000 tanner crabs from commercial catches around Kodiak and Shumagin Islands averaged 37 % and ranged from 7 to 75 %. In a later survey, Hicks (1982) reported prevalence and distribution of black mat syndrome in 38,394 male and 8,160 female tanner crabs from areas in northwestern Gulf of Alaska in his 1980 and 1981 studies. A marked difference in prevalence of black mat disease was found for legal-sized males in the Kodiak district. Offshore schools of crabs were more often infected and had markedly higher infection prevalences than males collected from inshore schools. Black mat syndrome infection prevalences reached 65 % in some offshore areas surveyed. The Chignik and South Peninsula area males were less frequently infected (4.9 % and 0.8 %, respectively) than male crabs sampled from the Kodiak fishery region. Interestingly, higher prevalence was observed in pot caught versus trawl collected tanner crabs. Hicks (1982) suggested this difference may reflect that pots selectively capture the older, larger crabs. A higher percentage of old and very oldshell tanner crabs had the disease. Hicks (1982) suggested there may be a relation between the syndrome and skip molting. Thus, prerecruit fishery crabs with the disease may not contribute to the fishery. Female tanner crabs caught during the 1980 and 1981 pot surveys had the highest overall prevalence of the disease, 50.1 % and 34.8 %, respectively. Furthermore, up to 94.7 % of barren females examined in the 1981 Kodiak pot survey were infected. However, whether the barren state is a matter of senescence or caused by black mat disease is unknown (Hicks, 1982). According to Sparks (1982a, 1985), the available information indicates black mat disease at epidemic levels in some areas in the Alaskan fishery.

In *Chionoecetes bairdi, Trichomaris invadens* progresses as a widespread invasion of internal organs with massive proliferation of hyphae, replacement or destruction of host tissue, and nearly complete absence of host defensive response to the invading pathogen. The infection is almost certainly fatal to infected crabs, but the time course of the disease and rate of transmission is unknown. Infections have not been studied under laboratory conditions (Sparks and Hibbits, 1979; Sparks, 1982a, b). Further, infected crabs undoubtedly do not molt, thus diseased sublegal crabs fail to attain legal size, and infected immature females do not undergo their molt to sexual maturity; hence they are a loss to population recruitment (Sparks, 1982a). Black mat disease has obvious implications to crab survival in the Alaskan fishery (Sparks and Hibbits, 1979; Sparks, 1982a, b, 1984); although an earlier report (van Hyning and Scarborough, 1973) indicated this seemed unlikely.

The gross appearance of the disease has been characterized by van Hyning and

Scarborough (1973); Sparks and Hibbits (1979); and Sparks (1982a, b, 1984). The first appearance of the disease are tiny focal black spots on the carapace, apparently quite easy to discern against the slightly iridescent orange exoskeleton. These spots enlarge and coalesce; eventually the whole dorsal carapace is overlayed by a dark, tar-like mass of tangled hyphae and fruiting bodies, and may spread to the ventral surface and appendages (Fig. 3-24, a). Occasionally, encrustation begins on the appendages or on both carapace and appendages. Eyes and eyestalks are often infected. There is no gross obvious evidence of fungal infection of the internal tissues as the hyphae are not pigmented. On gross dissection Sparks (1982a) noted the epidermis is more than normally adherent to the overlying exoskeleton.

Microscopically, the external hyphae are thick-walled and pigmented. Within the hyphal mass are perithecia (Fig. 3-24, b) and those that are mature accommodate numerous asci each of which contains 8 spores. Examination of unstained smears of epidermal and subepidermal tissues reveals abundant, non-pigmented hyphae. Histologically, tissue sections stained with GMS show the widespread distribution and massive proliferation of hyphae within the organs of infected crabs. While epidermal and subepidermal tissues are most often involved heavily, deeper infection of the fungus, suggested to be by extension via connective tissues (Sparks and Hibbits, 1979; Sparks, 1982a, b), can be expansive. In massive infections the connective tissues, blood vessels, etc. around major organs may be largely replaced by fungal hyphae. The hematopoietic tissue is necrotic, and mitoses are decreased or cease altogether. Striated (Fig. 3-24, c) and cardiac muscles are invaded, as are the walls of blood vessels and the gut. However, gut epithelium, midgut caeca, hepatopancreas acini and interstitial tissue, antennal gland, gonad and mandibular organ are not encroached. Degenerating, necrotic ova, are sometimes present in the ovary of heavily infected crabs. Small and large peripheral nerves are occasionally invaded, but infection of the brain and the thoracic ganglia have not been observed (Sparks, 1982a). The eyestalks and underlying tissues are frequently invaded with extensions along the connective tissues to the retina occasionally present, in which case the retina may be breached and destroyed. Sporadically, hemocytic inflammation and melanization has been noted in markedly degenerated retinal tissues (Sparks and Hibbits, 1979).

Changes in hemolymph cytology of *Chionoecetes bairdi* associated with black mat disease have been observed by Mix and Sparks (1980). They found a significant increase in eosinophilic granulocytes (EC) and a concomitant decrease in hyalinocytes (HC) in heavily diseased tanner crabs, compared to non-infected controls. Higher eosinophilic granulocyte counts also correlated with infection severity, based on histological criteria. Mix and Sparks could not attribute the shift in differential count to a specific cause, but suggested it could result from (i) increased mobilization of ECs in response to the fungal invasion, (ii) reduced time of maturation of the HCs to ECs with steady-state entry of HCs into circulation, and (iii) decrease in number of HCs in response to the fungal infection. Histologic data presented by Sparks and Hibbits (1979) and Sparks (1982a) do not support loss of hemocytes from circulation due to sequestering in tissues within hemocyte aggregations, as such aggregations are not part of the microscopic pathology for black mat disease. However, the observations of damage, necrosis and decreased mitotic activity (Sparks, 1982a) of the hematopoietic tissue of infected crabs would support the consideration that fewer hyalinocytes are being formed and, thus, entering the circulation. Therefore, the shift in differential could represent a situation where circulating EC numbers gradually

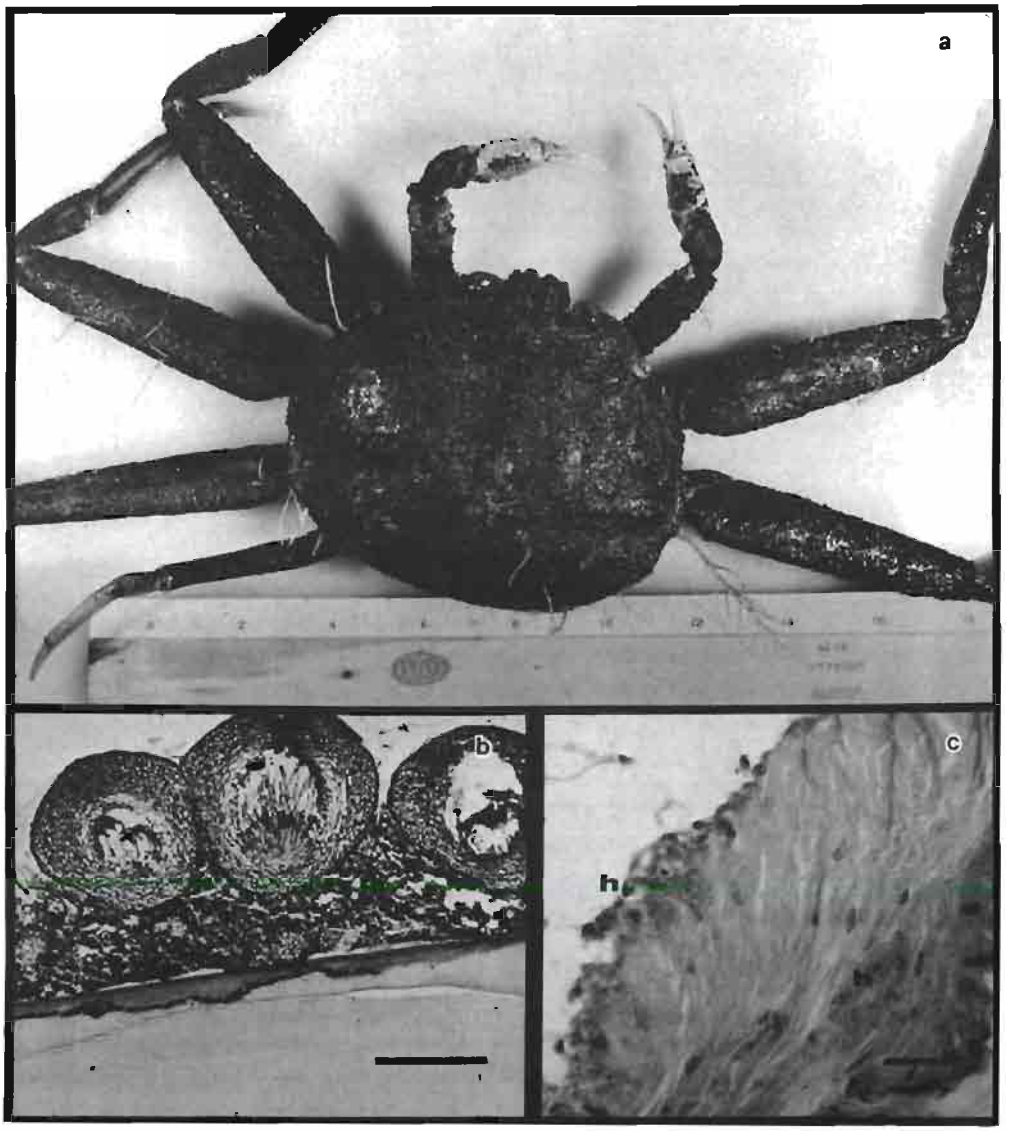

Fig. 3-24: *Chionoecetes bairdi* with black mat syndrome. (a) Gross appearance of a heavily encrusted tanner crab. (After Hibbits and co-authors, 1981. Reprinted with the permission of the National Research Council of Canada.) (b) LM section through carapace exoskeleton with external hyphal mat and fruiting bodies of *T. invadens;* GMS; bar = 200 μm. (After Sparks and Hibbits, 1979. Reprinted with the permission of Academic Press, Inc.) (c) LM of striated muscle tissue deep in the cephalothorax invaded by *T. invadens* hyphae (h); H&E; bar = 50 μm. (After Sparks, 1984.)

increase as a result of progressively fewer HCs being produced and entering circulation. Unfortunately, total hemocyte, total HC and total EC cell counts were not reported by Mix and Sparks (1980); these data would indicate if reduced output from the hematopoietic tissue could explain the differential shift observed in Black mat diseased tanner crabs. Data on serum or tissue chemistries comparing diseased and non-diseased tanner crabs

have not been reported. The results of such studies may provide additional insight into the pathogenesis of *Trichomaris invadens* in the tanner crab.

Hibbits and co-authors (1981) and Sparks (1982a, b, 1984) point out that both external and internal infection of crabs have always occurred together, suggesting a simultaneous external and internal development of the parasite in the host. These authors emphasize the great need for study of the disease under laboratory conditions so that further understanding of the means and rates of infection transfer, the mode of infection, the time course of the disease after infection, etc. can be established for this obviously unique microbial disease of crustaceans. Important not only theoretically, but also, and more significantly, because of its potential as an ecological factor in tanner crab populations and the productivity of the tanner crab fishery in Alaska (Sparks, 1985). Also of significance to the fishery, as pointed out by Hoskin (1983), is that the fungus invades the edible tissues in crab legs under externally infected cuticle areas. Fungus-infected crab meat is adulterated. The biochemical composition and other attributes of fungus-infected crab meat or the presence of a fungus-elaborated toxin in infected tissues have not been investigated (Hoskin, 1983).

Within the Zygomycotina, the Trichomycetes are a class of obligate fungal symbionts whose members attach to the chitinous lining of the hind-gut, and occasionally the foregut, in a variety of arthropods (Johnson, 1970; Johnson, 1983). In forms associated with marine crustaceans, the trichomycete fungi consist of branched and unbranched tubular filaments and attach to the host by a holdfast that does not penetrate the cuticle, thus the fungus is an epibiont. The relation is regarded as symbiotic (Johnson, 1970; Alderman, 1982). However, a recently described species penetrates the cells of the mid-gut and is considered as a cause of mortality to its larval host, the mosquito, *Anopheles hilli* (Alderman, 1982). Thus, true parasitic trichomycetes may also occur in marine Crustacea, but to date, none has been reported.

The family Ellobiopsidae is a heterogenous group of marine parasites, world-wide in distribution, that are predominantly found on pelagic Crustacea. The 2 best described genera are *Thallosomyces* and *Ellobiopsis* which comprise parasites of euphausides, mysids, copepods and occasionally carideans. The host specificity of the Ellobiopsidae is often low (Unestam, 1973). For example, Boschma (1959) lists 3 genera and 2 families of hosts attacked by *Thalassomyces racemosus*.

The Ellobiopsidae have been classified as protistans, colorless algae, fungi, protozoans or dinoflagellates (Boschma, 1949, 1959; Kane, 1964; Collard, 1966; Galt and Whisler, 1970; Unestam, 1973; Wing, 1975). Their affinities are not certain; they may be heterogenetic and polyphyletic (Unestam, 1973). The Ellobiopsidae are often listed as *incertae sedis* Collard (1966) and Wing (1975). These parasites are multinucleate protistans that have an external, relatively large and often visible grossly, reproductive mass, which is secured to the host by an internal haustorial system. Some ellobiopsids, attach to the host's surface rather than penetrate its cuticle. These forms are probably not parasites (Unestam, 1973). For those with an internal root system, the external portion is composed of proximal stems (trophomeres) that have terminal stems (gonomeres). Ellobiopsids are often found in a specific location on the host, such as ventral abdomen or eyes. Those that project from the ventral abdomen may be mistaken for egg cases (Johnson, 1983).

The internal haustorial system of ellobiopsids invades the host's tissues, and these absorptive roots may extend into the gonads or into the central nervous system where

degeneration and/or reabsorption of the organ may or may not occur. In general, ellobiopsids seem to be very well adapted to their hosts and to cause little tissue damage or detrimental effects. There is apparently no or minimal host response to the root system. Hormonal imbalance could be caused by species with root attachments in the nervous tissues (Johnson, 1983), but such an effect has not been demonstrated.

Acknowledgements. Drs. Albert Sparks and Phyllis Johnson are thanked for providing a number of photographs. The authors gratefully acknowledge the time, effort and expert assistance provided by Carol Larm in the procurement of papers, editing and preparation of the reference section. Also thanked for their help are Mineyo Shimojo and Lynn Miyashiro. Support for the work was provided by the Aquaculture Development Program, Department of Land and Natural Resources, State of Hawaii, USA.

3.2 DISEASES CAUSED BY PROTISTANS AND METAZOANS

T. R. MEYERS

This subchapter reviews the diseases of marine crustaceans caused by infectious protozoans and metazoans. Many of these agents have been included because they are parasites in the strict sense (Vol. I p. 19). However, disease by virtue of virulence does not result from infection by many of these parasites and is unknown for others from lack of study. Primary pathogens with epizootic or panzootic potential to produce significant negative deviation from the normal state (Vol. I: Kinne, 1980, p. 14) in crustacean hosts are discussed in detail. These include some of the most lethal crustacean diseases caused by members of the protozoans such as dinoflagellates, amoebae, and microsporidians. Equally devastating physiological and endocrinological disorders at the population level are caused by members of the Isopoda and Rhizocephala. Opportunistic parasites or ectocommensales in both the Protozoa and Metazoa may cause significant disease in crustaceans indirectly due to poor environmental or cultural conditions. Others may be capable of causing mortality in hosts but occur enzootically in often insignificant prevalences. Agents in these last 2 categories are considered as well but the reader is referred to other texts for greater detail (Overstreet, 1978; Provenzano, 1983; Sparks, 1985).

DISEASES CAUSED BY PROTISTANS

Agents: Dinoflagellata

The Dinoflagellida contain a curious collage of organisms ranging from photosynthetic forms such as red-tide organisms (better classified as algae) to obligate parasitic forms infecting crustacea, certain fishes (Brown, 1934; Weiser, 1949) and cephalopods (McLean and co-authors, 1987). The major feature of classification for these often unrelated-appearing organisms is the biflagellated grooved dinospore stage. *Blastodinium hyalinum* (Apstein, 1911; Chatton, 1920), *Syndinium* sp. (Chatton, 1910; Chatton, 1920; Jepps, 1937) and *Paradinium pouncheti* (Chatton, 1920; Jepps, 1937) infect the body cavities or guts of copepods in the North Sea and the Mediterranian Sea. Other external parasites such as 3 species of *Chytrodinium* (Cachon and Cachon, 1968) and 2 species of *Dissodinium* (Drebes, 1978; Elbrächter and Drebes, 1978) from France and the North Sea infect surfaces of copepod eggs. One unidentified peridinian parasite found in the Gulfs of Maine and Alaska infects the yolk of pandalid embryos (Stickney, 1978). Internal copepod parasites cause parasitic castration, and 2 species cause death of the host. Negative effects of such parasitism at the population level can be speculated upon but are as yet unknown.

Pandalid egg parasites may destroy the host egg and limit clutch sizes and recruitment (Holmes and co-authors, 1980). Major dinoflagellate pathogens include *Hematodinium perezi* infecting the hemolymph of *Carcinus maenas* and *Portunus depurator* in European waters (Chatton and Poisson, 1931) and the blue crab *Callinectes sapidus,* lady crab *Ovalipes oscellatus* and 2 other species of cancer crabs, *Cancer irroratus* and *C. borealis,*

from the mid-Atlantic Bight in North America (Newman and Johnson, 1975; MacLean and Ruddell, 1978). A similar but different dinoflagellate infects the hemolymph of the Tanner crabs *Chionoecetes bairdi* and *C. opilio* in Southeast Alaska and the Bering Sea causing Bitter Crab Disease (Meyers and co-authors, 1987). Another *Hematodinium*-like organism has also been reported to infect benthic amphipods (Johnson, 1986).

Blue crabs are known to die from experimental infections of *Hematodinium perezi,* and naturally infected individuals are lethargic sustaining deadloss after handling. Whether the disease exerts a negative effect on population numbers has not been established. The clinical effects of the parasite on the other reported crab species is assumed to be the same but is not documented. The pathogenesis of this parasite and its life cycle with spore production have not been described. Prevalence information is also lacking except that peak infections in the blue crab have reached 30 % in one sample. Although the disease is most prevalent in fall, it can be found during all months but has not been recorded in blue crabs from salinities less than 11 ppt (Newman, 1977).

The *Hematodinium* organism infecting benthic amphipods from the continental shelf of the northeastern United States has occurred in prevalences ranging from 1 to 67 % (Johnson, 1986). Some spore forms were observed but only histological material was available, so definitive morphologic determination of dinospore shape and whether biflagellation was present could not be determined. The parasite replicated in the hemocoel of infected amphipods representing 13 species with no apparent host response and was assumed to be lethal and possibly regulatory in amphipod populations (Johnson, 1986).

Bitter Crab Disease was first discovered in 33 % of the major commercially exploited Southeast Alaskan Tanner crab (*Chionoecetes bairdi*) populations in 1985 by Meyers and co-authors (1987). Clinical signs of the disease consist of an exaggerated pink coloration of the carapace, lethargy with deadloss and milky hemolymph. The causative agent is a dinoflagellate similar to, but morphologically different from, *Hematodinium* sp.; it replicates within the hemolymph of infected crabs resulting in population prevalences of up to 95 % in both sexes and all age classes. Virtually 100 % mortality occurs in naturally infected crabs with an accompanying chalky texture and bitter aftertaste in cooked crab meats that is unacceptable as a market product. The poor meat quality alone resulting from this disease has cost the crab industry in Southeast Alaska thousands of dollars yearly. Parasite infection results in a chronic wasting disease over an 11 month period produced by single-cell and plasmodial vegetative stages (Figs. 3-25 to 3-27) that infiltrate with the hemolymph into all major organs and tissues. Many crab hosts die within 5 to 6 months after infection but those surviving the entire vegetative period die within 24 to 48 hours following sporulation of the parasite.

Field and laboratory studies suggest that the disease follows a yearly cycle and may be related, in part, to increasing water temperature as the seasons progress. Vegetative stages (Figs. 3-28 to 3-31) occur in crab hosts from October through July with prespore stages (Fig. 3-32) developing from July through September. Sporulation of prespores, producing dinospore stages, generally follows within 10 to 14 days. Vegetative stages have typical mesokaryotic nuclear detail similar to *Hematodinium* sp. but no cytoplasmic trichocysts (Fig. 3-31). Despite the same microscopic and ultrastructural appearance of all vegetative stages, 2 types of biflagellated dinospores are produced, but only 1 type develops from any single crab host. The small spore type is elliptical (12.0 × 4.4 μm), rapidly motile, smooth

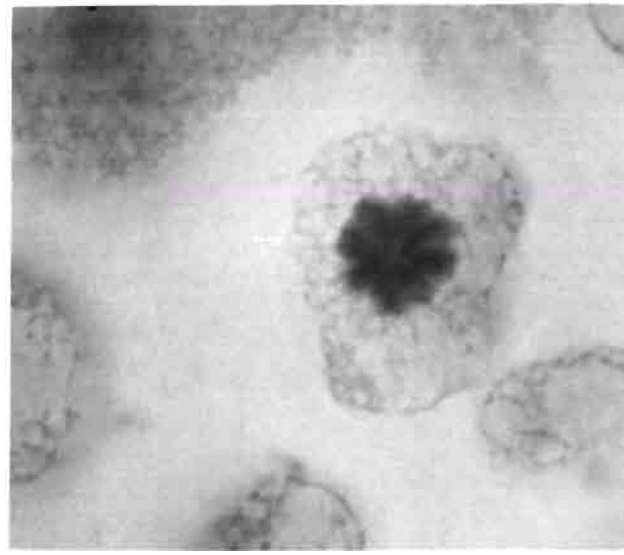

Fig. 3-25: *Chionoecetes bairdi*. Dinokaryon nuclear division of a dinoflagellate vegetative stage in infected crab tissues showing V-shaped pairs of chromosomes. Hematoxylin and eosin, × 5,000. (After Meyers and co-authors, 1987.)

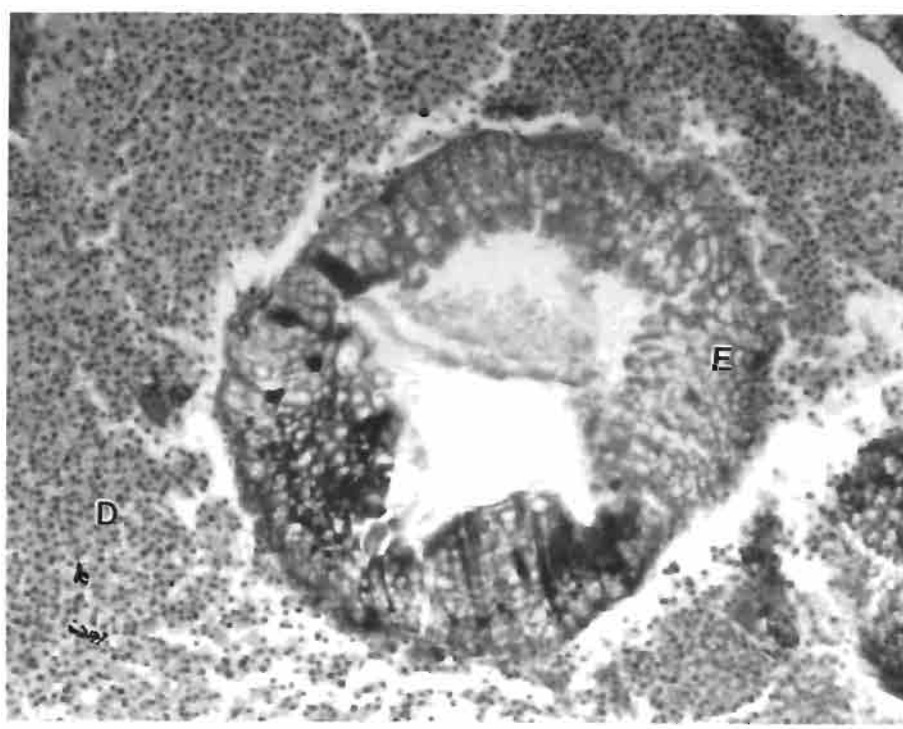

Fig. 3-26: *Chionoecetes bairdi*. Hepatopancreas from an infected individual with vacuolar degeneration and loss of nuclear staining in tubular epithelium (E) and replacement of interstitial vesicular connective tissue by myriad numbers of dinoflagellate vegetative stages (D). Hematoxylin and eosin. × 315. (After Meyers and co-authors, 1987.)

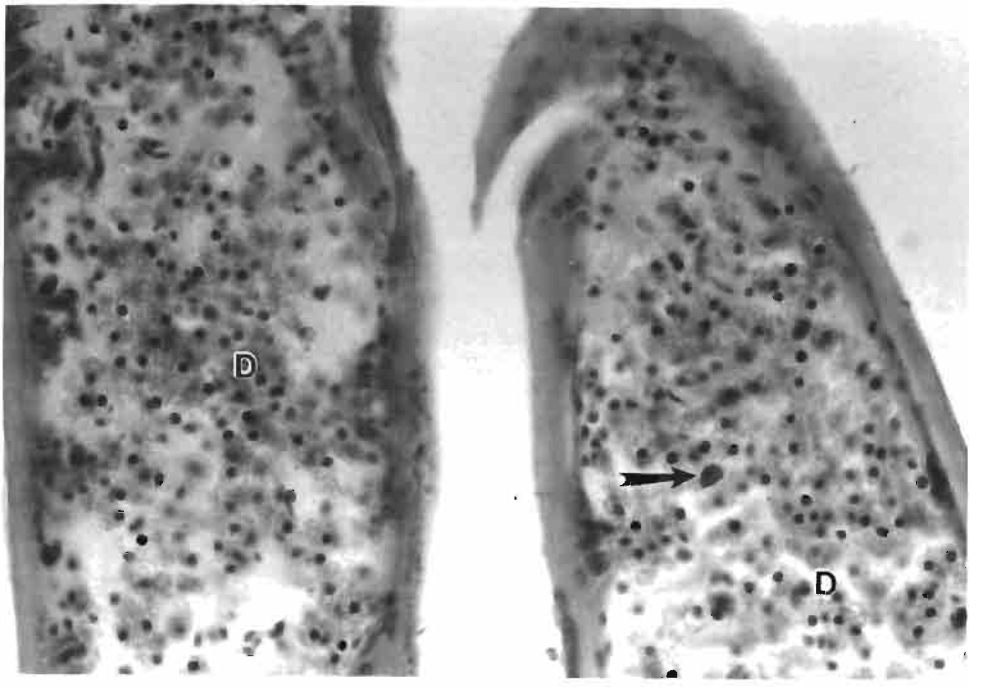

Fig. 3-27: *Chionoecetes bairdi.* Gill filament from an infected crab showing engorgement of vesicular spaces with dinoflagellate vegetative stages (D); host granulocyte (arrow). Hematoxylin and eosin, × 800. (After Meyers and co-authors, 1987.)

surfaced with a refractile body at the posterior end, and has a dense nucleus with wide swirls of chromatin and little nucleoplasm (Figs. 3-33 to 3-36). The nuclear-to-cytoplasmic ratio is less, with the cytoplasm containing fewer electron dense bodies and trichocysts than the larger spores. Small spores develop a bent corkscrew profile after 24 hours in culture that becomes more pronounced by 6 days (Fig. 3-34). The large spore type (Figs. 3-37 to 3-40) is oval (15.2 × 11.4 μm) and has a knobby surface with a slight lateral protrusion; this develops markedly by 9 days into a keeled structure that is beak-like at one end. The nuclear detail is similar to that of the vegetative stage, unlike the small spore nucleus, and the cytoplasm has abundant electron dense bodies and trichocysts connected to peripheral pores in the pellicle.

Infection of new Tanner crab hosts in August through September occurs putatively by one or both types of spores, or possibly by an infectious zygote formed by fusion of the 2 spore types; the latter being possible if the different spores represent male and female forms. The route of transmission and whether the molting cycle of the host enhances infection is a major part of the parasite's life cycle needing further investigation as does the potential cyclical nature of the disease. Contact transmission of the parasite does not appear to occur from crab hosts in the vegetative stage of the disease. But when injected into normal crabs, vegetative stages can produce detectable disease within 55 to 83 days post-inoculation. This would suggest that vegetative stages may be infectious if able to

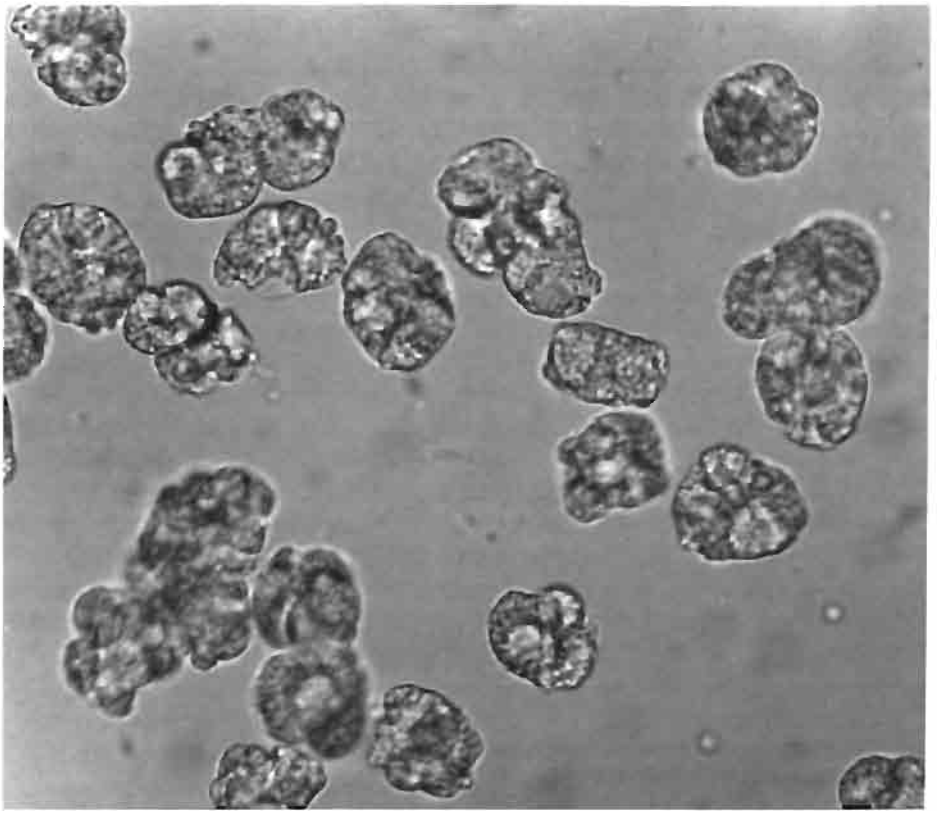

Fig. 3-28: *Chionoecetes bairdi*. Wet hemolymph smear of living dinoflagellate vegetative stages from an infected crab showing irregular shapes and variable size. × 2,000. (After Meyers and co-authors, 1987.)

enter the crab through a wound externally or within the gastrointestinal tract*. Whether vegetative stages could be infectious if ingested by susceptible crab hosts is not known but would account for the widespread infections in some populations and apparent efficiency of parasite transmission. Injection of vegetative stages into red king crabs *Paralithodes camtschatica* did not produce infections indicating this species was probably refractory to the parasite. Additional studies on host species susceptibility have not been performed.

Death of infected Tanner crabs may result from organ and respiratory dysfunction as tissues are replaced by the parasite. Loss of immunocompetence occurs as well with secondary bacterial and ciliate protozoal infections causing death of bitter crabs held in the laboratory. Vegetative stages of the parasite exude external droplets of a substance from pores within the pellicle (Fig. 3-30), speculated to be the cause of the bitter offlavor in cooked meats. The material could also be proteolytic and toxic in nature further contributing to meat degradation and death of the host. Biochemical analysis of this material and its

* Parasite prevalence is significantly greater in new shell vs old shell crabs suggesting infection by vegetative stages may be possible during crab molting in early spring (Meyers and co-authors, 1990).

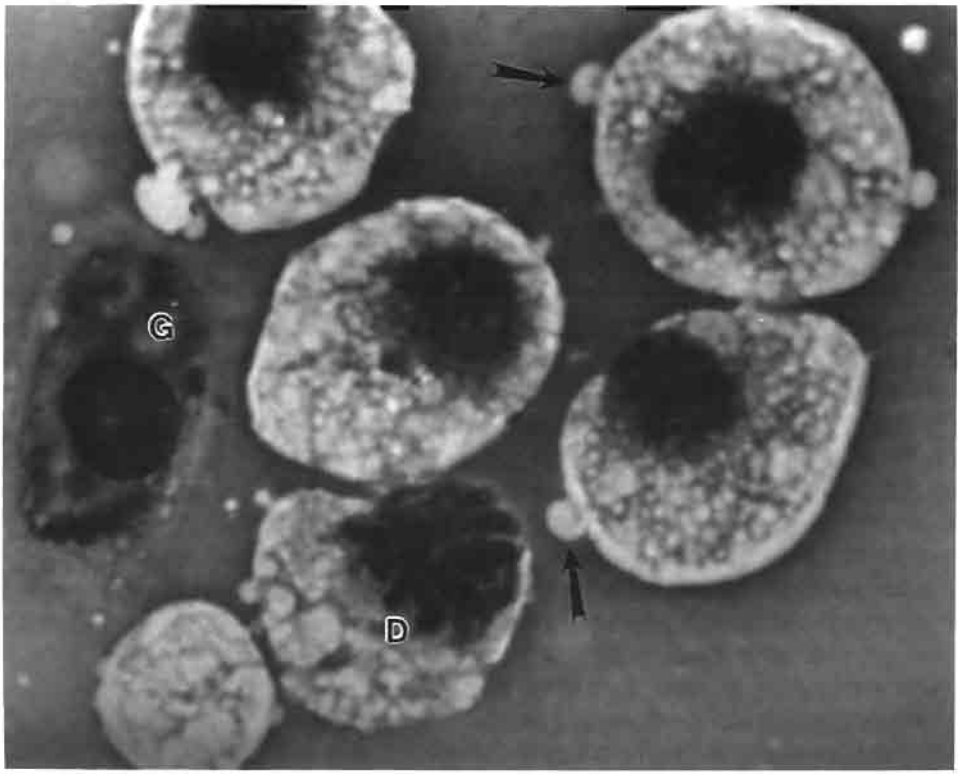

Fig. 3-29: *Chionoecetes bairdi.* Fixed hemolymph smear from an infected crab showing dinoflagellate vegetative stages and a host granulocyte (G). Note dinokaryon-type nuclear division (D) and droplet formation (arrows) on external surface of nonstaining pellicle. Diff-Quik, × 5,000. (After Meyers and co-authors, 1987.)

true role in the disease process remain to be determined. Although patchy, the distribution of Bitter Crab Disease in *Chionoecetes bairdi* populations in Southeast Alaska is widespread and has been detected recently in the Bering Sea *opilio* Tanner crab. Current field data and catch statistics in southeast Alaska suggest that the disease is spreading to previously uninfected *C. bairdi* populations.

Because the Bitter Crab Disease is eventually fatal in all infected crabs, the potential for serious decimation of crab populations and major economic losses is almost certain in those areas where the majority of crabs caught are infected with the dinoflagellate. There are methods being utilized to reduce the dissemination of this disease. Fishermen sort diseased crabs on the fishing grounds so that these animals may be returned where caught rather than be transported to another area and later discarded to infect other Tanner crab populations. Processors will not accept infected crabs and sort these out as well to be disposed of by burial, incineration or cooking before discharge into seawater. Areas having seriously diseased crab populations have also been closed to crab harvest. Perhaps the most promising method of disease management will depend upon whether the life cycle of the parasite is completed within a year in a single crab host. Should this be established, earlier harvest seasons could be enforced to take newly infected crabs before parasite infection progresses to the stage of meat degradation and crab death. All crabs would be

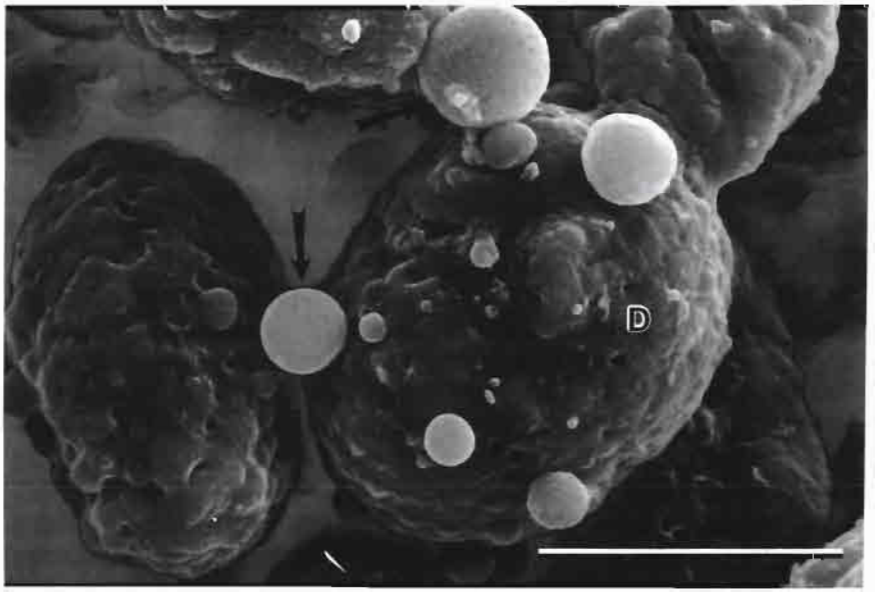

Fig. 3-30: Scanning electron micrograph of dinoflagellate vegetative stages (D) revealing irregular thecal surface with pores and exuding droplets of an unidentified material (arrows). Gold palladium, × 4,000. Bar = 10 μm. (After Meyers and co-authors, 1987.)

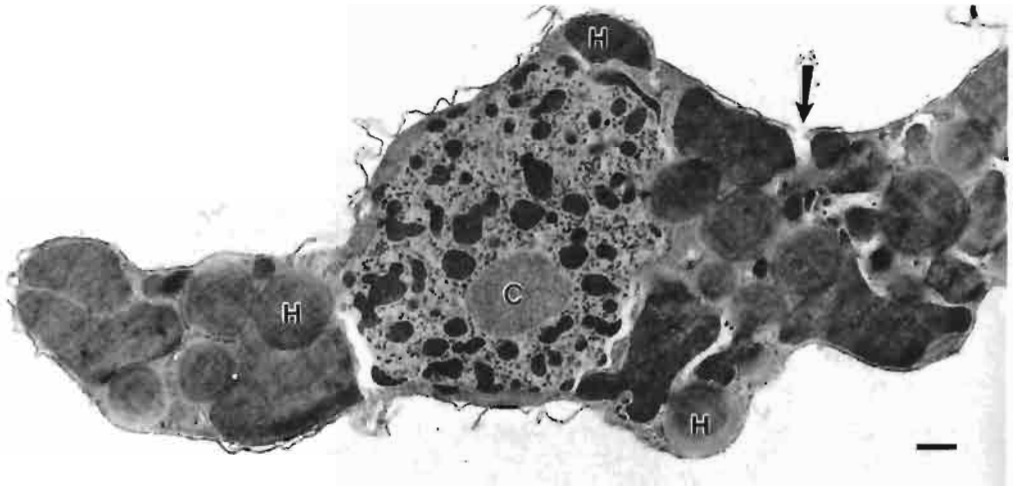

Fig. 3-31: Ultrastructure of dinoflagellate vegetative cell showing cytoplasmic invagination (C) in the nucleus, and numerous granular cytoplasmic inclusions (H) some of which cause outward bulging of the pellicle; a break within the pellicle integrity is visible (arrow). Uranyl acetate and lead citrate, × 7,000. Bar = 1 μm. (After Meyers and co-authors, 1987.)

marketable and the risk of disease dissemination from improper disposal of culled infected crabs would be greatly reduced. Bitter Crab Disease has already caused serious economic losses in the Tanner crab fishery in Alaska's southeast panhandle. The effect of this disease on the Bering Sea *opilio* fishery remains to be determined.

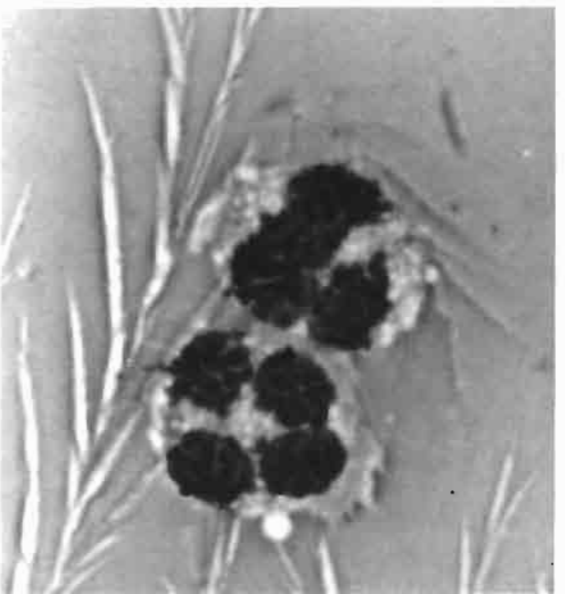

Fig. 3-32: Fixed crab hemolymph smear of dinoflagellate multinucleated prespore stages undergoing rapid cell division 9 days prior to sporulation into the smaller type of dinospore. Diff-Quik, × 5,000. (After Meyers and co-authors, 1987.)

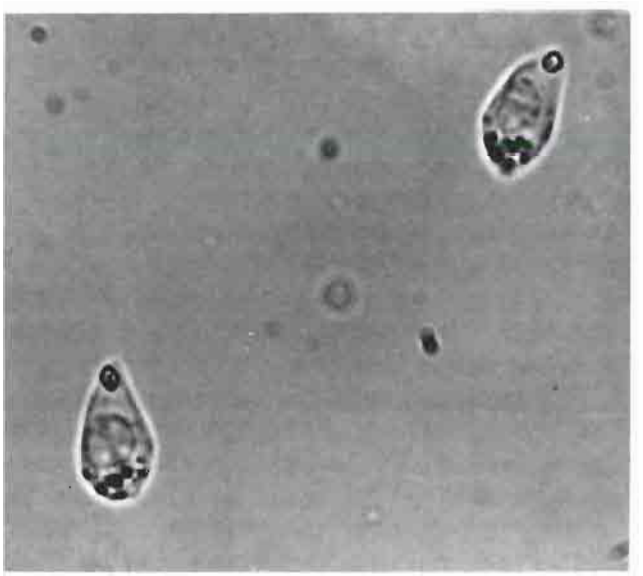

Fig. 3-33: Wet smear of the smaller type of live dinospores 2 days in saltwater culture after collection from an infected Tanner crab. Note refractile granule at distal posterior end. × 3,300. (After Meyers and co-authors, 1987.)

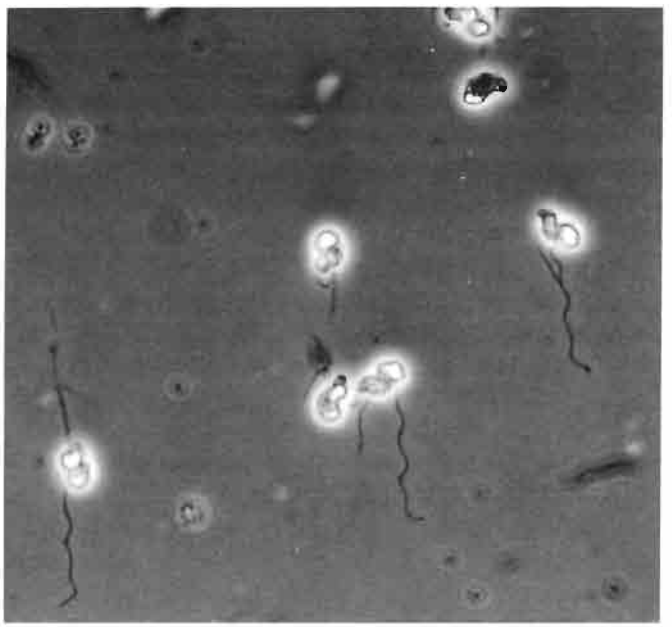

Fig. 3-34: Glutaraldehyde fixed spores from Fig. 3-33 after 11 days in saltwater culture demonstrating pronounced corkscrew shape and 2 obvious flagella. Phase contrast, × 1.320. (After Meyers and co-authors, 1987.)

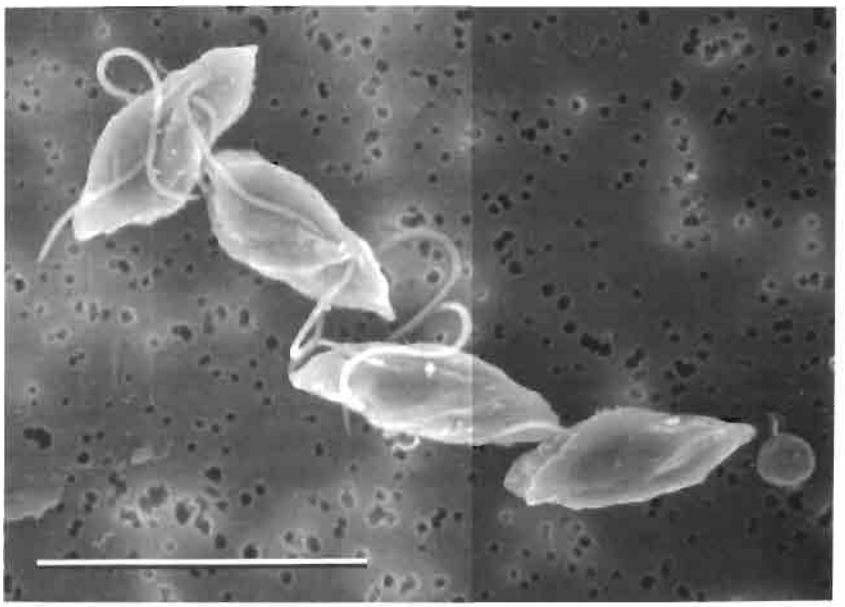

Fig. 3-35: Smaller dinospore type soon after sporulation showing a smooth surface with a small ventral keel having a groove that accommodates the trailing whiplash flagellum. The longer circumferential flagellum is attached anteriorly to the grooved keel. Gold palladium. × 4,200. Bar = 10 μm. (Original.)

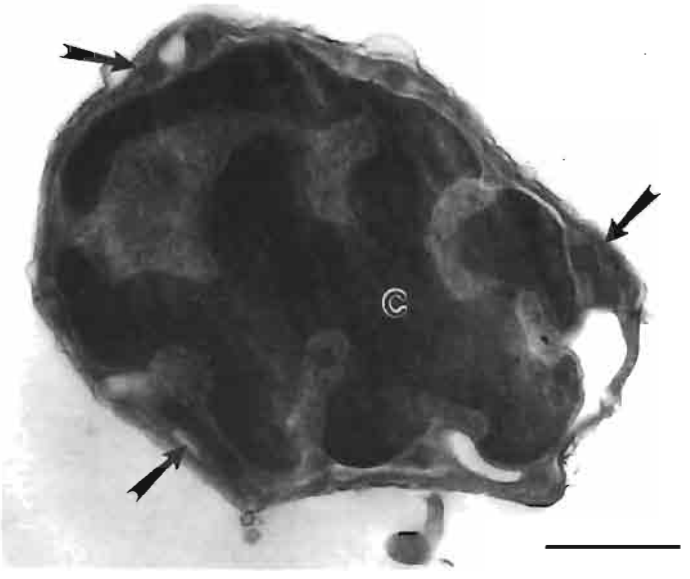

Fig. 3-36: Tangential section of small dinospore type from an infected Tanner crab immediately after sporulation showing nuclear chromatin (C) densely interconnected but not beaded and few trichocysts (arrows). Uranyl acetate and lead citrate, × 18,000. Bar = 1 μm. (After Meyers and co-authors, 1987.)

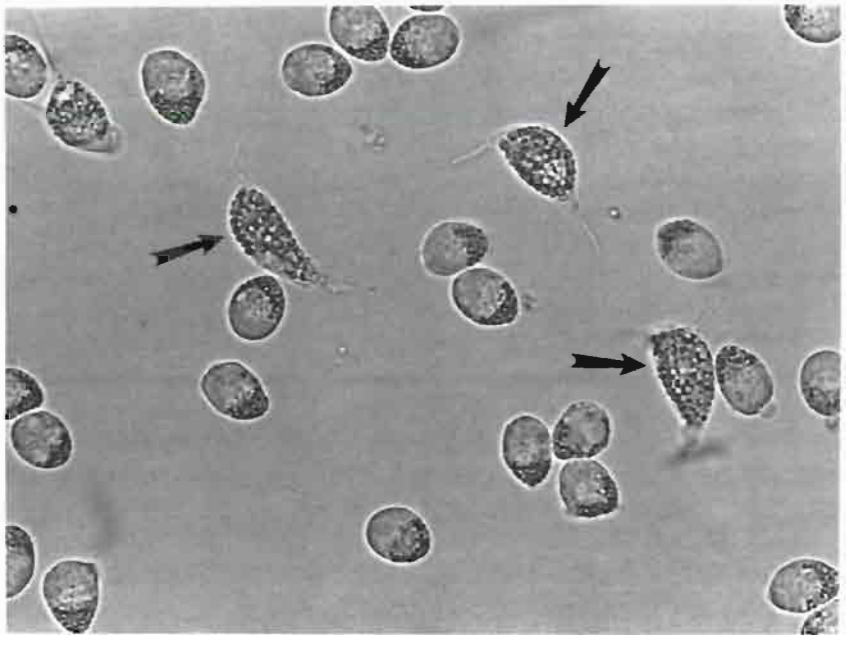

Fig. 3-37: Wet hemolymph smear of the large type of dinospores 24 hours after collection from an infected Tanner crab (slight lateral protrusion not shown); host granulocytes (arrows). × 2,000. (After Meyers and co-authors, 1987.)

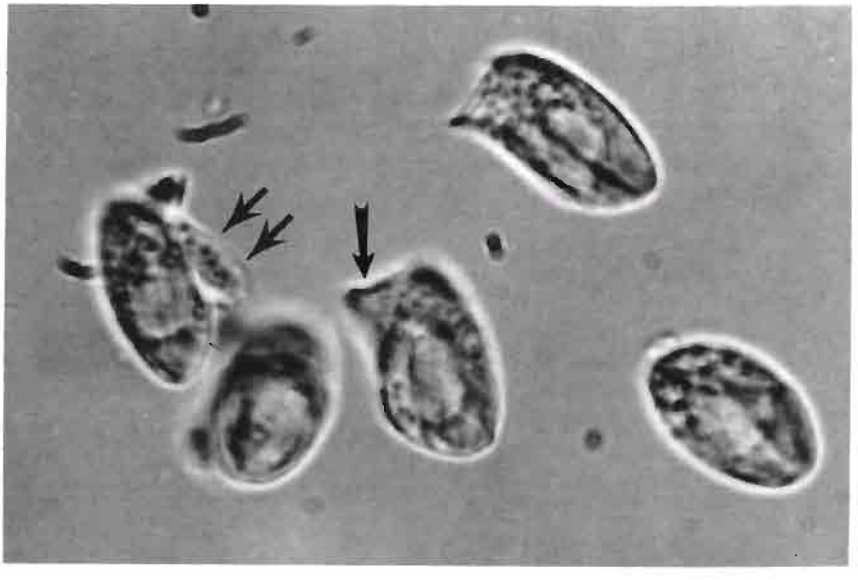

Fig. 3-38: Live dinospores from Fig. 3-37 after 11 days in hemolymph culture showing development of distinct lateral keel (double arrow) and beaked end (single arrow). × 3,300. (After Meyers and co-authors, 1987.)

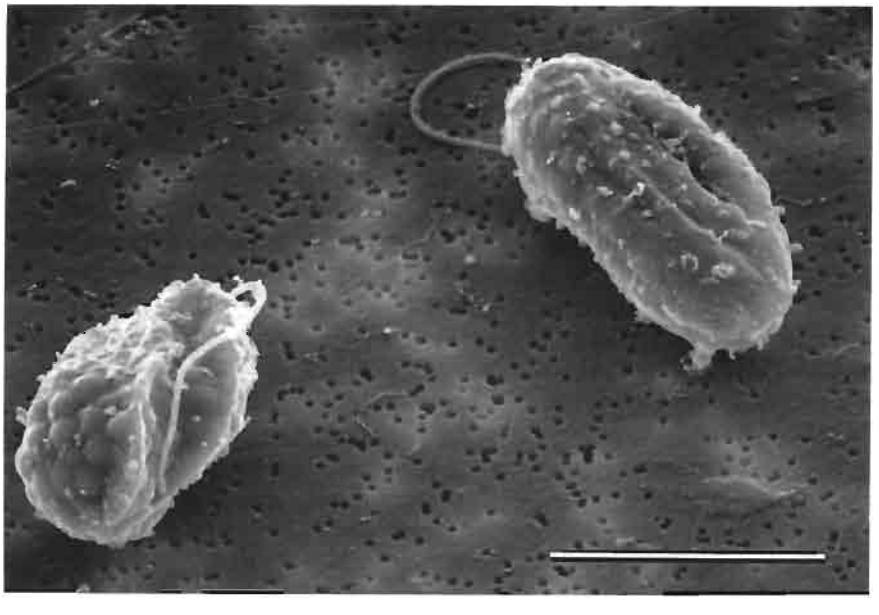

Fig. 3-39: Scanning electron micrograph of the large spore type soon after sporulation showing raised protuberances on the pellicle, lateral keel with groove and circumferential flagellum visible. Gold palladium, × 3,500. Bar = 10 μm. (Original.)

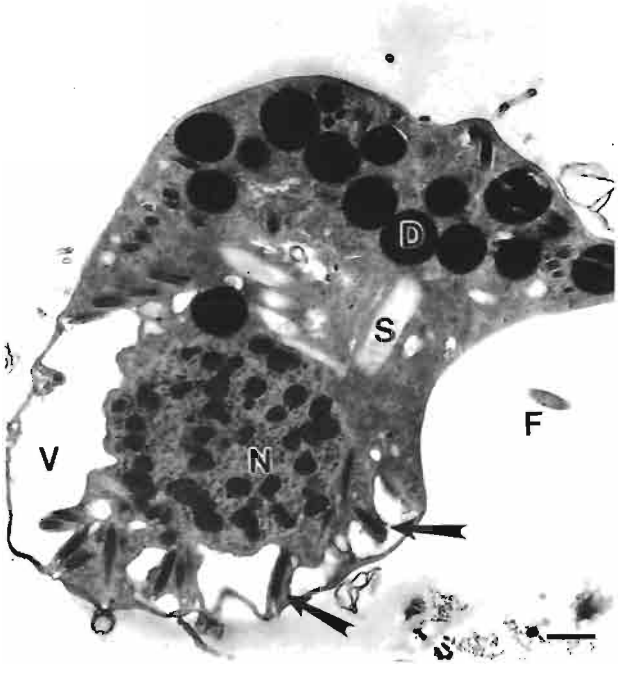

Fig. 3-40: Ultrastructure of the larger beaked dinospore after 10 days in hemolymph culture showing nucleus (N), electron dense bodies (D), trichocysts (arrows) connecting to pellicle surface via pores, flagellum (F), fibrillar polysaccharide-like material (S) associated with perinuclear space and endoplasmic reticulum and empty vacuoles (V). Uranyl acetate and lead citrate, × 6,500. Bar = 1 μm. (After Meyers and co-authors, 1987.)

Ellobiopsid parasites are believed to be dinoflagellates based on the discovery of flagellated spores (Galt and Whisler, 1970). A member of this group is *Thalassomyces californiensis* that parasitizes the Pacific shrimp *Pasiphaea emarginata* by penetrating the eyestalk and producing a root system that invades the brain, optic nerve and ventral nerve cord. This results in no apparent histopathological effects (Collard, 1966). Galt (1971) examined the development of *T. marsupii* parasitizing the amphipod *Parathemisto pacifica* from the San Juan Islands in Washington (USA). The root portion of the parasite eventually attaches to the 4th or 5th ganglion of the nerve cord and later an external stalked reproductive grape-like cluster penetrates through the ventral surface of the host. Protospores are released from the cluster and fall to the substrate where each spore develops 2 flagella and begins swimming. The motile spores are thought to be infectious but attempts to infect *P. pacifica* by their injection or by water exposure to the spores were unsuccessful (Galt, 1971). Both sexes of the amphipod are equally parasitized with overall prevalences of less than 0.2% that show no seasonality. Additional histopathological studies indicated the parasite causes hypertrophy of the ventral nerve cord with disorganization of nerve fibers. An associated host response of hemocyte infiltration around the parasite root and a thickening of the host cuticle at the point of protrusion of the reproductive structure were also noted (Hibbits — formerly Galt, unpubl., in Sparks, 1985).

Agents: Flagellata and Ciliophora

Members within these 2 groups of Protozoa are not generally regarded as serious crustacean primary pathogens. Some are symbionts (Vol. I, p. 18) turning into opportunistic pathogens when environmental conditions become poor for the host. Others are capable of tissue invasion but only through previous wounds in the cuticle.

A *Leptomonas*-like amoeboflagellate organism was reported in 64 % of 139 moribund and dead mysid stage larvae of brown shrimp *Penaeus aztecus* obtained from a hatchery in 1974 (Couch, 1978). The organism proliferated within the hemocoel and consequently infiltrated all vital organs and appendages. However, these shrimp also had concomitant viral and ciliate protozoan infections which severely compromised the hosts for subsequent flagellate infection and contributed to the overall mortality. This disease has not been reported in feral shrimp populations.

Zoothamnium sp., a stalked sessiline peritrichous ciliate, is a common ectocommensile on the gills of wild penaeid shrimp and can cause mortality of brown and white *P. setiferus* shrimp under intensive culture conditions (Overstreet, 1973; Lightner, 1975; Couch, 1978; Foster and co-authors, 1978). The predisposing factors for disease are heavy ciliate infestation with low dissolved oxygen and/or salinities (Johnson and co-authors, 1973).

The ciliate *Parauronema* sp. is another opportunistic agent parasitizing the hemocoel of protozoeal, mysid and juvenile stages of brown shrimp sustaining mortality at a commercial shrimp hatchery (Couch, 1978). However the role of this protozoan in causing the shrimp mortality was complicated by simultaneous host infections by a baculovirus and flagellate protozoan.

Synophyra sp., an apostome ciliate, is an ectocommensile which assumes an endoparasitic existence in both crabs and shrimps along the coasts of southeastern United States and France (Chatton and Lwoff, 1926, 1927, 1935; Johnson and Bradbury, 1976). *S. hypertrophica* is pathogenic for crab hosts of the genera *Macropipus* and *Ovalipes* in which trophonts and tomonts encysted in the gills cause tissue destruction and severe melanization of the cuticle and underlying tissues with loss of entire gill lamellae. Similar destruction of the carapace by this parasite has also been noted in postlarval juvenile crabs. An unidentified apostome reported by Couch (1978) causes a similar blackened gill condition in up to 30 % of penaeid shrimp along the northern Gulf of Mexico during spring and summer.

Other peritrichs such as *Lagenophrys callinectes* (Couch, 1966) and the suctorian *Ephelota* (Couch, 1978; Gucatan and co-authors, 1979) are gill and body ectocommensiles of blue crab and shrimp, respectively, that are potential stressors contributing to crustacean mortality in culture and holding situations. *L. callinectes* has been associated with blue crab mortality in floats or shedding tanks when crab densities were high (Couch, 1966).

An important facultative pathogen of marine crustaceans is *Paranophrys maggii,* a holotrichous ciliated protozoan, found in *Carcinus maenas* (Poisson, 1930) and *Cancer pagurus* (Bang, 1962; Groliere and Leglise, 1977) on the Brittany coast, France; in Dungeness crabs from Oregon (Armstrong and co-authors, 1981) and Washington, USA (Sparks and co-authors, 1982); in the marine isopod *Gnorimosphaeroma oregonensis* near Afognak Island, Alaska, USA (Hibbits and Sparks, 1983); and in adult and larval

American lobsters *Homarus americanus* from St. Andrews New Brunswick, Canada (Aiken and co-authors, 1973).

A *Paranophrys*-like ciliate has also been observed infecting the hemolymph and tissues of a moribund golden king crab *Lithodes aequispina* and a blue king crab *P. platypus* (Figs. 3-41, 3-44), held captive in flowing seawater for at least 2 months. Both

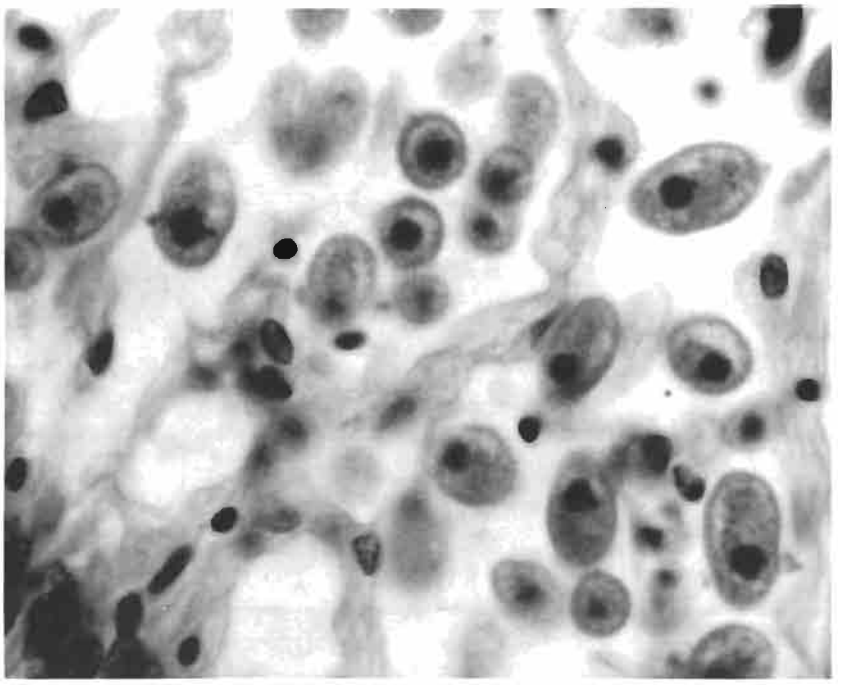

Fig. 3-41: *Paranophrys*-like ciliate infiltrating connective tissues beneath the epidermis of a *Paralithodes platypus* dying of the disease in the laboratory. Hematoxylin and eosin, × 3,800. (Original.)

king crabs had external injuries to the carapace and died; the blue king crab was also parasitized by the rhizocephalan barnacle *Briarosaccus callosus* (T. R. Meyers, unpubl.). Most investigators consider *Paranophrys* sp. a secondary invader requiring a primary portal of entry, usually a wound penetrating the exoskeleton. Consequently, this parasite can be responsible for mortalities in various species of Crustacea when held captive for periods of time since invariably some exoskeleton wounds occur from handling and aggressive crustacean behavior.

Once inside the crustacean host the ciliate invades the circulatory system proliferating profusely and consuming hemocytes as well as cells of vital organs and tissues. Massive destruction and dysfunction of major organ systems and hemocytopenia are the probable causes of host death (Sparks and co-authors, 1982). *Paranophrys* sp. has a prepatent period of 9 to 26 days after entry into a wound and can produce mortalities of up to 40 % in captive Dungeness crabs (Armstrong and co-authors, 1981). Typical clinical signs of infection are lethargy, anorexia and ataxia followed by death. Diagnostic features of the disease include cloudy hemolymph containing myriads of motile ciliates having a characteristic long trailing cilium. Examination of moribund Dungeness crabs infected with

Paranophrys sp. has shown microscopic tissue pathology characterized by nearly complete destruction of peripheral and tissue hemocytes accompanied by massive tissue infiltration of the ciliates with severe systemic necrosis of major organs (Sparks and co-authors, 1982). Although crowding of crustaceans in artificial holding facilities and reduced water quality are generally predisposing to infection by this parasite, in one case the disease has been observed in a Dungeness crab in the field (Sparks and co-authors, 1982) and also in feral *Carcinus maenas* (Poisson, 1930).

Agents: Amoebae

As a group, amoebae play a very minor role as crustacean pathogens with only one major agent reported. *Paramoeba perniciosa* is an important cause of mortality in the blue crab from Chesapeake and Chincoteague Bays and along the coast of North Carolina (USA) causing Gray Crab Disease. Other locations for the disease include South Carolina and Georgia (Sprague and Beckett, 1966, 1968; Sprague and co-authors, 1969). The parasite has one large vesicular nucleus with a large central endosome and an accessory body or Nebenkorper that is a diagnostic feature. Single amoebae generally come in small (3 to 12 μm) and large (15 to 35 μm) size classes in severe infections, but otherwise can occur in gradations of both. The clinical signs in infected crabs account for the name of the disease as the ventral carapace and hemolymph usually take on a gray coloration in severely infected individuals. Parasitized crabs in advanced stages of the disease are lethargic and die easily with hemolymph that does not clot and is clouded with myriads of the protozoan that replace all hemocytes.

Transmission of the parasite is considered to be by consumption of infected crab tissues, although some experiments have failed using this route (Couch, 1983). When ingested, *Paramoeba perniciosa* probably enter the new crab host through the midgut epithelium (Sparks, 1985). Experimental transmission of the parasite by injection suggests that another possible portal of entry is through external wounds in the carapace of both hard or recently molted soft-shelled crabs. Of note is that the parasite predominantly infects hemal spaces and connective tissues, thus occurring in the peripheral hemolymph only in terminal cases. Consequently, diagnosis of early disease would be missed using hemolymph smears and must be done using tissue sections (Johnson, 1977c).

Epizootics commonly occur in the spring during May and June (Couch and Tubiash, 1967; Sawyer, 1969) with parasite prevalences up to 35% that taper off by mid summer and fall. An exception reported by Couch (1983) occurred during October through February in Chincoteague Bay, Virginia (USA) where prevalences of infected crabs were as high as 20%. Generally, by winter hemolymph infections in hibernating crabs are not detectable and whether new infections start up each year or low grade tissue infections in these dormant crabs provide a reservoir for the disease is not known (Sawyer, 1969). In terminal cases of the disease the parasite invades all hemal spaces throughout the crab causing major tissue damage and lysis in adjacent connective tissues, hematopoietic tissues, the Y organ, epidermis and skeletal muscle. Hemocytes are also destroyed and virtually obliterated from the circulation in the most severe cases (Sawyer and co-authors, 1970; Johnson, 1977c). In light to moderate infections host response can consist of hemocyte infiltration and phagocytosis with encapsulation occurring less commonly. In some cases degenerating and lysed amoebae may predominate either free and/or phagocytized within hyaline hemocytes (Johnson, 1977c). In these instances almost all amoebae

may appear to be degenerate, possibly due to a humoral response. Despite this apparent effective dispatch of the parasite by the host some of these crabs still may die from apparent toxicity of the degrading masses of the amoebae. Physiological studies of infected crabs have shown a significant depletion of serum protein, hemocyanin and glucose indicating probable causes for loss of blood clotting ability, insufficient oxygen transport to the tissues and inability to compete with the parasite for nutrients (Pauley and co-authors, 1975). Crabs probably die from respiratory failure, organ dysfunction and nutrient deficiency.

Although potentially a devastating disease, the sporadic occurrence of Gray Crab Disease has not had a major economic impact upon the eastern US blue crab fishery. Other crustacean hosts for *Paramoeba perniciosa* include the rock crab *Cancer irroratus* and the American lobster *Homarus americanus* but the effects of parasitism have not been reported (Sawyer, 1976; Sawyer and MacLean, 1978).

Agents: Sporozoa

The sporozoan group of Protozoa is large and diverse with all members living as obligate histozoic intercellular or intracellular parasites infecting many different animal phyla. In Crustacea the Microspora are among the most common and serious of pathogens affecting many different host species. Other members of the Sporozoa such as gregarines, eugregarines and coccidians, though common, are not particularly harmful to their hosts. The gregarines, *Nematopsis ostrearum* and *N. prytherchi,* form innocuous spores in the oyster *Crassostrea virginica* (Sprague, 1949) which when eaten by their respective definitive crab hosts, *Panopeus herbstii, Eurypanopeus depressus* or *Eurytium limosume* (mud crabs) and the stone crab *Menippe mercenaria* release numerous sporozoites into the midgut. Trophozoites develop and attach to midgut epithelial cells and undergo reproduction with eventual gametogenesis and release of sporozoites from ruptured gametocysts into the environment along with feces causing no apparent harm to the host except a nutritional drain. Experiments comparing survival of parasitized and nonparasitized crabs showed slightly higher survival rates in nonparasitized animals (Sprague and Orr, 1955) but the difference was not considered significant. *Cephalolobus penaeus* (Kruse, 1959) is a eugregarine that undergoes a similar reproductive cycle in the pyloric stomach of brown and pink *P. duorarum* penaeid shrimps with no adverse effects on the hosts. Localized destruction of connective tissues surrounding the gut is caused by shizogonic reproduction of the coccidian *Aggregata eberthi* Labbe in *Macropipus depurator* from coastal waters of Europe.

However, the damage is not significant as to endanger the life of the crab host (Dobell, 1925). The gamogonic and sporogonic phases of the parasite cycle occur within the cuttlefish *Sepia officinalis* and typically cause little or no destruction of host tissues. Haplosporidians in crustaceans are few and, although some appear to have virulent potential, their prevalences are low and occurrences rare. An example of such is a *Haplosporidium*-like parasite found severely infecting moribund blue crabs from Chincoteague Bay, Virginia and coastal North Carolina, USA (Newman and co-authors, 1976). Although uninucleate and plasmodial stages filled hemal spaces throughout the affected crabs, the importance of the disease to crab populations appears insignificant since only 5 infected individuals were found. Much less virulent are the abundant spores of the

parasite *Haplosporidium louisiana* recorded in the gut wall of a single mud crab *Panopeus herbstii* in Louisiana, USA (Sprague, 1970). Despite their abundance, the only effect on the crab was a brown discoloration of the infected tissues. *Urosporidium crescens* does not infect crustaceans but instead is a hyperparasite of the microphallid trematode metacercariae *Microphallus basodactylophallus,* found encysted in the blue crab. However, because of the dark pigmentation given to the worms by the hyperparasite, the affected crabs are known as 'pepper' or 'buckshot' crabs and are aesthetically displeasing for human consumption (Overstreet, 1978).

Microsporidians are the most common parasites in Crustacea causing severe pathology in some host species (Sindermann, 1971). Well over 140 species of microsporidians are reported to parasitize all orders of Crustacea, consequently only representative parasites and the more important pathogens have been included here. A 2 volume review of microsporidian biology was published by Bulla and Cheng (1976, 1977), with a systematic survey of this group presented by Sprague (1977). The following discussion compares and contrasts the varying pathologies and effects of these parasites on different crustacean hosts. Although the life cycles of all crustacean microsporidians have not been completely established, the following generalized description (Reichenbach-Klinke and Elkan, 1965) will suffice in understanding further discussion points.

All microsporidians are intracellular parasites and produce spores as infectious stages. A host ingests the spore which attaches to a cell in the intestine of the host by extruding a single polar filament. The infective unit or amoebula is released from the spore and invades an epithelial cell where it divides forming schizonts. Schizonts eventually invade cells of the specific target tissues of the host where reproduction is completed and spores are formed within sporoblasts or pansporoblasts. The sporoblast produces varying numbers of spores depending upon the genus of microsporidian. In some cases the parasite replicates within the host cell nucleus rather than the cytoplasm. Infected cells and their nuclei may respond to the infection by marked hypertrophy in which the enlarged cells become cysts or xenomas containing myriad numbers of the parasite. Spores are released from the living host or after death, and are ingested by a new host to begin the cycle again. In amphipod hosts another major route of parasite transmission is transovarian (Bulnheim, 1975). Classification of microsporidia is determined by the number of spores produced within the spherical sporoblast. As examples, members of the genus *Nosema* have 1; of *Glugea,* 2, of *Thelohania,* 8, and of *Pleistophora,* 16 to 100.

Not all microsporidia are serious pathogens as seen in the brackish-water amphipod *Gammarus duebeni* infected with *Thelohania herediteria.* The parasite infects ovaries and muscle tissue of females but not males and is found only in the estuary of the Elbe River in the Federal Republic of Germany (Bulnheim, 1975). Infection causes no gross abnormalities but oocyte infection results in infection of developing embryos following egg fertilization after which predominantly female offspring are produced. The development of the parasite is believed to inhibit differentiation of the androgenic gland from which maleness is derived in Malacostracans (Bulnheim, 1975). No other significant negative effects concerning tissue pathology, survival, growth rate, fecundity, molting frequency, etc. have been observed associated with *T. herediteria* in *G. duebeni.* A similar sex-determining effect occurs with *Octosporea effeminans* infecting the ovaries and adipose tissues of *G. duebeni* occurring in the Elbe River estuary and the Baltic Sea (Bulnheim, 1975).

Microsporidians infecting penaeid shrimps are considerably more pathogenic causing 'cotton' or 'milky' disease characterized grossly by an opaque white abdomen. Infected shrimp are much less resistant to environmental stressors such as overcrowding and tend to remain in the estuaries rather than go offshore to reproduce (Overstreet, 1973). The most common microsporidian in the Gulf of Mexico and as far north as Georgia is *Ameson nelsoni* infecting 6 species of penaeids. Spores of this parasite are found replacing abdominal muscle fibers producing a whitened abdomen with deeply pigmented bluish-black chromatophores in the cuticle dorsally and dorso-laterally. Grossly, this discoloration appears similar to *Pleistophora* sp. infections occurring in brown, white and pink shrimp from Texas, Louisiana, Mississippi and Florida (USA). However, the pigmentation is more pronounced in *Pleistophora* sp. which also infects the heart, hepatopancreas, gills and stomach (Overstreet, 1973). Destruction of muscle bundles is not complete, as can be with *A. nelsoni,* since less than half are replaced by spores in *Pleistophora* sp. infections with atrophy and fibrosis as sequellae.

Thelohania duorara most commonly infects pink shrimp (Iversen and Manning, 1959) but can also be found infecting Brazilian *Penaeus brasiliensis* white and brown shrimps from Florida to Mississippi (Overstreet, 1973). Despite the usual cotton shrimp appearance, pansporoblasts and spores of this parasite occur in the interstices and in the outer surfaces of muscle bundles, hence complete destruction of muscle bundles does not occur. Invasion of connective tissue, muscle of the midgut and hindgut and hemocoels surrounding the hepatopancreas and hematopoietic organ also occurs. One of the more important penaeid microsporidians is *Agmasoma penaei* possibly responsible for an epizootic castration of 90% of the white shrimp along the Louisiana coast (USA) in 1919 (Viosca, 1945; Sprague, 1970). This parasite infects a variety of tissues and organs including gills. Infection of abdominal musculature involves superficial and internal fibers that can result in complete destruction in severe cases. Grossly, the cotton shrimp appearance prevails ranging from 1 cm whitened areas to complete involvement of the abdomen and cephalothorax (Lightner, 1975; Kelly, 1979).

Other microsporidians occurring in marine palaemonid, crangonid and pandalid shrimps predominantly invade abdominal musculature causing the typical muscle opacity and destruction. These cotton shrimp agents can also be serious pathogens as exemplified by *Indosporus octospora* infecting *Palaemon rectirostris* and *P. serratus* from the coast of France, and *P. serratus* and *P. elegans* off the coast of England and in the Black Sea of Romania, respectively. Parasite infections first appear in March eventually causing castration in females and apparently death of all infected animals by late fall (Sprague, 1970; Overstreet and Weidner, 1974).

Similar microsporidians occur in anomuran, branchyuran and lithodid crabs. Spores of *Thelohania paguri* were reported infecting the anomuran crab *Eupagurus bernhardus* within the abdominal spaces between the viscera rather than in the musculature (Perez, 1927). This parasite was reported from France but little is known about it since nothing has been reported subsequently.

A condition caused by microsporidian infection known as 'cottage cheese' disease occurs in lithodid king crabs from Bristol Bay and the Bering Sea off the coast of Alaska, USA (Sparks and Morado, 1985). Two different undescribed species of *Thelohania* cause this disease in both red king *Paralithodes camtschatica* and blue king *P. platypus* crabs in which massive numbers of spores appear as white curdy material invading all major

visceral organs, particularly the hepatopancreas, ovary, tegmental glands and the wall of the digestive system. Similar gross pathology, mostly confined to musculature including the heart, occurs in the golden king crab *Lithodes aeguispina* when infected by an unidentified species of microsporidian in the family Nosematidae (Sparks and Morado, 1985). These microsporidian infections are obviously lethal but so far have only been found at low prevalences ranging from 2 to 10%. There are several microsporidians infecting brachyuran crabs particularly the blue crab *Callinectes sapidus*, the green crab *Carcinus maenas* and a porcelain crab *Petrolisthes armatus*. There is not much information on many of these microsporidians which appear to be of minor importance due to their rarity or infection of a non-commercial species of crab.

Ameson michaelis is perhaps the most important and widespread microsporidian infecting the blue crab in low prevalences on the Atlantic and Gulf coasts of the United States. The parasite sporulates in muscle bundles throughout the body causing lysis of the myofibrils and the expected opaque and chalky gross appearance of the infected musculature. Severely infected crabs are lethargic, and often are found in shallow water with heavily fouled carapaces; eventually they die (Weidner, 1970; Overstreet, 1977). Infected crabs do not survive handling and sustain various other physiological abnormalities due to parasitism including a terminal hypoglycemia (Overstreet and Whatley, 1975; Findley and co-authors, 1981). Despite the information at hand, the economic importance of this parasite in blue crab populations still needs to be established.

DISEASES CAUSED BY METAZOANS

Agents: Helminthes

Although there are many host-parasite relationships regarding helminths and marine crustaceans there are few in which the pathological effects have been adequately described. Many such cases of symbiosis are basically harmless but undoubtedly some worms are capable of severely compromising or even killing their crustacean hosts. The following discussion mostly includes relations known to be pathogenic.

Turbellarians of the Rhabdocoela order are considered ectocommensales; however, *Kronborgia amphipodicola* is parasitic, infesting the body cavity of some ampeliscid amphipods and often kills its host (Christensen and Kanneworff, 1965). When first infesting an amphipod the larva develops in the hemocoel causing atrophy of the gonads and castration. Other similar species including *K. caridicola* infest shrimps such as *Eualus machilenta, Lebbeus polaris* and *Pacsiphaea tarda* (Kanneworff and Christensen, 1966).

Encysted digenetic trematode metacercarial stages have been reported in several marine crustacean hosts, including: penaeid shrimps from southeastern United States with *Opecoeloides fimbriatus* in the musculature and connective tissues (Overstreet, 1973); Dungeness crabs *Cancer magister* from Washington State (USA) with microphallid-like metaceriae in the nerves, brain, thoracic ganglion and lamina ganglionaria of the eye (Sparks and Hibbits, 1981; Meyers and co-authors, 1985); *C. magister* from Alaska (USA) with metacercariae in the connective tissues (Fig. 3-42); crangonid shrimp *Crangon alaskensis,* from Washington State with unidentified metacercariae in the nervous tissues (Morado and Sparks, 1983); several species of Gulf coast crabs with larvae of *Spelotrema* or *Microphallus* in the musculature, hepatopancreas and gonads (Overstreet, 1983); blue

Fig. 3-42: Trematode metacercaria encysted within connective tissues near the hepatopancreas of *Cancer magister* from southeast Alaska. Hematoxylin and eosin, × 400. (Original.)

crabs *Callinectes sapidus* from Rhode Island (USA) with unidentified microphallids within nervous tissues of the musculature and hepatopancreas (Melzian and Johnson, 1988). The potential pathogenicity of microphallid metacercariae in crustaceans was demonstrated (Stunkard, 1957) by exposing small green crabs *Carcinus maenas* to periwinkle molluscs *Littorina obtusata* or *L. saxatilis* releasing cercariae of *Microphallus similis*. The crabs became infested with thousands of metacercariae encysted in all tissues, especially the hepatopancreas; they died 10 to 20 days later. Although further histopathological studies were not performed to determine the definitive cause of death, and exposure levels were higher than what might occur in nature, clearly the worm was virulent for this crab host and possibly for other host species as well. Those metacercariae encysting within shrimp and crab nervous tissues cause compaction and necrosis of the neuropile and a hemocytic response in the blue and Dungeness crabs (Sparks and Hibbits, 1981; Morado and Sparks, 1983; Meyers and co-authors, 1985; Melzian and Johnson, 1988). Considering the importance of the tissues affected, severely parasitized hosts could become debilitated through disruption of nervous innervation to major organ systems as suggested by observed ataxia in one severely parasitized Dungeness crab (Sparks and Hibbits, 1981).

Marine Crustacea are not common hosts for monogenetic flukes but such relations do occur, primarily involving crustacean symbiotes of fish. Whether the crustacean is a true host for the worms or whether it acts as a substrate for dissemination to other fish hosts is a controversial subject. Regardless, the worms do no overt harm to the crustacean hosts.

Examples include: the parasitic isopod *Cymothoa excisa* which shares the monogenean *Choricotyle aspinachorda* with its host, the pigfish *Orthopristis chrysoptera; Udonella caligorum* which infests a variety of potentially parasitic copepods but most commonly caligids such as *Caligus praetextus* found on a variety of fish hosts (Overstreet, 1983). *U. caligorum* can also be found on fish gills and is equivocal regarding its true status as a monogenean due to significant morphological differences (Overstreet, 1983).

Agents: Cestoda

A common trypanorhynch cestode plerocercoid found in the 3 major commercial penaeid shrimp species from the Gulf of Mexico is *Prochristianella penaei.* The definitive host for the worm is the southern sting ray *Dasyatis sabina.* Prevalences and infection intensities reported include up to 94.4 % and 14.7 worms per white shrimp, 90.6 % and 6.2 worms per brown shrimp, and 97 % and 42 worms per pink shrimp (Kruse, 1959). Worm prevalences and intensities increase as the host increases in body size, but level off in brown shrimp reaching 13 to 15 mm carapace length and at 18 mm for white shrimp (Aldrich, 1965). Infection is more prevalent in juvenile shrimp from estuaries than in adults caught offshore (Aldrich, 1965). Obviously, the worm does not cause significant damage in the shrimp hosts, otherwise such high prevalences and intensities would cause massive mortality in some areas. Although some shrimp mortality could result from heavy infestations, they have not been recognized. The larvae are commonly found within the hepatopancreas of the host where they are thickly encapsulated by hemocytes and fibroblasts, and eventually destroyed. The centers of these granulomata are necrotic and melanized, and some adjacent hepatopancreatic tubules may be destroyed by granuloma formation. Worms migrating to the hemocoel apparently survive and are lightly encysted with significant melanization of the cyst wall usually on the side adjacent to the hepatopancreas (Sparks and Fontaine, 1973). It has been suggested that not penaeid shrimp, but rather callianassid mud shrimp, are the primary hosts for these trypanorhynch larvae. The latter are the preferred food of the sting ray definitive host and the host response in the mud shrimp is much less (Overstreet, 1983). Other similar trypanorhynchid larvae have been described from other penaeid shrimp species (Overstreet, 1983).

A single specimen of an unidentified trypanorhynchid plerocercoid was found loose within the hepatopancreas of a red king crab taken from southeast Alaska waters. No apparent host response was evident and the effect on the king crab host appeared to be harmless. This apparently was a rare occurrence since necropsies have been performed on many Alaskan king crabs without observing this worm (Meyers, unpubl.).

Coracidia of the cestode *Spirometra mansonoides* can infest up to 70 to 80 % of copepod populations of *Cyclops vernalis* causing developmental retardation by inhibition of molting and castration (Mueller, 1965). Interestingly, successive infestations of a stock result in lower parasite prevalences and host resistance which may include destruction of the oncosphere in the gut lumen or hemocoel (Mueller, 1965).

Agents: Nematoda

Marine Crustacea can also sustain juvenile stages of nematodes which generally appear to cause no major harm to the host. Most of these worms belong to the genus

Thynnascaris (originally referred to as *Contracaecum;* Norris and Overstreet, 1976) which are reported from mysid, pandalid and penaeid shrimps. A single female specimen of *Pandalus borealis* from Vancouver, British Columbia (Canada) revealed an unusual infestation of adult *C. aduncum* with large numbers of worms present in the hemocoel and embedded within the abdominal musculature. The effect on the host was an apparent inhibition of egg production as all other females collected from the same site were ovigerous (Margolis and Butler, 1954). The shrimp is not the normal definitive host for this worm, juveniles of which have been found in *P. borealis* from the Barents Sea. *Thynnascaris* sp. also have been reported during summer in up to 31 % of brown and white shrimp from Mississippi (USA) with mean intensities of 2.8 worms host^{-1}. Shrimp over 160 mm harbor 57 % of the worms (Overstreet, 1973) occurring in the cephalothorax, mostly in the hepatopancreas. No apparent pathology or mortality have been reported associated with these infestations. Second-stage juveniles of *C. spiculigerum* were used to experimentally infest the copepods *Cyclops vernalis* and *Tigriopus californicus* producing 6 to 10 worms host^{-1}. The infested copepods died within 1 to 6 days; during this period the worms increased in size from 329 μm to 350 μm but did not molt (Huizinga, 1966). Although the infestations were clearly lethal, the resulting pathology has not been described. A different spiruroid juvenile nematode, *Ascarophis* sp., infested up to 25 % American lobsters *Homarus americanus* off Cape Cod, USA (Uzmann, 1967). A mean number of 3.5 worms host^{-1} were coiled in cysts in and on the anterior wall of the rectum, but no pathological effects were specified. Juveniles of this same nematode genus also infest the crab *Hemigrapsus oregonensis* and may potentially increase mortality and reduce somatic growth (Poinar and Kuris, 1975). Adult worms of yet another nematode, *Rhabditis ocypodis,* occur on the gills and egg masses of the ghost crab *Ocypode quadrata,* and larvae of the same or similar species have been found unencysted within the striated muscle (Overstreet, 1983). Some negative effects on the host are likely but this possibility has not been examined.

Agents: Acanthocephala

Amphipods are one of the more common marine intermediate hosts for larval acanthocephalan symbiotes. The acanthor stage of *Leptorhyncoides thecatus* penetrates the intestinal wall of its amphipod host *Hyallella knickerbockeri* after having been ingested and encysts within the hemocoel. Should the cyst rupture, the larval parasite would be destroyed by a host foreign-body response from infiltrating hemocytes and walled off (DeGuisti, 1949). Cystacanth stages of *Tegorhynchus furcatus* and *Dollfusentis chandleri* also infest various marine and estuarine amphipods from Mississippi (USA), the former worm causing displacement of major visceral organs in the hemocoels of *Lepidactylus* sp. and *Haustorius* sp. Parasitized hosts are typically larger than non-parasitized individuals (Buckner and co-authors, 1978). Larval forms of *Corynosoma* sp. parasitize intestinal wall, heart and body musculature of the American lobster *Homarus americanus;* they are assumed to be a cause for low-level mortality (Montreuil, 1954).

Cystacanths of *Polymorphus botulus* commonly infest *Carcinus maenas* in England (Rayski and Garden, 1961). Cystacanths of a similar species, *P. major,* are found in prevalences of up to 37 % in *Cancer irroratus* from Maine appearing singly, or in clusters of as many as 15 white ovoid cysts attached to the midgut. A host response consisting of connective tissue proliferation and eosinophilic granulocyte infiltration occurs at the sites

of cyst attachment (Schmidt and MacLean, 1978). Sparks (1987) has also reported an unidentified cystacanth occurring singly in 15 of 805 *Paralithodes camtschatica* from Norton Sound, Alaska. The larvae were encysted just beneath the basement membrane of the midgut causing some compression of adjacent tissues but no other pathological effects or obvious host response. Other apparently innocuous occurrences of acanthocephalan larvae in other marine crustaceans also have been reported (Overstreet, 1983).

Agents: Nematomorphs

Nematomorphs, or horse hair worms, contain 2 orders one of which consists of pelagic marine worms with juveniles that parasitize crustaceans either following ingestion or by actively penetrating the host. *Nectonema agile* infests the shrimp *Palaeomonetes vulgaris* in Massachusetts (USA) waters causing castration (Born, 1967).

Agents: Nemertea

Members of this helminth group are phylogenetically more advanced than the previously discussed helminths and most are free living. However, certain members of the genus *Carcinonemertes* are important symbionts which predate on eggs of different marine crustaceans causing considerable loss of fecundity. The taxonomy of these worms is still a matter of discussion, but currently *C. errans* is considered host-specific for *Cancer magister*. Other carcino-nemertean egg predators on the crab species *Callinectes sapidus, Carcinus maenas,* and *Cancer anthonyi,* are considered to be *C. epialti* until further classified (Wickham, 1978). The nemertean on *Paralithodes camtschatica* and *Chionoecetes bairdi* has been named *Carcinonemertes regicides* n. sp. (Shields and co-authors, 1989). The recently reported egg predator of *Homarus americanus* has been placed in the genus *Pseudocarcinonemertes* (Aiken and co-authors, 1983). The actual number of different worm species in the genus *Carcinonemertes* is presently 6, one of which contains two subspecies (Shields and co-authors, 1989). However, host specificities are still under investigation (Kuris and Wickham, 1987).

Typically, juvenile *Carcinonemertes errans* occur on both male and female *Cancer magister* on the surfaces of arthrodial membranes, crevices of the eye sockets, bases of eye stalks, and setae-lined surfaces of the carapace. In male crabs, worms occur at the base of copulatory organs which may facilitate worm transfer to egg-bearing females (Wickham, 1980). Dormant juvenile worms may be sustained on adult crabs for months or years while awaiting host oviposition; they actively absorb dissolved primary amines leaking into the seawater from uncalcified portions of the host exoskeleton (Wickham, 1980; Roe and co-authors, 1981). Within 1 to 2 days after crab oviposition, juvenile worms migrate to the clutch and, using a piercing stylet, begin feeding on eggs at a rate of 0.5 to 1.5 eggs day^{-1} resulting in an average of 70 eggs eaten per worm during crab brooding. Indirect egg mortality may also result from the presence of feeding nemerteans that produce organic fouling of the sponge from worm feces and yolk leaking from punctured eggs, subsequently providing a substrate for fungal saprophytes. Worms grow from 0.5 to 10 mm in size and begin depositing their orange egg ribbons within the host clutch after 65 to 70 days.

The nemertean eggs hatch near-synchronic with the host eggs, and larvae are planktonic for up to 8 months before settling onto a new host crab (Wickham, 1980). Egg

predation by *Carcinonemertes errans* has been considered the dominant biological control of *Cancer magister* densities in the depressed fishery of central California, USA (Wickham, 1979, 1980). Overfishing is credited for seriously reducing crab numbers in the presence of high worm densities. The result was concentration of worm predation on the remaining crabs, thus increasing both worm densities and consequent crab losses (Wickham, 1979). Compensatory increased fishing pressure continued into northern California, Oregon and Washington having a similar effect of declining catches and increased worm densities on crab egg clutches (Wickham, 1979, 1986).

Carcinonemertes errans also occurred on egg clutches of *Cancer magister* in southeastern Alaska in 83 % of the females examined, but at low average densities of 21.6 worms crab^{-1} or about 0.009 worms per 1000 crab eggs compared to an average of 14 per 1000 in California's epidemic fishery (Wickham, 1980; Shirley and co-authors, 1984). Males had less than 1 worm crab^{-1} in less than 6 % of the crabs examined (Shirley and co-authors, 1984).

Other recent studies indicate that *Carcinonemertes regicides* may have been responsible for egg mortality of up to 100 % in *Paralithodes camtschatica* populations around Kodiak Island and Cook Inlet (Alaska, USA). Egg mortalities of 42 % with worm densities of up to 199/1000 eggs or 250,000/host were found in king crabs from southeast Alaska (Wickham and co-authors, 1985; Shields and co-authors, 1989). Although it is likely that Alaskan *P. camtschatica* populations have sustained major brood mortality from this predator, the episodes seem to have been localized and brief, with declines in egg mortality and worm numbers evident by 1985 (Kuris and Wickham, 1987).

Agents: Hirudinea

Many of the Hirudinea or leeches use marine Crustacea for dispersal and for incubation of cocoons. It has been suggested that some may parasitize certain crustacean hosts (Daniels and Sawyer, 1975; Burreson and Allen, 1978) but this has not been substantiated by clear evidence nor has there been any indication of associated pathology. Consequently, leeches will not be considered further in this subchapter.

Agents: Mollusca

Pathogenic and/or parasitic molluscs on marine Crustacea are almost virtually unknown since members of this group are generally free-living (an outstanding exception are freshwater bivalve mussels parasitic on fishes). However, small hipponicacean prosobranchs in the Indo-Pacific may live as true parasites on several gonodactylid stomatopods causing detrimental effects. Reaka (1978) described such effects by *Caledoniella montrouzieri* infesting up to 13 % of mantis shrimp *Gonodactylus viridis* collected near Thailand. Generally each host had a male and a female attached to or between its pereiopods with egg capsules often covering the pleopods. Hosts were usually smaller than uninfested individuals and did not reproduce. The snails fed on gill tissues of the shrimp, and molts were retarded with the snails apparently able to reattach to postmolted hosts. Although gill pathology almost certainly must result from the snail parasites, none was reported.

Agents: Copepoda

Many different groups within the Arthropoda are parasites and disease vectors of great pathogenic importance in terrestrial and aquatic environments. The numbers become less when the subject area narrows to parasites of marine crustaceans.

Most copepods inhabiting crustaceans have little specialized modifications (Overstreet, 1983). But exceptions do occur as in the choniostomatids, represented by *Rhizorhina ampeliscae*. This copepod inhabits the gills of ampeliscid gammarids with only its egg sacs protruding (Bacescu and Mayer, 1960). Embedded in this manner within host gill tissues, large numbers of copepods could be expected to cause significant tissue damage. However, no reports exist to substantiate this possibility. Another member of this copepod group is *Spohaeronellopsis monothrix* parasitizing the myodocopid ostracod *Pasterope pollex* in Massachusetts, USA (Bowman and Kornicker, 1967). When infesting a female ostracod that has not yet produced eggs, this parasite is noted for its egg mimicry in which eggs deposited by the female copepod are bound in an ovisac resembling ostracod eggs. Consequently, female host ostracods retain the parasite eggs as if they were their own. The copepod feeds by sucking hemolymph from the host and inhibits ovulation. Any further effects on the ostracod host are not mentioned.

Members of the copepod family Siphonostomatoidae are parasitic on marine Crustacea, including *Nicothoe astaci* which feeds on the gills of *Homarus gammarus* (Mason, 1959). Severely infested host gills become seriously damaged and misshapened. Recently molted *H. gammarus* are apparently more susceptible to infestation and pathologic consequences. Although *N. astaci* is likely to exert a negative effect on *H. gammarus,* there are no reports to support damage to the European lobster industry.

Agents: Isopoda

Cheng (1973) reviewed life cycles of isopods of the suborder Epicaridea, most of which are parasites during their larval and adult stages; morphologically they resemble free-living isopods. Briefly, larvae known as epicaridium hatch from eggs and must attach to a copepod intermediate host on which the parasite feeds and undergoes 6 molts encompassing 2 different larval stages, the microniscus and the cryptoniscus.

In the family Bopyridae the cryptoniscus leaves the copepod host and enters the branchial cavity of a shrimp where it molts becoming a bopyridium. The first larva to enter becomes a female; all subsequent larvae become neotenic males that cling to the female between her abdominal pleopods. Should several larvae reach a host simultaneously, all become females but only one matures. A brood pouch forms in the female where eggs hatch and larvae are released into ambient seawater synchronously with the molt of the parasite and that of the host.

Other isopods belonging to the families Cryptoniscidae and Entoniscidae produce cryptonisci that respectively take up residence in brood pouches or penetrate deeply within the body of a decapod host usually directly below the carapace within the hepatopancreas. Consequently, the latter entoniscids are considered true endoparasites (Overstreet, 1983) occurring in the hemocoels of anomuran, brachyuran and rarely macruran crabs. Adult females of both families do not resemble bopyrid isopods but are sac-like with little or no segmentation. According to van Arman and Smith (1970) the bopyrid *Probopyrus* sp.

infested up to 50% of the shrimp *Hippolysmata wurdemanni* in Biscayne Bay (Florida, USA). The gravid female and her neotenic male occupy one side of the branchial chamber producing an obvious laterally protruding mass. The parasites do not come in direct contact with gill tissue, but are walled off by a cuticular sheath continuous with the host cuticle overlying the protruding lesion. The resulting pathology includes compression atrophy of host gill and muscle tissues impinged upon by the parasite mass with thinning and hyperpigmentation from increased chromatophores of the cuticle covering the parasites (van Arman and Smith, 1970).

Other bopyrids, such as *Probopyrus pandalicola,* parasitized more than 1 to 5% (depending on season) of the grass shrimp *Palaemonetes pugio* in Aurora, North Carolina (USA). As with most bopyrid infestations, ovarian maturation of the female host is prevented. In *P. pandalicola* this may be due to nutrient depletion since the parasite may ingest up to 25% of the host's hemolymph per day (Walker, 1974, 1977). Testes in males are not affected (Beck, 1980). Another bopyrid, *Munidion irritans,* forms a similar lateral protrusion of a branchiostegite on the galatheid decapod *Munida iris.* The dorsal convex surface of the female parasite directly contacts gill filaments of the host causing a depression and tissue necrosis (Bursey, 1978). Necrotic areas of the gill are infiltrated by host hemocytes, connective tissue fibers become thickened and melanized nodules are formed. The cuticle overlying the parasite appears normal while the epidermis and underlying connective tissue are thickened and 'spongy' (Bursey, 1978). This condition is likely due to mechanical irritation from penetration of the parasite's mandibles into the blood sinuses of the branchiostegite during feeding. No other effects on the host have been reported.

Paguritherium alatum parasitized about 1% of the *Pagurus longicarpus* population near Woods Hole, Massachusetts (USA) (Reinhard, 1945). The parasite's long voluminous tubular body becomes encapsulated by a sheath derived from host hypodermis that connects to the outer environment at the site of initial parasite penetration for larval release. A long slender tube or pleon leading from the main body of the parasite exits through the sheath at the base of the crab's eyestalk rather than through the gills as in most other entoniscids. Most of the parasite consists of a massive brood chamber that replaces much of the hepatopancreas and greatly suppresses gonadal development, although the pleon is not reported to cause tissue damage.

Most entoniscids cause some degree of parasitic castration in the host with development of female secondary sexual characteristics in parasitized male crustaceans (Overstreet, 1983). *P. longicarpus* parasitized by *P. alatum* may be recognized grossly by their pale abdomen through which eggs and embryos within the parasite's brood chamber may be visible through the semitransparent tissue of the host. The sequestering effect of the host sheath appears to limit damage; this is not the case in some instances reported for another entoniscid, *Portunion conformis,* parasitzing *Hemigrapsus oregonensis.*

Occasional high prevalence in mortality of parasite postlarvae within *Hemigrapsus oregonensis* has been attributed to a poorly understood cellular thickening of the protective host sheath which is postulated to cause anoxia and/or starvation (Kuris and coauthors, 1980).

Agents: Cirripedia

Perhaps the most elaborately evolved and sophisticated symbiotes of marine Crustacea are the rhizocephalan barnacles noted for their invasiveness but general non-lethality in the natural host and endocrinological suppression of host fecundity at the population level. Rhizocephala are primarily symbiotes of decapod crustaceans but also occur in free-living barnacles (Bocquet-Vedrine, 1957, 1967; Pawlik, 1987) and in bivalve molluscs (Takahashi and co-authors, 1973; Elston and co-authors, 1985).

The generalized life cycle of symbiotic cirripedes was pieced together from the efforts of several investigators (Müller, 1863; Boschma, 1927, 1962; Reinhard, 1942a, b, c, 1956; Ichikawa and Yanagamachi, 1960) and is remarkably intricate. A starting point for discussion is the release of larvae from an adult sausage-shaped brood chamber or externa of the symbiote generally protruding from the abdominal area of a host crab. In most cases the larvae are released as nauplii resembling those of the Thoracica order of free-living barnacles, hence the barnacle classification. The non-feeding planktonic nauplii go through 4 to 5 molts developing finally into a cyprid stage which is either female or male. Female cyprids are smaller than males and represent the stage that attaches to a crab host via modified antennae. Once attached the cyprid molts again into the kentrogon stage with the valves containing a ball of germinal cells and a dart-like stylet. The cirripede cells inject into the crab host through the stylet and reach the visceral cavity of the host via the circulatory system. There the parasite cells divide forming a complex vastly dendritic system of rootlets, called the interna, which penetrate connective tissues throughout the crab and soak up nutrients from crab fluids. After several months (much sooner in some species) the interna reaches the ventral abdominal area where a rootlet bud forms a primordial female ovary surrounded by a brood chamber that penetrates the external cuticle of the crab host. Once emerged, the virginal externa, attached by a narrow stalk, remains small and inactive until fertilized by male cyprids released from a mature externa. Male cyprids inject spermatogonia into the mantle of the externa which migrate to seminal receptacles that produce sperm, becoming testes thereafter. Once fertilized the externa undergoes considerable growth and continues to produce eggs and release larvae. In some species of rhizocephala, nauplii molt through all stages while in the externa and are directly released as cyprids. Externae may alternate in releasing male and female larvae, or at times release both sexes.

The effects of rhizocephalan parasitism on host crabs are profound, primarily involving castration generally accompanied by endocrinological alterations that affect molting, behavior and secondary sexual characteristics (O'Brien and van Wyk, 1985). Little information is available in the literature regarding direct physiological and histopathological effects of rhizocephalan parasitism. The following discussion is not intended to review all rhizocephalans on crustaceans nor reiterate specific life history details for each species. For this information the reader is referred to Reinhard (1956), Lawler and Shepard (1979), O'Brien and van Wyk (1985), and Hoeg and Lützen (1985). Pathological or histological observations described for certain rhizocephalan species are considered in this treatise.

The only recognized rhizocephalan *Sylon* species is *S. hippolytes*; it parasitizes several shrimp species including *Spirontocaris lilljeborgi* in Norway and *Pandalus platyceros* in waters off British Columbia, Canada. This parasite releases cyprids directly from the externa. Lützen (1981a) has described the rootlet system in *S. lilljeborgi* as extensive,

surrounding the ventral nerve cord, occasionally invading the pleopods and invading the connective tissues of the hepatopancreas and those surrounding the thoracic ganglion. Rootlets also occur below the gonad and the heart. However, as with most Rhizocephala, the rootlets do not appear to invade any of the organs proper. The parasite eventually castrates the host such that existing gonads are largely preserved in their developmental state prior to infection but degenerate with inhibition of further development. *Sylon* sp., infesting up to 47% of *P. platyceros* near Vancouver Island, British Columbia (Canada), produces a similar rootlet system and histological appearance in this host (Bower and Boutillier, 1988).

As do several other rhizocephalans, *Peltogaster paguri* commonly parasitizes the hermit crabs *Pagurus bernhardus* and *P. pubescens* from Europe and the American North Atlantic, respectively. Reinhard (1942a, b, c) described the effects of this parasite on *P. pubescens* and a histological study of the parasite's development. As commonly found with some other rhizocephalans, female crabs (17.6%) had a higher prevalence of parasitism than males (14.2%). The overall parasite prevalence for both sexes of crabs was 13.7%, with 6.8% of infested crabs having multiple infestations. The parasite causes complete gonadal degeneration in females having externae but no effect on the testes of male crabs. The parasite also causes no change in secondary sex characteristics in either sex. Physiological and histochemical studies of parasitized hosts showed that they had much lower fat content in the hepatopancreas and in the whole body than normal crabs (Reinhard and von Brand, 1944). This was contrary to the elevated fat levels in host crabs reported by early investigators. The parasite apparently depletes fat from the host crab which is found in great abundance in eggs and developing nauplii and to a lesser degree in the externa and the rootlets of the interna.

One of the most detailed and thorough studies of rhizocephalan life history was presented by Ritchie and Hoeg (1981) for *Lernaeodiscus porcellanae* parasitizing the porcelain crab *Petrolisthes cabrilloi*. The life cycle is similar to that of *Peltogaster paguri* and *Sacculina senta*, however the externa does not always produce broods of one sex. The parasite castrates its host regardless of sex and produces female secondary sexual characteristics and behavior in parasitized male crabs. Male crabs will act like ovigerous females or parasitized females by treating the parasite externa as an egg clutch to the extent of keeping it clean and ventilated. Such males and females will also assist the externa in spawning larvae. Host crabs remain parasitized for life and damaged externae are lost, each leaving a scar, with apparent regeneration of new externae. Interestingly, healthy crabs avoid becoming parasitized by actively grooming the gills, thus preventing the settling of cyprids. As shown experimentally, crabs with damaged or impaired grooming appendages have a greater chance of becoming parasitized. Impairment can occur naturally from pre-molt condition, sickness or injury sustained from living in an unstable rocky substrate. Crabs from rocky shores had a parasite prevalence of 35% compared to 10% in crabs from other types of substrate. Experimental exposure of a competent crab host to several hundred cyprids caused physical exhaustion from frantic grooming behavior and eventual death from the stress. Whether this situation occurs in nature is not known.

Sacculina carcini parasitizes *Carcinus maenas*, and has the same life cycle (Day, 1935) as species of *Peltogaster* and *Lernaeodiscus*. Spermatogenesis is inhibited and vitellogenesis arrested in parasitized crabs apparently from hypertrophy and degeneration of the androgenic gland rather than from the observed invasion of the gonads by the rootlets

(Rubiliani and co-authors, 1980). In addition, secondary sexual characteristics are altered in both sexes with broadening of the abdomen and feminization of appendages in males, and narrowing of the abdomen and pleopod degeneration in females. These changes are obvious results of altered neuroendocrine control in the crab host caused by potential products produced from the rhizocephalan rootlets (Rubiliani and co-authors, 1980) and possibly some effect caused by invasion of the thoracic ganglion as the primary target organ and other parts of the nervous system including the brain. Nervous system histopathology included penetration of the neurilemma by rootlets accompanied by degeneration of the neuroglia and neuropile with pyknosis and karyorrhexis of perikarya. Nervous tissue degeneration also occurred without obvious invasion of the neuropile by the parasite, lending further credibility to a secretory product as the cause of tissue degeneration (Rubiliani and co-authors, 1980; Payen and co-authors, 1981). Andrieux and co-authors (1976) and Herberts (1978) identified 2 different specific protein fractions in the hemolymph of infested crabs, and Andrieux and co-authors (1981) produced delays in molting and changes in hemolymph protein profiles of normal crabs by injecting crude extracts of *Sacculina carcini* and hemolymph from infected crabs.

Additional studies by Rubiliani (1985) provided further evidence for neuroendocrine tissue degeneration from a rhizocephalan product rather than rootlet invasion. Aqueous extracts of rootlets from another sacculinid, *Loxothylacus panopei,* were injected into individuals of *Rhithropanopeus harrisii* and *Panopeus herbstii,* producing gonial and mesodermal degeneration and pyknosis in the testes, degenerative changes of the perikarya in the brain and thoracic ganglion, hyposecretion of the sinus glands, and 'signs of degeneration' in the androgenic gland. *P. herbstii* were more sensitive to the extract, possibly because they were taken from an area where the rhizocephalan does not naturally occur and were thus more naive (Rubiliani, 1985). This and other evidence presented for *L. porcellanae* in *Petrolisthes cabrilloi* and other related porcelain crabs suggest that a rhizocephalan parasite can restrict the range of other host species preventing competition with its primary host (Ritchie and Hoeg, 1981). Lützen (1981b) in another unrelated study showed evidence that shore crabs scarred by previous infestation with *Sacculina carcini* sustained a higher molting mortality than normal crabs when held in cages. Scarred crabs also were more lethargic and weakened, further implicating that sacculinid infestation in this host species can be fatal.

Loxothylacus texanus parasitizes *Callinectes sapidus,* and possibly 2 other hosts — *C. marginatus* and *C. ornatus* — in the Gulf of Mexico (Overstreet, 1978). The parasite typically infests immature crabs of both sexes and retards growth by preventing molting, thus producing dwarf or 'button' crabs. Few parasitized crabs are larger than 8 cm wide, compared to 14 cm in a normal crab. Castration of the host results in the usual changes in secondary sexual characteristics with both sexes developing a wide abdominal apron resembling a mature female crab. Males undergo further feminizing changes in their abdominal segments and musculature. Behavioral changes in both sexes include treatment of externae (from 1 to 8) as if an egg clutch. Parasitism only occurs in moderate-to-high salinities; some data suggest that externae will not emerge in salinities of 3 ppt and those emerged will take on water and burst (Ragan and Matherne, 1974). At 15 ppt, externae regress (Overstreet, 1978). Although negligible in the past, *Loxothylacus texanus* reached epizootic prevalences of up to 50 % in Mississippi Sound (USA) in 1965 and 1977 which conceivably reduced commercial stocks significantly (Overstreet, 1983). No other specific pathological effects of the parasite have been described.

Colonial rhizocephalans of the genus *Thompsonia* (Potts, 1915) parasitize various decapods but differ from the usual members of the Rhizocephala in several ways. The rootlets produce numerous small grapelike reproductive externae (1 to 4.5 × 1.1 mm) that emerge from the appendages and venters of the thorax and abdomen after the host molts. In 1 species these are easily dislodged. Host molting is not inhibited and no altered secondary sexual characteristics or behavior are produced in males or females, except that no parasitized females are berried (Hafele, 1911; Boschma, 1933; Phang, 1975).

A *Thompsonia* species parasitizing *Portunus pelagicus* was studied by Phang (1975) and produced several thousand of the small externae sometimes occurring on the dorsal surface of the abdomen in heavy infestations. A possible *Thompsonia* species was recently described by Johnson and co-authors (1986) parasitizing up to 52% of female and 33% of male *Paralithodes platypus* from Olga Bay, Kodiak Island (Alaska, USA). A single female *P. camtschatica* of 11 individuals examined was also parasitized. Parasitized crabs had no visible externae and no externally altered morphological features to indicate parasitism. Although parasitized females were mature, they lacked external evidence of any egg extrusion and had grossly abnormal ovaries. Male gonads were unaffected. Histological examination of affected crab tissues indicated parasite rootlets within hemal spaces of female pleopods and ovary, and in the hemal spaces of the following tissues in both sexes: connective tissues adjacent to the gut, the bladder, thoracic ganglia, antennal gland and hepatopancreas. Only 1 crab had rootlets actually invading the neuropile of the thoracic ganglion. Oocytes were either absent or in complete or partial degeneration in the ovaries of 88% of the parasitzed female crabs. This was in comparison to 46% of apparently normal crabs having the same oocyte condition. The intensity of rootlet invasion in parasitized crabs varied but in severe and moderate cases some rootlet degeneration and necrosis was nearly always present alongside normal appearing internal tissue. A foreign body-type host response with hemocyte infiltration around and within affected rootlets followed by melanization characterized the microscopic appearance. The data of Johnson and co-authors (1986) clearly indicate that the rhizocephalan was causing ovarian damage and probable reproductive impairment by prevention of mating and subsequent extrusion and attachment of fertilized ova. These authors further speculate that the blue king crab may not be the natural host for this parasite resulting in its failure to produce externae and a host reaction which may ultimately eliminate the infestation.

Lithodid crabs are also parasitized by the cosmopolitan peltogastrid rhizocephalan *Briarosaccus callosus,* first described on *Lithodes agassizii* from Florida and North Carolina, USA (Boschma, 1930). Other hosts include *L. antarcticus* and *Paralomis granulosa* from Antarctic and sub-Antarctic areas (Boschma, 1962); *L. murrayi* from the southwest Indian Ocean (Arnaud and Do-Chi, 1977); *L. couesi* from the Bering Sea (Boschma, 1970) and the Gulf of Alaska (Somerton, 1981); *Paralithodes camtschatica* from southeastern Alaska (Boschma and Haynes, 1969); *L. aequispina* from the Bering Sea (Boschma, 1962), Gulf of Alaska (McMullen and Yoshihara, 1970) and British Columbia (Sloan, 1984); and *P. platypus* from southeast Alaska (Hawkes and co-authors, 1985a). Both sexes of the crabs parasitized by *B. callosus* are castrated and males typically exhibit feminizing traits such as thick ventral growth of coxal setae (Fig. 3-54), enlarged convex abdominal aprons and ovigerous behavior towards the externae.

Several studies have been undertaken to determine the effects of *Briarosaccus callosus* on host crabs with the following results. Growth in parasitized crabs is inhibited as

evidenced indirectly by field data and supported by laboratory growth studies. Such growth suppression is a unique feature among peltogastrid rhizocephalans which generally allow host growth to continue and possibly to increase (O'Brien and van Wyk, 1985). Weights and condition factors of parasitized male blue and golden king crabs were considerably less than in the same size class of non-parasitized crabs, and the prevalence of parasitism was much greater in sublegal or smaller-sized crabs (Hawkes and co-authors, 1986a).

Laboratory studies of parasitized male *Paralithodes platypus* showed significant growth reductions as measured by reduced molt increments, and reduction in post-molt weight gains (Hawkes and co-authors, 1987). Speculated causes for growth inhibition include nutrient drain by the parasite and male feminization since growth rates are generally less in females than in males. However, parasitized crabs consumed less food on a daily basis and hence less energy would be available for somatic growth (Hawkes and co-authors, 1987). Also interesting was that parasitized golden king crabs *Lithodes aequispina* sustained significantly less growth suppression than did parasitized *P. platypus*. During this 6 month laboratory study no intermolt mortalities occurred in parasitized crabs and despite the often large size of externae, parasitized crabs were able to molt successfully with no significant parasite-related mortality. This suggests that the mortality rate may not be different between parasitized and non-parasitized crabs. Hemolymph from the 2 species of *Paralithodes* (red, blue) and 1 species of *Lithodes* (golden) king crabs parasitized by *B. callosus* was examined for parasite-induced changes. Changes observed, but not in all 3 species, included decreased osmolality and sodium and chloride concentrations and increased concentrations of protein, hemocyanin and glucose (Shirley and co-authors, 1986). Protein electropherograms of hemolymph taken from parasites of all 3 species were similar but hemocyanin was present in parasites taken from red and golden king crabs.

The respiratory pigment in *Briarosaccus callosus* is not hemocyanin but hemoglobin averaging about 5.8 mg ml^{-1} which imparts a bright red color to the hemolymph. Consequently, the parasite apparently absorbs small quantities of hemocyanin from its host but the circulatory systems of parasite and host appear discrete since no hemoglobin was detected in any of the host crab hemolymph (Shirley and co-authors, 1986). Differences in hemolymph among parasitized crab hosts were most noteworthy regarding higher compensatory hemolymph protein concentrations among other parameters in parasitized red and golden king crabs compared to their non-parasitized conspecifics and parasitized blue king crabs. These results and the lesser growth reductions noted above suggest the golden king and probably red king crabs may be less susceptible to the negative effects of *B. callosus* parasitism. This is further supported by lower parasite prevalences, fewer multiple externae on single crabs and a higher activity level in parasitized crabs of these 2 species when compared to the blue king crab. Near Juneau, Alaska (USA) during 1979 through 1985 parasite prevalences in commercial landings that included females and sublegal crabs of all 3 species ranged from $< 1\%$ in red king crabs, 20 % in golden king crabs and up to 76 % in blue king crabs (Hawkes and co-authors, 1985a, 1986b). Parasite prevalences tended to be greater in females than in males and in crab populations inhabiting glacial floured areas than in crabs from clear waters.

Gross observations of parasitized crab tissues during necropsy were similar among all 3 species of king crabs. Upon removal of the carapace the interna of *Briarosaccus callosus* is immediately visible as a strikingly emerald green dendritic mass with rootlets closely

adherant to all major organs and tissues in the visceral cavity including the bases of gills and muscle within the coxal joints. The rootlets do not appear to enter further into the leg musculature. Much of the hepatopancreas is replaced by the interna mass and gonads of both sexes are either severely atrophic or unable to be found (Figs. 3-43, 3-44). Newly parasitized crabs of either sex still without externae may be recognized by excessive growth

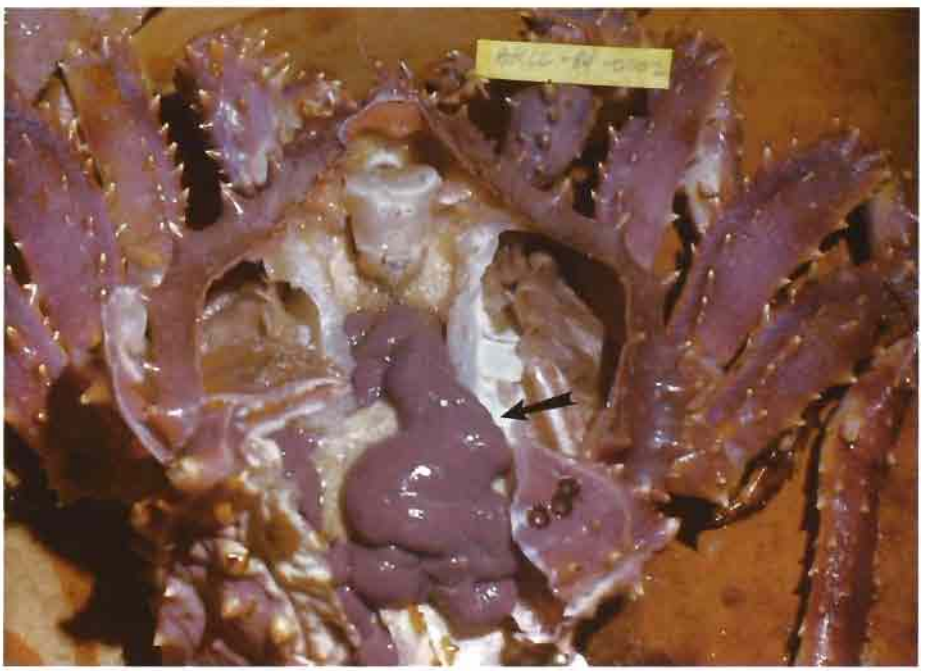

Fig. 3-43: *Paralithodes platypus*. Internal view of normal female showing ovarian development. (Original.)

of coxal setae. One such female, although berried, contained a parasite interna and atrophic ovary. Attempts at weighing the bulk of the rootlet mass from parasitized blue (N = 4) and golden (N = 13) king crabs resulted in conservative averages of 14.1 g and 21.6 g, respectively. The histological appearance of the parasite rootlets within host tissues consists of basophilic branching tubules, several cells in width having peripheral cells with large nuclei all surrounded by a thick refractile outer cuticle. A lumen is occasionally present containing proteinaceous material and cytoplasmic granules are prominent, retaining the green color that imparts the emerald hue in fresh material (Figs. 3-45, 3-46). Since the parasite utilizes hemoglobin as a respiratory pigment the green granules may represent biliverdin as the initial pigment resulting from the catabolism of hemoglobin. Interna rootlets infiltrated between host skeletal muscle bundles and within the connective tissues of the urinary bladder, hepatopancreas, pyloric stomach, midgut, rectum, ovary, testes and anterior vas deferens. Although atrophic, remnant gonads if found appeared to undergo normal oogenesis and spermatogenesis (Figs. 3-47, 3-48). Parasite rootlets have not been observed to penetrate epithelial boundries, thus entering organ lumina. This is not true for the thoracic ganglion and its nerve tracts in which rootlets penetrated the

Fig. 3-44: *Paralithodes platypus*. Internal view of female parasitized by *Briarosaccus callosus* showing green discoloration of tissues from the rootlets and atrophy of the ovary (yellow H-shaped organ). The barnacle parasitism was incidental to a systemic *Paranophrys*-like ciliate infection producing nodular foci of parasites and coagulated hemolymph (arrow) causing death of this crab. (Original.)

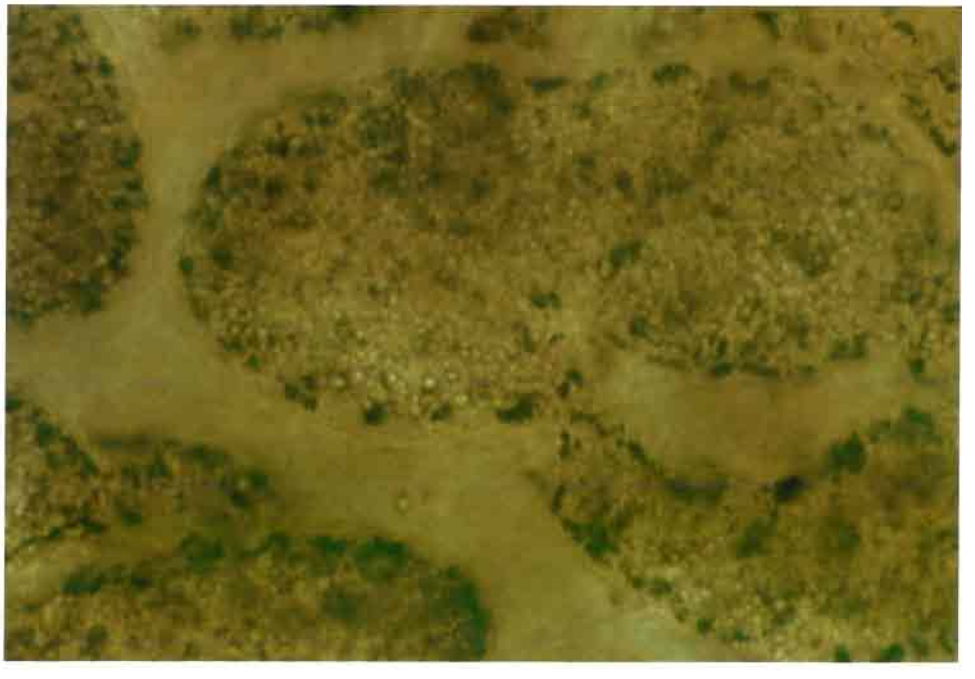

Fig. 3-45: *Briarosaccus callosus*. Fresh squash of live rootlets showing green biliverdin-like cytoplasmic granules and a thick pellicle, × 1,600. (Original.)

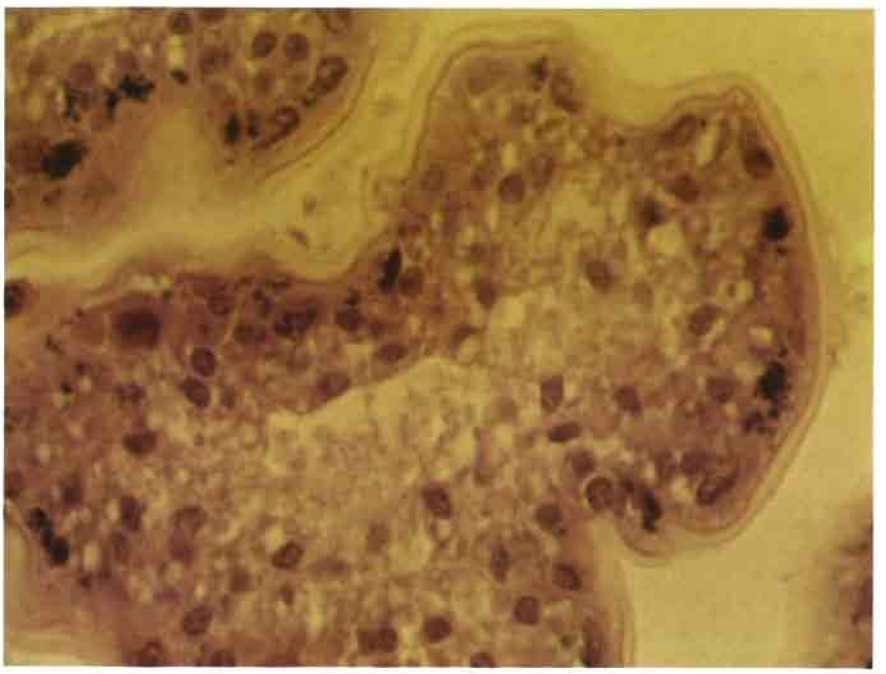

Fig. 3-46: *Briarosaccus callosus*. Rootlet histology consisting of a tubule having peripheral cells with large nuclei, a thick pellicle and cytoplasmic granules that retain the green coloration after fixation. Hematoxylin and eosin, × 4,000. (Original.)

neurilemma and neuropile. The neurilemma appeared hypertrophic and somewhat fibrotic accompanied by infiltration of granular hemocytes at the site of parasite invasion and between nerve fibers. However, there is almost no host response to the parasite once in the neuropile (Fig. 3-49). Occasional invasion of rootlets into the cerebral ganglion and optic nerve of the eyestalks also occurred with similar results.

Parasitized species of *Paralithodes* and *Lithodes* that have lost *Briarosaccus callosus* externae are recognized not only by altered secondary sexual characteristics but by the remnant leathery stalk on which the lost externa was attached seen protruding through the abdomen (Fig. 3-50). Unlike other rhizocephalans, *B. callosus* does not regenerate lost externae. Evidence for this has been demonstrated in the laboratory by holding crabs for a year or more after loosing their externae naturally or after these were removed by excision. During necropsy of scarred crabs the internae in some appeared dark brown and friable beginning at the point of stalk protrusion to the external environment extending inward (Fig. 3-51). Histologically this lesion consisted of degenerating and necrotic rootlets encapsulated by host hemocytes with melanization. This suggests that the parasite may begin to degenerate after the externa is lost which could be a far more life-threatening problem for the crab due to the severe foreign-body response to such a large mass of dying and dead parasite tissue. Externae on some crabs held in the laboratory underwent spontaneous senescent changes and dropped off leaving only the stalk (Fig. 3-52). This further suggests that the life span of the parasite may not be as long as the host's since externae are not regenerated and internae appear to degenerate in scarred crabs.

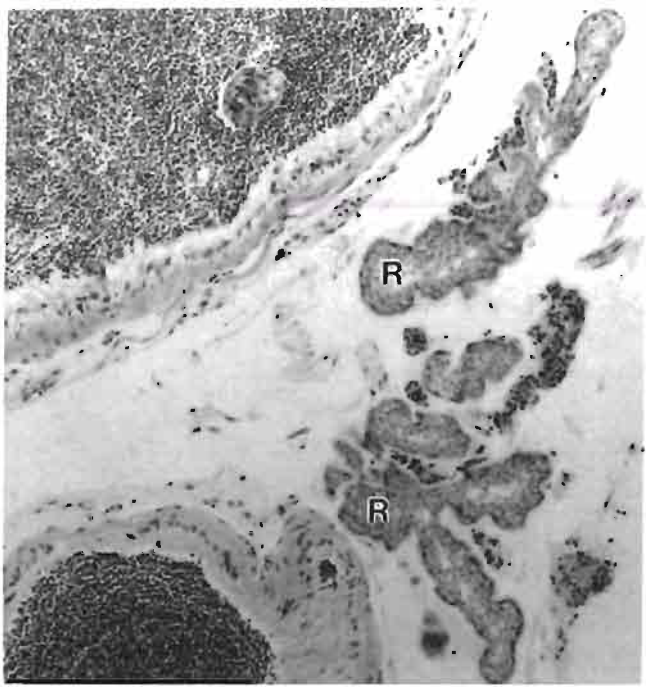

Fig. 3-47: *Paralithodes platypus*. Connective tissue of testes in male infiltrated by rootlets (R) of *Briarosaccus callosus*. Hematoxylin and eosin, × 256. (Original.)

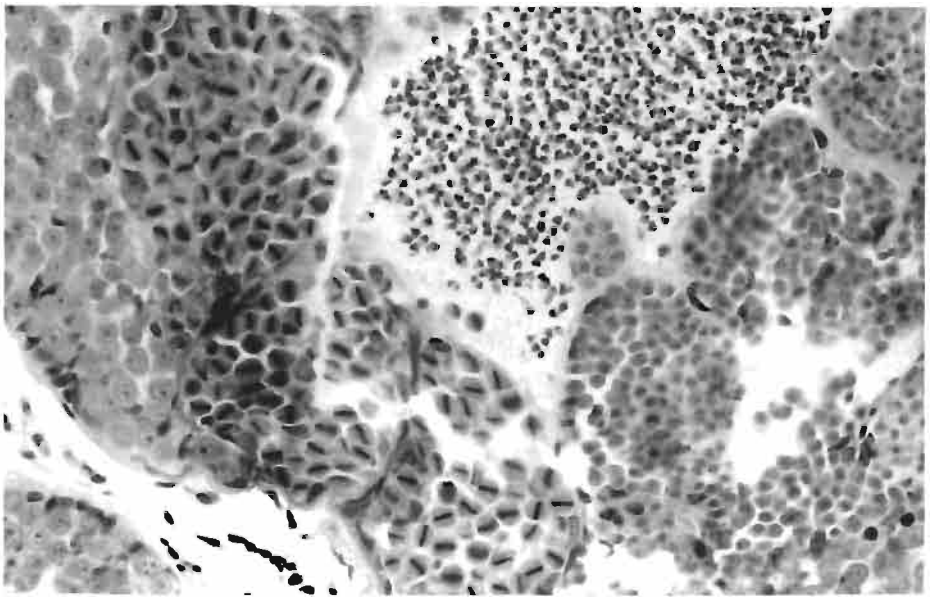

Fig. 3-48: Higher magnification of Fig. 3-47 showing normal spermatogenesis despite gross atrophy of the testes. Hematoxylin and eosin, × 640. (Original.)

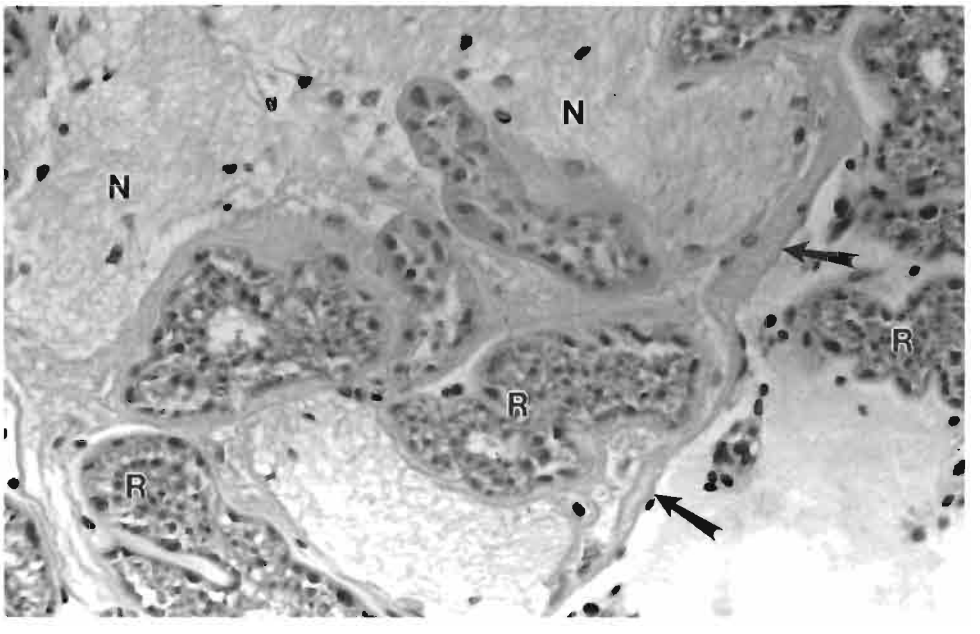

Fig. 3-49: *Paralithodes platypus*. Nerves of thoracic ganglion in individual infiltrated by rootlets (R) of *Briarosaccus callosus*. Note slight hypertrophy of neurilemma (arrows) and no host response to the parasite once within the neuropile (N). Hematoxylin and eosin, × 640. (Original.)

Fig. 3-50: *Paralithodes platypus*. Scarred individual showing a leathery brown stalk (arrow) at the attachment point of a previous externa of *Briarosaccus callosus*. The interna is still within the abdomen of the crab host. (Original.)

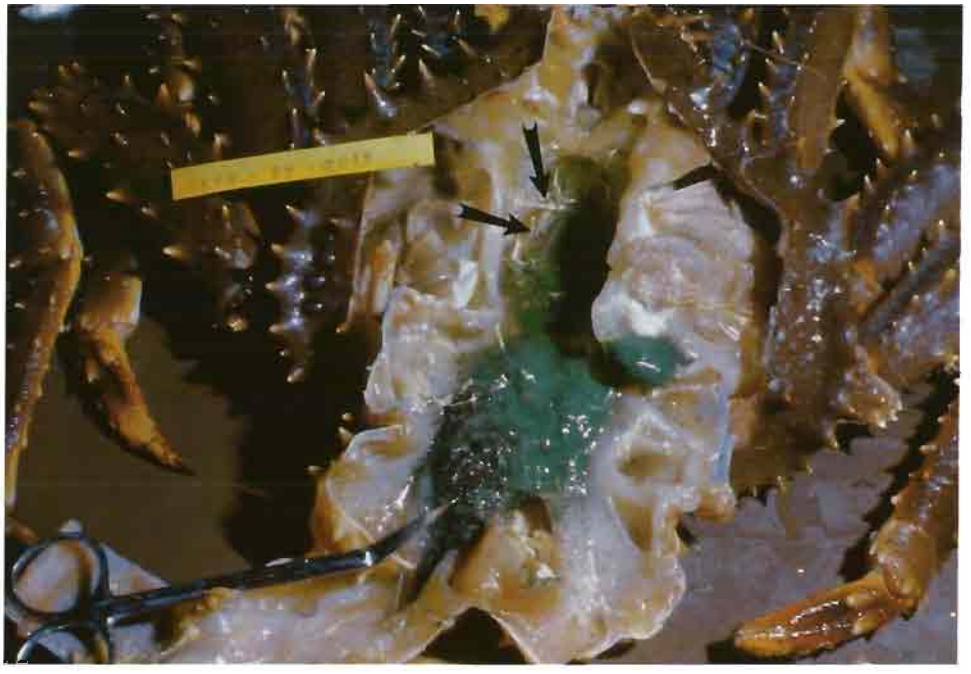

Fig. 3-51: *Paralithodes platypus*. Gross internal view of an individual with an external scar from a previous parasite externa of *Briarosaccus callosus*. Note brown friable hard mass of degenerating parasite rootlets extending inward from the internal attachment point (at tip of scissors). Nerves radiating from the thoracic ganglion (arrows) are visible. (Original.)

The life history of *Briarosaccus callosus* is not entirely known but some laboratory studies have been done regarding larval biology (Hawkes and co-authors, 1985b). The externa of this parasite is large, orange-to-red in color depending upon the maturity of embryos within and represents a double-chambered cylinder (Fig. 3-53). The internal chamber contains ova which move into the outer chamber as embryos mature causing the externa color to become a pale orange. At this time embryo development may be monitored using a Pasteur pipette to pull samples from the opening to the mantle cavity. Externa length ranges from 12 mm in a newly extruded virginal brood chamber (Fig. 3-54) to 77 mm in a mature parasite. Wet weights of mature externae (N = 33) may average 21 g (Hawkes and co-authors, 1985b). Externae on parasitized blue and golden king crabs release larvae mostly during the summer in 45 to 48 day intervals between broods. The host assists the parasite in spawning by dropping the abdomen and messaging the externa with its pleopods, a behavior similar to that reported for other rhizocephalan hosts. Once spawned, the outer chamber of the externa collapses and becomes transparent. Depending upon externa size about 3.1 to 3.9×10^5 larvae are released as Stage I nauplii that undergo molts to a final 4th stage before molting to a cyprid, all of which takes from 20 to 29 days at 6 to 8 °C (Figs. 3-55 to 3-57). Cyprids may survive for at least an additional 16 days and morphologically resemble those of other rhizocephalans (Reinhard, 1946; Yanagimachi, 1961). Size differences between 3 separate broods of cyprids indicated the largest (399 μm long) were probably males and the smallest (321 μm long) were females. No morphological

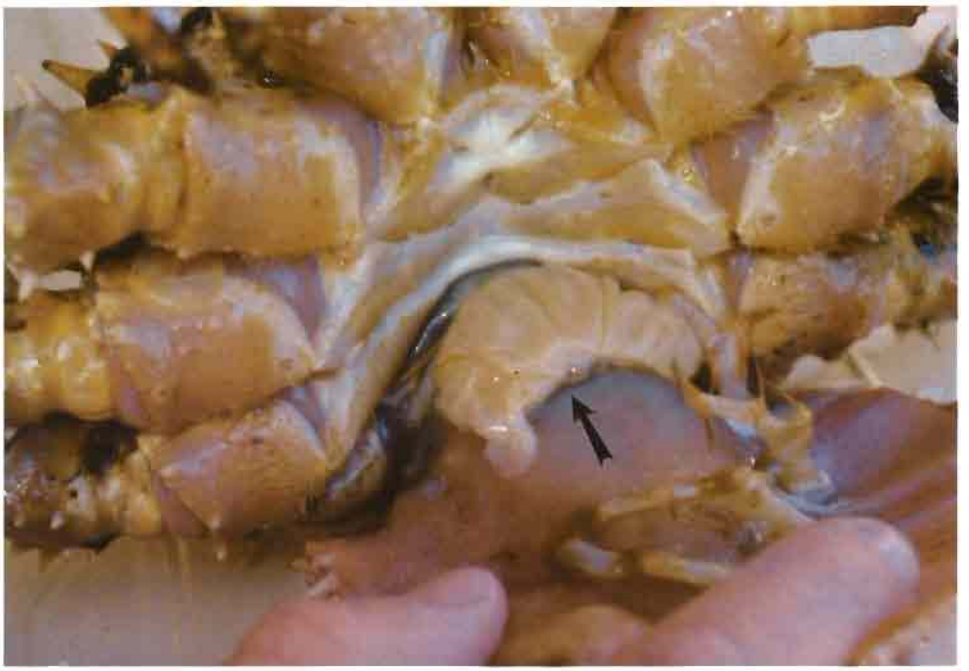

Fig. 3-52: *Lithodes aequispina*. Senescent externa of *Briarosaccus callosus* (arrow). Note blanched flaccid condition of the necrotic parasite tissues. (Original.)

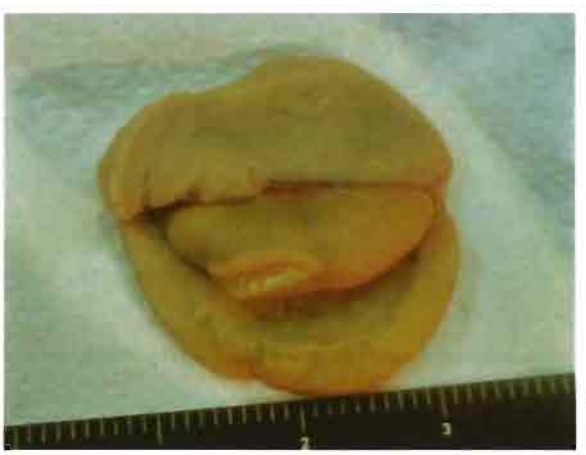

Fig. 3-53: *Paralithodes camtschatica*. Externa of *Briarosaccus callosus* surgically cut to show the double-chambered configuration. Scale in cm. (Original.)

differences were observed between parasite larvae from either the blue or golden king crabs, indicating the parasite was indeed conspecific on both hosts and probably on red king crabs as well (Hawkes and co-authors, 1985b). However, larvae from a parasite externa on a red king crab were reared only to the first naupliar stage. Many of the features above are different from other rhizocephalan species regarding the larger externa and

Fig. 3-54: *Paralithodes platypus*. Multiple parasitism by *Briarosaccus callosus* showing a small virginal externa next to a larger mature externa. Note red coloration of externae due to hemoglobin in parasite hemolymph and excessive development of ventral coxal setae on the host crab. (Original.)

Fig. 3-55: *Briarosaccus callosus*. First naupliar stage spawned directly from an externa on *Paralithodes platypus*. × 252. (After Hawkes and co-authors, 1985b.)

Fig. 3-56: Third larval stage of *Briarosaccus callosus* from the same brood in Fig. 3–55. × 252. (Original.)

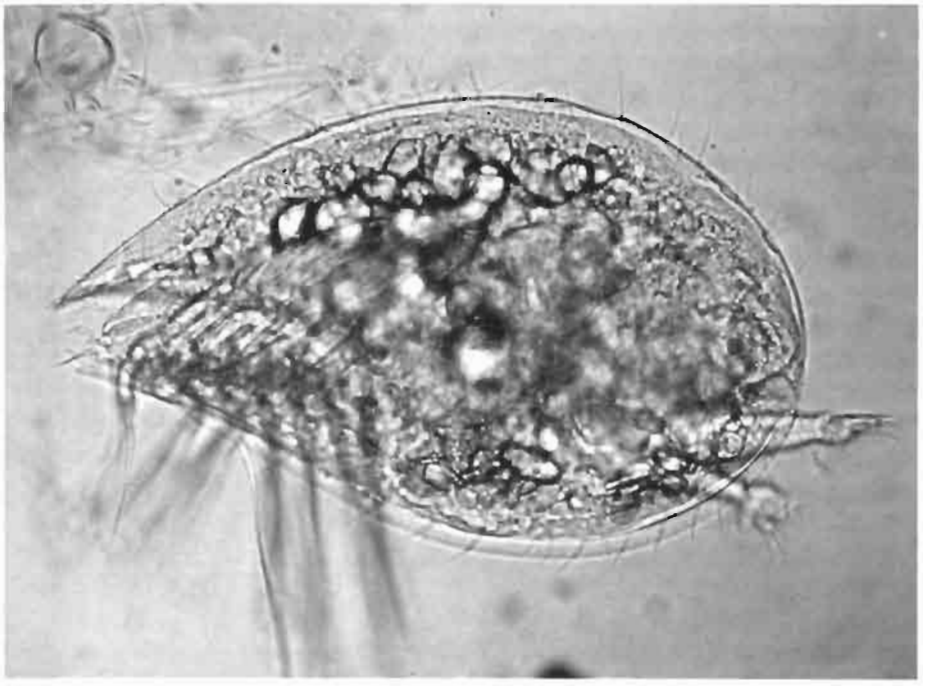

Fig. 3-57: *Briarosaccus callosus*. Cyprid stage from the same brood illustrated in Fig. 3-55. × 640. (After Hawkes and co-authors. 1985b.)

brood numbers, longer brood periodicity and slower larval development rate. The mode of host infection by the *Briarosaccus* cyprid and pathogenesis of the disease still are unknown needing further elucidation through infectivity studies in the laboratory.

Briarosaccus callosus parasitism in Alaskan *Paralithodes* and *Lithodes* populations has great potential to negatively impact already severely depressed populations. Parasitized crabs cannot contribute to recruitment due to castration of both sexes and feminized behavior of male crabs. Affected individuals also compete for space and food while disseminating the parasite to healthy individuals, and the fishery collects the larger healthy crabs leaving the parasitized individuals. Management of this problem may be possible if progressive thinking is applied through test fisheries such that all parasitized crabs incidentally harvested should be marketed regardless of sex or size. The parasite does not negatively affect meat quality of the crab host but knuckle meats are discolored by the green interna despite cooking.

3.3 DISEASES CAUSED BY PROLIFERATIVE LESIONS AND NEOPLASIA

J. A. Brock and D. V. Lightner

Within the past 20 years a few reports on proliferative lesions in crustaceans have appeared in which sufficient microscopic study was carried-out to warrant classification of the described pathologic change as tumor-like or neoplasia. Sparks (1972, 1985) reviewed the early published information on neoplasia in Crustacea and related forms, but from the descriptions given and the absence of microscopic documentation of the cytological nature of lesions it seems highly doubtful that any of these lesions were neoplastic (Sparks, 1972; Sparks and Lightner, 1973; Lightner and Brock, 1987). Indeed, even with the information documented over the past 10 years, the record clearly indicates that neoplasia is rare in Crustacea.

In this review only the more recent literature (1972 and later) is surveyed. In this context, 5 of the 8 cases or conditions discussed represent hyperplasia, hypertrophy or abnormal development, and 3 are clearly neoplastic lesions. All 8 lesions are reported from marine decapods, and literature reports of similar proliferative pathologic changes in other crustaceans are unknown to us.

The lesions or conditions reviewed here are as follows. A papilliform tumor-like growth in a pond-reared *Penaeus aztecus* was suggested by Sparks and Lightner (1973) to be a benign neoplasm. Hamartomas of the striated muscle of the 6th abdominal segment of wild postlarval *P. aztecus* and *P. setiferus* was thought to represent an apparent loss of normal growth regulation for a specific striated muscle (Overstreet and van Devender, 1978). There are 3 histopathological syndromes that are examples of unusual idiopathic hyperplasia or hypertrophy. The first is a proliferation of the epineural layer surrounding the ventral nerve cord of *Penaeus japonicus* (Lightner and co-authors, 1984). The second is an acellular proliferative lesion of the mid-gut, at 2 distinct sites within the organ, in *P. japonicus, P. plebejus* and *P. merguiensis* (Lightner and co-authors, 1984, 1985a). The third type of unusual hyperplasia occurs in Oka's lymphoid organ in the form of multiple proliferative centers of the lymphoid tissue, and apparent metastatic foci of these cells in other organ locations in the shrimp (Lightner and Brock, 1985; Lightner and co-authors, 1987a). The 3 neoplasms reported from marine decapods include a lymphosarcoma of hematopoietic tissue of adult pond-cultured *P. vannamei* infected with IHHN virus (Lightner and Brock, 1985, 1987), a putative carcinoma in the hind-gut of *Paralithodes camtschatica* (Sparks and Morado, 1987), and an embryonal carcinoma of developing embryos of tank-reared *Palaemon orientis* (Lightner and Hedrick, 1987).

A subadult *Penaeus aztecus* was collected from an earthen pond in Texas (USA) and submitted for evaluation because of a tumor-like growth on the right ventrolateral aspect of the 6th abdominal segment (Sparks and Lightner, 1973). The single cauliflower-like lesion, $8 \times 8 \times 9$ mm in size (Fig. 3-58, c, d), appeared to arise from the epidermis and subepidermal tissues of the shrimp. Histologically, the base of the mass was within the epidermis and although epithelial cells were hypertrophic and hyperchromatic, mitosis was not observed. The growth was covered by cuticle, uninterrupted with that covering the

Fig. 3-58: Hamartoma and a papilliform growth of penaeid shrimp. (a) Fourth through the 6th abdominal segments of a 13 mm long postlarval *Penaeus aztecus* with a large frayed hamartoma (arrow). (b) Histological cross section through a hamartoma (H) showing lateroventral extrusion of muscle bundles through the cuticle; Heidenhain's iron hematoxylin; bar = 50 μm. (a and b after Overstreet and van Devender, 1978. Reprinted with the permission of Academic Press, Inc.) (c) *P. aztecus* bearing a papilliform growth (arrow) on the 6th abdominal segment. (d) Gross appearance of the papilliform growth and adjacent normal body on cut section; note space (arrow) between the growth's connective tissue core and body musculature of the abdomen. (e) Histological section through a portion of the papilliform growth near its apical end. The core of the tumor is composed of loose vacuolated connective tissue (CT) stroma, and it contains numerous tegumental glands (T) basal to the epidermis its associated cuticle (arrows); H&E; bar = 250 μm. (c to e after Sparks and Lightner, 1973. Reprinted with the permission of Academic Press, Inc.)

normal body, although the cuticle over the apex of the mass was markedly thickened. Internally, the raised tissue (Fig. 3-58, e) was composed primarily of an expanded, well vascularized connective tissue stroma, scattered striated muscle fibers, and tegmental glands abnormal in number and location with respect to the inner surface of the cuticle. In concluding statements Sparks and Lightner noted that the lesion was aberrant new tissue, but the enlargement was under the control of normal growth regulating processes of the decapod host, and thus, probably represented a benign neoplasm.

Overstreet and van Devender (1978) documented hamartomas from the ventral aspect of the 6th abdominal segment of postlarval *Penaeus aztecus* and *P. setiferus,* sampled from an apparently polluted site in a Mississippi estuary (USA). This non-neoplastic overgrowth, the first and only report of hamartomas in marine Crustacea, was suggested to have resulted from "an interaction between a pollutant and the normal growth process of shrimp" (Overstreet and van Devender, 1978, p. 234). The lesion occurred in 33 postlarvae and involved 0.56% of 2,320 white shrimp, 0.08% of 26,238 brown shrimp and none of 4,573 pink shrimp examined. Evaluation of 21,331 juvenile-to-adult shrimp of the same species collected in similar areas failed to reveal the lesion (Overstreet and van Devender, 1978).

Grossly, the lesion is a ventral protuberance, obvious to the unaided eye and variable in size (Fig. 3-58, a). Histologically, the hamartomas were composed principally of U-shaped folds of the anterior oblique muscle Number 6 covered externally, in most specimens, by a thin layer of cuticle (Fig. 3-58, b). Apparently, stretched ventral nerve cord was present in the protruding striated muscle. Microscopically, cellular structure appeared normal in the hamartomas. Overstreet and van Devender (1978) explained the lesion development as an overgrowth of muscle tissue with a concomitant lack of growth of the encircling exoskeleton. The authors suggested that hamartoma-affected shrimp were more vulnerable to predation, thus, older shrimp were not found in wild-populations with the lesions. Overstreet and van Devender also noted that the putative benign neoplasm in a pond-cultured shrimp reported by Sparks and Lightner (1973) could represent a case of the chronic outcome of a hamartoma at the post larval stage. Exposure to pollution was offered as the probable etiologic factor mediating the muscle tissue overgrowth because hamartomas incidence was associated with sites considered to be polluted. Further cases of hamartomas in wild, experimental or cultured shrimp have not been reported, thus the above case is apparently unique.

In Hawaii and France, populations of cultured *Penaeus japonicus* had individual members exhibiting an unusual proliferative syndrome (Lightner and co-authors, 1984; Tsing and co-authors, 1985). The condition, termed 'gut and nerve syndrome' (GNS), was so named because of the principal microscopic lesions and its idiopathic nature (Lightner and co-authors, 1984). Gut and nerve syndrome was recognized in postlarvae through broodstock *P. japonicus.* Other species of penaeid shrimp (*P. stylirostris, P. vannamei* and *P. monodon*) cohabiting facilities with GNS-positive *P. japonicus* did not develop GNS lesions. The discovery of a reo-like virus from *P. japonicus* (Tsing and Bonami, 1986) in GNS-positive populations prompted the hypothesis that the pathology is caused by the reo-like virus (Tsing and co-authors, 1985). However, direct evidence is lacking of a cause-and-effect relationship for penaeid shrimp reo-like virus and the GNS micro-pathology.

Thirteen of 14 populations of *Penaeus japonicus* held in a variety of culture conditions in the Hawaiian study were positive for GNS. The presence of reo-like virus infection was

not reported for these groups of shrimp. However, at least one population studied retrospectively was infected (Lightner, 1988 his Chapter 3.1.21). The incidence of GNS in these shrimp populations ranged from 50 to 100 %. In the high-density rearing trial groups, GNS was associated with a subacute-to-chronic disease with 100 % cumulative mortality (Lightner and co-authors, 1984). Behavioral signs and gross pathology for shrimp in GNS-positive populations include lethargy, low stamina, reduced escape response, poor growth, focal-to-generalized abdominal muscle necrosis, increased susceptibility to epibiotic fouling organisms and opportunistic bacterial and *Fusarium solani* infections. Histologically, lesions were found in the anterior mid-gut and the epineurium of the ventral nerve cord. The anterior mid-gut basement membrane is thickened by a largely acellular layer of eosinophilic, PAS-positive material (Fig. 3-59, b). The increased thickness of the hypertrophied basement membrane measured at times up to 73 μm. Ultrastructurally, the lesion results from a tremendous hypertrophy of the inner layer of the basement membrane and a slight increase in thickness of the outer layer (Lightner and co-authors, 1984).

The nerve cord proliferative change is limited to the epineurium which is hyperplastic (Fig. 3-59, a) consisting of 2 to 12 essentially identical layers. The hyperplastic epineurium began at the first segmental ganglium posterior to the optic ganglia and extended to the ganglium at the first abdominal segment. Ultrastructurally, each repetitive layer is similar to a single, normal epineurium.

The cause of GNS is undetermined. Reo-like virus infection or a physiological etiology have been suggested, but data which confirm these factors as determinants have not been reported. In Hawaii, GNS-free *Penaeus japonicus* were not identifiable grossly, thus, studies that compared performance of GNS affected versus unaffected groups of *P. japonicus* could not be carried out.

A second form of mid-gut hypertrophy named 'mid-gut serosal hypertrophy' has been reported by Lightner and co-authors (1985a). The lesion differs from the mid-gut hypertrophy of GNS in hypertrophy location and shrimp species affected. At the microscopic and ultrastructural levels, a single, eosinophilic, PAS-positive amorphous layer (Fig. 3-59, c, d) forms in the serosa of the anterior mid-gut of *Penaeus merguiensis* and possibly other Asian penaeids. The histological, histochemical and ultrastructural characteristics for mid-gut serosal hypertrophy are similar to those of basement membrane hypertrophy of *P. japonicus*. The cause-and-effect relations of this lesion are also unknown. Interestingly, *P. merguiensis* with serosal hypertrophy are often infected by hepatopancreatic parvovirus (HPV), possibly HPV is related etiologically to the formation of the serosal lesion.

Another idiopathic, proliferative lesion has been detected in certain penaeid shrimp, particularly *Penaeus monodon,* and represents a hyperplasia of Oka's lymphoid organ and metastases of spherical clumps of hyperplastic cells from that tissue (Lightner and Brock, 1985; Lightner and co-authors, 1987a). The condition has tentatively been designated by Lightner and co-authors as 'Oka organ' hyperplasia and metastasis (OHM). Enlargement of the lymphoid organ is not associated with specific disease signs, however, cultured shrimp populations with affected individuals frequently display vague signs, and growth performance is substandard. The lesion has been observed in *P. monodon* and *P. penicillatus* cultured in Taiwan (Lightner and co-authors, 1987a) and in captive-wild *P. esculentus* in Australia (Paynter and co-authors, 1985); it has also been recognized in *P. vannamei, P. stylirostris* and *P. chinesis* (Lightner and Brock, unpubl.). The metastasis of hyperplastic cell clumps is, however, a feature most frequently observed in *P. monodon*.

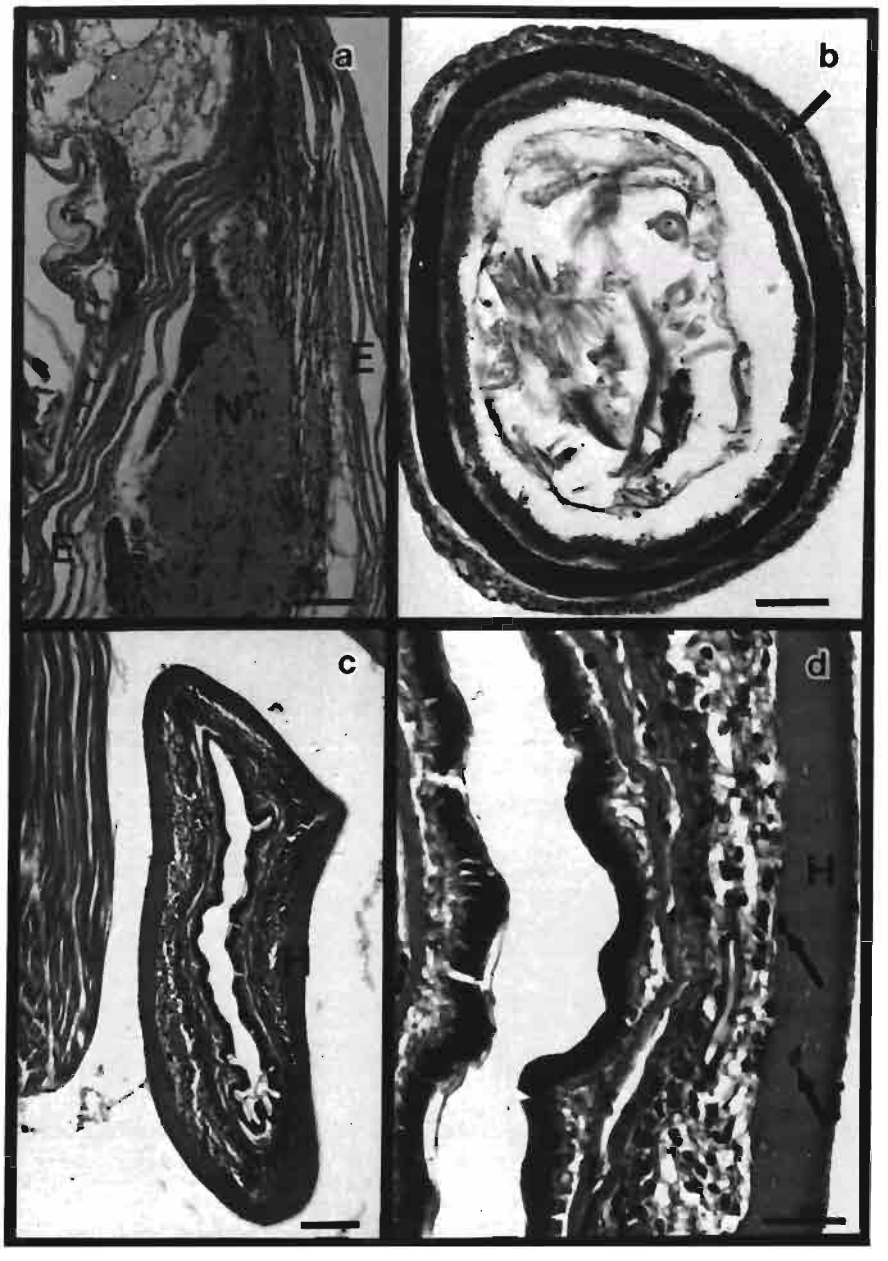

Fig. 3-59: Ventral nerve cord epineural hyperplasia and mid-gut basement membrane hypertrophy of penaeid shrimp. (a) Sagittal section of ventral nerve cord (N) in the gnathothorax of a juvenile *Penaeus japonicus* with 'gut and nerve syndrome' (GNS); the abnormal epineurium (E) covering the nerve cord is composed of multiple repeating layers, rather than the normal single layered epineurium; H&E; bar = 100 μm. (b) Cross section of the anterior midgut from the third abdominal segment of a juvenile *P. japonicus* with GNS; basement membrane (arrow) of the mucosal epithelium is hypertrophied to many times its normal thickness; PAS staining; bar = 50 μm. (a and b after Lightner and co-authors, 1984. Reprinted with the permission of Blackwell Scientific Publications Ltd.) (c and d) Cross section of the anterior midgut of a juvenile *P. merguiensis* with an idiopathic deposition of a hyaline material (H) on the serosal surface of the midgut; embedded within the deposit are cells (arrows) which presumably secreted the deposit; both H&E; bars = 50 μm (c) and 25 μm (d). (Originals.)

Grossly, in OHM the paired lymphoid organs are enlarged. Microscopically, the OHM lymphoid organ (Fig. 3-60, a) consists of both normal appearing lymphoid cells arranged in sheaths around a central hemolymph vessel and spherical clumps of hyperplastic cells or 'spheroids' that lack a central vessel (Fig. 3-60, c). The ratio of normal appearing lymphoid centers to abnormal 'spheroids' within the organ is variable between individual shrimp. In extreme cases the majority of the lymphoid organ is made-up of 'spheroids'. The enlargement of the lymphoid organ results from cellular hyperplasia. In some individuals ectopic 'spheroids' (Fig. 3-60, b) occur within the hypodermal connective tissues, gills, gonads, heart, antennal gland and striated muscle. Lightner and co-authors (1987a) suggest these arise by dissociation from the main lymphoid organ and are disseminated via the hemolymph. The cause of the condition is unknown as a specific etiologic agent has not been demonstrated within the lesions. Lightner and co-authors indicated that the lesion may be a peculiar or special type of inflammatory response. However, the cellular proliferation may not be inflammatory at all, but represent some other pathogenesis such as cellular hyperplasia in response to exocrine stimulation or increased functional demand. Additional study to understand the pathogenesis and cause of OHM in marine shrimp would provide insight into the nature of this unique and interesting lesion.

One adult male and one adult female *Penaeus vannamei,* in a sample of 20 shrimp collected for histological examination from an earthen pond at a research facility in Hawaii, had hematopoietic sarcomas (Lightner and Brock, 1987). Outwardly, the affected shrimp appeared normal and tumors were noted on histopathology examination. Both shrimp were infected by IHHN virus, and Cowdry-Type A inclusion bodies were present, although not abundant, in the neoplastic hematopoietic tissue. Histologically, Lightner and Brock observed that the hematopoietic nodules located near the base of the third maxillipeds were markedly enlarged. Smaller ectopic foci of normal and neoplastic appearing hematopoietic cells were present in the gills (Fig. 3-61, b), heart (Fig. 3-61, c) in the subcutis and in the haemocoel near and between bundles of striated muscle and in connective tissue adjacent to the lymphoid organ. Within hematopoietic tissue and dispersed between regions of normal appearing cells were nests of mitotically active, greatly enlarged, anaplastic cells (Fig. 3-61, a). Nuclei of these anaplastic cells were hypertrophied and contained one to several nucleoli. Abnormal metaphase mitotic figures with multiple mitotic spindles, multinuclear giant cells (up to 20 nuclei), synchronous mitosis of multinuclear giant cells and dipolar, tripolar or multipolar mitotic figures were a common feature (Fig. 3-61, a-c) in the lesion (Lightner and Brock, 1987). The authors conclude that the lesion is clearly neoplastic and classified it as a hematopoietic sarcoma.

A putative carcinoma was seen by Sparks and Morado (1987) in a wild-caught female *Paralithodes camtschatica,* collected in 1985 from the Kodiak Management area in Alaska. The lesion, recognized during gross dissection as a white, 20 × 12 mm lump, was attached to the ventral side of the anterior aspect of the hind-gut. Histologically, large pleomorphic, epithelioid-like cells with hypertrophic, irregular-shaped nuclei predominated the cellular profile of the lesion (Fig. 3-62). In some areas the apparently poorly differentiated epithelioid cells formed nests within the hind-gut wall. Hemocytes, especially eosinophilic granulocytes heavily infiltrated the mass, and encapsulation and phagocytosis of neoplastic cells were noted. Although classification of the tumor to cell origin was not possible, Sparks suggested the lesion is a probable carcinoma that arose from either the mid-gut, the hind-gut or the tegmental gland epithelium.

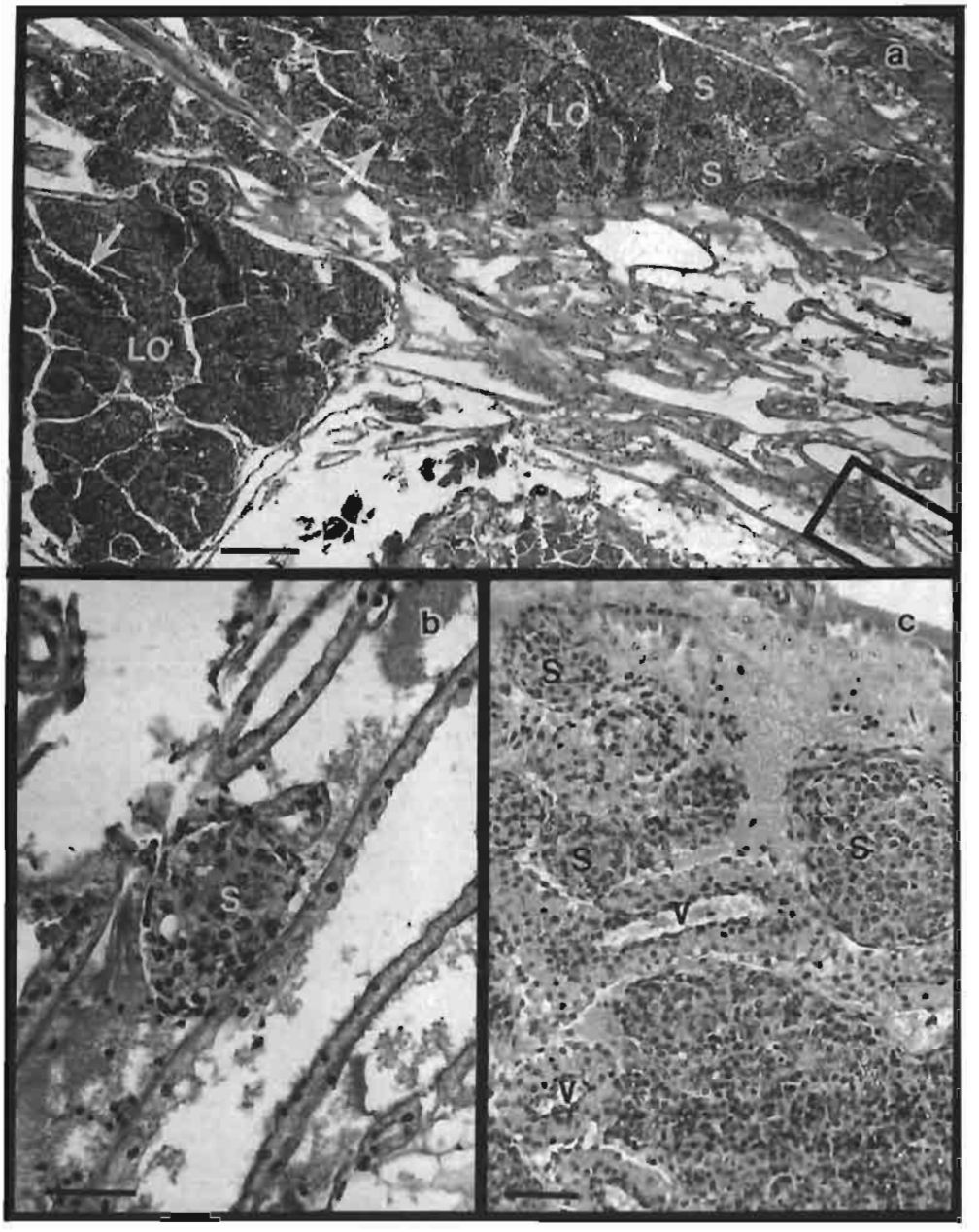

Fig. 3-60: Lymphoid (Oka) organ hypertrophy of penaeid shrimp. (a) Histological section of a greatly hypertrophied lymphoid organ (LO) of a subadult *Penaeus monodon;* normal LO vessels and sheath cells (arrows) are interspersed among abnormal spheroids (S) of sheath cells; H&E; bar = 200 μm. (b) Metastatic LO spheroid (S) from inset box in Fig. 3-60a lodged in the hemocoel adjacent to tubules of the antennal gland; H&E; bar = 50 μm. (a and b after Lightner and co-authors, 1987a. Reprinted with the permission of the Japanese Society of Fish Pathology, Tokyo.) (c) Higher magnification light micrograph of a hypertrophied LO showing normal LO tissue with vessels (V) surrounded by layers of sheath cells, as well as spheroids (S) which are aggregates of disassociated sheath cells that do not surround a central vessel; H&E; bar = 50 μm. (Original.)

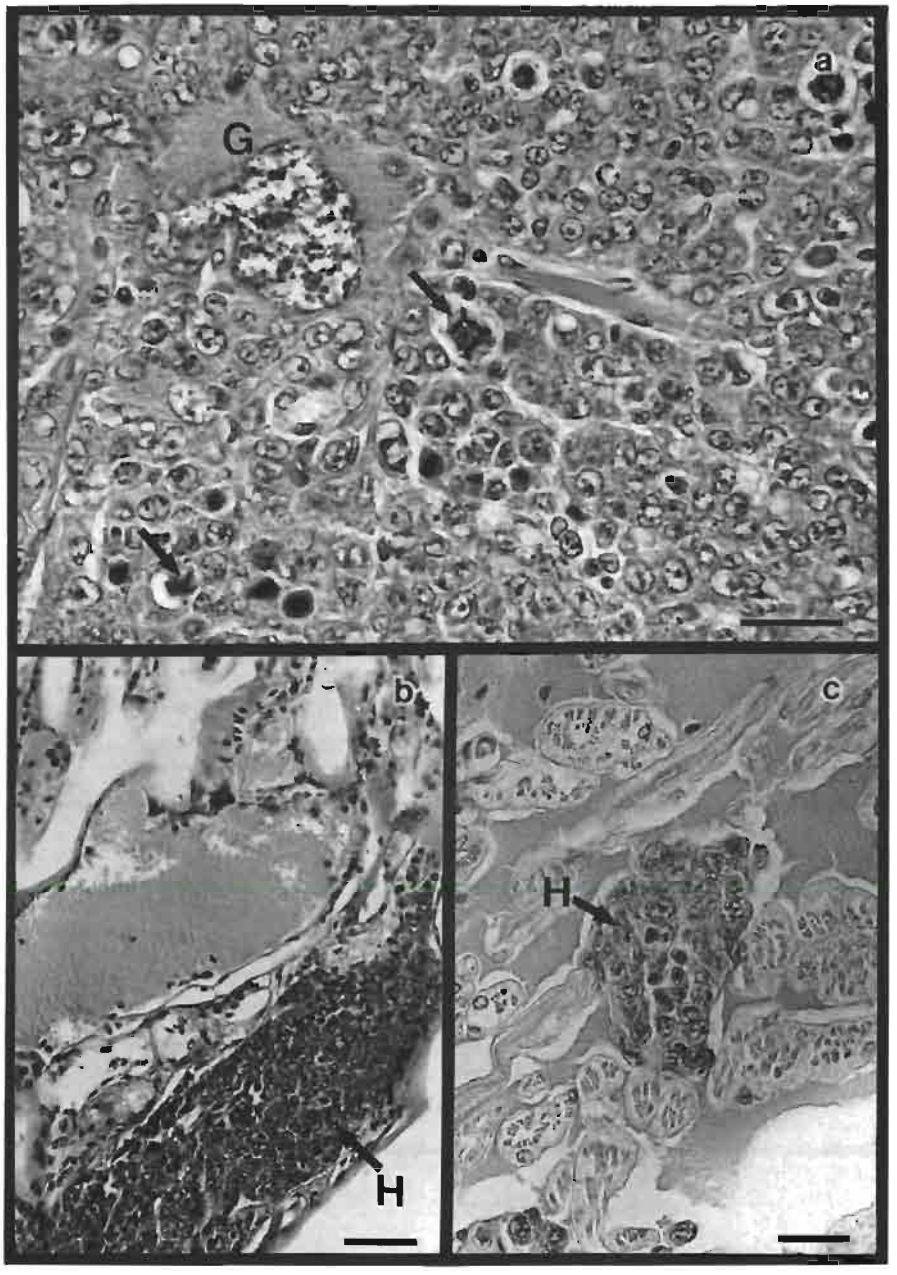

Fig. 3-61: Hematopoietic sarcoma of *Penaeus vannamei*. (a) Histological section of a sarcoma that arose from the hematopoietic nodules in the maxillipeds of an adult *Penaeus vannamei;* numerous anaplastic cells are present, many with nuclei that are highly variable in size, including 1 giant cell (G) that has a single enormously hypertrophied nucleus; also evident is a high incidence of dividing cells, some of which have bizarre polypolar mitotic figures (arrows); H&E; bar = 25 μm. (After Lightner and Brock, 1987. Reprinted with the permission of Academic Press, Inc.) (b) Section of gills from the same shrimp as in Fig. 3-15a showing a metastatic nodule of neoplastic hematopoietic tissue (H); H&E; bar = 50 μm. (Original.) (c) Another metastatic nodule of neoplastic hematopoietic tissue (H) in the heart; H&E; bar = 25 μm. (Original.)

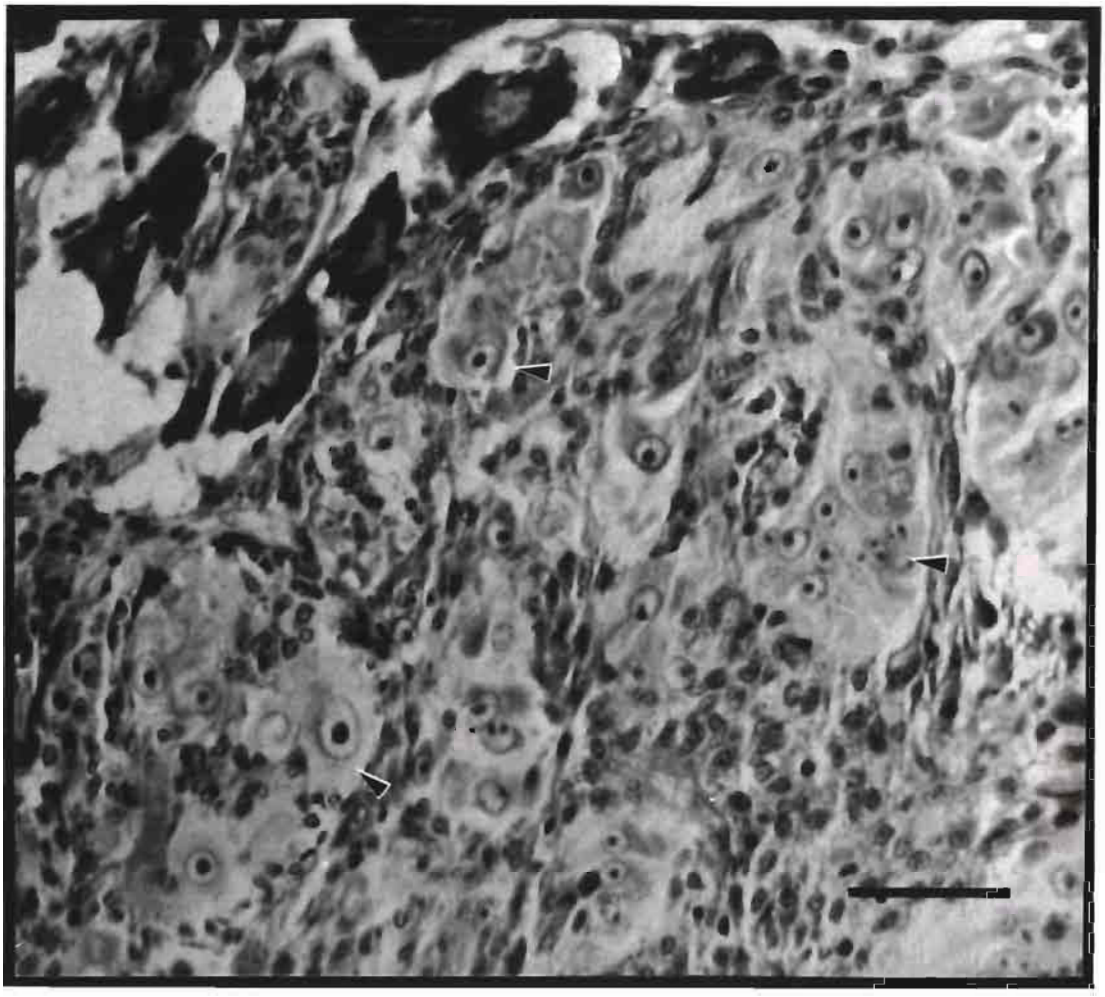

Fig. 3-62: LM section of the putative carcinoma-like neoplasm from the hind-gut of *Paralithodes camtschatica;* neoplastic cells (arrow heads) invading an area of tegmental glands (upper left); H&E; bar = 50 µm. (After Sparks and Morado, 1987. Reprinted with the permission of Academic Press, Inc.)

In a disease survey of shrimp in Taiwan Lightner and Hedrick (1987) found a neoplastic disorder in embryos of *Palaemon orientis.* About 20 % of the embryos histologically examined, from 3 brooding female *Palaemon orientis,* had neoplastic changes diagnosed as an embryonal carcinoma (Lightner and Hedrick, 1987). The shrimp, collected from a culture tank at a research facility, had not been stocked into the tank and probably entered the system via the incoming water. Embryos with neoplastic lesions were found on each of the 3 shrimp examined. Neoplastic lesions were not, however, observed in the tissues of the female shrimp or in tissues of another shrimp *(P. vannamei)* sampled from the rearing tank. Embryos with neoplastic lesions were slightly smaller than normal developing cohorts. The tissues of these embryos were disorganized and, except for the yolk, all parts appeared equally involved (Fig. 3-63, a, b). The neoplastic cells were

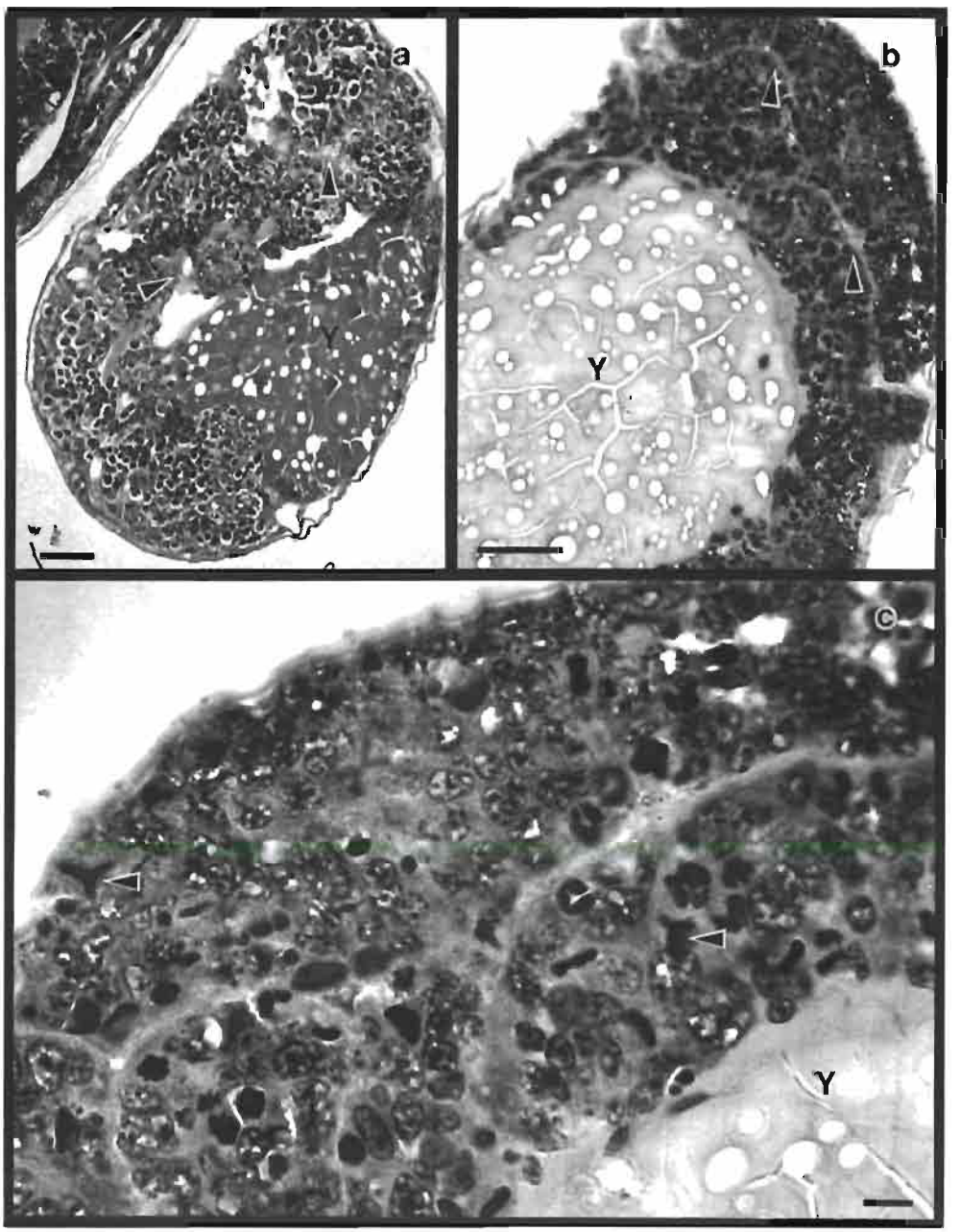

Fig. 3-63: Embryos of *Palaemon orientis* with an embryonal carcinoma. (a and b) Tissue organization is poor compared to unaffected embryos of the same stage, and except for the yolk mass (Y) and clefts (arrow heads) that mark the outline of limb buds, little normal tissue organization is apparent; H&E; bars = 50 μm. (c) Higher magnification of an embryonal carcinoma; neoplastic cells are undifferentiated, pleomorphic, and with generally hypertrophied nuclei; some nuclei possess one or more prominent nucleoli; dividing cells are very common, some have bizarre mitotic figures (arrow heads); H&E; bar = 10 μm. (a to c after Lightner and Hedrick, 1987. Reprinted with the permission of Inter-Research.)

undifferentiated pleomorphic cells with hypertrophic nuclei and one or more prominent nucleoli (Fig. 3-63, c). Mitosis were common and accompanied by bizarre multipolar metaphase figures (3-63, c). Necrotic cells and cells with intranuclear, Cowdry-Type A inclusion bodies were occasionally present.

The cause of the lesion was not determined. Lightner and Hedrick (1987) suggested potential etiologies to be chemical carcinogens, polyspermy or, possibly, IHHN virus infection.

In conclusion, unusual proliferative and neoplastic lesions have been recognized in marine decapod crustaceans, but the frequency of occurrence of neoplastic change is clearly rare. Six of the 8 conditions reviewed here were reported from marine shrimp in the genus *Penaeus*, one from a caridean shrimp and one lesion was recorded from a wild-caught red king crab. Interestingly, an association with concurrent virus infection has been reported for 4 of the 7 lesions noted from shrimp, but an etiological role for these viruses and the proliferative lesions observed is at best vague.

Future studies on the unusual proliferative pathologies of cultured marine shrimp, and on the putative role of virus infection in these and the neoplastic lesions would seem an area for productive research. Also, of considerable interest would be to determine why neoplasia seem to be a rare event in the members of the Class Crustacea.

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4. DISEASES OF CHAETOGNATHA

A. C. PIERROT-BULTS

The phylum Chaetognatha comprises 7 genera: *Sagitta, Eukrohnia, Pterosagitta, Krohnitta, Heterokrohnia, Spadella* and *Bathyspadella,* altogether including about 65 species. Chaetognatha are hermaphroditic and holoplanktonic animals, except for the genera *Spadella* and *Bathyspadella.* Chaetognatha live throughout the oceans, from coast to coast and from the surface to the bottom. They are most abundant in the epipelagic.

It is very difficult to keep chaetognaths under laboratory conditions, and consequently very little is known about their diseases. Chaetognaths are carnivorous, mainly feeding on copepods, but also on other prey, such as fish larvae and smaller chaetognaths. Fishes predate on chaetognaths which may act as intermediate hosts for fish parasites such as trematodes and nematodes, e.g., the trematode *Hemiuris levinseni* on cod, or the nematode *Anisakis* sp. on herring.

The organisms found on or in chaetognaths are not very 'host' specific. 'Parasitism' as defined in Vol. I, p. 19 (Kinne, 1980) has only rarely been established in symbiotes of chaetognaths. Whether the symbiotes actually behave as phoronts, commensals, parasites or mutualists requires detailed investigation in most cases. Actual diseases of chaetognaths, their aetiology, diagnosis and prevention are topics barely touched upon in the literature. The references to 'parasitism' in this chapter, as well as in the pertinent scientific literature concern all kinds of associate fauna, rather than relations based on demonstrated agent-host relationships.

Considering the fact that chaetognaths are a unique phylum, not easily placed in the animal kingdom, it is surprising that only quite common 'parasites' are found and that a specific parasite or associate fauna has not developed. Reimer and co-authors (1971) state that chaetognaths serve as reservoir or additional second hosts for the parasites. Weinstein (1972) suggests that the low diversity of parasites in *Sagitta elegans* may be due to the limited variety of its prey and to a difficulty of parasitic organisms with complex life cycles (e.g., transfer between several different types of hosts) in colonizing an extensive 3-dimensional environment.

The ratio of parasitic infection in chaetognaths is higher in some areas compared to others. Jarling and Kapp (1985) found $\pm$ 0.2 % infected specimens both on the Patagonian shelf and in the Mid-Atlantic Great Meteor Bank region and about 3.2 % infected specimens in the upwelling region off North-West Africa. Hutton (1954) reported infection rates of 0.09 % to 1.15 % in Caribbean chaetognaths. Weinstein (1972) found infection rates of 0.13 to 4.8 % with trematodes, 0.5 % with ciliates and 0.02 % with cestodes and nematodes in populations from Atlantic Canadian waters.

Alvariño (1965) and Mazzoni (1986) suggested that the incidence of parasitism is usually greater in nearshore populations than in their oceanic counterparts.

The effect of the parasites in chaetognaths is not very severe, most authors report little or no direct effect on mortality, growth or reproduction. Fig. 4-1 shows *Sagitta hexaptera*

Fig. 4-1: *Sagitta hexaptera* from the Mid-North Atlantic; a trematode larva is clearly seen through the transparent body wall. (Original.)

from the North Atlantic containing a trematode larva. The larva is clearly seen from the outside and makes the specimen more visible than non-infected ones. This causes a higher mortality through stronger predation (Pearre, 1976), and change of niche selection of infected chaetognaths also results in higher mortality (Pearre, 1979). Nematodes affect productivity by damaging male products or ovaries by their movements (Grey, 1931; Reeve and Cosper, 1975). The effects of bacterial infection seem to be frequently lethal (Nagasawa and co-authors, 1985).

DISEASES CAUSED BY BACTERIA

Nagasawa and Nemoto (1984) described 2 diseases (X-diseases) in *Sagitta crassa*, isolated and maintained in the laboratory. One specimen was kept alive for 24 days; it laid a total of 343 eggs from the 8th to the 23rd day after sampling. Its main daily food ration was 10.4 copepods (*Acartia* sp.). The specimen suddenly died after minor tail damage. Examination under SEM showed the epithelium to be degenerated; the authors concluded that death was due to an unknown disease caused by bacterial infection of the skin-epithelium. The second type of bacterial infection caused sticking together of hairs of the ciliary organs. These organs are important for feeding; their malfunction may cause problems, and infected individuals may die of starvation. About 1 % of the specimens were thus infected. Nagasawa and co-authors (1984) found *S. crassa* in laboratory culture which ceased feeding and became opaque. Opaque parts contained a mass of *Flexibacter*-like bacteria between muscle fibres. Also they harboured large numbers of bacteria attached to their skin.

Nagasawa (1985) reports tumour-like swellings on the side of one *Sagitta crassa* (1 specimen out of 12,853) from Tokyo Bay which consisted of a massive bacteria aggregate; bacteria were also dispersed over other areas of the body surface. Bacterial infection was also found by Nagasawa and co-authors (1984) in preserved *S. pacifica* and *S. nagae* collected in Suruga Bay, and in living *S. helenae* from the Atlantic Ocean. The percentage of the population invaded by bacteria decreased with increasing water depth, from 13.3 % at 10 m to 1.6 % at 75 m to 0 % at 100 m (n = 200). The bacteria occurred mainly on the body surface causing head or tail damage. Nagasawa and Nemoto (1985) reported that in unhealthy *S. crassa* bacterial colonization prevailed before death.

Bacterial infection in chaetognaths from Suruga Bay caused head damage, while bacterial infection of chaetognaths from Tokyo Bay revealed abnormal knotty and flabby specimens (Nagasawa and co-authors, 1985). Here, the infection was mainly internal in muscle tissue. The authors also state that feeding activity and reproductive behaviour were far lower in these abnormal specimens than in normal ones. Mean body length of abnormal individuals was smaller than that of normal ones. About 12 % of the population was infected in July 1979 and about 4 % in June 1982. The authors suggest that bacterial infection is far more serious and causes more damage than metazoan parasitic infestations in chaetognaths.

DISEASES CAUSED BY PROTOZOANS

Furnestin (1957) mentioned unidentified Protozoa in *Sagitta minima, S. hexaptera* and *S. enflata* from Maroccan waters. Øresland (1986) found 1 Protozoa specimen in the head and 3 specimens in the body coelom of *S. setosa* from the English Channel.

Agents: Amoeba

Grassi (1882, cited in Weinstein, 1972) reported *Amoeba pigmentifera* and *A. chaetognathi* in the tail coelom of *Sagitta enflata, S. bipunctata, S. serratodentata* and *Spadella cephaloptera* from the Mediterranean Sea. Van Oye (1918) found Grassi's unicellular endoparasites in the caudal coelom of *S. enflata, S. hexaptera, S. trichodermis* van Oye, 1918 *(= S. neglecta), S. regularis* and *S. bedoti* from Indonesian waters. The

mentioned amoebas were placed in *Paramoeba* by Janicki (1912, 1932), who found *P. pigmentifera* in *S. enflata* and *P. chaetognathi* in *S. bipunctata* from the Gulf of Naples, while no parasites were seen in *S. serratodentata* and *S. hexaptera* from the same samples. The infection rate of *S. enflata* was higher than that of other species but in general, infection rates of chaetognaths were low compared to those of, for example, molluscs. *P. pigmentifera* was also found on the tail fin of *S. enflata* by Ramult and Rose (1946) and in the tail coelom of the same species by Ghirardelli (1950), both in Mediterranean samples. Hamon (1957) found *Janickina pigmentifera* on *S. bipunctata* off the Algerian coast.

Agents: Flagellata

Several authors report flagellate parasites in chaetognaths. Van Oye (1918) mentioned a flagellate as epibiontic ectoparasite. *Trypanoplasma sagittae,* described by Hovasse (1924) from the intestine of *Sagitta* sp., was placed in the genus *Trypanophis* by Rose and Hamon (1950) who found it between cells of the intestinal wall of *S. lyra, S. hexaptera* and *S. decipiens* from Algerian waters. Hamon (1951, 1957) recorded it from the same area in *S. lyra.*

Agents: Gregarinia

Gregarines in chaetognaths were reported as early as 1917 by Ikeda. Ramult and Rose (1946) found the gregarine *Lankasteria* sp. Mingazzini in the digestive tract of *Sagitta enflata* from the Mediterranean Sea. Furnestin (1957) described a monocystid gregarine, probably *Lankasteria* sp. from the gut of *S. friderici,* and may be the same or a related species in *S. bipunctata*; in addition, an unidentified, probably gregarine, specimen was found in the body coelom of *S. hexaptera,* all off the Maroccan Atlantic coast.

Hamon (1951) described a new genus and species for a polycyst gregarine: *Tricystus planctonis* living in or between epithelial cells of the intestine wall of *S. lyra.* She found a similar form in *S. bipunctata.* Dollfus and co-authors (1954) mentioned a tetraphyllid on a distomatous larva in the body coelom of *Sagitta enflata.*

Agents: Ciliophora

Ikeda (1917) was the first to describe the mouthless holotrichous ciliate *Metaphyra sagittae* from the coelom of a chaetognath. Van Oye (1918) mentioned a peritrichous ciliate as an epibiontic ectoparasite. Ramult and Rose (1946) found a ciliate very much like *M. sagittae* in the body coelom of *Sagitta enflata* from the Mediterranean Sea. They mentioned also unidentified apostomous ciliates in digested copepods. Furnestin (1957) saw an unidentified protozoan resembling *M. sagittae* in *S. minima* and *S. enflata* off the Maroccan coast. Stadel (1958, cited in Weinstein, 1972) reported this protozoan for *Eukrohnia hamata* from the Antarctic and placed it tentatively in the genus *Opalinopsis.* Weinstein (1972) found it in *S. elegans* from the North-western Atlantic, with levels of infestation raising with increasing chaetognath length, 45 % of the individuals >40 mm were infected. The body of infected chaetognaths was flaccid and swollen, ovaries were considerably shortened, maybe because of infestation during or following spawning. Jarling and Kapp (1985) report *M. sagittae* in *S. tasmanica* from N. W. African upwelling waters.

DISEASES CAUSED BY METAZOANS

Agents: Trematoda

Trematodes are the most frequently found parasites of Chaetognatha (see Table 4-1). Mostly there is 1 specimen per host, rarely 2. Trematodes are usually found in the trunk coelom near the ovaries, sometimes in the gut. The trematodes may cause atrophy or complete disappearance of ovaries (Elian, 1960; Reeve and Cosper, 1975; Øresland, 1986). Pearre (1976) observed that prey selection differed between infested and non-infested members of the same species although the amount of food was the same. This author mentions gigantism caused by parasites, leading to lowered fecundity and lengthened generation time, but Nagasawa and Marumo (1984) found that the mean body length of infested specimens was nearly the same as that of non-infested specimens of *S. crassa* from Tokyo Bay.

Pearre (1979) studied niche-modification in *Sagitta minima, S. friderici* and *S. enflata* from the north-west Mediterranean. Infested individuals dwelled higher in the water column than non-parasitised conspecifics; they were overdispersed, lived longer and became larger and more conspicuous than non-parasitized conspecifics, thus rendering the infested individuals more visible to sight-hunting fish predators.

Køie (1979a, 1983) studied the life-cycle of *Derogenes varicus* (Müller, 1784). The first intermediate host is the snail *Natica* sp., second intermediate hosts are copepods, ctenophores and chaetognaths (Fig. 4-2). *Sagitta* is probably infested by consuming infested copepods, as already mentioned by Apstein (1911). Pearre (1976) found some evidence that *Paracalanus* sp. is the parasite vector. *D. varicus* may mature in the chaetognath but the eggs are only released into the water after the death of the chaetognath or ingestion by the final host (members of various fish families or cephalopods). The sizes of *D. varicus* in chaetognaths are intermediate between those found in fishes and the smallest sizes found in *Calanus finmarchicus* (Gunner, 1736) in the same area. Some of the *D. varicus* in the chaetognaths were egg-producing; no egg-producing trematodes were seen in copepods (Weinstein, 1972).

Køie (1979a) did not succed in experimental infestation of *Sagitta elegans* after exposure to free-swimming cercariae; she found a natural infestation of *S. elegans* in the Kattegat. Species of *Natica* and *Sagitta* infested with *Derogenes varicus* are only found in the Atlantic and adjacent seas while infested fish are found all over the world except in warm shallow waters of low latitudes. According to Køie (1983) the life cycle of *Hemiurus communis* appears to be similar to that of *D. varicus* with, among others, copepods as second intermediate hosts through which the chaetognaths are infested; various fish species act as final hosts.

Weinstein (1972) found egg-producing *Hemiuris levinseni* in *Sagitta elegans* from Canadian Atlantic waters. The chaetognaths become infested in spring and more heavily in their second autumn (up to 6 parasites host^{-1}) while showing hyperbenthic distribution. The cod *Gadus morhua* is supposed to be the main final host of *H. levinseni,* but it was also found in *Scomber scombrus* and in the four-bearded rockling *Enchelyopus cimbrus*. The first intermediate host could not be established. Also the life cycle of *Brachyphallus crenatus* (Rudolphi, 1802) seems to be similar to that of *Derogenes varicus* (Køie, 1983) in that the unencysted metacercaria develops in copepods, which then may be eaten by a chaetognath.

Table 4-1
Trematods found in chaetognath species

Chaetognath	Trematod	Sea area	Source
Sagitta sp.	Unidentified	North Sea	Busch (1851)
	Unidentified	Off Scotland	Scott (1896)
	Hemiuris sp.	North Sea	Busch (1851)
	H. communis	Kattegat	Køie (1983)
	Opechona bacillaris	North Sea	Burfield (1927)
	O. bacillaris	Kattegat	Køie (1979b)
	Adolescaria progastrica	Black Sea	Sinitzin (1911)
	A. appendiculata	Black Sea	Sinitzin (1911)
S. bipunctata	Unidentified	Mediterranean Sea	Gourret (1884)
	Unidentified	Black Sea	Moltschanoff (1909)
	Derogenes sp.	Caribbean	Dawes (1958)
	Derogenes sp.	South Atlantic	Montero (1974)
	Derogenes sp.?	Off Marocco	Furnestin (1957)
	Hemiuris sp.?	Off Marocco	Furnestin (1957)
	Aphanuris stossichi	Mediterranean Sea	Dollfus (1960)
S. crassa	*Tergestia* sp.	Tokyo Bay	Nagasawa and Marumo (1984)
S. elegans	Unidentified	Off Scotland	McIntosh (1890, 1927)
	Derogenes varicus	English Channel	Lebour (1916)
	D. varicus	Wadden Sea	Künne (1952)
	D. varicus	North Sea	Pearre (1979)
	D. varicus	Kattegat	Køie (1979a, 1983)
	D. varicus	N-W Atlantic	Weinstein (1972)
	Derogenes sp.	North Atlantic	Zaika and Kolesnikov (1967)
	Derogenes sp.	White Sea	Kulachkova (1972)
	Opechona bacillaris	English Channel	Lebour (1916)
	O. bacillaris	North Sea	Reimer and co-authors (1971)
	Hemiuris communis	Off Scotland	Meek (1928)
	H. communis	White Sea	Kulachkova (1972)
	H. communis	N-W Atlantic	Weinstein (1972)
	H. levinseni?	Off Scotland	Myers (1956)
	Didimozoids	North Sea	Reimer and co-authors (1971)
	Brachyphallus crenatus	N-W Atlantic	Linton (1927)
	B. crenatus	White Sea	Kulachkova (1972)
S. enflata	Unidentified	S-W Atlantic	Boltovskoy (1981)
	Hemiuris sp.	Mediterranean Sea	Ramult and Rose (1946)
	H. rugosus?	Mediterranean Sea	Pearre (1976, 1979)
	Accacladium sp. or *Tetrochaetus* sp.?	Off Madras (India)	Dollfus and co-authors (1954)
	Cercaria sp.	Off S-E Australia	Thomson (1947)
	Metacercaria owreae	Caribbean	Hutton (1954); Suarez-Caabro (1955)
	M. sagittae	Mediterranean Sea	Dollfus (1960)
S. euxina	Unidentified	Black Sea	Moltschanoff (1909)
	Derogenes sp.	Black Sea	Elian (1960)
S. friderici	Unidentified	S-W Atlantic	Boltovskoy (1981)
	Derogenes sp.? or *Hemiuris* sp.?	Off Marocco	Furnestin (1957)
	H. rugosus?	Mediterranean Sea	Pearre (1976, 1979)
	Ectenurus virgula?	S-W Atlantic	Jarling and Kapp (1985)
S. hexaptera	*Derogenes* sp.?	Off Marocco	Furnestin (1957)
	Hemiuris sp.?	Off Marocco	Furnestin (1957)
	Metacercaria owreae	Caribbean	Hutton (1954); Dawes (1958, 1959)

Table 4-1 (continued)

Chaetognath	Trematod	Sea area	Source
S. hispida	*Hemiuris* sp.	S-W Atlantic	Almeido Prado (1961)
S. lyra	*M. owreae*	Caribbean	Hutton (1954)
S. minima	*H. rugosus?*	Mediterranean Sea	Pearre (1976, 1979)
S. setosa	Unidentified	North Sea	Leuckart and Pagenstecher (1858)
	Unidentified	Off Scotland	McIntosh (1890, 1927)
	Derogenes varicus	English Channel	Lebour (1916)
	D. varicus	Wadden Sea	Künne (1952)
	D. varicus	English Channel	Øresland (1986)
	Opechona bacillaris	English Channel	Lebour (1916)
	Hemiurus communis	Off Scotland	Meek (1928)
	Lecithochirium sp.	English Channel	Øresland (1986)
	Adolescaria progastrica	English Channel	Øresland (1986)
	Monascus filiformis	English Channel	Øresland (1986)
	Didimozoid	English Channel	Øresland (1986)
S. tenuis	*Hemiuris* sp.	Caribbean	Dawes (1958)
Pt. draco	*Metacercaria owreae*	Caribbean	Suarez-Caabro (1955)
Spadella sp.	Unidentified	Off Scotland	McIntosh (1890, 1927); Scott (1986)
Sp. cephaloptera	Unidentified	Normandy coast	Claparède (1963)
	Lecithaster sp.? or *Lecithochirium* sp.?	N-E Atlantic	Dollfus (1960)

Køie (1975) described the life cycle of *Opechone bacillaris*. The first intermediate host is the prosobranch mollusc *Nassarius pygmaeus* (Lamarck), the cercariae then enter ctenophores, small medusae or chaetognaths. The most common final host is the mackerel *Scombrus scombrus*. Experimental infestation was carried out using *Sagitta elegans*; these were always lying on the bottom of the dish and the cercariae easily penetrated their body wall and moved to the posterior end of the coelom. How the free-swimming cercariae in nature are able to locate and enter free-swimming chaetognaths is not clear. Køie (1975) did not succeed in feeding cercariae to chaetognaths although most did swallow copepods. Lebour (1916) sometimes found metacercariae of *Opechone* sp. in the alimentary canal of chaetognaths and suggested that the parasites are swallowed and afterwards move through the intestinal wall into the body coelom. Apparently, the presence of metacercariae in the water did not seem to affect chaetognaths and medusae (Køie, 1975).

The first intermediate host of *Metacercaria owreae* Hutton, 1954 is most probably a bottom-dwelling mollusc and the final host the sun fish *Mola mola* (Dawes, 1958, 1959).

Dollfus (1960) reviewed the older literature on trematode parasites in Chaetognatha and tried to apply modern species names. The trematodes found in Chaetognatha belong to the Families Hemiuridae Looss, 1899 *(Hemiuris, Derogenes)*; Lecithochiriidae Skrjabin and Guschanskaja, 1954 *(Lecithochirium)*; Accacoelioidae Looss, 1912 *(Accacladium, Tetrochaetus)*; Lepocreadiidae Nicoll, 1934 *(Opechona)* and Fellodistomidae Nicoll, 1909 *(Adolescaria, Monascus)* (Table 4-1).

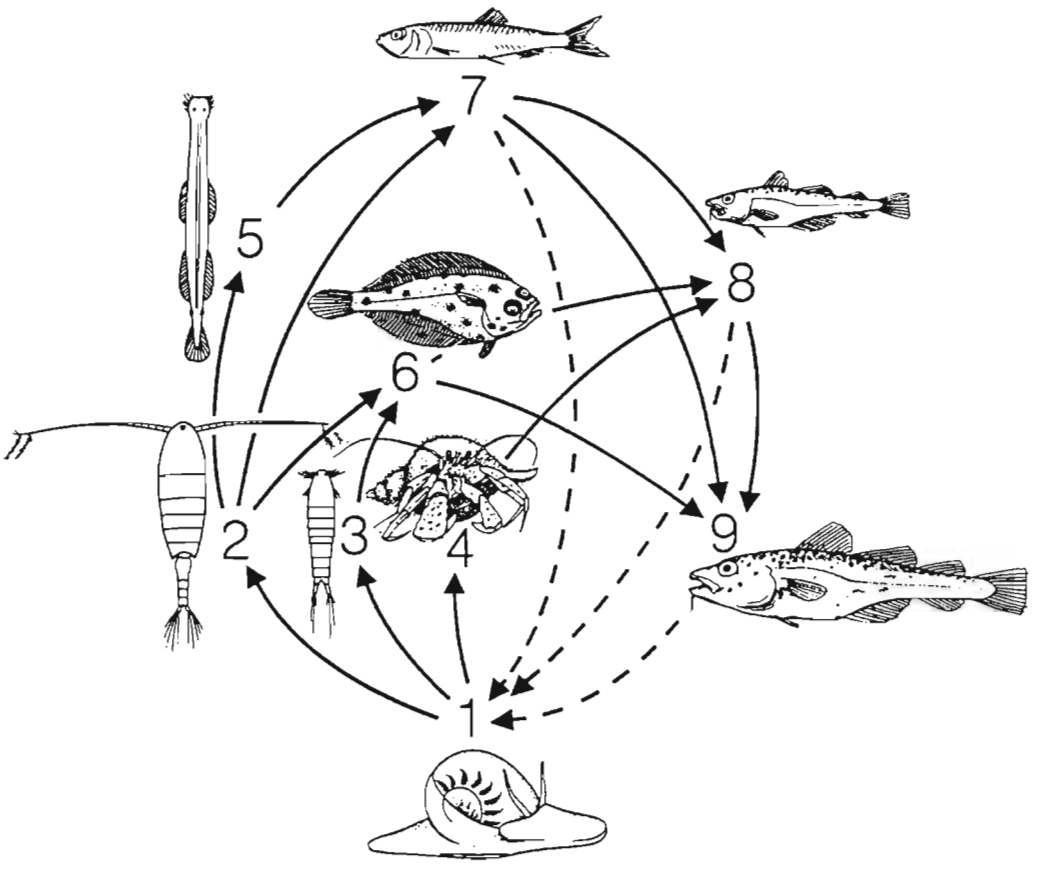

Fig. 4-2: *Derogenes varicus*. Life cycle (1) *Natica* spp.; (2) calanoid copepod; (3) harpactoid copepod; (4) hermit crab (or other decapods); (5) *Sagitta* spp.; (6) small fishes; (7) planktophagous fishes; (8) benthophagus and piscivorous fishes and *Sepia officinalis*; (9) piscivorous fishes, e.g., large cod. (After Køie, 1979a.)

Agents: Cestoda

Chaetognaths appear to be infrequent hosts for cestodes; very few cestodes have been documented to occur in Chaetognatha. Grey (1931) found a Cysticercus in the trunk coelom of *Sagitta friderici* off the Society Islands. Vanucci and Hosoe (1952) detected a cestode larva in *Pterosagitta besnardi (= P. draco)* from the South Atlantic, and Dawes (1958) found a very young larval cestode in *S. tenuis* from the Caribbean.

A procercoid cestode larvae was reported in the trunk coelom of *Sagitta setosa* from the English Channel by Øresland (1986). Scolex-type larvae (Trephyllidae) were mentioned by Dollfus and co-authors (1954) in *S. enflata* off the Madras coast. Reimer and co-authors (1971) reported *Scolex pleuronectis* Muller, 1788 in *S. elegans* from the North Sea. Kulachkova (1972) found *Scolex pleuronectis* and *Pseudophyllita* sp. in *S. elegans* from the White Sea. She established infection rates ranging from 0.02 to 0.4 %, usually being higher in spring than in other seasons. Weinstein (1972) found *Scolex pleuronectis* in *S. elegans* from Atlantic Canadian waters; the infection rate was 0.02 %.

Agents: Nematoda

Nematodes have been found frequently in Chaetognatha (Table 4-2). They are mostly indicated as larval ascaroids; often identification to species is not possible, as larval ascaroids of the different genera are very similar. Mostly they are placed in the genus *Contracoecum*.

Van Banning (1967) found *Contracoecum* larvae in *Sagitta* sp. from the North Sea, in samples from 50 to 56°N. He looked for *Anisakis* larvae, but did not find any. Reimer and co-authors (1971) found larvae of *Contracoecum* sp. and *Anisakis* sp. in *S. elegans* from the North Sea; *Anisakis* larvae were found only at latitudes higher than 57°N, so apparently herring is infected at higher latitudes.

Mazzoni (1986) reported *Contracoecum* sp. larvae in *Sagitta friderici*. *S. gazellae, S. tasmanica* and *Eukrohnia hamata* from the South Atlantic. Infestation rates were 0.13%, 0.08%, 0.82% and 0.14% respectively. Only the near-shore populations of *S. tasmanica, S. friderici* and *Eukrohnia hamata* were infested. *S. tasmanica* was infested only in waters of less than 33.7‰ S. Weinstein (1972) found an infestation rate of 0.02% with *Contracoecum* sp. in *S. elegans* from Atlantic Canadian waters.

Camillanus trispinosus was found in the intestine of *Sagitta friderici* off the Society

Table 4-2
Nematodes found in chaetognath species

Chaetognath	Nematode	Sea area	Source
Sagitta sp.	Nematodes	Off Scotland	McIntosh (1890, 1927); Scott (1896)
	Ascaris sp.	Mediterranean Sea	Pierantoni (1914)
	Contracoecum sp.	North Sea	van Banning (1967)
S. elegans	*Ascaris* sp.	English Channel	Lebour (1916)
	Contracoecum sp.	North Sea	Reimer and co-authors (1971)
	Contracoecum sp.	White Sea	Kulachkova (1972)
	Contracoecum sp.	N-W Atlantic	Weinstein (1972)
	Anisakis sp.	North Sea	Reimer and co-authors (1971)
S. enflata	Nematodes	Off S-E Australia	Thomson (1947)
S. euxina	Nematodes	Black Sea	Moltschanoff (1909)
	Hysterothylacium aduncum	Black Sea	Elian (1960)
S. friderici	*H. aduncum*?	S-W Atlantic	Jarling and Kapp (1985)
	Contracoecum sp.	S-W Atlantic	Mazzoni (1986)
	Camillanus trispinosus	Society Islands	Grey (1931)
S. gazellae	*Contracoecum* sp.	S-W Atlantic	Mazzoni (1986)
S. hispida	Nematodes	N-W Atlantic	Reeve and Cosper (1975)
S. setosa	*Ascaris* sp.	English Channel	Lebour (1916)
	Ascaris sp.	North Sea	Burfield (1927)
	H. aduncum	English Channel	Øresland (1986)
Eukrohnia tasmanica	*Contracoecum* sp.	S-W Atlantic	Mazzoni (1986)
S. hamata	*Contracoecum* sp.	S-W Atlantic	Mazzoni (1986)
Spadella sp.	Nematodes	Off Scotland	McIntosh (1890, 1927)

Islands (Grey, 1931). Grey found the nematodes extremely vigorous and thinks that the presence of nematodes in the tail coelom renders impairs the male sexual products, as the sperm morulae are broken up by the permanent movement of the parasite. Reeve and Cosper (1975) mention that the nematodes move around in the body coelom of *S. hispida* pushing the ovaries to the side.

Agents: Polychaeta

Pierrot-Bults (unpubl.) found *Sagittella kowalewskii* Wagner, 1872 attached to the body wall of *Sagitta lyra* (in 1 case of *S. hexaptera*) in the mid North Atlantic Ocean. Whether the polychaete is an ectoparasite, a passenger or a scavenger of the chaetognath is uncertain. *S. kowalewskii* was also found free in the same samples. Since chaetognaths are extremely delicate and die easily if damaged, a polychaete attached to their body wall might cause immediate death. That polychaetes are found only on *S. lyra* (with 1 exception) points more to a host/parasite relation than to feeding on dead or dying chaetognaths which would be expected to be indiscriminate. Feigenbaum (1979) reports *Typloscolex* sp. as a predator or ectoparasite of *Sagitta enflata* from the Gulf Stream.

Agents: Copepoda

Thomson (1947) found a copepod attached to *Sagitta decipiens* in Australian waters, and Ghirardelli (1948) reported copepods on *S. hispida* and *S. bedoti* in the Mediterranean Sea. Reimer and co-authors (1971) report parasitic copepods in different stages of development in the coelom of *S. elegans* from the North Sea.

ABNORMALITIES

Abnormal growth and tumor-like appendages on chaetognaths of the Japan Sea have been established by Nagasawa and Marumo (1979). Ghirardelli (1948) described the regenerating capacities of chaetognaths: while tail and fins sometimes regenerate, muscles do not; if a chaetognath is wounded the wound simply heals. The abnormal specimens pictured and described by d'Orbigny (1843) as *Sagitta diptera* and *S. triptera* may have suffered from bacterial damage instead of showing artefacts due to fixation or preservation.

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5. DISEASES OF ECHINODERMATA*

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The phylum Echinodermata is a very ancient group of exclusively marine animals. Recent echinoderms belong to 5 different classes: Crinoidea (ca. 700 species), Holothuroidea (ca. 1,100 species), Echinoidea (ca. 900 species), Asteroidea (ca. 1,800 species), and Ophiuroidea (ca. 2,000 species). They show conspicuous differences from all other invertebrates, such as radial (pentamerous) symmetry and presence of a water-vascular (ambulacral) system, and exhibit great diversity in structure and function (e.g., Ludwig and Hamann, 1889–1907; Hyman, 1955; Jangoux and Lawrence, 1982). Only a few species of echinoderms are of direct economic importance (Sloan, 1985). Gonads of some species of regular echinoids are consumed as delicacies by people in several places distributed all over the world. Holothuroids, also called 'trepang' or 'bêche de mer', are fished commercially in the whole Indo-West Pacific area; however, their utilization as seafood is restricted mainly to eastern Asia.

Representatives of most marine phyla are known to live in close association with echinoderms (for reviews see Ludwig and Hamann, 1889–1907; Hyman, 1955; Barel and Kramers, 1977; Jangoux, 1984, 1987a–d). Authors who reported such associations focussed in most cases on associated species — their taxonomy and morphology — and discussed very briefly, if at all, the true nature of the host-associate relation. Consequently, many associated species were termed parasitic or semi-parasitic when they were in reality commensal or simply phoretic (see Volume I, pp. 18 and 19). So far, most reports on biotic diseases of echinoderms refer to animal agents. Disease agents are not necessarily parasites in the classical sense, i.e., associated organisms which are "provided with host substances that are essential for their nutritional requirement" (Baer, 1951, p. 6). Numerous echinoderm associates may exert detrimental effects although they do not feed on the host's tissues or fluids. In this context Kinne's (1980, p. 19) definition of parasites is more satisfactory as it does not imply an obligatory trophic relation but only a detrimental effect. According to Kinne "parasitism involves an intimate coexistence of heterospecific organisms which is characterized by the fact that one of the organisms involved (the parasite) obtain benefits (e.g., energy, matter) at the expense of the other (the host); parasitism often tends to result in demonstrable negative effects in the host. The metabolic dependence of the parasite may be facultative (facultative parasite) or obligate (obligate parasite)". I have adopted that definition and used it in this review in a very broad sense, considering disease agents (parasites *sensu lato*) to represent any kind of a potentially harmful associate which affects, if even slightly, the host's tissues or internal fluids (i.e., coelomic and hemal fluids). Although a great deal of echinoderm parasites have been known for more than 100 years, there is little published information on host-parasite

* This chapter is based on reviews previously published in the journal "Diseases of Aquatic Organims" (Jangoux, 1987a–d).

relations or on the effect of parasitism on echinoderm life-cycles. Interestingly, echinoderms — while rather intensively parasitized — never act as parasites themselves.

Immune defense mechanisms of echinoderms have been rather intensively studied during the past 10 years. Cellular defense mechanisms act through phagocytic coelomocytes whose ability to phagocytose and/or to wall off the unwanted material entering the echinoderm body cavities is well-known (Smith, 1981; Bang, 1982; Karp and Coffaro, 1982; Dybas and Fankboner, 1986). Cell-types similar to coelomocytes have been seen also in experimentally altered echinoderm body walls (pathological alteration or reaction to allografts: Höbaus, 1980; Karp and Coffaro, 1982; Gilles and Pearse, 1986; Maes and co-authors, 1986). These cells massively invade the altered areas producing an inflammatory-like reaction. Moreover, coelomic fluids of echinoderms possess naturally-occurring humoral factors such as hemolysin and hemagglutinin (Ryoyama, 1973, 1974; see also Bang, 1982; Canicatti and co-authors, 1987) and show bactericidal and fungicidal properties (Wardlaw and Unkles, 1978; Service and Wardlaw, 1984, 1985; Turton and Wardlaw, 1987; Wardlaw and co-authors, 1988). Some echinoderm species produce antiviral and antifungal substances (Shimizu, 1971; and Shimada, 1969, respectively).

I shall first consider the different groups of potential biotic disease agents — ranging from Bacteria to Chordata — which are known to affect echinoderms, and then consider the location and effects of these agents as well as the reaction and sensitivity of echinoderms to pathogens. Definitive information on the biotic diseases of echinoderms is still rare. Consequently, I have chosen to follow the general format of the treatise in considering in this overview all external and internal echinoderm associates whose detrimental effects were demonstrated or documented, as well as intradigestive symbiotes that act or may potentially act as parasites. The latter still require critical investigation as to their role — benefital, neutral or detrimental — in relation to the echinoderms' life, i.e., its ecological potential and health status.

DISEASES CAUSED BY MICROORGANISMS

Agents: Bacteria

Non-pathological bacteria occur naturally in echinoderms, for example, gut-associated bacteria of some echinoids (e.g., Unkles, 1977, Guerinot and Patriquin, 1981 who studied regular echinoids; De Ridder and co-authors, 1985 who reported on spatangoid echinoids) or subcuticular bacteria observed in most echinoderms (e.g., Holland and Nealson, 1978). However, healthy echinoderms never harbor bacteria in their coelomic fluid which is normally sterile (Bang and Lemma, 1962; Wardlaw and Unkles, 1978; Kaneshiro and Karp, 1980). According to Bang and Lemma, bacteria-infected coelomic fluid occurs in *Asterias forbesi* when this asteroid undergoes autotomy or is traumatized dermally. They reported also that such infections generally prevail when *A. forbesi* is collected from stagnant water, and disappear progressively after the asteroid had been returned to running sea-water. Bang and Lemma noted moreover that coelomic-fluid infection is accompanied by weight loss (presumably due to loss of coelomic fluid) correlated with the intensity of the infection.

Experimental infection of coelomic fluid of healthy asteroids or echinoids showed that bacterial (as well as viral, i.e., exotic virus) suspensions are cleared rather quickly from the

body-cavity of echinoderms (Bang and Lemma, 1962; Coffaro, 1978; Wardlaw and Unkles, 1978; Kaneshiro and Karp, 1980; Yui and Bayne, 1983). The elimination of bacteria appears to be chiefly the consequence of the activity of phagocytic coelomocytes (Johnson, 1969; Johnson and co-authors, 1970; Johnson and Chapman, 1971; Kaneshiro and Karp, 1980). Antibacterial activities of coelomocytes are not restricted to phagocytosis: some echinoid coelomocytes release mucins which immobilize microorganisms entering the coelom (i.e., vibratile cells: Johnson, 1969) or produce bactericidal substances (i.e., red spherule cells: Johnson, 1969; Johnson and Chapman, 1971; Wardlaw and Unkles, 1978; Messer and Wardlaw, 1980; Service and Wardlaw, 1985; Wardlaw and co-authors, 1988). The bactericidal substances produced by the red spherule cells are naphthoquinone pigments belonging to the spinochrome (echinochrome) group (Service and Wardlaw, 1984; Maes and co-authors, 1986).

Individuals of several species of littoral regular echinoids suffered from a spectacular disease — the bald-sea-urchin disease (Table 5-1) — causing conspicuous lesions on the body surface (Fig. 5-1). Generally this disease develops as follows (Johnson, 1971b; Maes and Jangoux, 1984; Maes and co-authors, 1986; Roberts-Regan and co-authors, 1988): (1) the epidermis surrounding some spine bases turns green or purplish-black; (2) spines and other appendages are lost and the green (or purplish-black) epidermis and its underlying dermal tissue become necrotic; (3) epidermis and superficial dermal tissue are lost and circular-to-elongate denuded test areas are formed; (4) the upper layer of the skeleton is partially destroyed. When of limited size, the diseased individuals may recover: the affected skeleton is simply eliminated (Fig. 5-2), and the body wall tissues and outer appendages are regenerated. According to Maes and Jangoux (1984) death occurs either with lesions extending over a large area (more than 30 % of the total body surface) or with deep lesions involving test perforations (see also Gilles and Pearse, 1986). Affected echinoids develop a conspicuous inflammatory-like reaction around the area concerned. There is a massive migration of coelomocytes — phagocytic cells and red spherule cells — around and within the affected area (Johnson, 1971b; Maes and Jangoux, 1984; Gilles and Pearse, 1986; Maes and co-authors, 1986).

The bald-sea-urchin disease is communicable. Pieces of necrotic tissues initiate the disease when painted on experimentally produced injuries of the outer body surface of healthy echinoids (Maes and Jangoux, 1984; Gilles and Pearse, 1986). Maes and Jangoux also showed that the disease is not species specific: it is easy to experimentally infect regular echinoids of several different species.

The causative agent of the bald-sea-urchin disease is of bacterial nature (Maes and Jangoux, 1985; Gilles and Pearse, 1986). Gilles and Pearse isolated 14 different bacterial strains from lesions of diseased *Strongylocentrotus purpuratus*. They demonstrated that only the isolates of *Vibrio anguillarum* and *Aeromonas salmonicida* — 2 well-known pathogenic marine bacteria — were able to initiate lesion formation in the laboratory. Both Maes and Jangoux (1984) and Gilles and Pearse (1986) concluded from experimental infectivity tests that a stress such as physical injury is necessary for the formation of characteristic lesions. As suggested by Maes and co-authors (1986), lesions may possibly be caused by bacteria that are resistant to the antibacterial substances naturally produced by the echinoids, namely the spinochrome pigments conveyed by the red spherule cells.

Several authors reported mass mortalities of echinoids, presumably due to the bald-sea-urchin disease. Mass mortalities affected 60 to 95 % of *Strongylocentrotus franciscanus*

Table 5-1
Records of bald-sea-urchin disease (After Maes & Jangoux 1984; expanded)

Species	Geographical area	Source
Allocentrotus fragilis	NE Pacific (California) ('red spot disease')	Boolootian and co-authors (1959), Giese (1961)
Arbacia lixula	Western Mediterranean Sea (French coast)	Höbaus and co-authors (1981), Maes and Jangoux (1984)
Cidaris cidaris	Western Mediterranean Sea (French coast)	Fenaux, in Maes and Jangoux (1984)
Echinus esculentus	NE Atlantic Ocean (Brittany coast)	Nichols (1979), Maes and Jangoux (1984)
Paracentrotus lividus	Western Mediterranean Sea (Alicante, Spain; French coast; S. Italy and Sicily; Rijeka, Yugoslavia), NE Atlantic Ocean (Brittany)	Höbaus and co-authors (1981), Boudouresque and co-authors (1980, 1981), Azzolina (1983), Maes and Jangoux (1984), Maes and co-authors (1986)
Psammechinus miliaris	English Channel (Normandy, France)	Maes and Jangoux (1984), Maes and co-authors (1986)
Sphaerechinus granularis	Western Mediterranean Sea (French coast), NE Atlantic Ocean (Brittany)	Höbaus and co-authors (1981), Maes and Jangoux (1984)
Strongylocentrotus droebachiensis	NW Atlantic Ocean (Nova Scotia)	Scheibling and Stephenson (1984), Roberts-Regan and co-authors (1988)
Strongylocentrotus franciscanus	NE Pacific Ocean (California)	Johnson (1971b), Pearse and co-authors (1977), Pearse and Hines (1979)
Strongylocentrotus purpuratus	NE Pacific Ocean (California)	Johnson (1971b), Gilles and Pearse (1986)

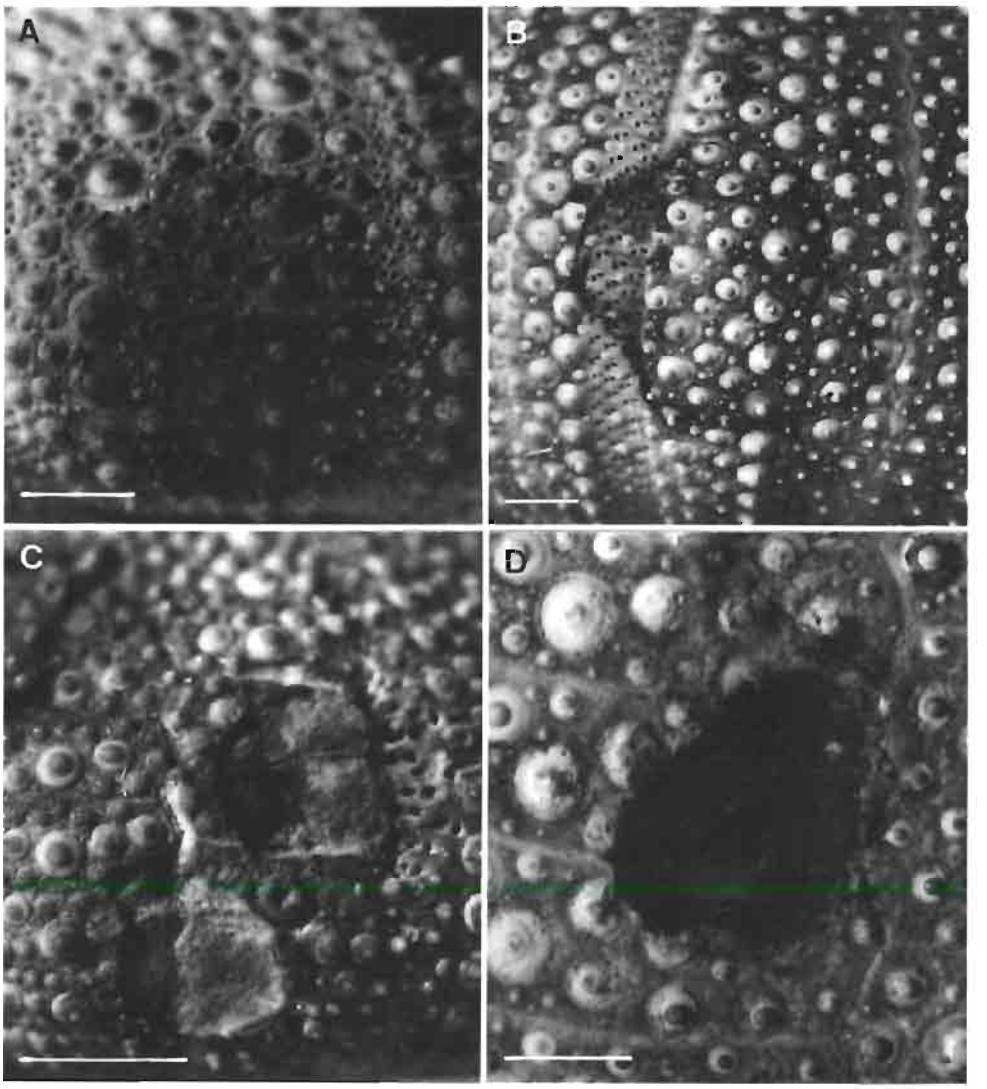


Fig. 5–1: Traces produced by bald-sea-urchin disease on the echinoid test skeleton (bar = 5 mm). A: *Paracentrotus lividus;* greenish-colored area (early infection) B: *Echinus esculentus;* greenish-colored area (late infection). C: *Psammechinus miliaris;* drepressed test area. D: *Echinus esculentus;* perforated test area. (After Maes and Jangoux, 1984.)

(Pearse and co-authors, 1977) and 10 to 75 % of *Paracentrotus lividus* (Boudouresque and co-authors 1980, 1981). Azzolina (1983) noted that mortality of *P. lividus* is higher in shallow water, and that diseased individuals are more numerous during summer. However, as noted by Gilles and Pearse (1986), they are also reports of lesions on occasional individuals in populations of otherwise healthy echinoids (e.g., Pearse and co-authors, 1977; Maes and Jangoux, 1984). Bacteria are not the causative agent of disease causing mass mortalities of the echinoid *Strongylocentrotus droebachiensis* along the Atlantic coast of Canada (e.g., Jones, 1985). Although characteristic body-wall lesions do sometimes

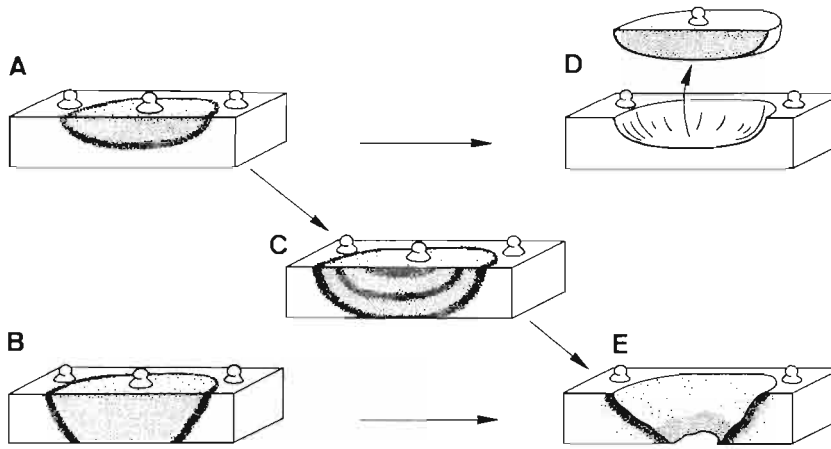


Fig. 5–2: Effects of bald-sea urchin disease on the echinoid skeleton. A: Superficial infection. B and C: Lethal infections. D: elimination of affected skeletal layer (usually followed by echinoid recovery). E: Perforation of skeleton involving death of the echinoid. (After Maes and Jangoux, 1984.)

occur on these echinoids (Scheibling and Stephenson, 1984), these appear to be symptoms of chronic, superficial infections from which echinoids generally recover (Roberts-Regan and co-authors, 1988). According to the latter authors, the increased incidence of lesions in species of regular echinoids during or after outbreaks of acute disease may reflect secondary bacterial and/or fungal infections (see also Gilles and Pearse, 1986). Whether or not bacteria causing the bald-sea-urchin disease may also induce echinoid mass-mortality remains to be proven.

Scattered information on other types of bacterial infection can be found in the literature. Olmsted (1917) noticed several small dark brown-green spherical masses of up to 1.5 mm in diameter in the body cavity of almost all individuals of the holothuroid *Synaptula hydriformis* examined. These masses consisted of 'bacterial parasites' belonging to the genus *Mycrocystus*. Mortensen (1935) observed granular epidermal swellings on the echinoid *Calveriosoma gracile*. According to him these swellings are very probably of bacterial nature. Delavault and Leclerc (1969) briefly reported a bacterial disease affecting *Asterina gibbosa* under aquarium conditions. Diseased asteroids show patches of epidermal necrosis which progressively unite and finally cause death. The echinoid *Diadema antillarum* was demonstrated to be susceptible to clostridial infection in laboratory conditions *(Clostridium parfringens* and *C. sordellii),* the infection being highly virulent (Bauer and Agerter, 1987).

Agents: Fungi

Mortensen (1909, 1928, 1936), Mortensen and Rosevinge (1910), and Koehler (1911, 1912) reported a very peculiar disease that affects several species of Antarctic cidaroid echinoids (genera *Rhynchocidaris* and *Ctenocidaris*). The disease is caused by an agent *(Echinophyces mirabilis)* which according to Mortensen (1909) is likely a fungus. The pathogen lives in the echinoid's primary spines which are much more slender and fragile than those of healthy echinoids (Fig. 5-3). As infected individuals are typically smaller than

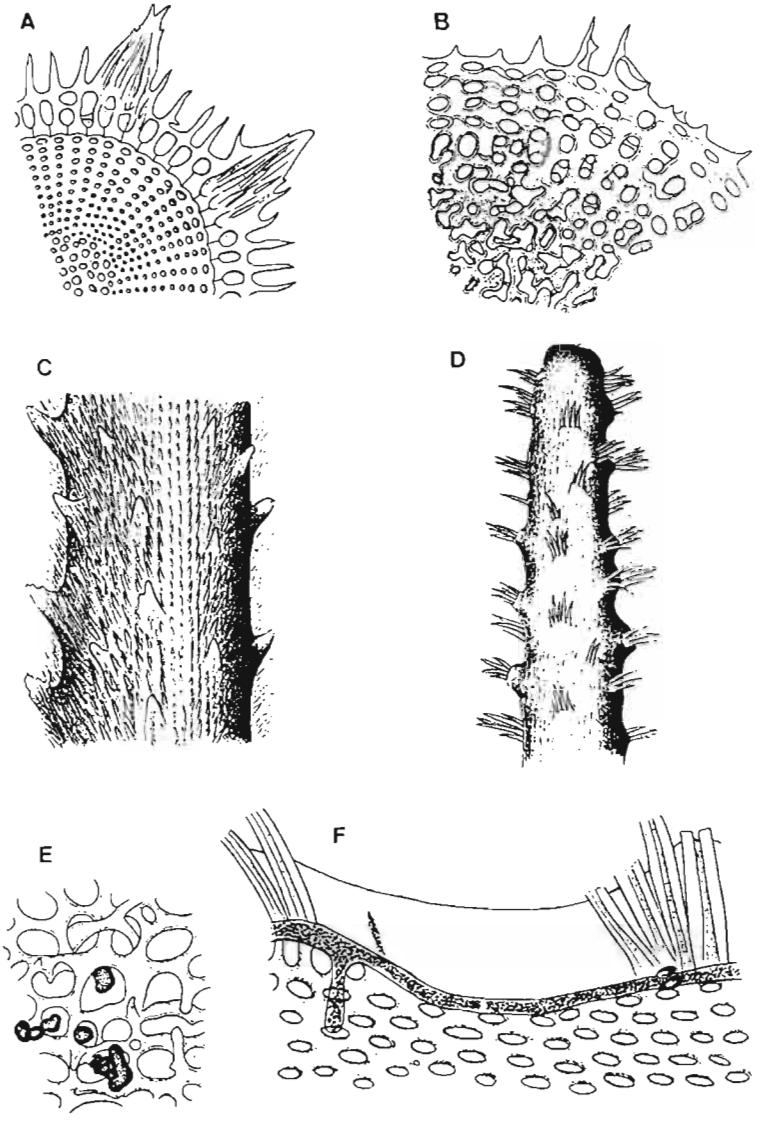


Fig. 5–3: Infection of primary spines of the cidaroid echinoid *Rhynchocidaris triplopora* by the fungus *Echinophyces mirabilis*. A and B: Cross-sections through (A) healthy and (B) infected primary spines. C and D: Outer views of (C) healthy and (D) infected primary spines. E and F: Aspect and location of parasite within a primary spine. (After Mortensen, 1909.)

healthy ones, Mortensen concluded that the parasite interferes with growth and dwarfs the specimens. He pointed out that the presumed fungus had no castrating effect but caused a very unusual abnormality on the echinoid test: in infected individuals the genital pores — presumably together with the genital or apical plates — are not in their usual place in the apical system but displaced to the edge of the peristome; consequently, new (?) genital ducts are formed leading to the peristomeal pores. Mortensen made no proposal as to how the parasite could 'move' the genital pores. He noted, however, that a few specimens had

their genital pores in the middle of the interambulacra. All this suggests that the fungus could perhaps modify normal echinoid test growth (new skeletal plates of the test always differentiate just at the outer edge of the apical plates and grow when migrating down; Märkel, 1981). In other words, the abnormalities observed could mean that the apical plates of infected echinoids lose their specificity in behaving like any other test plates and that they consequently migrate downwards. The position of genital pores — in the middle of the interambulacra or near the peristome — would consequently differ according to whether the echinoid was infected immediately following metamorphosis or later during juvenile growth.

No other fungal disease has been reported from echinoderms, except that Mortensen (1940) briefly described a fungus-like organism within the primary spines of *Diadema antillarum*. It causes hypertrophy of the calcareous mass of spines and an irregular growth of their edge. Johnson and Chapman (1970b) briefly reported the occurrence of fungi on and within the regenerating spines of some individuals of *Strongylocentrotus franciscanus*. Laboratory experiments by Turton and Wardlaw (1987) showed that the marine yeasts *Metschnikowia zobelli* and *Rhodotorula rubra* have definite pathogenicity for the echinoid *Echinus esculentus* when injected into its body cavity. They suggested the yeasts act by depressing the host defense mechanisms thereby allowing secondary bacterial infection to kill the echinoid (there is no data documenting the echinoid pathogenicity of these yeasts under natural conditions).

Agents: Cyanophyta

In a single specimen of the echinoid *Echinus acutus*, collected off Bergen (Norway), Mortensen and Rosevinge (1934) found the body wall to be locally deprived of appendages and epidermis. Patches of denuded test areas showed a typical blue-green color. According to the authors the disease was caused by the blue-green alga *Dactylococcopsis echini*. Actually the alga was identified on smears made from necrotic tissues but was not recognized in histological sections; the pathogenic nature of *D. echini* remains to be proven. The disease possibly destroys the calcified tissue and produces an intense reaction of amoeboid cells which invade massively the necrotic area. This symptomatology is similar to that of the bald-sea-urchin disease (p. 441).

DISEASES CAUSED BY PROTOZOANS

Agents: Flagellata and Sarcodina

As stated by Barel and Kramers (1977), the flagellate *Oikomonas echinorum* — noted by Cuénot (1912) in the general body cavity of most European echinoids — is nothing else than a particular type of coelomocyte known as 'vibratile cell'. According to Lecal (1980) the bodonid flagellate *Cryptobia antedonae* occurs in the coelomic fluid of *Antedon bifida*, but no information is given on the relation between crinoid and flagellate. Unidentified flagellates were noted also by Chesher (1969) in the body cavity of the spatangoid *Meoma ventricosa*.

An unidentified amoeboid protozoan infesting the ovaries of the echinoid *Arbacia lixula* near Naples (Italy) was recorded by Janssens (1903). The protozoan actively ingests ovocytes, with the parasitized echinoids remaining seemingly healthly.

Mass mortalities of the echinoid *Strongylocentrotus droebachiensis* occurred along the Atlantic coast of Nova Scotia, Canada, from 1980 to 1983 (Miller and Colodey, 1983; Scheibling and Stephenson, 1984). These mortalities were proven to result from a parameobiasis due to the species *Paramoeba invadens* (Jones, 1985; Jones and co-authors, 1985b; Jones and Scheibling, 1985). The symptomatology was described by Jones and co-authors (1985a) who reported that diseased echinoids show loss of peripheral muscle function in tube feet, spine, and mouth. The symptoms are diffuse and include general infiltration of tissues by coelomocytes, reduction of the numbers of red and white spherule cells, and incomplete clotting. Cultures of *P. invadens* reproduce the disease of healthy echinoids by both injection and water-borne route, and the pathogen appears to be highly host specific (Jones and Scheibling, 1985; Jellett and co-authors, 1988b). There is a direct relationship between water temperature and echinoid paramoebiasis (Scheibling, 1984; Scheibling and Stephenson, 1984). Jellett and Scheibling (1988a) reported that the growth rate of *P. invadens* in monoxenic cultures is maximal at 15 to 20°C which corresponds to annual sea temperature maxima in natural environment (see also Jellett and Scheibling, 1988b). Pathogenesis of paramoebiasis could possibly results from either the activation of autolysis in echinoids or the production of degradative enzymes by *P. invadens* (Jellett and co-authors, 1988a).

Agents: Sporozoa (Apicomplexa)

Apicomplexa parasitize only holothuroids and spatangoid echinoids. So far they have never been observed in crinoids, asteroids or ophiuroids. About 23 species are know from echinoderms, among them 22 gregarines and 1 coccidian (Table 5-2). They are undoubtedly many more species of Apicomplexa infesting echinoderms; numerous authors reported the occurrence — either in the hemal system or in the coelomic cavity — of cysts belonging to undescribed species (e.g., Hérouard, 1902; Changeux, 1961; Chesher, 1968, 1969; Brownell and McCauley, 1971; Jespersen and Lützen, 1971; Massin, 1984).

Echinoderms are infested mainly by members of 3 gregarine genera: *Cystobia*, *Lithocystis* and *Urospora* (Family Urosporidae, Order Eugregarinida; see Levine, 1977). Deposit-feeding echinoderms are very sensitive to gregarine infestations (Table 5-2). They may infest themselves simply by swallowing sediment that contains mature cysts. The cysts are broken down and sporozoites are liberated owing to the physico-chemical properties of the digestive fluid.

A more or less prolonged stay in the host's hemal system appears to be necessary for most gregarines infesting deposit-feeding holothuroids (Minchin, 1893; Cuénot, 1912; Pixell-Goodrich, 1925; Changeux, 1961). As shown by Changeux in *Holothuria* spp. simultaneously infested by 3 different species of *Cystobia*, throphozoites and subsequent cysts colonize particular hemal aras (see also Lützen, 1968b). Changeux (1961) claimed that the location may be considered a species-specific characteristic. With the exception of the species *Cystobia holothuriae*, said to be either intrahemal or intracoelomic (Changeux, 1961; Kroll and Jangoux, 1989; respectively), most of the life cycle of *Cystobia* spp. occurs within the holothuroid hemal system. Growth of the *Cystobia* trophozoite progressively occludes the host's hemal lacuna, and a peculiar hemal outgrowth is formed that looks like a bell-clapper protruding into the coelomic cavity. The clapper represents the so-called 'stalked gregarine' (Fig. 5-4). It is formed by an evagination of the underlying hemal lacuna whose distal end encloses an enlarged trophozoite or cyst. Mature cysts generally

Table 5-2
Parasitic sporozoans (Apicomplexa) from echinoderms. Hosts: E, echinoids; H, holothuroids (Compiled from the sources indicated)

Sporozoan[1]	Hosts	Location in host	Geographical area	Source
GREGARINIA				
Cystobia grassei	*Holothuria tubulosa*, *Holothuria stellati* (H)	Gut-associated hemal system; coelomic cavity	Mediterranean Sea (Banyuls)	Changeux (1961)
Cystobia holothuriae	*Holothuria tubulosa*, *Holothuria stellati* (H)	Gut-associated hemal system; coelomic cavity	Mediterranean Sea (Banyuls, Naples, Nice)	Schneider (1858), Mingazzini (1891), Minchin (1893), Changeux (1961); Kroll and Jangoux (1989)
Cystobia irregularis	*Holothuria forskali* (H)	Hemal system	English Channel (Plymouth)	Minchin (1893), Woodcock (1902, 1904, 1906)
Cystobia schneideri	*Holothuria tubulosa*, *Holothuria polii*, *Holothuria stellati* (H)	Gut-associated hemal system; coelomic cavity	Mediterranean Sea	Mingazzini (1891), Changeux (1961); Kroll and Jangoux (1989)
Cystobia stichopi	*Parastichopus tremulus* (H)	Radial hemal system (dorsal radius only)	North Sea (Oslo Fjord)	Lützen (1968b), Jespersen and Lützen (1971)
Diplodina gonadipertha[2]	*Cucumaria frondosa* (H)	On and in gonads	Barents Sea (Kola Bay)	Djakonov (1923)
Goniospora mercieri[3]	*Labidoplax digitata* (H)	Gut associated hemal system; coelomic cavity	N. E. Atlantic (Arcachon Bay)	Cuénot (1912), Barel and Kramers (1977)
Lithocystis brachycerus	*Chirodota laevis* (H)	Gut-associated hemal system; coelomic cavity	NW Atlantic (St. Andrews, New Brunswick)	Pixell-Goodrich (1925)
Lithocystis cucumariae	*Pawsonia saxicola* (H)	Respiratory trees	English Channel (Plymouth)	Pixell-Goodrich (1929)
Lithocystis foliacea	*Echinocardium cordatum* (E)	Coelomic cavity	Mediterranean Sea (Naples); English Channel (Plymouth, Wimereux)	Pixell-Goodrich (1915), Coulon and Jangoux (1987)
Lithocystis latifronsi	*Brisaster latifrons* (E)	Coelomic cavity	NE Pacific (off Oregon coast)	Brownell and McCauley (1971)
Lithocystis microspora	*Spatangus purpuratus* (E)	Coelomic cavity	English Channel	Pixell-Goodrich (1915)
Lithocystis minchini	*Pawsonia saxicola* (E)	Body wall (coelomic side)	English Channel (Plymouth)	Woodcock (1906), Pixell-Goodrich (1929)

Table 5-2 (continued)

Sporozoan[1]	Hosts	Location in host	Geographical area	Source
GREGARINIA (continued)				
Lithocystis oregonensis	*Brisaster latifrons* (E)	Coelomic cavity	NE Pacific (off Oregon coast)	Brownell and McCauley (1971)
Lithocystis schneideri	*Echinocardium cordatum* (E)	Coelomic cavity	NE Atlantic (Arcachon); Mediterranean Sea (Naples); English Channel (Cabourg, Dunkerque, Plymouth, Wimereux)	Giard (1876), Cuénot (1891, 1892, 1912), Léger (1896, 1897), Pixell-Goodrich (1915), De Ridder and Jangoux (1984), Coulon and Jangoux (1987, 1988)
Urospora chirodotae	*Chirodota laevis* (H)	Gut-associated hemal system	Barents Sea (Murmansk); NW Atlantic (St. Andrews, New Brunswick)	Dogiel (1906), Pixell-Goodrich (1915), Théodoridès and Laird (1970)
Urospora echinocardii	*Echinocardium cordatum*, *Spatangus purpureus* (E)	Coelomic cavity	English Channel (Plymouth)	Pixell-Goodrich (1915)
Urospora intestinalis	*Cucumaria japonica* (H)	Gut-associated hemal system	NW Pacific (Peter the Great Bay)	Bogolepova (1953, quoted by Changeux 1961)
Urospora neapolitana	*Echinocardium cordatum* (E)	Coelomic cavity	Mediterranean Sea (Naples); English Channel (Wimereux)	Pixell-Goodrich (1915), Coulon and Jangoux (1987)
Urospora pulmonalis	*Cucumaria japonica* (H)	Respiratory trees	NW Pacific (Peter the Great Bay)	Bogolepova (1953, quoted by Changeux 1961)
Urospora synaptae	*Leptosynapta galliennei*, *Leptosynapta inhaerens* (H)	Gut-associated hemal system; coelomic cavity	NE Atlantic (Arcachon, Morgat, Roscoff)	Cuénot (1891, 1892, 1912), Barel and Kramers (1970)
COCCIDIA				
Ixoreis psychropotae	*Psychropotes longicauda* (H)	Mostly gut-associated hemal-system	NE Atlantic (Bay of Biscay : deep sea)	Massin and co-authors (1978)

[1] Specific names according to Pixell-Goodrich (1915–1929) and Levine (1977). [2] Generic position unclear. [3] Possibly synonym of *Urospora synaptae* (see Barel and Kramers 1977).

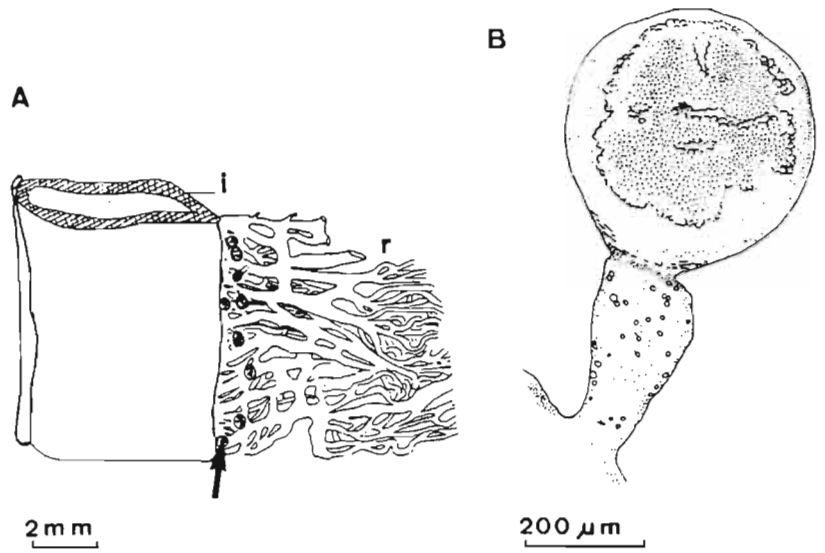


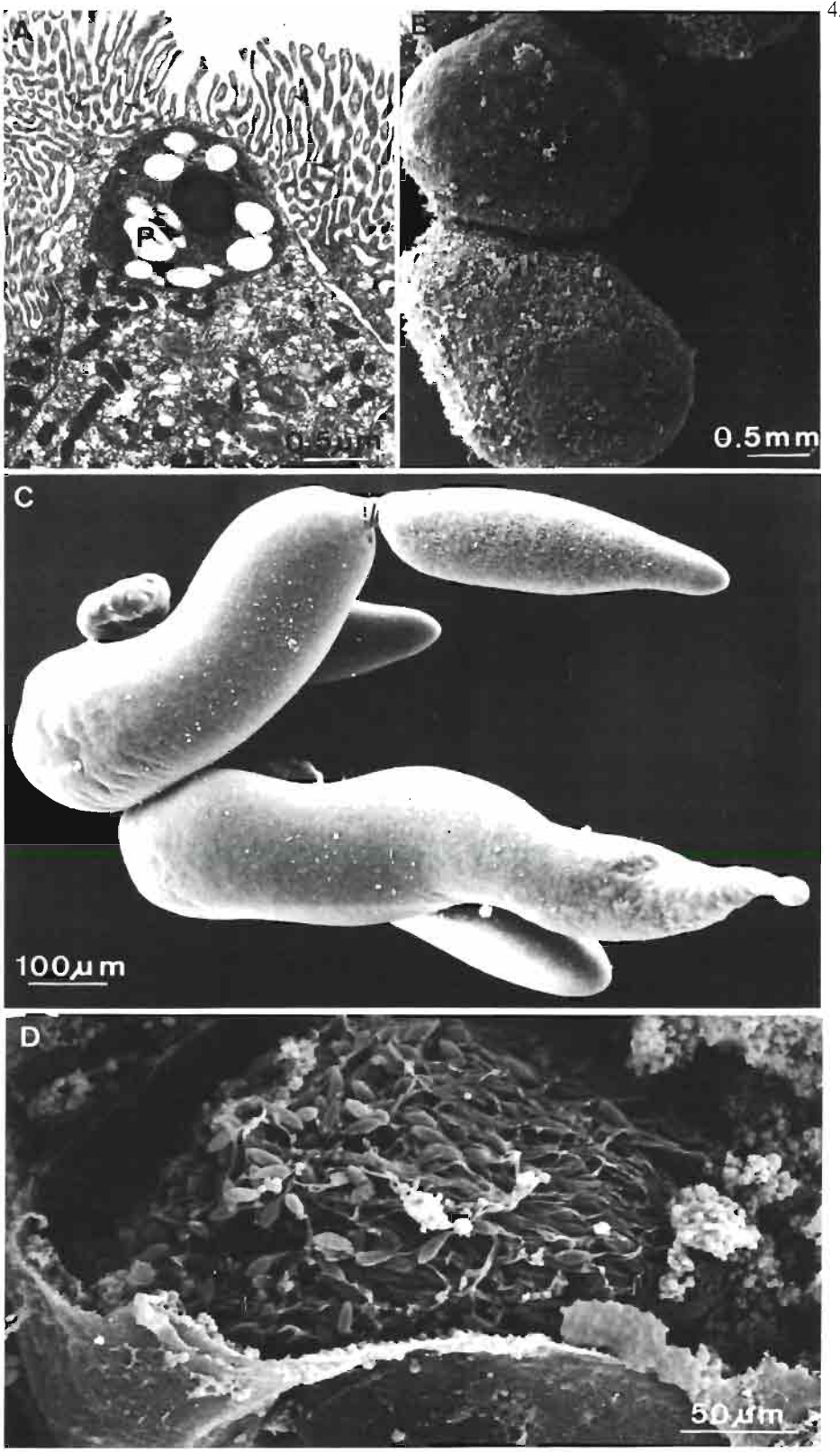
Fig. 5–4: *Cystobia grassei,* a stalked gregarine from the holothuroid *Holothuria tubulosa.* A: Section of gut and associated hemal system showing location of the cyst (arrow). (i) intestine; (r) rete mirabile. B: Stalked gametocyst. (After Changeux, 1961.)

reach the coelomic cavity either by the stalk breaking off or by tearing off its distal end. Cysts in the coelomic cavities frequently are embedded in brown-bodies (Briot, 1906a; Arvy, 1957; Changeux, 1961; Kroll and Jangoux, 1989).

Other parasitic gregarines from deposit-feeding holothuroids (*Lithocystis* spp. and *Urospora* spp.) do not produce 'hemal stalks'. *Urospora chirodotae* is known only from the gut-associated hemal system of *Chirodota laevis,* and seemingly does not occur in its coelomic cavity (Dogiel, 1906; Pixell-Goodrich, 1925). In contrast, *U. synapta* and *L. brachycercus* (Cuénot, 1912 and Pixell-Goodrich, 1925, respectively) occur in both the gut-associated hemal system and the coelomic cavity of their host, pre-mature cysts and even trophozoites being often present in the coelomic cavity.

In most gregarines from spatangoids, the whole life cycle occurs within the coelomic cavity (Fig. 5-5). Gametocysts are often embedded in brown-bodies (De Ridder and Jangoux, 1984; Coulon and Jangoux, 1987) as is also the case with gregarines from holothuroids. However, a peculiar and conspicuous host reaction occurs with spatangoid gregarines (Léger, 1897; Pixell-Goodrich, 1915; Fauré-Fremiet, 1926; Brownell and McCauley, 1971; De Ridder and Jangoux, 1984; Coulon and Jangoux, 1987, 1988). Some trophozoites are massively attacked by the host's coelomocytes which surround them. Coelomocytes progressively change their shape thus attaining a sharp-pointed appearance. The coelomocytes are so numerous and their reaction so intense that the parasite rapidly

Fig. 5–5: Life-stages of *Lithocystis* sp., a gregarine parasite of the spatangoid echinoid *Echinocardium cordatum.* A: Sporozoite within digestive cell of gastric caecum; (P) paraglycogen granule. B: Intracoelomic brown body containing gregarinean cysts. C: Intracoelomic gamonts in syzygyan process. D: Gametocyst with mature spores (B and C after De Ridder and Jangoux, 1984; A and D courtesy of C. De Ridder.)


A
P
0.5μm
B
0.5mm
C
100μm
D
50μm

takes on the appearance of a minute pin cushion. Such coelomocyte behavior has been interpretated either as a normal defense reaction against encysting trophozoites (Léger, 1897) or as a reaction only against necrotic trophozoites (Pixell-Goodrich, 1915; Brownell and McCauley, 1971). De Ridder and Jangoux (1984) reported that coelomocyte reaction could as easily affect single trophozoites as paired gamonts, depending presumably on gregarine motility. They suggested such reaction might be a way of preventing the formation of cysts. According to Coulon and Jangoux (1987) paired gamonts are more sensitive to coelomocytes than single trophozoites. The coelomocyte reaction always leads to the death of the gregarines: it results in the complete isolation of the trophozoites or syzygies from the surrounding environment; the gregarine cuticle is deformed and its micropores are obliterated while uric acid crystals develop and enlarge in the gregarine cytoplasm (Coulon and Jangoux, 1988; Coulon and co-authors, 1988).

Both Pixell-Goodrich (1915) and Coulon and Jangoux (1987) reported that several gregarine species may co-infest spatangoid echinoids. The latter authors recognized up to 5 species in individuals of a North Sea population of *Echinocardium cordatum*, viz. 3 intracoelomic species and 2 intrahemal species. Infestation takes place through the digestive cells of the gastric caecum where early growth of trophozoites occurs. Trophozoites reach then either the ambulacral hemal lacunae or the body cavity depending on the species. Differential sensitivity to coelomocytes appears to occur according to the species of intracoelomic gregarines (Coulon and Jangoux, 1987).

According to Brownell and McCauley (1971) the gonads of some gregarine-infested *Brisaster latifrons* contain 'encysted sporozoites' of *Lithocystis* sp. The authors strongly doubted that such sporozoite encystment is part of the normal life-cycle of the parasite as these sporozoites generally appear necrotic or partly decomposed. They noted, however, that the infestation of the gonad may be so high that its normal structure is changed and that it is filled with phagocytes and cell debris rather than germ cells.

A few gregarines parasitize suspension-feeding holothuroids of the genus *Cucumaria* (Woodcock, 1906; Djakonov, 1923; Pixell-Goodrich, 1929). Infestation presumably takes place through the respiratory current; cysts or spores lying on the sediment would be sucked up with the current through the cloaca and thus into the respiratory trees. Of the 2 gregarine species studied by Woodcock (1906) and Pixell-Goodrich (1929) *Lythocystis cucumariae* passes through its whole life cycle in the wall of the respiratory trees, while *Lythocystis minchini* is enclosed throughout most of its life cycle in cup-like outgrowths formed by the host's mesothelium and connective tissue on the inner side of the holothuroid's body wall. The gregarine agent of *Cucumaria frondosa,* investigated by Djakonov (1923), occurs only in the host's gonad. While Djakonov did not observe early infestation stages, he described almost the whole gregarine life-cycle, from growing trophozoite to dehiscent cyst. Small trophozoites mostly attach to the coelomic wall of the gonad, while enlarged (growing) trophozoites are located within the gonad's hemal lacunae. Cysts at different stages of sporulation lie exclusively inside the gonadal lumen. When mature, sporozoites are liberated into the gonad and discharged outside through the gonoduct. Host reaction takes place either within the hemal lacunae or within the gonadal lumen, since Djakonov reported the occurrence of invading coelomocytes that sometimes destroy the gregarines. He also reported that the parasite partially destroyed the gonad, the degree of destruction depending on the intensity of infestation.

The single species of Coccidia which parasitizes echinoderms, *Ixoreis psychropotae,* is

known from the deep-sea holothuroid *Psychropotes longicauda* (Massin and co-authors, 1978). Like most of the other holothuroid sporozoans, *I. psychropotae* was found in the gut-associated hemal system where it may occur in very high number (Fig. 5-6).

Apicomplexa presumably are present in most deposit-feeding echinoderms although only rather few species have been recorded. Even with the most common species information on their life cycle, biology and host effects is very limited. For instance, how do most echinoderms expel sporozoan cysts? Authors agree that ripe cysts of spatangoid gregarines are simply embedded into coelomic brown bodies which are liberated only at the echinoid's death. This implies that ripe cysts may have to wait several years before being able to start a new cycle (Coulon and Jangoux, 1987). Presumably holothuroid

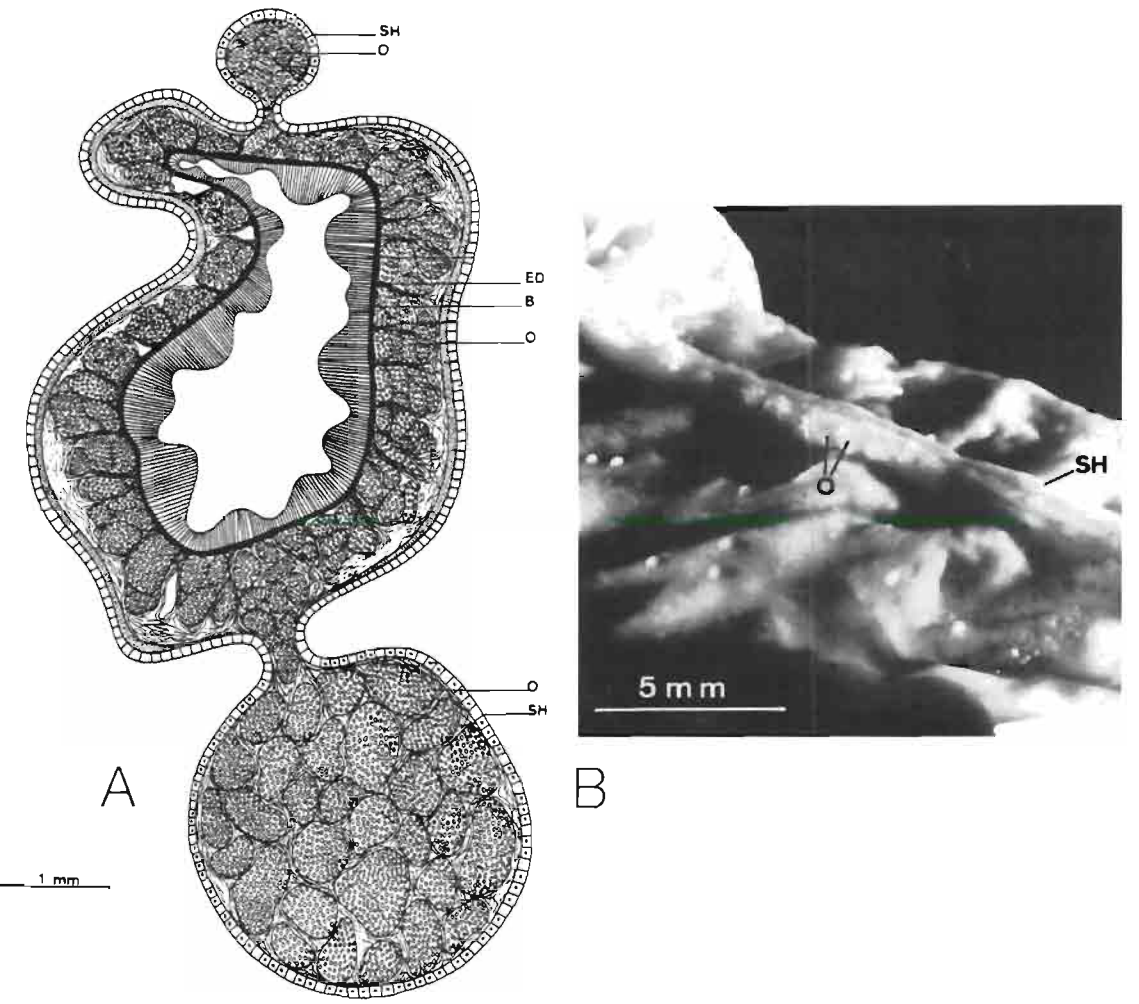


Fig. 5–6: *Ixoreis psychropotae*, a coccidian parasite of the deep-sea holothuroid *Psychropotes longicauda*. A: Diagramatic cross-section through intestine showing coccidian cysts in hemal lacunae and hemal vessels of the gut. B: Outer view of gut wall of an infested individual; (B) basal lamina. (ED) digestive epithelium; (O) coccidian cysts; (SH) hemal vessels. (After Massin and co-authors, 1978.)

gregarines are eliminated more rapidly as their hosts easily — sometimes seasonally — eviscerate. A variation in infestation levels, correlated with the seasonal evisceration of *Stichopus tremulus*, was reported by Jespersen and Lützen (1971) for an undescribed species of sporozoan inhabiting the hemal lacunae of the stomach. Undoubtedly, sporozoans induce an echinoderm immune response, for instance the coelomocyte reaction of spatangoids against free stage of gregarines (such reaction appears to occur also against some gregarine trophozoites infesting the body cavity of holothuroids; Kroll and Jangoux, 1989). Intracoelomic gametocysts are presumed to be inocuous while intracoelomic free stages are harmful, i.e., they do compete for energy supply (Coulon and Jangoux, 1987). The detrimental effect of gregarines will be thus directly linked to the number of free stages housed by the echinoderms, occurrence of cysts meaning only that individuals have suffered from gregarinose in the past.

Whether or not a host's reaction occurs against intrahemal free stages of gregarines is not documented. However, tissue (connective tissue) reactions were seen around intrahemal cysts (Pixell-Goodrich, 1929; Changeux, 1961). When mass-infestation occurs (such as those described in holothuroids by Pixell-Goodrich, 1925; Massin and co-authors, 1978; and Kroll and Jangoux, 1989) the hemal lacunae are everywhere distended by trophozoites and/or cysts, and the hemal fluid can no longer circulate. One could consider that formation of 'stalked gregarines', as seen in some holothuroids parasitized by *Cystobia* spp. (see Changeux, 1961; Kroll and Jangoux 1989), is actually a host reaction which removes parasites from the circulating hemal fluid.

Agents: Sporozoa (Ascetospora)

A single species of Ascetospora, the haplosporidian *Haplosporidium comatulae*, is known to parasitize echinoderms (LaHaye and co-authors, 1984). It was found in the gut hemal lacunae of the tropical Pacific comatulid *Oligometra serripinna*. Infested individuals harbored several life-history stages of the haplosporidian. The parasites have an obvious detrimental effect on their host in causing a marked reduction of the thickness of the gut wall.

Agents: Ciliophora

Ciliates associated with echinoderms have been reported by many authors. They live within the digestive system of some echinoderms — regular echinoids, crinoids, and synaptid holothuroids — as well as in the respiratory trees of a few holothuroids (for a detailed bibliography see Barel and Kramers, 1977). According to Powers (1935) and subsequent authors, these ciliates are clearly non-pathogenic and must be considered entocommensal protozoans strictly depending on the echinoderm digestive biotope for their survival.

The only ciliate which definitely acts as echinoderm parasite belongs to the species *Orchitophrya stellarum*, originally described by Cépède (1907a, b) who found it in the testes of the asteroid *Asterias rubens* (Fig. 5-7) (see also Bouland and co-authors, 1987). Since then, *O. stellarum* has been reported several times from asteroid gonads (Table 5-3). The ciliate parasitizes mostly male gonads in which it causes a progressive breakdown of germinal tissue. Most of the infested male asteroids observed by Vevers (1951) were completely castrated. Female gonads may also be affected but they are seemingly not

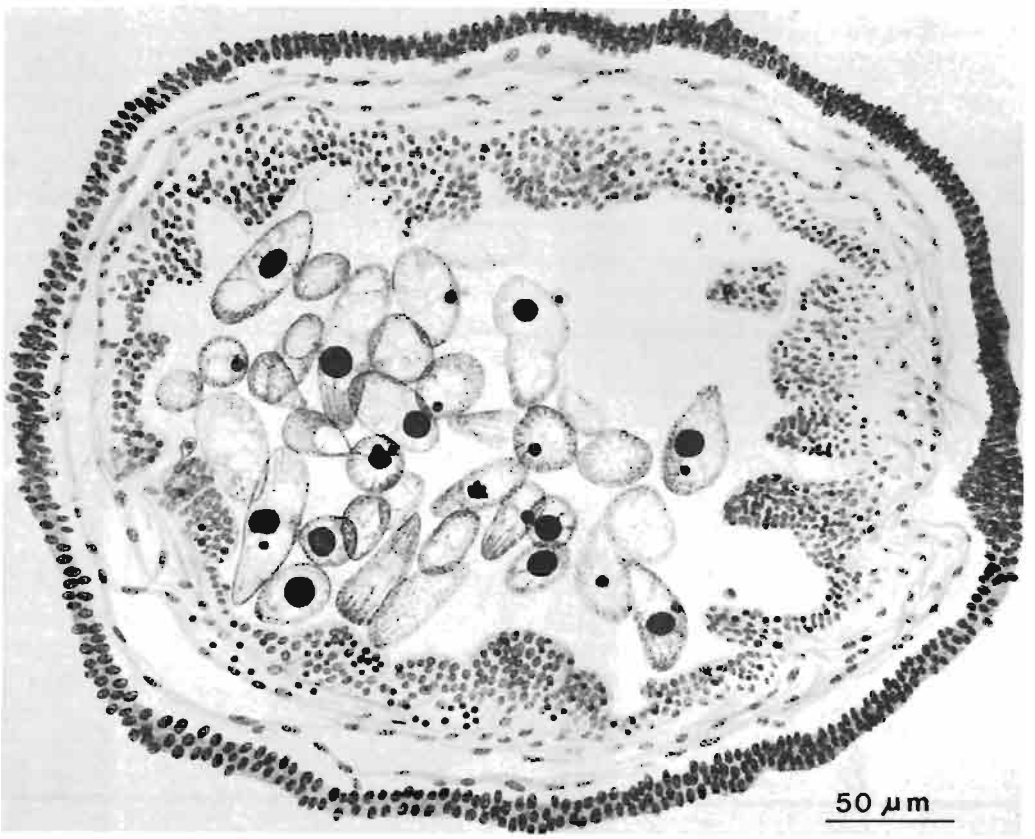


Fig. 5–7: *Asterias rubens*. Cross-section of testes showing infestation by the ciliate protozoan *Orchitophrya stellarum*. (After Cépède, 1910.)

destroyed as their eggs are said to be fertilizable (Smith, 1936). However, Burrows (1936) noted that spawned eggs often have ciliates within their membrane: the ciliates move around the yolk and consume it, apparently being unable to leave the egg membrane. Investigations by Bouland and Jangoux (1988) showed that *O. stellarum* disorganize the germinal epithelium and phagocytize germ cells, and that infested testes are massively invaded by the host's phagocytic cells that also phagocytize germ cells. They suggested that the phagocytic cells do not react against invading ciliates but against host tissues damaged by the parasites.

In view of the economic interest in controlling populations of oyster- or mussel-eating asteroids, attempts were made to experimentally infest *Asterias* spp. by *O. stellarum* but these were all unsuccessful (Cépède, 1910; Piatt, 1935; Burrows, 1936). A peculiar indirect effect of the infestation by *O. stellarum* was reported by Childs (1970) and Bang (1982), i.e., clumping of coelomocytes on glass did not occur in individuals of *Asterias forbesi* in which the ciliate was present in the gonads. According to Taylor and Bang (1978) and Bang (1982), recovery from infestation and return to normal glass-induced clumping of coelomocytes generally occur within 10 to 15 days under laboratory conditions.

Other ciliates claimed as echinoderm parasites have been noted. Ball (1924) briefly

Table 5-3
Infestation of asteroid gonads by the ciliate *Orchitophyra stellarum* (Compiled from the sources indicated)

Host	Incidence of parasitism	Geographical area	Source
Asterias forbesi	9 % of male asteroids infested (no data on female asteroids)	NW Atlantic (Long Island Sound)	Piatt (1935)
	Male asteroids (43 infested/326 investigated); female asteroids (4 infested/382 investigated)	NW Atlantic (Milford, Connecticut)	Burrows (1936)
	Infestations of male asteroids varied from 1 to 20 % depending on locality	NW Atlantic (Long Island Sound)	Galtsoff and Loosanoff (1939)
Asterias rubens	Male asteroids (3 infested/3000 investigated); no female asteroid infested	English Channel (Boulogne, Wimereux)	Cépède (1907a, b, 1910)
	1 to 28 % of the asteroid population infested according to season; no female asteroid infested	English Channel (Plymouth)	Vevers (1951)
	1 to 15 % of male asteroids infested	North Sea (Belgium; Netherlands)	Jangoux and Vloebergh (1973); Bouland and Jangoux (1988)
Asterias vulgaris	25 % female asteroids infested	NW Atlantic (Prince Edward Island)	Smith (1936)
	33 % of male asteroids infested; no female asteroids infested	NW Atlantic (Gulf of Maine)	Lowe (1978)
Sclerasterias richardi	0 to 25 % of the asteroids collected infested according to season	Mediterranean Sea (off Calvi, Corsica)	Febvre and co-authors (1981)

described new species of ciliates, supposedly parasites of the gut and the gonads of regular echinoids (*Diadema* sp., *Echinometra* sp., *Toxopneustes* sp.), and André (1910) and Cuénot (1912) noted the occurrence of the hymenostomatous holotrich *Cryptochilum echini* in gut, coelomic cavity and gonads of the echinoids *Paracentrotus lividus* and *Psammechinus miliaris*. No attempt was made to prove definitely the parasitic nature of these ciliates. Ciliates also occur in the coelomic cavity of *Lytechinus variegatus* which similarly harbors in its coelom individuals of the turbellarian *Syndysirynx franciscanus*. According to Jennings and Mettrick (1968) ciliates form the bulk of the diet of the turbellarians.

Experimental infestations of the coelomic cavity of *Asterias rubens* by the ciliate *Anophrys* sp. were performed by Bang (1975, 1982). When the asteroids were injected with the blood of crabs (genera *Carcinus* or *Cancer*) infested by *Anophrys* sp., the parasites were effectively cleared from the coelom within 6 h. After subsequent injections, the parasites were both cleared and lysed in less than 1 h.

DISEASES CAUSED BY PROTOPHYTANS (ALGAE)

Mortensen (1897) and Mortensen and Rosevinge (1910) described a unicellular green alga, *Coccomyxa ophiura,* that parasitizes 2 species of ophiuroids from the Danish seas, *Ophiura texturata* and *Ophiura albida,* and can also infest *Ophiura sarsi* (Mortensen, 1933a). The first signs appear on the aboral surface of disc and arms where the algae form small subepidermal patches of green color. The algae are generally located within the organic meshes of the calcareous plates of ophiuroids. According to the above-mentioned authors the algae progressively dissolve the skeletal plates forming irregular holes in which dense masses of algal cells occur. The algal masses grow into conspicuous subepidermal green cushions which progressively unite. Soon afterwards, the epidermis disintegrates inducing loss of arms and/or perforations of the disc, followed by death. Similar diseases caused by the closely related alga *Coccomyxa astericola* occur in the asteroids *Hippasteria phrygiana* and *Solaster endeca* (Mortensen and Rosevinge, 1933).

According to Johnson and Chapman (1970b) the regenerating spines of an abormally pale *Strongylocentrotus franciscanus* carried heavy internal infections by the diatom *Navicula* aff. *endophytica*. They suggested that this unusual infection could be linked to a shortage of red spherule cells. These cells are indeed thought to act as 'general disinfectant' in wounded and regenerating areas preventing tissue colonization by foreign cells (see also Johnson and Chapman, 1970a; Karp and Coffaro, 1982; Maes and co-authors, 1986). The single observation by Lawrence and Dawes (1969) of *Fosliella farinosa* (red coralline alga) growing over the spine epiderm of the echinoid *Heterocentrotus trigonarius* might also be related to a shortage in red spherule cells. A similar assumption might be made regarding the observation by Mortensen (1943a) of a brownish mass of incrusting algae (*Melobesia* sp.) partly covering the body surface of the echinoid *Temnopleurus hardwicki*.

Mass mortalities of the spatangoid echinoid *Echinocardium cordatum,* related to a dinoflagellate bloom, occurred along the east coast of Ireland (Helm and co-authors, 1974). These is no evidence that mortality was induced by dinoflagellate toxins. According to Helm and co-authors it resulted from decomposition of the bloom in conjunction with calm, sunny weather. These circumstances may have caused oxygen depletion in the substrate occupied by *E. cordatum*. Individuals which did not die in their burrow emerged

and succumbed to exposure or predation. A similar bloom-related mass mortality was reported by Cross and Southgate (1980) for the echinoid *Paracentrotus lividus*.

DISEASES CAUSED BY UNIDENTIFIED AGENTS

Mass mortalities of littoral asteroids and echinoids have been reported repeatedly since the early eighties. One can reasonably assume they were caused by as yet unidentified bacteria or protistans.

Mass mortalities of the asteroid *Heliaster kubinji*, a top carnivore in the rocky intertidal zone of the Gulf of California, were recorded by Dungan and co-authors (1982). Individuals initially exhibit whitish lesions on their aboral surface. The lesions rapidly enlarge until the entire animal fragments. High concentrations of bacteria are found in the lesions but it is not known whether bacterial infection is the primary cause of death. The authors hypothesize that prolonged elevated temperature, perhaps in conjunction with other factors, renders the asteroid increasingly susceptible to infection by an as yet unidentified pathogen. Mortality of *H. kubinji* approached 100 %, and within 2 weeks the asteroids had virtually disappeared from the study site.

A widespread and conspicuous mass mortality of the echinoid *Diadema antillarum* occurred in the Caribbean in 1983–1985 (Lessios and co-authors, 1983, 1984a, b; Bak and co-authors, 1984; Murillo and Cortés, 1984; Hughes and co-authors, 1985; Hunte and co-authors, 1986). Population densities of *D. antillarum* were reduced to 1 to 6 % of their previous levels (Lessios and co-authors, 1984a). Other species of echinoid remained unaffected, indicating a high level of agent specificity. Bak and co-authors (1984) and Hughes and co-authors (1985) briefly described the symptomatology of the disease: (1) accumulation of colorless mucus on many spines, especially at their tip; (2) development of dermal lesions at spots over the test and the peristome; (3) break and/or loss of spines; (4) progressive exposure of the whole skeleton and decomposition of the remaining tissue. Death of affected individuals occurred after 4 days or so from the first visible change. According to Hunte and co-authors (1986), echinoids with test diameter between 20 and 40 mm were more severely affected than smaller or larger individuals. Most authors agree in considering the causative agent to be a water-born pathogen transported by oceanic currents (see, e.g., Bauer and Agerter, 1987). Lessios and co-authors (1984a) pointed out that the effects of the causative mortality agent extended over a geographic area of ca 3.5 million km^2, causing the most widespread epidemic ever documented for marine invertebrates.

DISEASES CAUSED BY METAZOANS

Agents: Mesozoa

The Mesozoa, a small group of uncertain taxonomic affinity, comprise about 50 species of minute animals parasitic on marine invertebrates. One species, *Rhopalura ophiocomae*, parasitizes ophiuroids. Its most frequent host is the small cosmopolitan incubating amphiurid *Amphipholis squamata* (see Caullery and Mesnil, 1901; Kozloff, 1969; Rader, 1982) but it may — if very rarely — also affect other ophiuroid species, namely *Ophiothrix fragilis* and *Ophiura albida* (respectively Fontaine, 1968; Bender, 1972). *R. ophiocomae* is mostly known from European localities (Atlantic coast of France,

North Sea, northwest Mediterranean Sea; for reviews see Kozloff, 1969; Barel and Kramers, 1977), and also from 2 Pacific localities along the coast of Washington (Kozloff, 1969; Rader, 1982).

Structure and life cycle of *Rhopalura ophiocomae* were studied intensively at the beginning of this century, mainly by Caullery and Mesnil (1901) and Caullery and Lavallée (1908, 1912) (Fig. 5–8). Mature adults of *R. ophiocomae* are free living. Adults, either male or female, develop in *Amphipholis squamata* and are emitted through the ophiuroid's bursal slits. Their life span is short (a few days), and they give rise to ciliated larvae. These infesting larvae penetrate the ophiuroid bursal slits and intimately contact the outer epithelium of the bursae. Soon afterwards, small parasitic 'plasmodia' occur within the

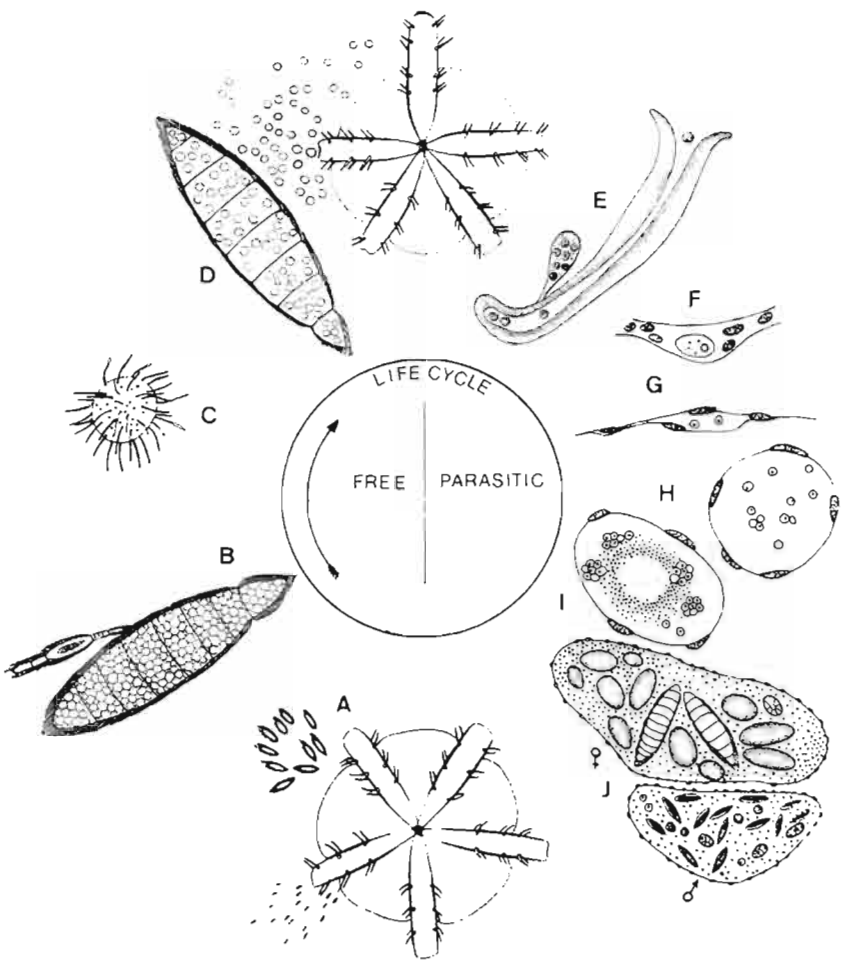


Fig. 5–8: *Rhopalura ophiocomae*. Life-cycle of a mesozoan parasite of the ophiuroid *Amphipholis squamata* (not to scale). (A) Male and female mesozoans emitted through the genital slits of an infested ophiuroid; (B) fertilization; (C) infesting mesozoan larvae; (D) release of infesting larvae from a female mesozoan; (E) infestation of ophiuroid bursae; (F) penetration of the larvae in the bursal epithelium; (G to I) developing plasmodia; (J) male and female plasmodia. (After Caullery and Lavallée, 1912.)

outer epithelium of the bursae. Subsequently, plasmodia migrate to the coelomic side of the bursae where they remain close to the ovaries. At that time plasmodia often protrude into the coelomic cavity. They are completely surrounded by an epithelial layer presumably formed by host mesothelium (Caullery and Mesnil, 1901; Rader, 1982). Whether each plasmodium derived from a whole larva or from one or more cells of that larva is not known. The plasmodia grow and some of them move along the coelomic lining. Fully developed plasmodia consist of an enlarged cytoplasmic (?) mass surrounded by an epithelium of host origin. Each plasmodial mass contains numerous small nuclei (the 'plasmodic' or 'vegetative' nuclei), some germ cells (sometimes called 'agametes') and a few embryos at different developmental stages. These are either males or females, embryos of both sexes within the same plasmodium being exceptional. When mature, the plasmodium presumably disintegrates and numerous adult *R. ophiocomae* are emitted into the outer medium through the host's bursal slit.

The pathogenicity of *Rhopalura ophiocomae* is unquestionable. Its most obvious effect is the regression of host ovaries, while the testes — as noted by several authors — remain functional (*Amphipholis squamata* is hermaphroditic). The parasite does not consume the ovaries; these regress as soon as small plasmodia invade the bursal wall. Ovarian regression implies that infested ophiuroids never harbor incubated embryos. Other consequences of the disease are a decrease in the ophiuroid's regenerative abilities, as well as probably a decrease in its growth rate (Rader, 1982).

Agents: Parazoa

There are only 2 sponge species known to parasitize echinoderms. Clark (1896, 1898) reported the occurrence of a *Grantia*-like species firmly attached to the outer body surface of several individuals of the holothuroid *Synapta vivipara.* The sponges always were seen at the base of the holothuroid buccal tentacles. Antarctic ophiuroids of the genus *Ophiurolepis* are very often parasitized by the sponge *Iophon radiatus* (Mortensen, 1936; Fell, 1961). The parasite fixes itself on the ophiuroid, and infestation is generally very extensive, the whole disc and the basal parts of the arms being involved.

As shown by Mortensen (1932), the peculiar sponge *Microcordyla asteriae* described by Zirpolo (1926) as an ectoparasite of the asteroid *Coscinasterias tenuispina,* actually represents a globiferous pedicellaria of the echinoid *Sphaerechinus granularis.* The pedicellariae probably were detachted in a defensive reaction of *S. granularis* (globiferous pedicellariae of echinoids autotomize easily).

Agents: Cnidaria

Several sea anemones attach to the body surface of echinoderms. Gravier (1918) noted the occurrence of the actinid *Sicyopus commensalis* partly embedded in the body wall of the deep-sea holothuroid *Pseudostichopus villosus.* Kropp (1927) reported echinoids of the genus *Diadema* with the sea-anemone *Aiptasia tagetas* firmly attached to their body surface near the anal cone. Other cnidarians may incidentally infest echinoderms, namely hydrozoans which live attached to the stem or the cirri of crinoids. Four crinoid-associated hydrozoans are known: *Calycella syringa, Cuspidella* sp., *Lafoea fruticosa* and *Stegopoma fastigiata* (Clark, 1921). A case of symbiosis between the

hydrozoan *Hydractina vallini* and several species of the Antarctic ophiuroid genus *Theodoria* has been reported by Smirnov and Stepanyants (1980). This symbiosis recalls the one between Antarctic ophiuroids and sponges. The single known case of hydrozoans living on asteroid was reported by Madsen (1961) who recorded unidentified athecate hydroids attached to the peristome of the deep-sea asteroid *Eremicaster gracilis*.

Agents: Turbellaria

While Turbellaria are mainly free-living, each order has developed representatives living in close association with other organisms. Symbiotic turbellarians were reviewed by Jennings (1971) (see also Stunkard and Corliss, 1951) who noted that echinoderms represent preferential shelters for turbellarians. Table 5-14 lists symbiotic turbellarians living with echinoderms; of the 67 species, 9 are Acoela; 61, Rhabdocoela (53 species belonging to the family Umagillidae); and 1, Polycladida. With very few exceptions (*Euplana takewakii* and *Acholades asteris;* respectively, Kato, 1935; Hickman and Olsen, 1955), almot all echinoderm-associated turbellarians live either within the digestive tract or within the coelomic cavity of their host. Symbiotic turbellarians have been reported for each echinoderm group, but most of these associates live in aspidochirote holothuroids (mainly Holothuriidae and Stichopodidae) or in regular echinoids. As noted in Table 5-4, massive turbellarian infestations occur rather frequently in echinoderms. There is, however, no information on the effect of parasitic Turbellaria on the echinoderm life cycle.

Gut-associated umagillids may either occur all along the digestive tract (Smith, 1973) or be more or less restricted to some digestive areas (Barel and Kramers, 1970; Shinn, 1981; Cannon, 1982; see also Table 5-4). Holt and Mettrick (1975) reported that *Syndisyrinx franciscanus* from the gut of *Strongylocentrotus purpuratus* feeds mostly on associated ciliates, harbored by the digestive tract of the echinoid. Snyder (1980) could determine neither beneficial nor detrimental effects due to the occurrence of gut-associated umagillids. He concluded that these symbiotes should be considered simply commensals. In contrast Shinn (1981) reported that the gut-associated umagillids always compete with their host for nutrients and thus may exert adverse effects. He noted that all the umagillids studied by him ingest intestinal host tissue — one of them subsisted entirely on that tissue (see also Cannon, 1982). Shinn suggested that gut umagillids parasitize their host to varying degrees. Giese (1958) noted that infestation level of *S. franciscanus* in the gut of *S. purpuratus* remains constant throughout the year and does not differ whatever the size, sex or gonadal stage of the echinoid. In contrast, *Wahlia pulchella* inhabiting the intestine of *Stichopus californicus* display a distinct annual cycle of infestation related to the annual feeding cycle of its host (worms do not occur in *S. californicus* in fall and winter when the host's viscera are resorbed) (Shinn, 1986b). According to Shinn (1980, 1983b), egg capsules of the gut-associated *S. franciscanus* leave the host gut with fecal material. Embryogenesis within capsules lasts approximately 2 months, and fully-formed embryos (infesting embryos) can survive in their capsule for about 10 additional months (Fig. 5-9). Embryos hatch after the capsules were ingested by an echinoid. Hatching is induced by some property of the host's digestive fluid and performed presumably owing to a hatching enzyme secreted by the embryos (Shinn, 1983b, 1986a).

Coelomic umagillids swim in the host's body cavity, seemingly without any particular intracoelomic location. They directly depend on their host for nutrition — they ingest the

Table 5-4

Turbellarians associated with echinoderms. Turbellarian species names according to Cannon (1982). Hosts: A, asteroid; C, crinoid; E, echinoid; H, holothuroid; O, ophiuroid (Compiled from the sources indicated)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
I. Acoela					
Aechmalotus pyrula	*Eupyrgus scaber* (H)	Digestive tract and respiratory trees	–	Barents Sea (Murmansk coast)	Beklemishev (1915)
Aphanastoma pallidum	*Myriotrokus rinki* (H)	Digestive tract	–	Barents Sea (Murmansk coast)	Beklemishev (1915)
Aphanastoma sanguineum	*Chirodota laevis* (H)	Posterior part of the digestive tract	–	Barents Sea (Murmansk coast)	Beklemishev (1915)
Avagina glandulifera	*Spatangus purpureus* (E)	Digestive tract	Many echinoids infested	English Channel (Plymouth)	Westblad (1953)
Avagina incola	*Echinocardium cordatum*, *Echinocardium flavescens*, *Spatangus purpureus* (E)	Digestive tract (including siphon)	5 % of the echinoid population infested (Leipert); 3 to 14 worms echinoid^{-1} (50 echinoids investigated) (Westblad)	NE Atlantic (Millport); North Sea (Norwegian coast); English Channel (Plymouth)	Leipert (1902, 1904), Westblad (1948, 1953)
Avagina vivipara	*Echinocardium cordatum* (E)	Esophagus	2 to 5 worms echinoid^{-1} (18 infested/68 investigated)	SW Indian Ocean (Ralph's Bay, Tasmania)	Hickman (1956)
Faerla echinocardii	*Echinocardium flavescens* (E)	Unspecified	–	? North Sea (Norwegian coast)	Dorjes (1972)
Meara stichopi	*Parastichopus tremulus*, *Mesothuria intestinalis* (H)	Anterior intestine; coelomic cavity	1 to 8 worms holothuroid^{-1} (Westblad)	North Sea (Norwegian coast: Herdla, Trondhjem, Oslofjord)	Westblad (1926, 1949), Jespersen and Lützen (1971)
Octocoelis chirodota	*Chirodota laevis* (H)	Anterior part of digestive tract	–	Barents Sea (Murmansk coast)	Beklemishev (1915)
II. Rhabdoecoela (f. umagillidae)					
Anoplodiera voluta	*Parastichopus tremulus* (H)	Digestive tract (anterior part)	Up to 90 worms holothuroid^{-1} (Westblad)	North Sea (Norwegian coast: Herdla, Trondhjem, Oslofjord)	Westblad (1926, 1930), Jespersen and Lützen (1971)

Table 5-4 (continued)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
II. Rhabdoecoela (f. umagillidae) (continued)					
Anoplodiera sp.	*Holothuria arenicola* (H)	Pharynx	Average infestation: 7 worms holothuroid^{-1} (9 infested/12 investigated)	Tropical W Atlantic (Bermuda)	Snyder (1980)
Anoplodiopsis gracilis	*Holothuria forskali* (H)	Coelomic cavity	Up to 9 worms holothuroid^{-1} (11 infested/47 investigated) (Wahl 1906)	Mediterranean Sea (Naples)	Wahl (1906, 1909), Westblad (1953)
Anoplodium chirodotae[1]	*Chirodota pellucida* (H)	Coelomic cavity		White Sea	Sabussow (1900, quoted by Barel & Kramers, 1977)
Anoplodium evelinae	Unidentified holothuroid	Coelomic cavity	Up to 50 worms holothuroid^{-1}	SW Atlantic (Brazil: Santos Bay)	Marcus (1949)
Anoplodium graffi	*Holothuria impatiens* (H)	Coelomic cavity	–	Mediterranean Sea (Naples)	Monticelli (1892), Westblad (1953)
Anoplodium hymanae	*Parastichopus californicus* (H)	Coelomic cavity	1 to 51 (average 15) worms holothuroid^{-1} (25 infested/27 investigated)	NE Pacific (Washington coast: Cowlitz Bay)	Shinn (1983a; see also Shinn 1985b)
Anoplodium longiductum	*Actinopyga* sp. (H)	Unspecified	–	SW Indian Ocean (Madagascar: Nossi-Bé)	Hyman (1960)
Anoplodium mediale	*Stichopus japonicus* (H)	Coelomic cavity	–	NW Pacific (Japan: Hiroshima)	Ozaki (1932)
Anoplodium myriotrochi	*Myriotrochus rinckі* (H)	Digestive tract	Infestation frequent	Arctic Seas (Spitzbergen)	Barel and Kramers (1977)

Table 5-4 (continued)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
II. Rhabdoecoela (f. umagillidae) (continued)					
Anoplodium parasita	*Holothuria tubulosa*, *Holothuria polii*, *Holothuria stellati* (H)	Coelomic cavity; rarely digestive tract or respiratory trees	1–4 worms holothuroid^{-1} (16 infested/26 investigated) (Changeux); infestation rate 80 % (Kroll and Jangoux)	Mediterrancan Sea (Banyuls, Naples, Trieste)	Schneider (1858), Monticelli (1892), Briot (1906b), Wahl (1906), Westblad (1953), Changeux (1961), Kroll and Jangoux (1989)
Anoplodium ramosum	*Stichopus variegatus* (H)	Unspecified (endoparasitic)	–	SW Indian Ocean (Madagascar: Nossi-Bé)	Hyman (1960)
Anoplodium stichopi	*Parastichopus tremulus* (H)	Coelomic cavity	Up to 30 worms holothuroid^{-1} (Westblad)	North Sea (Norwegian coast)	Bock (1926), Westblad (1926), Jespersen and Lützen (1971)
Anoplodium tubiferum	*Holothuria forskali* (H)	Digestive tract	–	English Channel (Plymounth)	Westblad (1953)
'*Anoplodium*' sp.[1]	*Leptosynapta bergensis*, *Leptosynapta galliennei*, *Leptosynapta inhaerens* (H)	Digestive tract	Rather frequent	NE Atlantic (Plymouth, Roscoff)	Cuénot (1912), Barel and Kramers (1970, 1977), Kramers (1971)
Bicladus metacrini	*Metacrinus rotundus* (C)	Digestive tract	'Occur in enormous number'	N Pacific (Japan: Sagami Sea)	Kaburaki (1925)
Cleistogamia heronensis	*Holothuria atra*, *Holothuria leucospilota* (H)	Anterior to midpart of digestive tract	–	Great Barrier Reef (Australia)	Cannon (1982), Jennings and Cannon (1987)
Cleistogamia holothuriana	*Actinopyga mauritiana* *Actinopyga echinites* (H)	Digestive tract	50 worms in a single individual (Faust)	Indian Ocean (Andaman Islands, (Madagascar: Nossi-Bé)	Faust (1924, 1927), Baer (1938), Hyman (1960)
Cleistogamia longicirrus	*Stichopus chloronotus*, *Stichopus horrens*, *Stichopus variegatus* (H)	Anterior to midpart of digestive tract	–	Great Barrier Reef (Australia)	Cannon (1982), Jennings and Cannon (1987)
Cleistogamia louftia	*Holothuria* sp.	Unspecified (endoparasitic)	–	Red Sea	Khalil (1938, quoted by Stunkard and Corliss 1951)

Table 5-4 (continued)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
II. Rhabdoecoela (f. umagillidae) (continued)					
Cleistogamia pallii	*Bohadschia argus* (H)	Anterior to midpart of digestive tract	–	Great Barrier Reef (Australia)	Cannon (1982)
Cleistogamia pulchra	*Actinopyga echinites, Actinopyga lecanora, Actinopyga miliaris* (H)	Midpart of digestive tract	–	Great Barrier Reef (Australia)	Cannon (1982)
Cleistogamia pyriformis	*Holothuria impatiens* (H)	Anterior part of digestive tract	–	Great Barrier Reef (Australia)	Cannon (1982)
Desmote antarcticus	*Promachrocrinus kerguelensis* (C)	Intestine	Up to 3 worms crinoid^{-1}	W coast of Antarctic Peninsula	Shinn (1987)
Desmote inops	*Florometra serratissima* (C)	Digestive tract	10 to 30 worms crinoid^{-1} (49 infested/60 investigated)	NE Pacific (British Columbia: satellite Channel)	Kozloff (1965)
Desmote vorax	*Heliometra glacialis* (C)	Unspecified (endoparasitic)	1 to 20 worms crinoid^{-1} (9 infested/100 investigated)	Barents Sea (Kola Bay)	Beklemishev (1916)
Fallacohospes inchoatus	*Florometra serratissima* (C)	Digestive tract	2 to 15 worms crinoid^{-1} (59 infested/60 investigated)	NE Pacific (British Columbia: satellite Channel)	Kozloff (1965), Shinn (1986a)
Macrogynium ovalis	*Isostichopus badionotus* (H)	Coelomic cavity; digestive tract	15 worms holothuroid^{-1} (average number; 36 holothuroids investigated) (Snyder)	Tropical W Atlantic (Bermuda)	Meserve (1934), Snyder (1980)
Monticellina longituba[2]	*Holothuria impatiens, Holothuria polii* (H)	Coelomic cavity	–	Mediterranean Sea (Naples)	Westblad (1953)
Notothrix inquilina	*Mensamaria thompsoni* (H)	Digestive tract (anterior part)	Up to 22 worms holothuroid^{-1} (51 infested/121 investigated)	Tasmania	Hickman (1955)
Ozametra arborum	*Stichopus japonicus* (H)	Digestive tract	–	NW Pacific (Japan: Hiroshima)	Ozaki (1932)
Ozametra sp.	*Parastichopus californicus* (H)	Digestive tract	–	Pacific coast of N America	Kozloff in Shinn (1983a)

Table 5-4 (continued)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
II. Rhabdoecoela (f. umagillidae) (continued)					
Parafallacohospes bransfieldensis	*Promachrocrinus kerguelensis* (C)	Intestine	Up to 4 worms crinoid^{-1}	W coast of Antarctic Peninsula	Shinn (1987)
Paranotothrix queenslandensis	*Actinopyga echinites, Actinopyga miliaris, Bohadschia argus, Holothuria atra, Holothuria hilla, Holothuria impatiens, Holothuria leucospilota, Stichopus chloronotus, Stichopus horrens, Stichopus variegatus, Thelonota ananas* (H)	Posterior part of digestive tract	–	Great Barrier Reef (Australia)	Cannon (1982), Jennings and Cannon (1987)
Seritia elegans	*Parastichopus tremulus* (H)	Digestive tract (anterior part)	Rather frequent	North Sea (Norwegian coast)	Westblad (1926, 1953), Jespersen and Lützen (1971)
Seritia striata	*Stichopus mollis* (H)	Digestive tract (anterior part)	–	Tasmania	Hickman (1955)
Syndesmis alcalai	*Heterocentrotus mammillatus* (E)	Digestive tract and coelomic cavity	–	NW Pacific (Philippines: Sumilon Island)	Komschlies and Vande Vusse (1980a)
Syndesmis compacta	*Echinometra oblonga* (E)	Digestive tract and coelomic cavity	–	NW Pacific (Philippines: Cebu Province)	Komschlies and Vande Vusse (1980b)
Syndesmis dendrastomum	*Dendraster excentricus* (E)	Digestive tract	Up to 23 worms echinoid^{-1} (Smith); worms consistently present in large number (Orihel)	E Pacific (California; Washington State)	Stunkard and Corliss (1950, 1951), Orihel (1952), Smith (1973), Shinn (1981)

Table 5-4 (continued)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
II. Rhabdoecoela (f. umagillidae) (continued)					
Syndesmis echinorum	*Echinus acutus, Echinus esculentus, Paracentrotus lividus, Psammechinus microtuberculatus, Psammechinus miliaris, Sphaerechinus granularis, Strongylocentrotus droebachiensis* (E)	Digestive tract and coelomic cavity	Infestation rate highly variable (see Barel and Kramers 1977)	European Seas	Silliman (1881), François (1886), Cuénot (1891), Shipley (1901), Briot (1906b), Westblad (1926), Barel and Kramers (1970, 1977), Lama Seco and Rodriguez Babio (1978), Kozloff and Westervelt (1987)
Syndesmis aff. *echinorum*	*Strongylocentrotus droebachiensis, Strongylocentrotus pallidus* (E)	Digestive tract	–	NE Pacific (Washington: San Juan Island)	Shinn (1981)
Syndesmis glandulosa	*Diadema setosum, Echinothrix calamaris* (E)	Digestive tract and coelomic cavity	–	SW Indian Ocean (Madagascar: Nossi-Bé)	Hyman (1960), Komschlies and Vande Vusse (1980a)
Syndesmis mammillata	*Echinometra oblonga* (E)	Digestive tract and coelomic cavity	–	NW Pacific (Philippines: Negros Oriental Province)	Komschlies and Vande Vusse (1980a)
Syndesmis philippinensis	*Echinometra oblonga* (E)	Digestive tract and coelomic cavity	–	NW Pacific (Philippines: Negros Oriental Province)	Komschlies and Vande Vusse (1980a)
Syndesmis sp.	*Evechinus chloroticus, Heliocidaris erythrogramma* (E)	Digestive tract	–	New Zealand	McRae (1959)

Table 5-4 (continued)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
II. Rhabdoecoela (f. umagillidae) (continued)					
Syndisyrinx antillarum	*Diadema antillarum, Lytechinus variegatus, Echinometra viridis* (E)	Digestive tract and coelomic cavity	60 worms echinoid^{-1} (average number; 3 infested/9 investigated) (Snyder); up to 205 worms echinoid^{-1} (350 investigated/475 investigated) (Nappi and Crawford)	Tropical Atlantic (off Florida: Bermuda, Jamaica)	Powers (1935), Stunkard and Corliss (1951), Mettrick and Jennings (1969), Snyder (1980), Nappi and Crawford (1984)
Syndisyrinx atriovillosa	*Spatangus purpureus* (E)	Digestive tract	–	English Channel (Plymouth)	Westblad (1953)
Syndisyrinx franciscanus	*Strongylocentrotus franciscanus, Strongylocentrotus purpuratus, Strongylocentrotus droebachiensis, Strongylocentrotus pallidus, Lytechinus anamesus, Allocentrotus fragilis, Lytechinus variegatus* (E)	Digestive tract and coelomic cavity	Often up to 30 worms in infested echinoid (Lehman, Shinn); maximum 3 worms echinoid^{-1} (5 infested/75 investigated) (Giesel); 29 worms echinoid^{-1} (average number) (Jones and Canton)	Pacific coast of N America (California, Washington), Tropical W Atlantic (Jamaica)	Lehman (1946), Stunkard and Corliss (1951), Giese (1958), Hyman (1960) Jennings and Mettrick (1968), Barnes (1969), Mettrick and Jennings (1969), Jones and Canton (1970), Mettrick and Boddington (1972), Holt and Mettrick (1975), Shinn (1981, 1983b)
Syndisyrinx pallida	*Echinocardium cordatum* (E)	Digestive tract	1 to 4 worms echinoid^{-1} (10 infested/68 investigated)	Tasmania (Ralph's Bay)	Hickman (1955)
Syndisyrinx punicea	*Heliociaris erythrogramma, Amblypneustes ovum* (E)	Digestive tract	Infestation very frequent; up to 18 worms echinoid^{-1}	SE Indian Ocean (Tasmania: Ralph's Bay)	Hickman (1956)

Table 5-4 (continued)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
II. Rhabdoecoela (f. umagillidae) (continued)					
Umagilla forkalensis	*Holothuria forskali* (H)	Digestive tract	Up to 14 worms holothuroid^{-1} (29 infested/47 investigated) (Wahl 1909)	Mediterranean Sea (Naples); North Sea (Norwegian coast); English Channel (Plymouth)	Wahl (1906, 1909), Westblad (1953)
Wahlia macrostylifera	*Isostichopus tremulus*, *Parastichopus badionotus* (H)	Digestive tract and coelomic cavity	15 worms holothuroid^{-1} (average number) (33 infested/36 investigated) (Snyder)	North Sea (Norwegian coast); Tropical W Atlantic (Bermuda)	Westblad (1926, 1930), Jespersen and Lützen (1971), Snyder (1980)
Wahlia pulchella	*Stichopus californicus* (H)	Anterior part of intestine (attached by the pharynx to host's digestive epithelium)	2 to 5 worms holothuroid^{-1} (infestation level: 62 to 100 % in spring and summer; 0 % in fall and winter when host's viscera are resorbed)	NE Pacific (coast of Washington)	Shinn (1986b), Kozloff and Shinn (1987)
Wahlia stichopi	*Stichopus chloronotus*, *Stichopus horrens*, *Thelonota ananas* (H)	Anterior to midpart of digestive tract	–	Great Barrier Reef (Australia)	Cannon (1982)
III. Rhabdocoela (f. acholadidae and pterastericolidae)					
Acholades asteris	*Coscinasterias calamaria* (A)	Encysted in tube feet wall	20 or more worms asteroid^{-1} (216 infected/267 investigated)	SW Indian Ocean (Tasmania: D'Entrecasteaux Channel)	Hickman and Olsen (1955)
Pterastericola australis	*Patiriella calcar* (A)	Pyloric caeca	Up to 10 worms asteroid^{-1} (28 infested/407 investigated)	Hasting Point (New South Wales, Australia)	Jennings and Cannon (1985), Cannon (1986)
Pterastericola fedotovi	*Pteraster militaris*, *Pteraster obscurus*, *Pteraster pulvillus* (A)	Unspecified (endoparasitic)	–	Barents Sea (Murmansk); White Sea (Kandalaksha Bay)	Beklemishev (1916), Karling (1970)

Table 5-4 (continued)

Turbellarian	Host	Location in host	Remarks	Geographical area	Source
Rhabdocoela (f. acholadidae and pterastericolidae) (continued)					
Pterastericola ramosa	*Luidia australiae* (A)	Pyloric caeca	–	W Pacific (Queensland: Moreton Bay)	Cannon (1986)
Pterastericola sprenti	*Ophidiaster granifer* (A)	Pyloric caeca	–	W Pacific (Great Barrier Reef: Heron Island)	Cannon (1986)
Pterastericola vivipara	*Acanthaster planci*, *Anthenea acuta* (A)	Pyloric caeca	Infested asteroid may have large number of worms	W Pacific (Australia: central Great Barrier Reef)	Cannon (1978), Jennings and Cannon (1985), Cannon (1986); Cannon and Jennings (1988)
Triloborhynchus astropectenis	*Astropecten irregularis* (A)	Pyloric caeca	5 to 10 worms per pyloric caecum in infested asteroid	North Sea (Norwegian and Swedish coasts); English Channel (Plymouth)	Bashirudin and Karling (1970), Jennings and Cannon (1985)
Triloborhynchus psilastericola	*Psilaster andromeda* (A)	Pyloric caeca; coelomic cavity (juvenile forms)	Infestation frequent (sometimes more than 10 worms asteroid^{-1})	North Sea (Oslofjord)	Jespersen and Lützen (1972)
IV. Polycladida					
Euplana takewakii	*Ophioplocus japonicus* (O)	Bursae	20 infested/200 investigated	NW Pacific (Japan: Mitsui)	Kato (1935)

[1] Species of doubtful validity (Shinn pers. comm.).
[2] Synonym of *Umagilla forskalensis*, according to Cannon (1982).

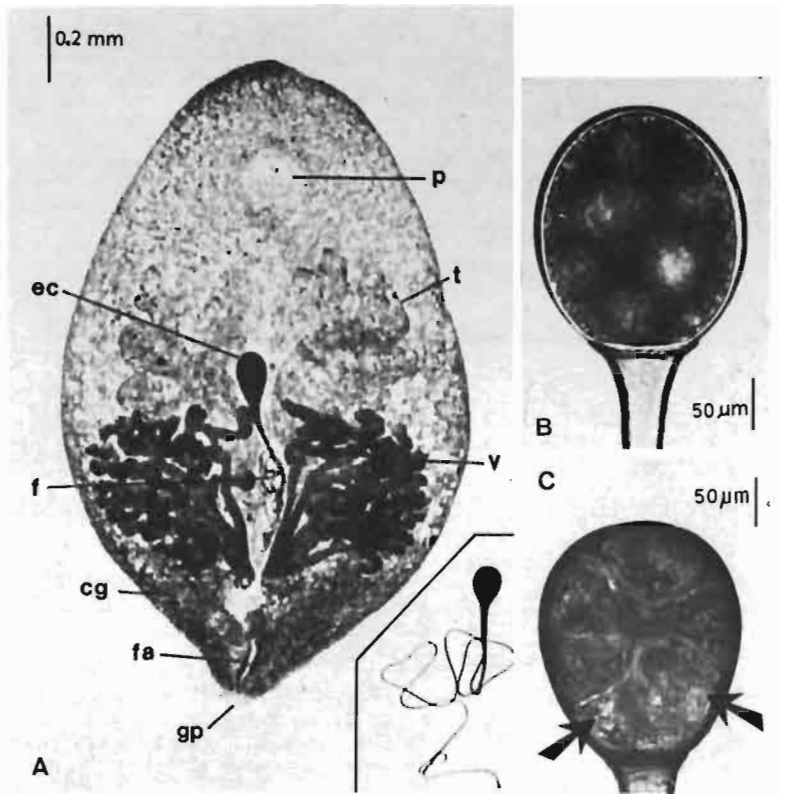


Fig. 5–9: *Syndisyrinx franciscanus,* a symbiotic turbellarian from the intestine of echinoids (*Strongylocentrotus* spp.). A: Ventral view of a live adult individual; (cg) cement glands; (ec) bulb of egg capsule; (f) filament of egg capsule; (fa) female antrum; (gp) location of common gonophore; (p) pharynx; (t) left testis; (v) vitellaria. Insert: egg capsule showing bulb and filament. B: Bulb of a newly produced egg capsule. C: Bulb of a 2 month old egg capsule containing 6 fully developed embryos (arrows). (After Shinn, 1983b.)

host's coelomic fluid together with coelomocytes (Jennings and Mettrick, 1968; Shinn, 1983b) — or on other coelom-associated organisms such as ciliates (Mettrick and Jennings, 1969; Jennings, 1980). Egg-capsules of intracoelomic umagillids of holothuroids frequently occur within brown bodies (Briot, 1906a, b; Arvy, 1957; Changeux, 1961; Jespersen and Lützen, 1971; Shinn, 1983b, 1985a). Up to 1,000 eggs capsules may occur in the body cavity of a single infested holothuroid (Kroll and Jangoux, 1989). They are thought to be released into the outer medium through host evisceration (Changeux, 1961, Jespersen and Lützen, 1971). Shinn (1985a) reported, however, that brown bodies containing egg capsules of the coelom-associated *Anoplodium hymanae* may pass out of intact hosts — the holothuroid *Parastichopus californicus* — through any of a series of pores that connect the coelom to the posterior end of the rectum. Embryogenesis of *A. hymanae* lasts about 1 month, and embryos remain quiescent in their capsule until they are ingested by a holothuroid (developed embryos can survive in their capsules for 10 to 11 month; Shinn, 1985b). Hatching is stimulated by some property of the host's digestive fluid. Larvae penetrate the wall of the posterior intestine or, more commonly, that of the respiratory

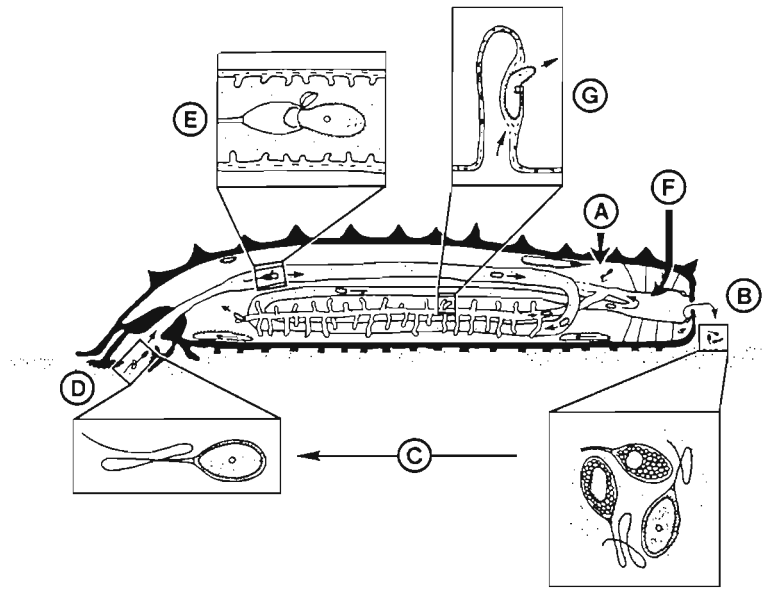


Fig. 5–10: *Anoplodium hymanae*. Life cycle of a coelom-associated umagillid from the holothuroid *Parastichopus californicus*. (A) Release of umagillid egg capsules into the host's coelom: (B) ensheathment of egg capsules into brown bodies; (C) completion of embryos' development outside the host; (D) ingestion by the new host of egg capsules containing embryos; (E) hatching of larvae in the upper intestine; (F) migration of larvae towards the respiratory trees; (G) larvae penetrate the wall of the respiratory trees and enter the coelom. (After Shinn, 1985b.)

trees to reach the coelom (Fig. 5-10). As demonstrated by Shinn (1985b), the size of *A. hymanae* infesting *P. californicus* varies seasonally and is correlated with the seasonal feeding behavior of the host. According to Kroll and Jangoux (1989), a similar life cycle appears to characterize *Anoplodium parasita* infesting *Holothuria tubulosa* in the Mediterranea. They reported, moreover, that a rather high proportion of egg capsules (ca. 20 %) hatch in the host's body cavity and thus considered the possibility that some individuals of *A. parasita* may undergo a complete life cycle within their host.

Investigations by Shinn (1983b, 1985a, b, 1986b) on echinoderm-associated umagillids showed that hatchings are not adversely affected by the host's digestive fluid whatever the final location of the worms in the host. However, adult worms of coelom-inhabiting species are killed by the host's digestive fluid but appear to have some mean of avoiding attack by coelomocytes. Considering the number of species of umagillids that are reported to inhabit both the coelom and gut of the host (Table 5-4), careful re-examination is needed "to determine if the worms clearly are adapted to inhabiting very different sites in their hosts, or whether the reports are the results of improper dissection techniques" (Shinn, 1985b, p. 213).

Non-umagillid rhabdocoels associated with echinoderms have been reported only from asteroids (Table 5-4). The acholadid *Acholades asteris* was always found encysted in the connective tissue layer of the tube feet of *Coscinasterias calamaria*. Nothing is known on the life cycle of this aberrant rhabdocoel. All pterastericolids found thus far were associated with asteroid pyloric caeca on which they feed (feeding on energy-rich epithelial

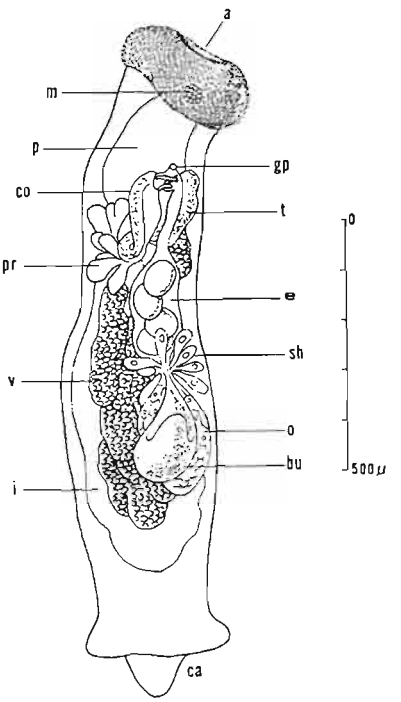


Fig. 5–11: *Triloborhynchus psilastericola.* Ventral view of a turbellarian parasite of the pyloric caeca of the asteroid *Psilaster andromeda.* (a) Entrance to apical organ; (bu) bursa; (ca) caudal adhesive disk; (co) copulatory bulb; (e) egg capsule in uterus; (gp) common genital pore; (i) intestine; (m) mouth; (o) ovary; (p) pharynx; (pr) prostatic glands; (sh) shell glands; (t) testis; (v) yolk glands. (After Jespersen and Lützen, 1972.)

cells; Cannon, 1975, 1978, 1986; Jennings and Cannon, 1985) (Figs 5-11 and 5-12). According to Jennings and Cannon (1985), the occurrence of pterastericolids is independent of host size and sex. They noted that the worms neither affect the host's reproductive potential nor produce any marked damages to the asteroid's pyloric caeca. Digestion in asteroid pterastericolids is predominantly intracellular (their gut is deprived of gastrodermal glands), and Jennings and Cannon (p. 211) suggest this would be "an adaptative simplification related to the particular diet of host storage and digestive cells which provides all necessary dietary components plus the enzymes necessary for digestion".

There is no information on the type of relation between echinoderms and acoel turbellarians although such associations occur frequently (Table 5-4). The only polyclad species known to be an echinoderm parasite, *Euplana takewakii,* feeds on ophiuroid's gonads, the gonads of infested bursae always being castrated (Kato, 1935).

Deposit-feeding echinoderms infest by ingesting bottom sediment holding egg capsules with embryo (e.g., Shinn, 1985b). According to Shinn (1986a) suspension-feeding hosts (i.e., crinoids) would infest by eating free-swimming umagillid larvae rather than by eating egg capsules. No information came to the reviewer attention as to how plant-eating hosts (i.e., regular echinoids) and carnivorous hosts (i.e., asteroids) are infested by turbellarians.

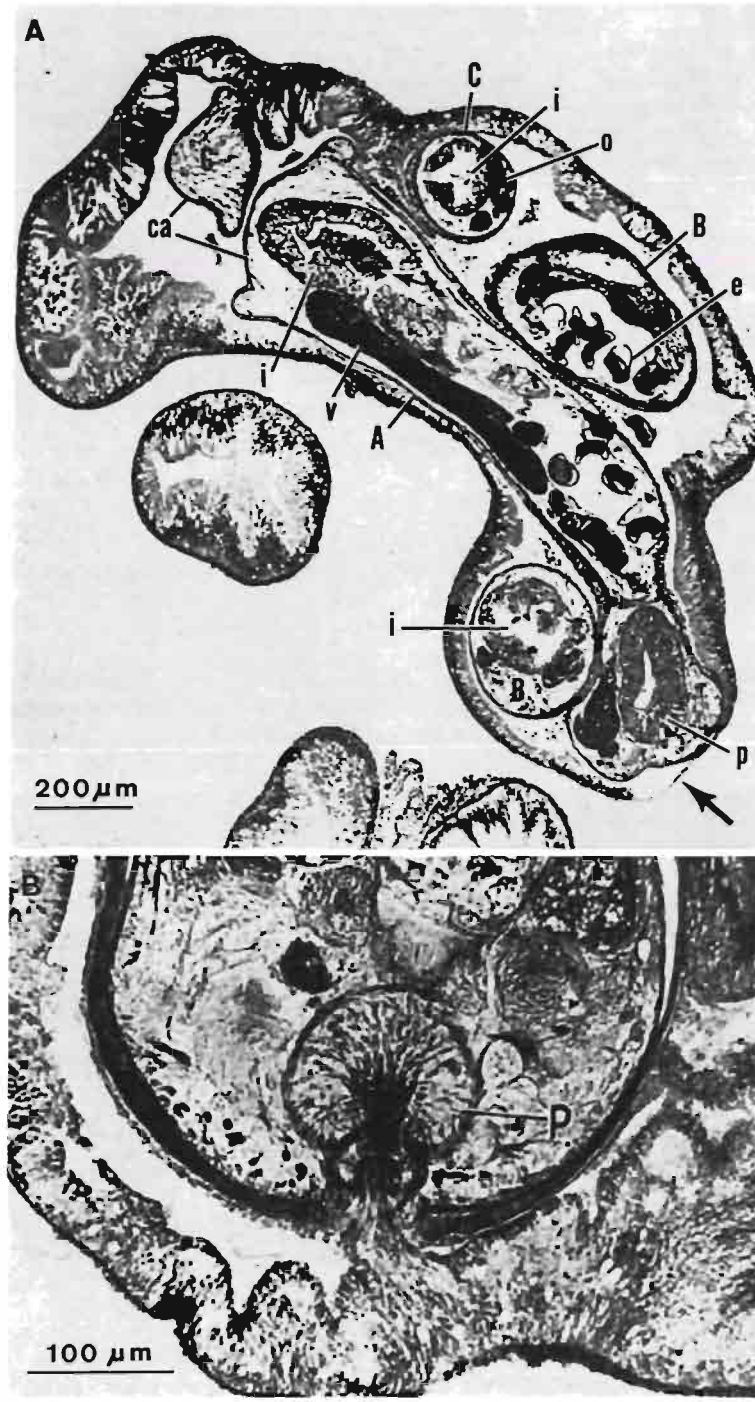


Fig. 5–12: Infestation of asteroid pyloric caeca by pterastericolid turbellarians. A: *Psilaster andromeda;* section through pyloric diverticulum containing 3 specimens (A, B, C) of *Triloborhynchus psilastericola;* (ca) caudal adhesive disk; (e) egg capsule in uterus; (i) intestine; (o) ovary; (p) pharynx; (v) yolk glands;large arrow: area of pyloric diverticulum demolished by specimen; small arrow: piece of ingested tissue from pyloric diverticulum. B: *Acanthaster planci;* section through a pyloric diverticulum showing an individual of *Pterastericola vivipara* ingesting pyloric tissues; (P) pharynx. (A after Jespersen and Lützen, 1972; B after Cannon, 1978.)

Agents: Trematoda

Trematodes reported from echinoderms are listed in Table 5-5. Unidentified metacercariae were noted by Schneider (1858), in the body cavity of *Holothuria tubulosa;* by Schurig (1906), in the gut of a deep-sea echinoid; by Ohshima (1911), in stomach and mesenchyme of a planktonic holothuroid larva; by Mortensen (1921b), in gonads of the Japanese echinoid *Mespilia globulus;* and by Johnson (1971a), in gonads of *Strongylocentrotus purpuratus.*

Echinoderms generally act as second intermediary host (Fig. 5-13). The echinoderm's reaction to invading cercariae or to encysted metacercariae is largely unknown. According to Prévot (1966a) host tissues form a 'xenocyst' of dense connective tissue around metacercariae (Fig. 5-14), but Køie (1976) reported that infested ophiuroids do not respond to trematode cysts. Effects of metacercariae on their echinoderm host appear to be rather unimportant. According to Køie (1976), heavily infested *Ophiura albida* tend to autotomize their arms. This is presumably linked to cysts' location at the joints between the arm vertebrae. One may also suggest that, when heavily infested, the jaw muscles of echinoids become less functional (Table 5-5; *Zoogonus mirus* and *Zoogonus* sp.); thus the cysts may affect echinoid feeding.

The location of metacercarian cysts (in muscles or within the body wall) may partly explain why relatively few species of echinoderm-infesting trematodes have been recorded. Whatever the cause, it seems rather obvious that echinoderms are very suitable intermediary host for marine digenic trematodes. Not only do echinoderms occur frequently in very dense populations, but some of their representatives also form part of the diet of many fishes. As seen in Table 5-5, most echinoderm-infesting trematodes are known from echinoids and ophiuroids which constitute the most frequent echinoderm prey for fishes. The role of echinoderms as potential vectors of trematode-caused fish diseases requires further attention.

Agents: Nematoda

Rather few nematodes have been reported to occur in echinoderms (e.g., Fig. 5-15). In addition to the species listed in Table 5-6, unidentified — and presumably undescribed — nematodes were found inside the body (mostly the coelomic cavity) of various echinoderms: Antarctic asteroids *Hymenaster perspicuus* and *Diplasterias luetkeni* (Ludwig, 1903); echinoids *Echinus esculentus* and *Brissopsis lyrifera* (respectively Shipley, 1901; Brattström, 1946); holothuroids *Leptosynapta* spp., *Holothuria* spp. and *Aslia lefevrei* (respectively Monticelli, 1892; Briot, 1906a; Hérouard, 1923); and North Sea ophiuroids *Asteronyx loveni* and *Ophiura albida* (respectively Jungersen, 1912; Mortensen 1921a). Intense infestations by juvenile nematodes also occurred within the digestive wall of the abyssal holothuroids *Kolga hyalina, Trochostoma thompsoni* and *Elpidia glacialis* (Danielssen and Kören, 1882; Massin pers. comm.).

As seen from Table 5-6, echinoderm-associated nematodes are mostly juveniles. Echinoderms presumably act as intermediary host, the primary host being fishes. This was suggested by Ward (1933) and demonstrated by Pearse and Timm (1971) who identified the primary host of the echinoid parasite *Echinocephalus pseudouncinatus* as the California horned shark *Heterodontus francisci.* Host reactions were noted only by Pearse and Timm (1971) who reported the encystment of juvenile nematodes within echinoid gonads.

Table 5-5
Parasitic trematodes from echinoderms. Hosts: C, crinoid; E, echinoid; H. holothuroid; O, ophiuroid (Compiled from the sources indicated)

Trematode	Host	Location in host	Primary host	Remarks	Geographical area	Source
Diphterostomum brusinae	*Antedon mediterranea* (C)	Inside crinoid calyx (within connective tissue strings)	Several species of benthic fishes	1 to 15 trematodes crinoid^{-1}; first intermediary host would be a gastropod mollusc (*Nassa* sp., *Natica* sp.)	Mediterranean Sea (Marseille)	Prévot (1966a, see also Palombi 1930)
Fellodistomum fellis[1]	*Ophiura albida, Ophiura sarsi* (O)	In wall of digestive sac	Fishes (*Anarrhicas lupus, Platessa platessa)* (Mortensen)	1 to 13 trematodes ophiuroid^{-1}; the first intermediary host is bivalve *Nucula nucula* (Chubrik)	Barents Sea (Kola Bay); North Sea (Gullmarfjord)	Tauson (1917), Mortensen (1921a), Chubrik (1952, see also Barel and Kramers 1977)
Himasthla leptosoma	*Leptosynapta gallienei, Leptosynapta inhaerens* (H)	In body wall, at base of buccal tentacles; sometimes within coelomic brown bodies	Sea birds *(Tringa variabilis, Calidris leucophoea)*	Alternative intermediary hosts: bivalve *Scrobicularia tenuis,* polychete *Arenicola marina,* sipunculid *Phascolosoma vulgare* (Cuénot 1912)	NE Atlantic (Arcachon, Roscoff)	Cuénot (1892, 1912), Timon-David (1938)
'Metacercaria' psammechini	*Psammechinus microtuberculatus, Sphaerechinus granularis* (E)	In muscles of Aristotle's lantern	Presumably echinoid-eating fishes of the family Labridae	Infestation may be very heavy	Mediterranean Sea (Banyuls, Marseille)	Timon-David (1934, 1938)
Monorchis monorchis	*Antedon mediterranea* (C)	Inside crinoid calyx (within connective tissue strings)	Sparid fishes, in particular *Spondyliosoma cantharus*	5 to 60 trematodes crinoid^{-1} (17 infested/151 investigated)	Mediterranean Sea (Marseille)	Prévot (1966a, b)
?Nidrosia ophiurae[2]	*Ophiura sarsi* (O)	In gonads	–	–	North Sea (Trondhjemfjord)	Mortensen (1933a)

Table 5-5 (continued)

Trematode	Host	Location in host	Primary host	Remarks	Geographical area	Source
Paralepidapedon hoplognathi	*Anthocidaris crassispina* (E)	Mostly in gonads; also in muscles of Aristotle's lantern and in ampullae of tube feet	Fish *Hoplognathus punctatus*	1 to 66 trematodes echinoid^{-1} (22 infested/29 investigated)	Misaki (Japan)	Shimazu and Shimura 1984
Proctoeces maculatus	*Anthocidaris crassispina, Diadema setosum, Hemicentrotus pulcherrimus* (E)	In gonads	–	–	Misaki (Japan)	Shimazu and Shimura (1984)
Protoeces sp.	*Strongylocentrotus intermedius* (E)	In gonads	–	Gastropod *Haliotis discus hannai* is alternative intermediary host	Japan Sea (off Maehama, Hokkaido)	Shimazu (1979)
?Tetrarhynchus holothuriae[3]	*Molpadia* sp. (H)	Body wall (?)	–	–	NE Indian Ocean (Malaysian coast)	Shipley (1903)
Zoogonoides viviparus[4]	*Ophiura albida, Ophiothrix fragilis* (O), *Leptosynapta gallienei, Leptosynapta inhaerens* (H)	In gonads and coelom wall of ophiuroids; in body wall of holothuroids (at base of buccal tentacles)	–	Might also occur in *Mysis* sp.	NE Atlantic (Arcachon, Roscoff)	Cuénot (1892, 1912)
Zoogonoides viviparus	*Ophiura albida;* rarely *Ophiura texturata* and *Ophiura robusta* (O)	Mostly between arm vertebrae (natural infestation); also within disc (gonads, water vascular system, mesenteries) (experimental infestation)	Fishes: posterior intestine and rectum of plaice, flounder, dab and long rough dab from Øresund	First intermediary host is gastropod *Buccinum undatum, O. albida* is the most important second intermediary host. 1 to 30 trematodes ophiuroid^{-1}; up to 250 in experimentally infested ones	North Sea (Kattegat, Øresund)	Køie (1976)

Table 5-5 (continued)

Trematode	Host	Location in host	Primary host	Remarks	Geographical area	Source
Zoogonus mirus	*Arbacia lixula, Paracentrotus lividus, Sphaerechinus granularis* (E)	In muscles of Aristotle's lantern	Fishes: *Labrus merula* (natural infestation), *Blennius gattorugine* (experimental infestation)	1 to 30 trematodes echinoid^{-1}; heavy infestation with *P. lividus*, slight infestation with *A. lixula*	Mediterranean Sea (Banyuls, Marseille)	Timon-David (1933, 1934, 1936, 1938)
Zoogonus rubellus	*Arbacia punctulata* (E)	Presumably in muscles of Aristotle's lantern	Eel (natural infestation); toadfisch (experimental infestation)	First intermediary host gastropod *Nassa obsaleta;* usual second intermediary host: polychete *Nereis virens*. Experimental use of *A. punctulata* as alternative second intermediary host was partly successful	NW Atlantic (Woods Hole)	Stunkard (1941, see also Stunkard 1938)
Zoogonus sp.	*Psammechinus miliaris* (E)	In muscles of Aristotle's lantern	–	1 to 36 trematodes echinoid^{-1}	North Sea (Boulogne, Wimereux)	Stunkard (1941)

[1] Previously identified by Tauson (1917) as *Adolescaria ophiurae*.
[2] The parasite has been tentatively ascribed to trematodes by Mortensen; it causes destruction of infested gonads.
[3] Described as encysted larvae of cestode (Shipley 1903).
[4] Identified by Cuénot (1892, 1912) as *Cercaria capriciosa*.

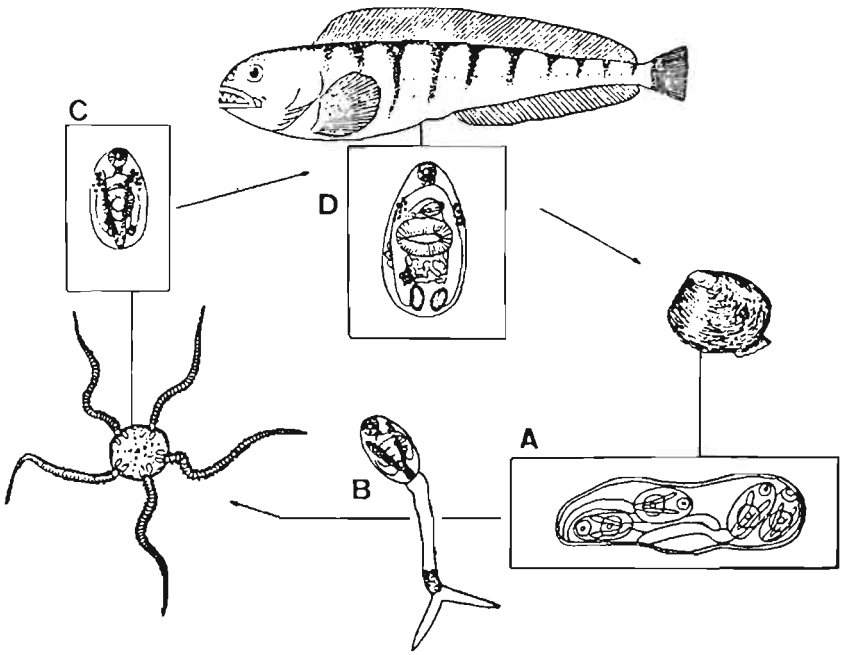


Fig. 5–13: *Fellodistomum fellis*. Life cycle of a marine digenic trematode with 2 intermediary hosts: bivalve mollusc *Nucula tenuis* and ophiuroid *Ophiura sarsi*. (A) Redia; (B) cercaria; (C) metacercaria; (D) late metacercaria and adult worm. (After Chubrik, 1952.)

The cyst is host-produced and made of dense connective tissue. Effects of nematodes on their hosts are obvious when the worms destroy the echinoderm's body wall, an injury reported by Ludwig (1903), Ward (1933) and Rubstov (1977). Another, less conspicuous effect was noted by Pearse and Timm (1971) on gonads of *Centrostephanus coronatus*: growing juvenile nematodes progressively invaded the gonadal tubules (small juveniles are confined to the gonad wall) and negatively affected host gametogenesis. Gametogenesis is suppressed in the infested tubules, especially above the parasite, viz. in the oral or distal part of the tubule. Pearse and Timm suggested that encysted juveniles block the passage through the tubules of some hormonal substance that regulates echinoid gametogenesis.

The infestation level by *Echinomermella grayi* — an endocoelomic nematode infesting the echinoid *Echinus esculentus* — is always very low, and the parasites do not show evidence of seasonality in either size or egg maturity (Comely and Ansell, 1988). *Echinomermella matsi* occurs in the body cavity of the echinoid *Strongylocentrotus droebachiensis* and was said to be the critical factor terminating echinoid outbreaks in Norwegian waters (Hagen, 1985, 1987). *S. droebachiensis* would be the final host of the worm (Hagen, 1987). According to Jones and Hagen (1987), the gonad of infested echinoids are notably reduced in size suggesting some degree of parasitic castration. They suggested moreover that echinoid die-off could be linked to the damages caused to host's tissues when large number of nematode larvae migrate towards the exterior.

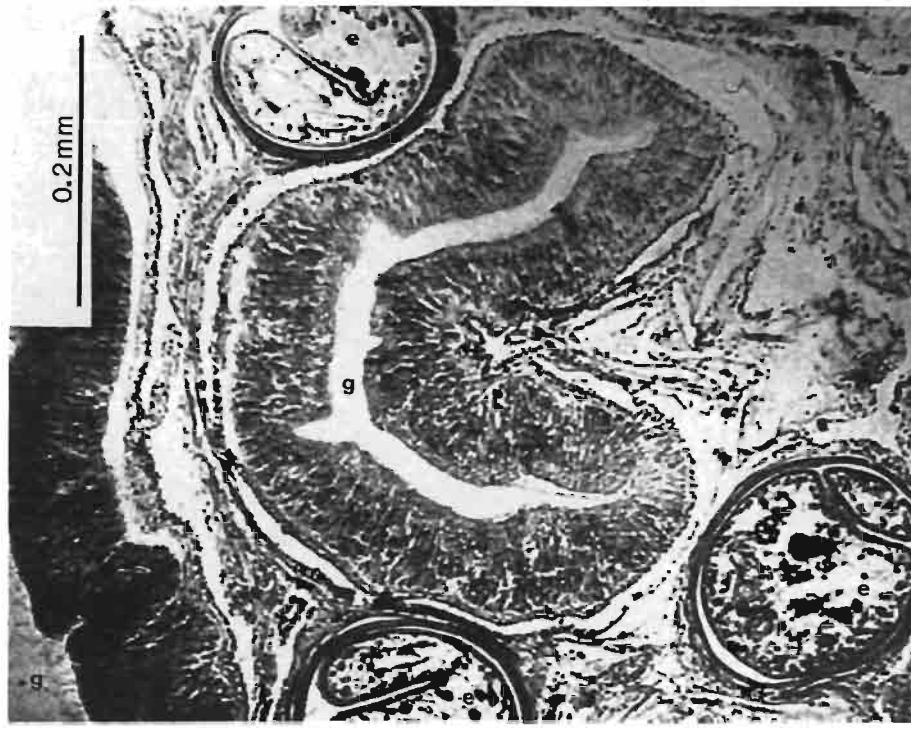


Fig. 5-14: *Monorchis monorchis* (Trematoda). Metacercariae encysted within connective tissue strings of the calyx of the comatulid crinoid *Antedon mediterranea*. (e) encysted metacercariae; (g) gut of the crinoid. (After Prévot, 1966a.)

Agents: Mollusca Gastropoda

Gastropods living symbiotic with echinoderms belong almost exclusively to the family Eulimidae. According to Warén (1984) there are about 800 species (43 genera) of extant eulimids all of which, except 2, being associated with echinoderms. Table 5-7 lists both ecto- and endoparasitic eulimids (species classified as ectoparasites clearly behave as parasites or intertain morphological relations with their host which imply parasitism).

Most ectoparasitic eulimids live attached to the echinoderm's body surface, by either their snout or their proboscis (Vaney, 1915; Warén, 1984). They feed on the host's tissues or fluids using their proboscis which penetrates more or less deeply into the echinoderm's body wall or crosses it to reach the coelomic cavity, the water-vascular system, the perihemal system, or the hemal system. However, unattached ectoparasites also occur, e.g., *Pulicicochlea calamaris* and *Vitreobalcis temnopleuricola* which browse over the epidermis of the echinoids *Echinothrix calamaris* and *Temnopleurus toreumaticus* (Ponder and Gooding, 1978; Fujioka, 1985; respectively) and *Peasistilifer nitidulus* which moves over the entire body surface of *Holothuria atra*, puncturing periodically the body wall of its host (Hoskin and Cheng, 1970).

Some attached ectoparasitic eulimids are said to feed exclusively on echinoderm dermal tissues. Among them are those belonging to the gallicole genus *Stilifer* (Tullis and Cheng, 1971; Warén, 1980a) (Fig. 5-16), as well as representatives of the genera

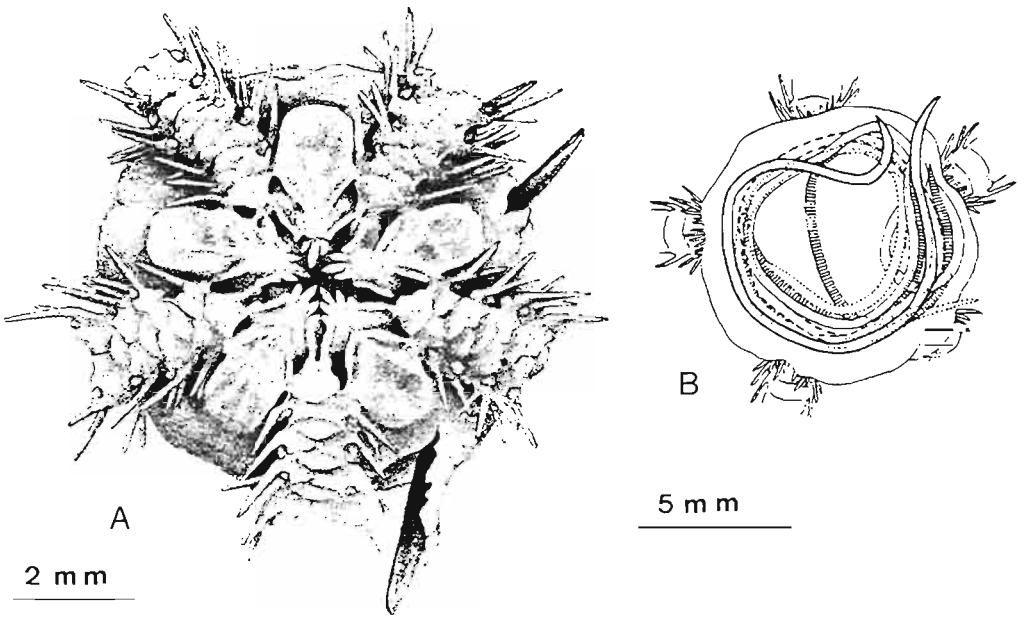


Fig. 5–15: *Thalassonema ophioctinis* a nematode parasite of the ophiuroid *Ophiocten amitinum*. A: Oral view of ophiuroid showing ends of nematode protruding through the wall of disc. B: Five nematodes coiled within the ophiuroid body cavity. (After Ward, 1933.)

Pelseneeria (see Koehler and Vaney, 1908) and *Monogamus* (see Lützen, 1976). These authors reported that the proboscis is inserted into the dermis but they do not discuss the way in which the dermal tissue is ingested. Dermal-tissue-feeding eulimids may induce conspicuous host reactions (Lützen, 1976) — the formation of swollen areas which are basically disorganized outgrowths of the connective tissue upon which the parasite feeds (Fig. 5-17).

Fluid-feeding ectoparasitic eulimids have been reported also (e.g., Warén, 1981d). According to Bacci (1948) the proboscis of *Melanella comatulicola* reaches the arm's coelomic canal of its crinoid host and sucks up coelomic fluid. Cabioch and co-authors (1978) found that *Balcis alba* — a temporary holothuroid ectoparasite — penetrate the host's body wall via its proboscis. Aquarium observations have shown that the proboscis does not seek out a specific organ or tissue. It moves actively within the holothuroid coelomic cavity and pumps off coelomic fluid. Aquarium observations further revelaed that the point of penetration of the proboscis is not restricted to any part of the body surface. In the field, however, it was invariably located immediately below the buccal tentacles. Smith (1984) observed that the proboscis of *B. alba* is infolded when penetrating the holothuroid integument, and that the proboscis epithelium releases secretory material which appears to bring about a rapid loosening of the host's connective tissue. Fluid-feeding was inferred also with *Echineulima* spp., *Ophieulima minima* and *Peasistilifer edulis,* as the proboscis of individuals of these species was observed inserted into the host's body cavity (respectively Lützen and Nielsen, 1975; Warén and Sibuet, 1981; Hoskin and Warén, 1983). One may wonder, however, if the coelomic fluid together with coelomocytes can ensure sufficient nutrients for parasites. As noted by Lützen and Nielsen (1975),

Table 5-6
Parasitic nematodes from echinoderms. Hosts: A, asteroid; E, echinoid; O, ophiuroid (Compiled from the sources indicated)

Nematode	Host	Location in host	Remarks	Geographical area	Source
Ananus asteroideus	*Diplopteraster perigrinator* (A)	Coelomic cavity	One nematode in each asteroid arm	Antarctic seas (off Kerguelen Islands)	Rubstov (1977)
Echinocephalus pseudouncinatus	*Arbacia punctulata* (E)	Gonads	Only juvenile nematode observed	NW Atlantic (Woods Hole)	Hopkins (1935), Millemann (1951)
Echinocephalus pseudouncinatus	*Centrostephanus coronata* (E)	Gonads	Most infested echinoids had several juvenile nematodes in each of their 5 gonads (142 infested/213 investigated)	E Pacific (Southern California: Santa Catalina Island)	Pearse and Timm (1971)
Echinomermella grayi	*Echinus esculentus* (E)	Coelomic cavity	1 to 4 nematodes echinoid^{-1}; infestation relatively rare	Around British Isles	Gemmill (1901), Gemmill and von Linstow (1902), Irving (1910), Ritchie (1910, see also Barel and Kramers 1977), Comely and Ansell (1988)
Echinomermella matsi	*Strongylocentrotus droebachiensis* (E)	Coelomic cavity	Infestation level: 6 to 56 %	North Sea (Vestfjorden, Norway)	Hagen (1983, 1985, 1987), Jones and Hagen (1987)
Marimermis kerguelensi	*Hippasteria hyadesi* (A)	Coelomic cavity	–	Antarctic seas (off Kerguelen Islands)	Rubstov and Platanova (1974)
Onchaleimus echini	*Echinus esculentus* (E)	Digestive tract	–	–	Leydig (1854)
Thalassonema ephiacanthis	*Ophiacantha antarctica* (O)	Coelomic cavity	–	Antarctic seas	Rubstov (1985)
Thalassonema ophioctinis	*Ophiocten amitium* (O)	Coelomic cavity	1 to 5 juvenile nematodes ophiuroid^{-1} (4 infested/37 investigated)	SW Indian Ocean (South Africa: Glendower Beacon)	Ward (1933)

Table 5-7
Parasitic gastropods from echinoderms. Species names of gastropods according to Warén (1984) (Compiled from the sources indicated)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
I. Parasites of crinoids					
Annulobalcis marshalli	*Crotalometra rustica*	Attached between arm ossicles	2 specimens known from single host	New Zealand (off Mayor Island)	Warén (1981a)
Balcis devians	*Antedon bifida*	Attached to base of pinnule	Only 1 specimen found	North Sea (Plymouth)	Fretter (1955)
Eulima ptilocrinida	*Ptilocrinus pinnatus*	Proboscis deeply inserted inside of the crinoid calix	–	NE Pacific (off British Columbia)	Bartsch (1907)
Goodingia varicosa	*Capillaster multiradiatus*	Attached to aboral side of arms	4 specimens known from 2 hosts	NE Indian Ocean (New Guinea)	Lützen (1972b)
Melanella comatulicola	*Antedon mediterranea*	Attached to pinnules, also to calyx or anal cone	1 to 18 gastropods crinoid^{-1} (27 infested/ 65 investigated)	Mediterranean Sea (Naples, Banyuls)	Graff (1874), Bacci (1948), Changeux (1956)
Mucronalia capillastericola	*Capillaster multiradiatus*	Attached to the oral side of arms	–	Indian Ocean (Red Sea, Singapore)	Bartsch (1909), Fishelson (1973, 1974)
Tropiometricola sphaeroconchus	*Tropiometra afra macrodiscus*	Galls on arms	–	Japan Sea (Honshu)	Habe (1974, 1976), Warén (1981b)
II. Parasites of holothuroids					
Balcis acicula	*Stichopus chloronotus*	Body surface or coelomic cavity	–	Tropical W Pacific (Fiji, Hawaii, Palao)	Habe (1952)
Balcis alba	*Neopentadactyla mixta*	Body surface, near tentacles	Up to 6 gastropods on single host (aquarium observation)	NE Atlantic (Irish coast)	Cabioch and co-authors (1978)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
II. Parasites of holothuroids (continued)					
Balcis catalinensis	*Holothuria arenicola*	Body surface or stomach	Stomach of infested hosts harbors 9 to 26 gastropods according to holothuroid size; percentage of infestation 66 to 100 % depending on locality	Tropical E Pacific (Mexico: Bay of La Paz)	Brand and Ley (1980)
Balcis intermedia	*Holothuria glaberrima*	Firmly attached to outer body surface	1 to 3 gastropods holothuroid^{-1} (12 infested/35 investigated)	Tropical E Pacific (Mexico: Vera Cruz)	Caso (1968)
Diacolax cucumariae	*Cucumaria mendax*	Parasite protrudes outside host body with its rostrum deeply inserted into the holothuroid's coelomic cavity	Only 1 specimen known	Southern Atlantic (51° 10′ S, 64° 15′ W)	Mandahl-Barth (1945)
Enteroxenos bouvieri	*Holothuria atra*	Coelomic cavity	–	Tropical W Pacific (New Caledonia)	Risbec (1953)
Enteroxenos oestergeni	*Parastichopus tremulus*	Mostly hanging in coelomic cavity, attached to esophagus, rarely to stomach or intestine. Some live free in coelomic cavity	5 gastropods holothuroid^{-1} (average number) (537 infested/1515 investigated) (Lützen)	North Sea (Scandinavian coast)	Bonnevie (1902), Oestergren (1938), Lützen (1979)
Enteroxenos parastichopoli	*Parastichopus californicus*	Hanging in coelomic cavity, attached to esophagus	Ca. 3 gastropods holothuroid^{-1} (average number) (37 infested/244 investigated) (Lützen)	NE Pacific (Washington: Puget Sound)	Tikasingh (1961, 1962), Kincaid (1964), Lützen (1979)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
II. Parasites of holothuroids (continued)					
Entocolax chirodotae	*Chirodata pellucida*	Hanging in coelomic cavity, attached to body wall (anterior part)	–	Sea of Japan	Skarlato (1951)
Entocolax ludwigi	*Myriotrochus rinki*	Hanging in coelomic cavity, attached to body wall (anterior part)	–	Behring Sea (Lorenz Bay)	Voigt (1888)
Entocolax rimskykorsacovi	*Myriotrochus mitsukurii*	Hanging in coelomic cavity, attached to intestine	–	Sea of Japan	Ivanov (1945a, quoted from Lützen 1979)
Entocolax schiemenzi	*Chirodota pisani*	Hanging in coelomic cavity, attached to body wall	2 infested holothuroids observed (1 gastropod holothuroid^{-1})	SE Pacific (Chile: Calbuco)	Ludwig (1897, 1898), Voigt (1901)
Entocolax schwanwitschi	*Myriotrochus eurycyclus*	Hanging in coelomic cavity, attached to intestine	1 to 22 gastropods holothuroid^{-1} (10 % of investigated hosts infested; Heding and Mandahl-Barth)	Barents Sea (Kola Bay)	Schwanwitsch (1914), Heding and Mandahl-Barth (1938), Andersen (1971)
Entocolax trochodotae	*Trochodota purpurea*	Hanging in coelomic cavity, attached to body wall (anterior part)	–	SW Atlantic (Falkland Islands)	Heding (1934)
Entoconcha mirabilis	*Oestergrenia digitata*	Hanging in coelomic cavity, attached to ventral hemal vessel of intestine	–	Mediterranean Sea	Baur (1864) (see also Koehler 1895, Lützen 1979)
Gasterosiphon deimatis	*Deima blackei*	In coelomic cavity; proboscis penetrates the intestine hemal system while siphon opens to outer medium across the host's body wall	Only 2 specimens known from a single host	NE Indian Ocean (Bay of Bengal)	Koehler and Vaney (1903, 1905)
Megadenus cantharelloides	*Stichopus chloronotus*	Presumably attached to digestive wall or body wall	Only 2 specimens known from a single host	Tropical Indian Ocean (Aldabra)	Humphreys and Lützen (1972)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
II. Parasites of holothuroids (continued)					
Megadenus holothuricola	*Holothuria mexicana*	Attached to wall of respiratory trees	Only 2 specimens known from a single host	Tropical Atlantic (Bahamas)	Rosén (1910)
Megadenus oneirophantae	*Oneirophanta mutabilis*	Swellings in intestine	The 2 hosts investigated had 3 and 9 gastropods	North Atlantic (deep sea)	Bouchet and Lützen (1980)
Megadenus voeltzkowi	*Holothuria pardalis*	Attached to peri-esophagial ring (presumably water-vascular ring)	Only 1 specimen known	Tropical W Indian Ocean (Zanzibar)	Schepman and Nierstrasz (1914)
Megadenus sp.	*Holothuria atra*	Cloaca	1 to 3 gastropods holothuroid^{-1} (8 infested/1359 investigated)	NE Indian Ocean	Jones and James (1970)
Melanella muelleriae	*Actinopyga mauritiana, Holothuria pervicax, Holothuria cinarescens, Holothuria arenicola*	Projecting from body wall	Some individuals infested	Central Indian Ocean (Aldabra)	Sloan and co-authors (1979)
Molpadicola orientalis	*Molpadia* sp.	Coelomic cavity	–	Okhotsk Sea (deep sea)	Grusov (1957)
Mucronalia variabilis	*Synapta ooplax*	Free on host body surface, or in host digestive tract	–	SW Indian Ocean (Zanzibar)	Vaney (1913), Schepman and Nierstrasz (1914)
Paedophorus dicoelobius	*Eupyrgus pacificus*	In Polian vesicles or respiratory trees	12 gastropods collected from 3 infested holothuroids (80 investigated)	NW Pacific (Peter the Great Bay)	Ivanov (1933, 1937)
Peastilifer edulis	*Holothuria edulis*	Attached to body surface	–	Tropical W Pacific (Great Barrier Reef)	Hoskin and Warén (1983)
Peastilifer nitidula	*Holothuria atra*	Free on body surface	1 to 5 gastropods holothuroid^{-1} (200 infested/400 investigated)	Tropical W Pacific (Hawaii, NE Australia, New Caledonia)	Hoskin and Cheng (1970), Hoskin and Warén (1983)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
II. Parasites of holothuroids (continued)					
Pisolamia brychius	*Oneirophanta mutabilis*	Attached to body surface	26 gastropods collected from 17 infested holothuroids (111 investigated)	NE Atlantic (Bay of Biscay, deep sea)	Bouchet and Lützen (1976, 1980)
Prostifer subpellucida	*Bohadschia argus*, *Holothuria* sp.	Galls in body wall	–	NE Indian Ocean (N Australia: Yonge Reef)	Warén (1980b)
Scalaribalcis angulata	*Holothuria cinerascens*	Galls in body wall	–	SE Pacific (Easter Island)	Mandahl-Barth (1949), Warén (1980b)
Thyonicola americana	*Eupentacta quinquesemita*, *Eupentacta pseudo-quinquesemita*	Hanging in coelomic cavity, attached to posterior intestine	Overall incidence of infestation: 41 % (1 to several hundred parasites host^{-1}, Wright; infestation highly variable according to the host populations, Byrne)	NE Pacific (US and Canadian coasts)	Tikasingh (1961), Wright (1974), Byrne (1985)
Thyonicola dogieli	*Cucumaria miniata*, *Cucumaria japonica*, *Cucumaria obunca*	Hanging in coelomic cavity, attached to posterior intestine	–	NE Pacific (?)	Ivanov (1945b, quoted from Lützen 1979)
Thyonicola mortenseni	*Thyone serrata*	Hanging in coelomic cavity, attached to posterior intestine	About 40 gastropods in a single holothuroid	SE Indian Ocean (off Cape of Good Hope)	Mandahl-Barth (1941)
III. Parasites of echinoids					
Euchineulima eburnea	*Chaetodiadema granulatum*, *Astropyga radiata*, *Astropyga pulvinata*, *Heterocentrotus mammillatus*, *Heterocentrotus trigonaria*	Attached to oral side of body surface	1 to 4 gastropods echinoid^{-1}	Tropical Indo-Pacific	Lützen and Nielsen (1975)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
III. Parasites of echinoids (continued)					
Euchineulima mittrei	*Echinothrix diadema, Echinothrix calamaris, Diadema setosum, Diadema mexicanum, Diadema savignyi*	Attached to oral side of body surface	1 to 6 gastropods echinoid^{-1}	Circumtropical	Lützen and Nielsen (1975)
Euchineulima ponderi	*Parasalenia gratiosa*	Attached to peristome	Only 2 specimens from single host	Tropical W Pacific (Great Barrier Reef: Lizard Island)	Warén (1980a)
Luetzenia asthenosoma	*Asthenosoma* sp.	Attached to peristome	Only 2 specimens from single host	SW Pacific (Australia: New South Wales)	Warén (1980b)
Megadenus cysticola[1]	*Stylocidaris tiara*	Galls in primary spines	1 to 7 gastropods echinoid^{-1}	E Indian Ocean (off Ceylon)	Koehler (1924, 1927), Koehler and Vaney (1925)
Monogamus entopodia	*Echinometra mathaei*	Tube foot wall	21 gastropods from 10 infested echinoids	Red Sea (Gulf of Aqaba)	Lützen (1976)
Monogamus interspinea	*Echinometra mathaei*	Buried in skin	2 gastropods from 2 infested echinoids (55 investigated)	SW Indian Ocean (Amboina)	Lützen (1976)
Monogamus parasaleniae	*Parasalenia gratiosa*	Galls in spines	2 gastropods from single host	Tropical Pacific (Tonga Islands)	Warén (1980b)
'Mucronalia' sp.	*Stylocidaris tiara, Stereocidaris indica*	Attached to body surface, producing conspicuous test deformations	–	E Indian Ocean (Ceylon, Bay of Bengal)	Koehler (1927)
Pelseenaria media	*Echinus affinis*	Attached to body surface	–	NE Atlantic (off Azores: deep sea)	Koehler and Vaney (1908)
Pelseenaria minor	*Echinus affinis*	Attached to body surface	–	North Sea (Banc de Seine)	Koehler and Vaney (1908)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
III. Parasites of echinoids (continued)					
Pelseenaria profunda	*Genocidaris maculata*	Attached to body surface	11 echinoids infested (several hundred investigated)	NE Atlantic (off Azores: deep sea)	Koehler and Vaney (1908)
Pelseenaria stilifera	*Strongylocentrotus droebachiensis, Echinus esculentus*	Attached to body surface	4 gastropods from single host (Ankel)	Baltic Sea (Kristinenberg)	Ankel (1938), Mortensen (1940)
Pulicochlea calamaris	*Echinothrix calamaris*	Free on body surface	Rather frequent infestation	Tropical W Pacific (Hawaii, Papua New Guinea, New Caledonia)	Ponder and Gooding (1978)
Pulicochlea fusca	*Diadema setosum*	Free on body surface	Numerous gastropods collected	Tropical W Pacific (Papua New Guinea and adjacent islands)	Ponder and Gooding (1978)
Robillardia cernica	*Echinometra mathaei, Echinometra insularis*	Attached to wall of rectum	1 to 2 gastropods echinoid^{-1} (54 infested/185 investigated)	Indian Ocean (Red Sea, Mauritius, Amboina); SW Pacific (Easter Island)	Gooding and Lützen (1973)
Sabinella infrapatula	*Ogmocidaris benhami*	Attached to body surface, close to periproct	Only 1 individual found	SW Pacific (New Zealand: off Major Island)	Warén (1981a)
Sabinella troglodytes[2]	*Eucidaris tribuloides*	Galls in primary spines	Infestation relatively rare (33 infested/1467 investigated) (McPherson)	Tropical Atlantic (Cape Verde Islands, Florida)	Thiele (1925), Pilsbry (1956), McPherson (1968)
Trochostilifer mortenseni	*Prionocidaris australis*	Galls in primary spines	1 gall with 2 gastropods in each infested echinoid	Tropical W Pacific (New Caledonia)	Warén (1980b)
Vitreobalcis temnopleuricola	*Temnopleurus toreumaticus*	Attached to body surface	Infestation rate varied from 5.3 to 50% depending on host population and season	Inland Sea (Japan)	Fujioka and Habe (1983), Fujioka (1984, 1985)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
IV. Parasites of asteroids					
Apicalia palmipedis	*Palmipes rosaceus*	Attached to body surface (oral side)	1 to 2 gastropods per infested asteroid	NW Indian Ocean (Ceylan, Singapore)	Koehler (1910), Koehler and Vaney (1912), Warén (1981b)
Asterolamia cingulatus	*Craspidaster hesperus*	Attached to side of body (marginal plates)	–	NW Pacific (Hong Kong)	Warén (1980b)
Asterolamia hians	*Astropecten indicus*	Attached to aboral body surface, among paxillae	–	Tropical Pacific (Great Barrier Reef)	Warén (1980b)
Asterophila japonica	*Pedicellaster magister, Ctenodiscus crispatus, Leptasterias polaris, Leptasterias arctica*	Coelomic cavity attached to the body wall	10 to 29 % asteroids infested depending on locality (Hoberg and co-authors)	N Pacific (off Japan, Asiatic coast, Alaska)	Randall and Heath (1911), Grusov (1965), Hoberg and co-authors (1980)
Paramegadenus arrhynchus	*Anthenoides rugulosus*	Open gall on body surface (aboral side)	–	Tropical W Pacific (Philippines: near Cebu)	Kanazawa & Habe (1979), Warén (1980b)
Paramegadenus scutellicola	*Stellaster incei*	On tube feet	–	Tropical W Pacific (Great Barrier Reef)	Warén (1980b)
Parvioris equestris	*Stellaster incei*	Attached to body surface (marginal plates)	–	Indo-West Pacific (Andaman Islands, Java Sea, Great Barrier Reef)	Koehler (1910), Koehler and Vaney (1912), Warén (1981b)
Parvioris mortoni[3]	*Archaster typicus*	Attached to body surface (aboral or lateral side)	1 to 4 gastropods asteroid^{-1} (75 infested/ 396 investigated)	NW Pacific (Hong Kong)	Morton (1976), Warén (1981b)
Stilifer astericola	*Heliaster cumingi*	Gall in body wall	Up to 5 gastropods asteroid^{-1}	E Pacific (Galapagos)	Lützen (1972a)
Stilifer inflatus	*Linckia laevigata*	Gall in body wall	Only 1 specimen known	Tropical W Pacific (Great Barrier Reef)	Warén (1980a)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
IV. Parasites of asteroids (continued)					
Stilifer linckiae	*Linckia multifora*	Gall in body wall	1 to 2 galls asteroid^{-1} (54 infested/665 investigated) (Davis)	Tropical Indo-West Pacific (Oman Sea, Ceylon, Great Barrier Reef, Hawaii)	Sarasin and Sarasin (1887), Davis (1967), Tullis and Cheng (1971), Lützen (1972a), Warén (1980a)
Stilifer ophidiastericola	*Ophidiaster cribrarius, Ophidiaster lorioli, Ophidiaster confertus, Ophidiaster granifer*	Gall in body wall	–	Tropical Indo-Pacific (Indonesia to SW Japan)	Habe (1976)
Stilifer ovoideus[4]	*Certonardoa semiregularis, Ophidiaster granifer, Tamaria dubiosa*	Gall in body wall	–	Tropical Indo-Pacific (Indonesia to SW Japan)	Hirase (1927, 1932), Lützen (1972a), Habe (1976)
Stilifer utinomi	*Linckia guildingi, Linckia laevigata*	Gall in body wall	–	Tropical W Pacific (Great Barrier Reef, SW Japan)	Habe (1952) Lützen (1972a)
Stilifer sp.	*Ophidiaster granifer*	Gall in body wall	Up to 4 gastropods asteroid^{-1} (8 infested/26 investigated)	Tropical W Pacific (Guam)	Yamaguchi and Lucas (1984)
Thyca callista	*Phataria unifascialis, Pharia pyramidata*	Attached to body surface	1 to 3 gastropods asteroid^{-1}, infestation rather rare	Tropical E Pacific (coast of Mexico and central America)	Berry (1959), Shasky (1961), Bertsch (1975)
Thyca crystallina	*Linckia multifora, Linckia laevigata*	Attached to body surface	Infestation rate variable: from 14 to 62 % depending on localities	Tropical Indo-West Pacific (Indonesia, Papua New Guinea, Great Barrier Reef, Fiji)	Egloff (1966), Elder (1979), Warén (1980a), Bouillon and Jangoux (1984), Egloff and coauthors (1988)
Thyca ectoconcha	*Linckia multifora, Linckia guildingi*	Attached to body surface	Infestation rate ca 3 % (MacNae and Kalk)	Indian Ocean (Ceylon, Mozambique coast)	Sarasin and Sarasin (1887), MacNae and Kalk (1962)

Table 5-7 (continued)

Gastropod	Host	Location in host	Remarks	Geographical area	Source
IV. Parasites of asteroids (continued)					
Thyca stellasteris	*Stellaster equestris*	Attached to body surface	–	Indian Ocean (Andaman Islands, West Australia, Red Sea)	Koehler (1910), Koehler and Vaney (1912), Warén (1980a)
V. Parasites of ophiuroids					
Fuscapex ophiocanthicola	*Ophiocantha* sp.	Attached to body surface (oral side), covering bursal slits	3 gastropods from single host	SW Pacific (off Kermadec Islands, deep sea)	Warén (1981a)
Ophieulima armigeri	*Ophiomusium armigerum*	Attached to body surface (oral side), near bursal slits	Up to 5 gastropods ophiuroid^{-1} (23 infested/ more than 3000 investigated)	NW Atlantic (off Virginia)	Warén and Carney (1981)
Ophieulima fuscoapicata	*Ophiactis profundi*	Attached to body surface (radial shields)	2 gastropods from single host	SW Pacific (off Kermadec Islands, deep sea)	Warén (1981a)
Ophieulima minima	*Ophiactis abyssicola*	Attached to body surface (aboral side)	–	N Atlantic (deep-sea: off Ireland, off Iceland, Bay of Biscay)	Warén and Sibuet (1981)
Ophioarachnicola biformis	*Ophioarachna incrassata*	Attached to body surface (oral side of arm)	Only 1 gastropod found	Tropical W Pacific (Salomon Islands)	Warén (1980b)
Punctifera ophiomoerae	*Ophiomoeris projecta*	Open galls (aboral side of the disc)	2 gastropods from single host	SW Pacific (off Kermadec Islands: deep sea)	Warén (1981a)

[1] Generic position unclear (see Warén 1980b).
[2] Identified as *Mucronalia nidorum* by Pilsbry (1956) and McPherson (1968) (see Warén 1980b).
[3] Identified as *Eulima shoplandi* by Morton (1976) (see Warén 1981b).
[4] Identified as *Stilifer celebensis* by Hirase (1927, 1932) (see Warén 1980a).

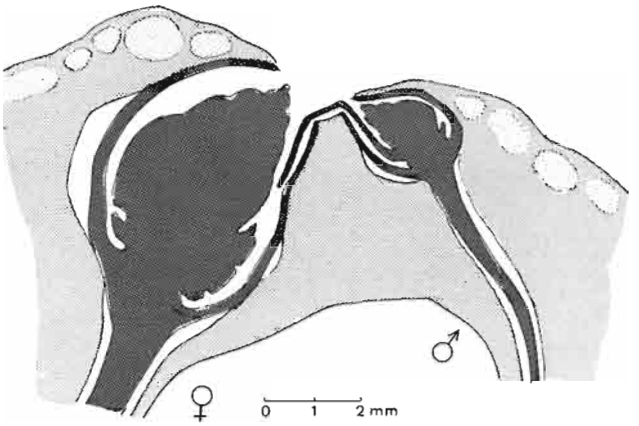


Fig. 5–16: *Stilifer linckiae*. Position of 2 specimens of a parasitic gastropod in a gall in the arm of the asteroid *Linckia laevigata*. (After Lützen, 1972a.)

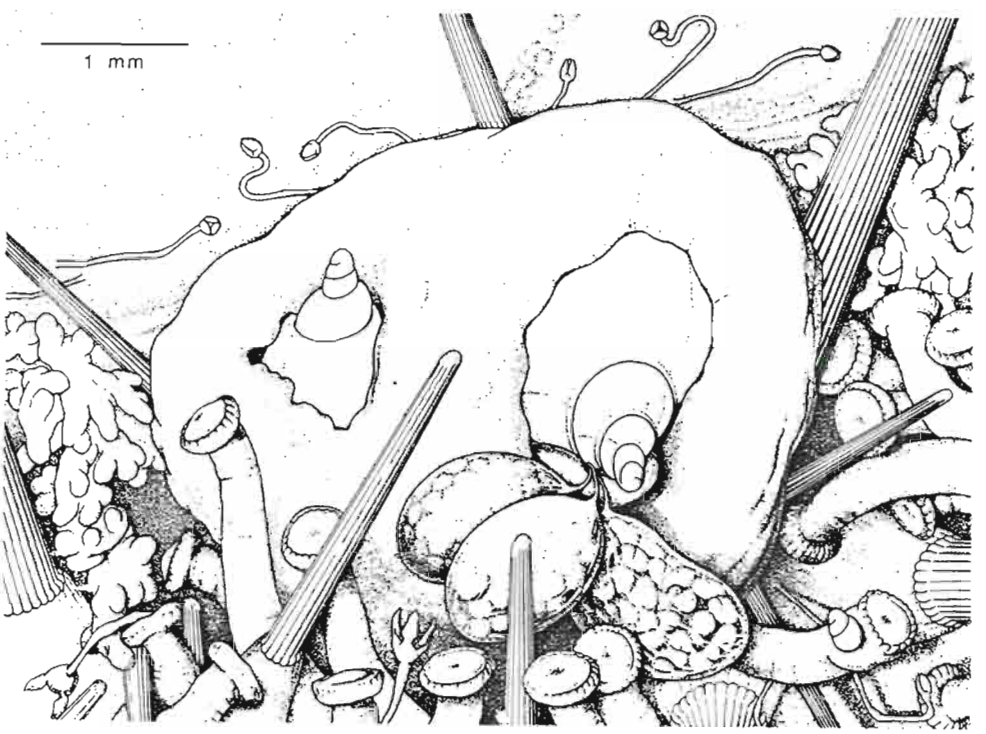


Fig. 5–17: *Monogamus entopodia*. Male and female (with 3 egg capsules) of a parasitic gastropod in a transformed tube foot of the echinoid *Echinometra mathaei*. (After Lützen, 1976.)

additional predation upon internal organs presumably occurs. Other fluid-feeding eulimids insert their proboscis into the hemal lacunae of holothuroids or asteroids (the hemal system has energy-rich content). Such a symbiosis has been documented by Bouchet and Lützen (1976, 1980) who studied relations between *Pisolamia brychius* and the deep-sea

holothuroid *Oneirophanta mutabilis* (Fig. 5-18), and by Egloff and co-authors (1988) who reported on the penetration of the radial hemal system of the asteroid *Linckia laevigata* by the proboscis of *Thyca crystallina*. Ectoparasitic gastropods may also feed directly on internal organs (i.e., digestive organs); this has been suggested by Warén (1980b) for 2 species of *Asterolamia*.

Little information is available on the feeding biology of intradigestive eulimids. An unusual feeding habit was reported for 2 unattached species of holothuroid-associated snails, *Mucronalia variabilis* and *Balcis catalinensis,* symbiotic with *Synapta ooplax* and *Holothuria arenicola* (Vaney, 1913; Brand and Ley, 1980). The snails move freely on the body surface of their host but may enter the host's digestive tract in order to feed by puncturing the digestive wall. The presence of several individuals of *B. catalinensis* in the stomach of *H. arenicola* does not cause significant effects on the absorption efficiency of

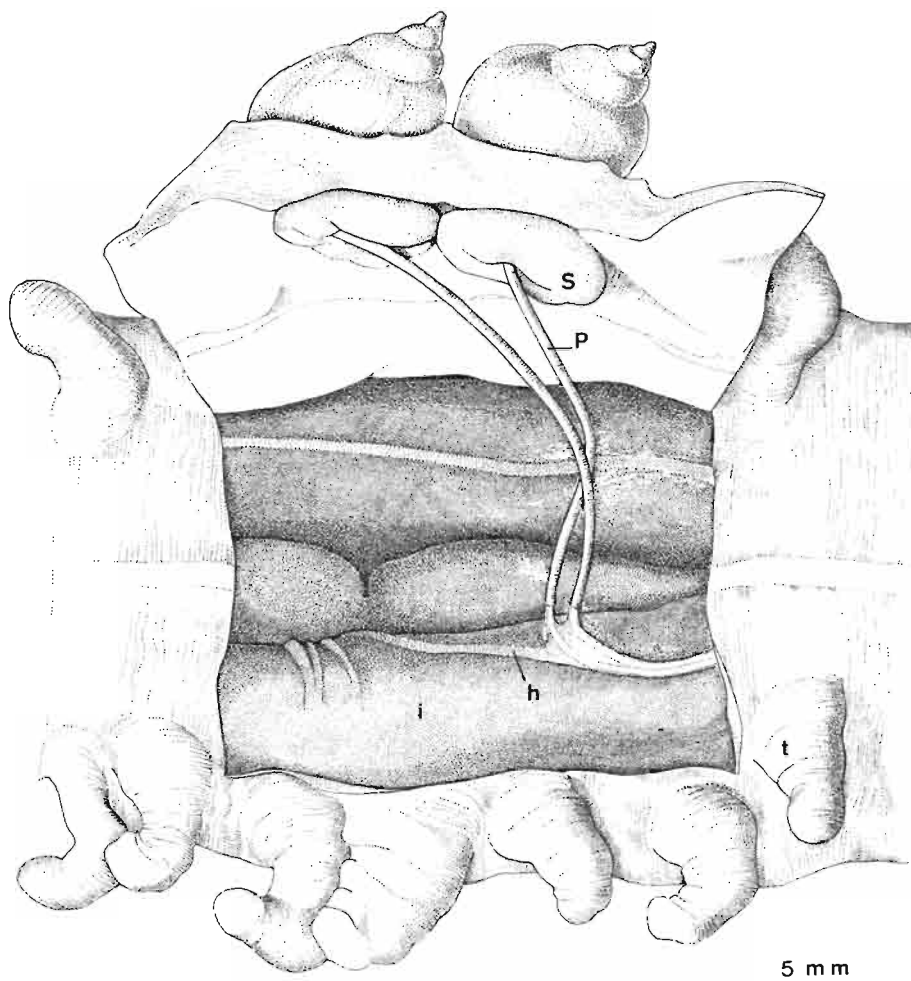


Fig. 5–18: *Pisolamia brychius,* a blood-sucking gastropod parasite of the deep-sea holothuroid *Oneirophanta mutabilis*. (i) Intestine of holothuroid; (h) hemal vessel of holothuroid; (P) proboscis; (S) snout; (t) tube-foot. (After Bouchet and Lützen, 1976.)

the host (Brand and Ley, 1980). Gooding and Lützen (1973) provide evidence that *Robillardia cernica* which inhabits the rectum of the echinoid *Echinometra insularis* feeds on host gonads, using its elongated proboscis. *Megadenus oneirophantae* lives in cyst-like swellings in the digestive wall of a deep-sea holothuroid. According to Bouchet and Lützen (1980), it supposedly feeds on content of the digestive hemal lacunae. A most peculiar feeding habit is that of *Megadenus cantharelloides*: it attaches to the digestive wall of *Stichopus chloronotus* — the visceral mass of the parasite protruding into the digestive cavity — with its proboscis crossing both digestive wall and coelomic cavity and penetrating the host's body wall, in order to feed on dermal tissue (Humphreys and Lützen, 1972).

Oral feeding by intracoelomic eulimids has been inferred only for *Gasterosiphon deimatis* which inserts its proboscis into digestive hemal lacunae (Koehler and Vaney, 1903). Other intracoelomic forms (viz. the aberrant *Entocolax* and allied genera, i.e., the former Entoconchidae; e.g., Tikasingh and Pratt, 1961; Lützen 1968a, 1979; Byrne, 1985) are believed to derive their energy from the host's coelomic fluid by direct absorption of nutrients through their body wall. Intracoelomic parasitic gastropods occur only in asteroids (eulimid genus *Asterophila*) and in holothuroids (eulimid genera *Diacolax, Enteroxenox, Entoconcha, Gasterosiphon, Molpadicola, Paedophorus, Thyonicola*) (Table 5-7; Figs 5-19 and 5-20). Most of them are attached to the coelomic side of either digestive tract or body wall of their host by a hollow stalk or by a siphon. Although some authors have suggested that feeding could take place through that duct (e.g., Heath, 1910; Tikasingh, 1962), such a hypothesis has not been accepted generally.

Harmful effects of parasitic gastropods are not restricted to their feeding activities, Ectoparasitic eulimids may produce clearly definable attachment lesions (Lützen and Nielsen, 1975; Lützen, 1976; Elder, 1979). Host reactions produce conspicuous soft swellings of the dermal tissue in parasitized echinoid tube feet (Lützen, 1976) and in infested crinoid pinnules (Bacci, 1948; Fretter, 1955). Gallicole eulimids (e.g., *Stilifer* spp., *Puctifera ophiomoerae, Tropiometricola sphaeroconchus;* Table 5-7) produce spectacular hard swellings or galls in the body wall of asteroids, ophiuroids and crinoids (see also Grygier, 1988). These galls resemble those induced by myzostomids on crinoid arms. Whether or not they involve particular modifications of host skeleton has not been investigated. According to Davis (1967), *S. linckiae* suppresses the autotomizing capability of the asteroid arm in which it is located. Other gallicole species modify primary spines of cidaroid echinoids (Koehler and Vaney, 1925; Koehler, 1927; Pilsbry, 1956; Warén, 1980b). In most cases the snails bore into the distal part of spines which then enlarges. Sometimes spine-dwelling gastropods induce a conspicuous regression, the spine being reduced to its swollen basal part (Koehler and Vaney, 1925). Nothing is known of the feeding habits of these spine-associated eulimids. The host skeleton is also affected by non-gallicole species. Fishelson (1973, 1974) reported that *Mucronalia capillastericola* attaches to crinoid arms and causes a pronounced twist and degeneration of the arm skeleton above the place of attachment. Egloff and co-authors (1988) indicated that a partial alteration of ambulacral ossicles and a loss or reduction in size of ambulacral ampullae occur at the place where the proboscis of *Thyca crystallina* penetrates by the body wall of the asteroid *Linckia laevigata*. Koehler and Vaney (1912) and Vaney (1913) drew attention to the particular gaps occurring in the marginal skeleton of asteroids infested by *Parvioris equestris*. According to them, the absence of marginal plates is the consequence of the early attachment of parasitic snails which had inhibited skeletal growth. Eulimids parasitiz-

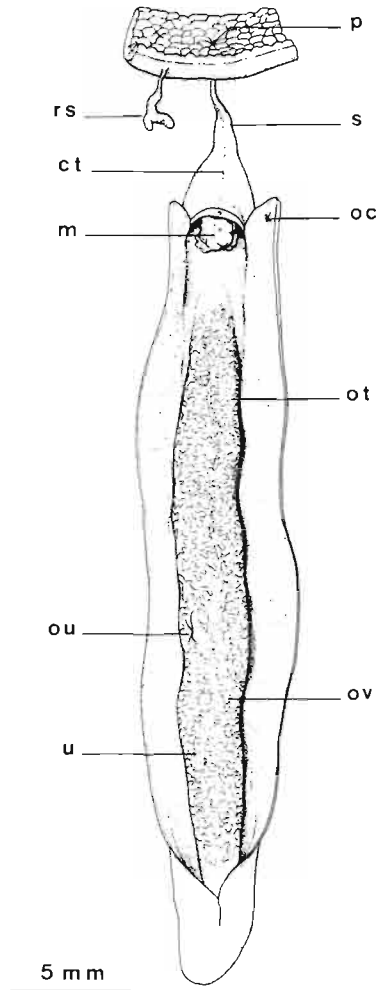


Fig. 5–19: *Enteroxenos oestergreni*, an intracoelomic gastropod parasite of the holothuroid *Stichopus tremulus*. (ct) Ciliated tubule; (m) modified male implanted in receptaculum masculinum; (p) pit in the wall of host's esophagus; (rs) remains of stalk of another individual; (s) stalk; (oct) opening of ciliated tubule; (ot) ovarian tubules; (ou) opening of uterus; (ov) oviduct; (u) uterus. (After Lützen, 1979.)

ing cidaroid echinoids may induce conspicuous test swellings implying deformations of the test skeleton (Döderlein, 1906; Koehler, 1927) (Fig. 5-21). Pyriform test deformations caused by a *Mucronalia*-like species were reported by Mortensen (1943a) for the echinoid *Salmacis bicolor*. According to Byrne (1985) the intracoelomic eulimid *Thyonicola americana* for the most part did not appear to affect its holothuroid host *(Eupentacta quinquesemita)*. She noted, however, that heavily parasitized hosts could be detected by their apparent inability to keep their tentacles fully retracted and that, in some cases of mass infestation, the parasites may interfere with the use of holothuroid's tentacles for suspension feeding.

That parasitic eulimids may produce partial castration of the host was considered by

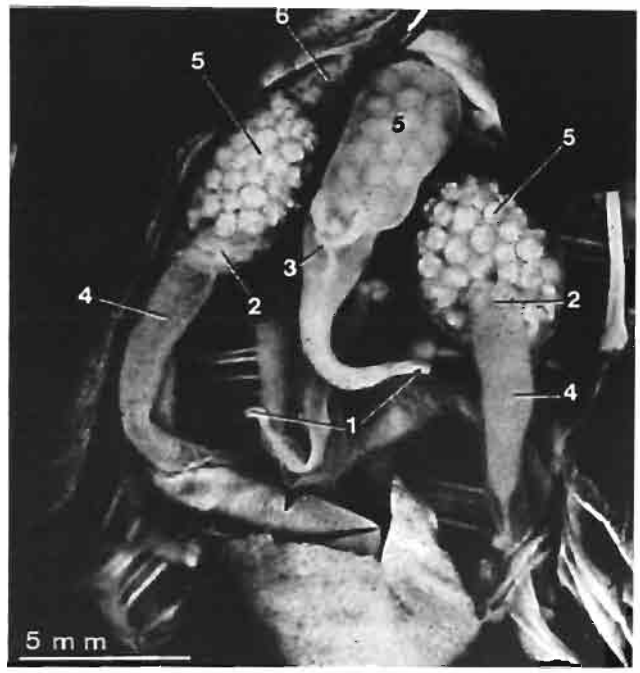


Fig. 5–20: *Myriotrochus rinki*. Holothuroid parasitized by 3 ovigerous specimens of the intracoelomic gastropod *Entocolax ludwigi*. (1) Oral end; (2) ovary; (3) oviduct; (4) part of the body containing intestine; (5) pseudopallium with egg capsules; (6) siphon. (After Lützen, 1979.)

Gooding and Lützen (1973). They found the size of the gonads in echinoids infested by *Robillardia cernica* to be usually smaller than with uninfested echinoids. According to Heding and Mandahl-Barth (1938), intracoelomic *Entocolax* spp. may castraste their host, while Lützen (1979) reported that *Enteroxenox oestergreni* is not likely to influence the fecundity of the holothuroid *Stichopus tremulus*.

Few host reactions have been reported from echinoderms parasitized by gastropods, except the production of dermal swellings and galls. This does not imply that echinoderms do not react to snail infestations. A particular host-parasite relation must be noticed, namely the constant presence of a host envelope surrounding the intracoelomic entoconchid eulimids from holothuroids (Vaney, 1913; Tikasingh, 1962; Wright, 1974). This envelope consists of an outer mesothelial layer and of an inner connective tissue layer. Considering the as yet unsolved question of entoconchid nutrition, it would be worth investigating whether or not host hemal lacunae occur within the inner layer of the envelope. A similar envelope was found around individuals of *Asterophila japonica*, an intracoelomic entoconchid from asteroids (Hoberg and co-authors, 1980). The mesothelial cover surrounding intracoelomic gastropods should occur presumably around any part of the parasitic snail which permanently crosses the coelomic cavity (a mesothelial covering of the parasite proboscis has been noticed by Warén, 1980b, for *Asterolamia hians*).

So far there are practically no data indicating that parasitism by eulimids can seriously alter the echinoderm life cycle. Eulimids do not — or only exceptionally — produce host castrastion, nor do they have any measurable effect on the biology of their host, not even

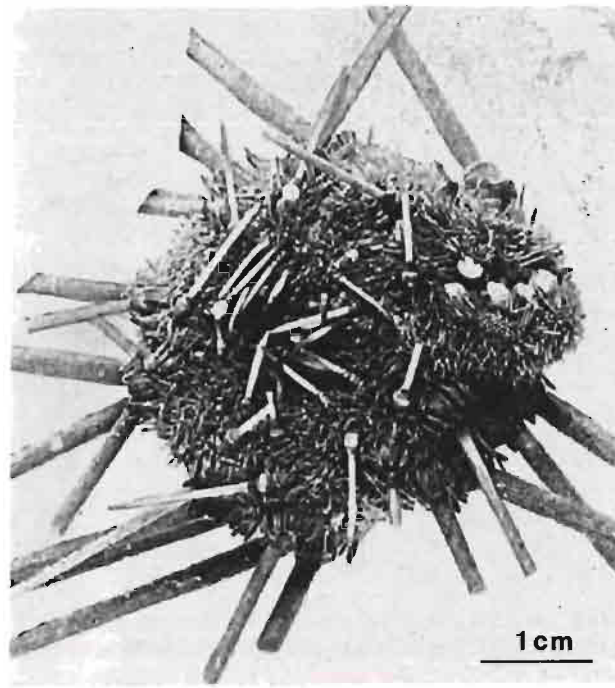


Fig. 5–21: *Stereocidaris tricarinata*. Oral view of cidaroid echinoid showing test deformations produced by parasitic gastropods (*Stilifer* sp.). (After Döderlein, 1906.)

when mass infestations occur. (It may be presumed, however, that eulimids involving skeletal deformations are rather constraining for the echinoderms.) All these facts suggest that the ecological consequences of parasitism due to eulimid gastropods may be quite limited for the echinoderm involved.

Agents: Mollusca Bivalvia

Bivalves associated with echinoderms have been recorded almost exclusively from echinoids and holothuroids (Boss, 1965). Most echinoid-associated bivalves are simply attached to the host's spines through byssal threads (e.g., Gage, 1966; Barel and Kramers, 1977). However, Bernard (1895, 1896) described a species *(Scioberetia australis)* which lives in the brood pouches of the Antarctic spatangoid *Abatus cavernosus*. According to Bernard, only the female of *A. cavernosus* without *S. australis* had developing embryos in their brood pouches. This might imply that the bivalves inhibit the development of embryos or prevent their settlement in brood pouches.

A few bivalve species live ectosymbiotic on synaptid holothuroids. They attach to the synaptid body surface through their spade-shaped creeping foot. It is generally agreed that creeping bivalves do not affect their host, except that they may slightly erode its skin (Anthony, 1916; Popham, 1940). Three species of endosymbiotic bivalves have been reported from holothuroids. There is almost no information on the relations between *Holothuria fuscocineria* and *Entovalva major* which is only said to supposedly live in the

holothuroid cloaca (Bruun, 1983). *Entovalva mirabilis* and *Cycladoconcha amboinensis* inhabit small pouches dug into the digestive wall of synaptid holothuroids (see Voeltzkow, 1890 and Schepman and Nierstrasz, 1914, and Spärck 1931, respectively). These 3 species are presumably typical suspension feeders.

Agents: Entoprocta

A few Loxosomatidae appear to be relatively common symbiotes of crinoids and ophiuroids, especially in polar and subpolar areas. Mortensen (1910, 1911) reported the occurrence of *Loxosomella antedonis* on cirri of *Poliometra prolixa* and *Heliometra glacialis*. *L. antedonis* appears to secrete a cement on the surface of the crinoid cirri and then attach to its host. The ophiuroid *Amphiocnida pilosa* often supports individuals of *Loxosoma* sp. attached to various places of the oral side of its disc and arms (Mortensen, 1924). According to Moyano and Wendt (1981) the entoproct *Barentsia discreta* may attach to the bivium of the Antarctic holothuroid *Psolus charcoti*.

Agents: Annelida Polychaeta

Symbiotic polychaetes were reviewed by Paris (1955) and Clark (1956), both authors stating that parasitic polychaetes rarely affect echinoderms. While numerous species are known to live ectocommensally on echinoderms, only 3 cases of parasitism have been reported with polychaetes. According to Monticelli (1892) the eunicid *Ophryotrocha puerilis* occurs in the coelomic cavity of the holothuroid *Ocnus planci* from Naples (Italy). Ganapati and Radhakrishna (1962) noted that 50% of the holothuroid *Molpadia* sp. investigated harbored the small hesionid *Ancistrocyllis* sp. either in the digestive tract or respiratory trees. The only case of unequivocal parasitism is that of the lumbrinereid *Ophiuricola cynips* which forms myzostomid-like galls at the base of the arms of the deep-sea ophiuroid *Ophioglypha tumulosa* (= *Ophiura irrorata*) (see Ludwig, 1905). According to Ludwig, the galls are rather large and partly protrude into the host's coelomic cavity.

Agents: Annelida Myzostomida

The class Myzostomida (about 140 described species) occupies a peculiar place among echinoderm symbiotes. They are polychaete-related aberrant annelids with a small flattened body several mm in length. Their most extraordinary feature is their intimate association with echinoderms: there are no free-living myzostomids, nor is there any species associated with other phyla. Myzostomids mostly infest crinoids, but a few are known from asteroids and ophiuroids. Myzostomids are highly differentiated, both morphologically and ecologically. Their almost obligatory association with crinoids (they even infested now extinct crinoids; e.g., Meyer and Ausich, 1963; Arendt, 1985) suggests that they are an ancient group which evolved together with crinoids. Myzostomids may be considered a unique case of co-evolution involving a whole class of organisms (e.g., Clark, 1921; Stummer-Traunfels, 1926; Prenant, 1959).

About 30 species of myzostomids have been recorded as echinoderm parasites (Table 5-8), the remaining species being frequently referred to 'free-living'. However, these latter species are ectocommensals which generally live unattached on the echinoderm body

Table 5-8

Parasitic myzostomids from echinoderms. Myzostomid classification and species names according to Jägersten (1940). Hosts: A, asteroid; C, crinoid; O, ophiuroid. Records of conspicuous deformations caused by unidentified myzostomids were reported on several occasions (e.g. Carpenter 1889 for *Acitinometra notata*). Speel and Dearborn (1983) noted that each of the 96 individuals of *Promochocrinus kerguelensis* they observed harbored 1 to 3 myzostomid cysts (Compiled from the sources indicated)

Myzostomid	Host	Location on/in host	Remarks	Geographical area	Source
I. Proboscidea					
Myzostomum beardi	*Perissometra flexilis* (C)	Galls on arms	Only 1 worm gall^{-1}	NE Indian Ocean (Arafura Sea)	Graff (1887)
Myzostomum belli	*Endoxocrinus alternicirrus* (C)	Galls at base of arms	Only 1 worm gall^{-1}	NW Pacific (S Philippines)	Wheeler (1896)
Myzostomum cryptopodium	*Metacrinus interruptus* (C)	Galls on arms	–	Indian Ocean (Bay of Bengal?)	Wheeler (1896)
Myzostomum deformator	*Endoxocrinus alternicirrus* (C)	Galls in pinnules	2 worms (♂, ♀) gall^{-1}	NW Pacific (SE Philippines)	Graff (1884)
Myzostomum eremita	*Metacrinus moseleyi* (C)	Galls at base of arms and pinnules	Only 1 worm gall^{-1}	NW Pacific (SE Philippines)	Wheeler (1896)
Myzostomum pentacrini	*Endoxocrinus alternicirrus* (C)	Slight galls extending into 3 to 6 arm segments	1 to 3 worms gall^{-1}	NW Pacific (SE Philippines)	Graff (1884)
Myzostomum taeniatum	*Zygometra mertoni* (C)	Juvenile in cysts on pinnules; adults free-living	1 worm cyst^{-1}	NW Indian Ocean (Aru Islands)	Remscheid (1916)
Myzostomum tenuispinum	*Pachylometra inaequalis, Perissometra flexilis, Charitometra basicurva, Charitometra incisa* (C)	Conspicuous galls extending into 2 to 3 arm segments	2 worms (♂, ♀) gall^{-1}; several galls on each infested host	Tropical W Pacific (SE Philippines, Fiji Islands and Kermadec Island)	Graff (1884)
Myzostomum willemoesi	*Pachylometra inaequalis, Perissometra flexilis, Charitometra basicurva* (C)	Galls in pinnules	2 worms (♂, ♀) gall^{-1}	Tropical W Pacific (Fiji and Kermadec Islands); NE Indian Ocean (Arafura Sea)	Graff (1884, 1887)
Myzostomum sp.	*Lamprometra palmata* (C)	In sac-like uncalcified cysts on pinnules	1 worm cyst^{-1}	NW Indian Ocean (Australia: Northern Territory)	Grygier (1988)

Table 5-8 (continued)

Myzostomid	Host	Location on/in host	Remarks	Geographical area	Source
II. Pharyngidea					
Asteromyzostomum arcticum	*Urasterias lincki* (A)	Attached in ambulacral groove	–	Arctic seas	Wagin (1954, in Grygier 1988)
Asteromyzostomum multiplicatum	*Crossaster papposus* (A)	Attached in ambulacral groove	–	Arctic seas	Wagin (1954, in Grygier 1988)
Asteromyzostomum witjasi	*Psilaster pectinatus* (A)	Attached in ambulacral groove	–	Arctic seas	Wagin (1954, in Grygier 1988)
Asteriomyzostomum sp.	*Labidiaster* sp. (A)	Attached to body surface (between arms)	–	Antarctic seas	Grygier (1988)
Asteriomyzostomum asteriae	*Sclerasterias neglecta, Sclerasterias richardi* (A)	In hypertrophied pyloric caeca (proximal part of the caeca)	1 to 3 worms infested asteroid^{-1}	Mediterranean sea	Marenzeller (1895a, b), Stummer-Traunfels (1903)
Asteromyzostomum fisheri	*Tosia leptoceramus* (A)	Coelomic cavity, loosely attached to body wall	–	Tropical E Pacific (off South California)	Wheeler (1904)
Cystimyzostomum clarki	*Metacrinus rotundus* (C)	Subcutaneous cysts on underside of arms	1 worm cyst^{-1}; up to 7 cysts host^{-1}	N. Pacific (Japan: Sagami Sea)	McClendon (1906)
Cystimyzostomum cysticolum	*Anthometra adriani, Amphimetra discoidea, Comactinia meridionalis* (C)	Subcutaneous calcified cysts on upper side of arms	2 worms (♂, ♀) cyst^{-1}; no more than 1 cyst on each host arm	Cosmopolitan (Caribbean; Red Sea; Aru Islands; Eastern coast of Japan)	Graff (1884), McClendon (1906), Remscheid (1916), Fishelson (1973, 1974)
? Cystimyzostomum ijimai	*Tropiometra macrodiscus, Capillaster multiradiatus* (C)	Subcutaneous cyst on calyx (upper surface)	1 to 2 worms cyst^{-1}	Indo-West-Pacific (Sagami Sea, Japan; Red Sea)	Hara and Okada (1921), Fishelson (1973, 1974)
? Cystimyzostomum inflator	*Neocomatella pulchella, Adelometra angustiradia* (C)	Subcutaneous calcified cysts at arm base (upper surface)	2 worms (♂, ♀) cyst^{-1}	Circumtropical (Papua; Barbados)	Graff (1884)
Cystimyzostomum murrayi	*Horaeometra duplex, Stiremetra breviradia, Adelometra angustiradia* (C)	Subcutaneous stalked and calcified cysts on calyx (upper surface)	2 worms (♂, ♀) cyst^{-1}	Circumtropical (Papua; Kermadec Islands; Barbados)	Graff (1884)

Table 5-8 (continued)

Myzostomid	Host	Location on/in host	Remarks	Geographical area	Source
II. Pharyngidea (continued)					
? Cystimyzostomum platypus	*Comanthina schlegeli* (C)	Subcutaneous cyst on calyx (upper surface)	1 worm cyst^{-1}	W Pacific (Philippines: off Samboangan)	Graff (1887)
Cystimyzostomum robustum	*Metacrinus rotundus* (C)	Cysts on arms	1 worm cyst^{-1}	NW Pacific (Japan: Sagami Sea)	Hara and Okada (1921)
Mesomyzostoma katoi	*Comanthus japonicus* (C)	Gonads (genital pinnules)	–	Sea of Japan	Okada (1933)
Mesomyzostoma reichenspergi	*Amphimetra discoidea* (C)	Coelomic cavity of arms	Only 2 individuals known	NW Indian Ocean (Aru Islands)	Remscheid (1916)
Protomyzostomum astrocladi	*Astrocladus coniferus* (O)	Encysted in gonads	–	NW Pacific (Sagami Sea)	Fedotov (1925)
Protomyzostomum polynephris	*Gorgonocephalus arcticus, Gorgonocephalus eucnemis, Gorgonocephalus caputmedusae* (O)	Bursae and gonads	10 to 20 worms host^{-1}	North Sea (Scandinavian coast); Barents Sea	Fedotov (1912, 1914, 1916), Barel and Kramers (1977)
Protomyzostomum sagamiense	*Gorgonocephalus* sp. (O)	Bursae and gonads	–	NW Pacific (Sagami Sea)	Okada (1922)
Pulvinomyzostomum pulvinar	*Leptometra phalangium, Antedon bifida* (C)	Digestive tract	Infestation level: 10 to 20 % (Jägersten)	Mediterranean (Banyuls; Naples); NE Atlantic (Roscoff)	Prouho (1892), Wheeler (1896), Jägersten (1940), Barel and Kramers (1977)

surface. As pointed out by Stummer-Traunfels (1926), 3 types of parasitic myzostomids may be distinguished depending on whether they are gallicole, cysticole or endoparasitic. Endoparasitic species feed on the host's tissues, while cysticole and gallicole species are suspension feeders which divert the water current produced by the host's ambulacra (galls and cysts are most often located near the ambulacral grooves and always have 2 apertures allowing passage of directional water-currents) (Prenant, 1959). With rather few exceptions (e.g., *? Cystomyzostomum ijimai* and *Mesomyzostoma katoi;* respectively Fishelson, 1974; Okada, 1933), parasitic myzostomids mostly infest bathyal echinoderms (in 200 to 1200 m water depth; Stummer-Traunfels, 1926). This fact explains why these parasitoses have been so anecdotally documented.

Gallicole species dig into the dermal tissue of crinoid arms or pinnules (Fig. 5-22A, B) and build more or less spacious intradermal cavities, always located under skeletal ossicles. The cavities sometimes are very complicated, with internal partitions (e.g., in *Myzostomum tenuispinum;* Graff, 1884). Myzostomid galls rather often harbor a pair of individuals. Gallicole species were termed 'deformative Arten' by Stummer-Traunfels (1926); they induce conspicuous deformation of the host's ossicles which considerably enlarge and adjust their shape to that of the myzostomid's shelter (Graff, 1884; Wheeler, 1896; Stummer-Traunfels, 1926). Cysticole species behave differently in that they build peculiar, stalked or unstalked cysts which are always subepidermal (i.e., located outside the host's skeleton) and protrude into the external medium (Fig. 5-22C). In many cases the cyst wall is reinforced by a pavement of minute skeletal plates (Graff, 1884; Stummer-Traunfels,

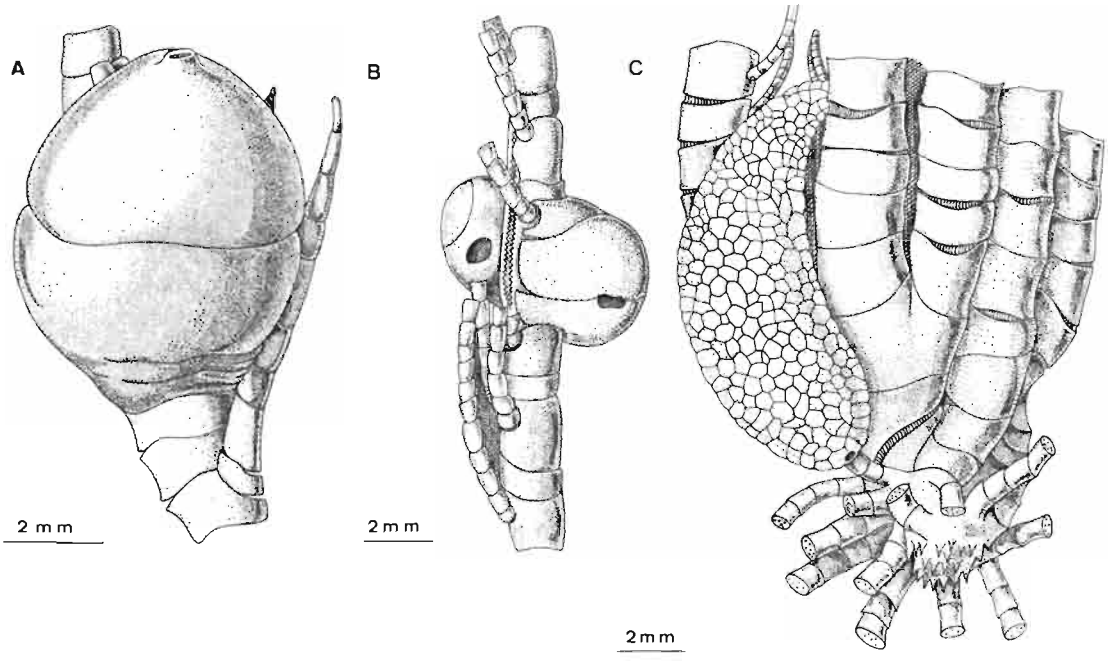


Fig. 5–22: Gallicole and cysticole myzostomids of crinoids. A: *Myzostomum deformator;* gall in the pinnules of *Endoxocrinus alternicirrus*. B: *Myzostomum tenuispinum;* gall in the arms of *Pachylometra inaequalis*. C: *Cystomyzostomum murrayi;* cyst on the arms of *Stiremetra breviradiata*. (Redrawn from Graff, 1984.)

1926). Both gallicole and cysticole myzostomids induce major host reactions affecting the crinoid skeleton either by modifying size and shape of skeletal ossicles or by inducing the formation of additional skeletal plates. Such host reactions invite further investigations.

Among the 8 species of endoparasitic myzosotomids, 4 infest ophiuroid or crinoid gonads (respectively *Protomyzostomum* spp. and *Mesomyzostoma katoi*). They cause at least partial castration of their host. According to Remscheid (1916), *Mesomyzostoma reichenspergi* infests gonads, while according to Prenant (1959) it affects only the arm coelom of its crinoid host and feeds on coelomocytes. *Asteriomyzostomum asteriae* (Fig. 5-23) is said to feed on the host's digestive contents; its occurrence supposedly increases the

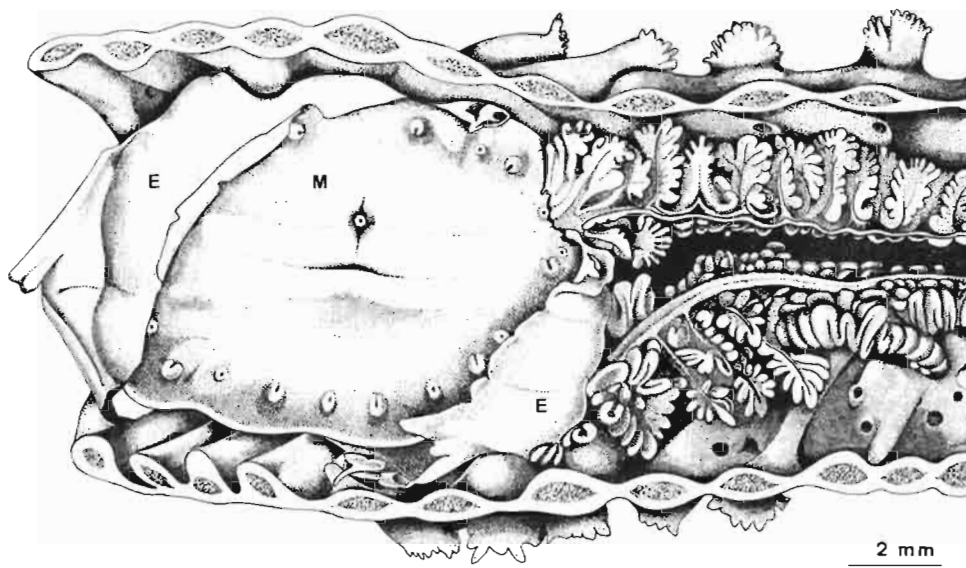


Fig. 5–23: *Asteriomyzostomum asteriae*, a myzostomid parasite of the pyloric caeca of the asteroid *Sclerasterias neglecta*. (E) Enlarged and deformed part of pyloric caeca harboring the parasite; (M) myzostomid. (Redrawn from Stummer-Traunfels, 1903; slightly modified.)

propencity of asteroids to autotomize their arms (Marenzeller 1895a, b). *Pulvinomyzostomum pulvinar* might be more properly classified as 'semi-parasitic'. It inhabits the crinoid's digestive tract (anterior part) living intimately attached to the host's digestive epithelium without causing injury but diverting the flow of food particles entering the crinoid's mouth (Prenant, 1959; West and West, 1976).

Agents: Tardigrada

The tardigrade *Tetrakentron synaptae* lives attached to the body surface of the synaptid holothuroid *Leptosynapta galliennei* (Cuénot, 1892, 1912; Van der Land, 1975; Kristensen, 1980). This symbiosis has been reported only from Brittany (Roscoff and adjoining areas) where it is common. Very high infestation rates occur in various populations, viz. 45, 80 and even 100% (respectively Barel and Kramers, 1970; Kristensen, 1980; Cuénot, 1912). The number of *T. synaptae* per holothuroid is highly variable,

from 2 to 3 up to 270 individuals (respectively Cuénot, 1912; Kristensen, 1980). The latter author noted that infestation occurs only from May to October (eggs and juveniles are seen in June and July). Kristensen presented photographic evidence that *T. synaptae* punctures the epidermal cells of *L. galliennei* and sucks out their contents; such behavior indicates a parasitic nature of the tardigrad-holothuroid association. Another tardigrad *(Actinarchus doryphorus)* occurs on the body surface of the small European clypeasteroid echinoid *Echinocyamus pusillus*. According to Schulz (1935), *A. doryphorus* is a facultative parasite.

Agents: Crustacea Copepoda

There is little information regarding the relations between echinoderms and closely associated copepods. This is paradoxical given the high numbers of copepods recorded as echinoderm symbiotes (see, e.g., Humes, 1986). Table 5-9 lists species presumed to be parasites. Although many ectoparasitic copepods of echinoderms have been reported in the literature, the parasitic nature of these associations has been proven for only 4 species, viz. *Cancerilla tubulata, Scottomyzon gibberum, Asterocheres lilljeborgi,* and *Ophiopsyllus reductus* (respectively Carton, 1968; Röttger, 1969; Röttger and co-authors, 1972; Emson and co-authors, 1985). *A. lilljeborgi* is not particularly adaptated to parasitic life. It is a motile copepod which uses its siphon-shaped mouth to feed on the skin of the asteroid *Henricia sanguinolenta*. *S. gibberum* mainly lives on or near the pedicellarial rosettes of *Asterias rubens* where it seems to feed more deeply in the host tissues (Fig. 5-24). *O. reductus* lives firmly attached to its ophiuroid host and feeds on surface tissues. As for endoparasitic species, Pyefinch (1940) concluded that *Ophioica asymmetrica* found in the bursae of ophiuroids does not feed on the host's tissues, and Changeux (1961) reported — based on casual observations — that the intracoelomic species *Allantogynus delamarei* 'browses' on the holothuroid's mesothelium.

Host reactions against several species of parasitic copepods have been documented. Parasitism by *Scottomyzon gibberum* induces the infested asteroid to produce dermal outgrowths which progressively embed the copepods (Röttger, 1969). Ophiuroid hosts tend to produce a fibrous, sometimes calcified cyst around symbiotes which live in their bursae (Pyefinch, 1940; Heegard, 1951). However, encystment does not occur with all bursal-infesting species (Stephensen, 1935a). Mortensen (1933b) recorded unidentified copepods inducing gall formation in the dorsal wall of both *Ophiomitrella corynephora* and *O. hamata*. Paterson (1958) reported that cysts, presumably produced by the host, form around the intracoelomic copepod *Cucumaricola notabilis*. According to her, small spherical or oval cysts contain juvenile parasites. These cysts are attached to the coelomic wall either near the circumpharyngeal calcareous ring or on the cloaca near the insertion of the respiratory trees; large amorphous cysts containing mature copepods lie detached in the coelomic cavity. Jungersen (1914) reported that the ophiuroid *Asteronyx loveni* produces cysts around intracoelomic copepods. These cysts are attached on the ophiuroid's coelomic wall. Other intracoelomic copepods have been noted to live often 'free in the coelomic cavity' without seemingly inducing any host reaction (e.g. intracoelomic copepods of holothuroids). However, some bursal- or coelom-infesting copepods may produce conspicuous swellings of the body wall of ophiuroids. Actually these swellings follow the outline of the parasite and do not result from a particular host reaction (Mortensen and Stephensen, 1918; Stephensen, 1933; Goudey-Perrière, 1979).

Table 5-9
Parasitic copepods from echinoderms. Hosts: A, asteroid; C, crinoid; E, echinoid; H, holothuroid; O, ophiuroid (Compiled from the sources indicated)

Copepod	Host	Location in host	Remarks	Geographical area	Source
I. Harpacticoida					
Metis holothuriae	*Actinopyga agassizi* (H)	Coelomic cavity	–	Tropical Atlantic (Bahamas)	Edwards (1891), Humes (1980a)
Tisbe furcata	*Ocnus planci* (H)	Coelomic cavity	Almost 100 % of holothuroids investigated were infested (Monticelli)	Mediterranean Sea (Naples)	Monticelli (1892), Humes (1980a)
Tisbe holothuriae	*Holothuria stellati* (H)	Digestive tract (anterior part)	–	Mediterranean Sea (Banyuls)	Humes (1957)
II. Cyclopoida (mostly Siphonostomata)					
Allantogynus delamarei	*Holothuria tubulosa*, *Holothuria poli*, *Holothuria stellati* (H)	Anterior part of coelomic cavity fixed on mesothelium (occur also in wall of pharynx, gonads, tentacular ampullae, or Polian vesicles)	*H. tubulosa* and *H. stellati*: about 2 copepods holothuroid^{-1} (82 infested/117 investigated); *H. poli* is only occasional host	Mediterranean Sea (Banyuls, Villefranche)	Changeux (1958, 1961), Stock and Kleeton (1963), Humes (1980a)
Arthrochordeumium appendidulosum	*Astrocharis gracilis* (O)	Galls at arm base	At least 2 copepods (♂, ♀) found	NW Pacific (Philippines: Mindanao)	Mortensen and Stephensen (1918), Boxshall (1988)
Arthrochordeumium asteromorphae	*Asteromorpha koehleri* (O)	Galls at arm base	Only 1 copepod found	NE Indian Ocean (Amboina)	Stephensen (1933), Boxshall (1988)
Asterocheres lilljeborgi	*Henricia sanguinolenta* (A)	Free on outer body surface	Up to 24 copepods asteroid^{-1}	NE Atlantic (Swedish coast: Gullmarfjord)	Röttger and co-authors (1972), Brun (1976)

Table 5-9 (continued)

Copepod	Host	Location in host	Remarks	Geographical area	Source
II. Cyclopoida (mostly Siphonostomata) (continued)					
Botulosoma endoarrhenum	*Echinaster purpureus* (A)	Respiratory papulae (♀ copepod); coelomic cavity (♂ copepod)	Several dozen copepods asteroid^{-1}	SW Indian Ocean (Madagascar: Tuléar)	Carton (1974)
Calvocheres engeli	*Hygrosoma hoplacantha* (E)	Galls on spine at adoral side	Two infested echinoids with 6 and 3 copepods, respectively	NE Indian Ocean (off Celebes, Timor Sea)	Stock (1968a)
Calvocheres globosus	*Calveriosoma gracile, Sperosoma quincunciale* (E)	Galls on secondary spines or on spines of adoral side	Not more than 1 ♀ copepod gall^{-1} (2 and 5 galls observed on 2 infested hosts)	N Indo-Pacific (off Philippines; in Halmaheira Sea; off SE Japan)	Hansen (1902), Stephensen (1935b), Stock (1968a)
Calvocheres oblongus	*Hygrosoma petersi* (E)	Galls on spines	–	N Pacific (SE of Japan)	Stephensen (1935b)
Calypsarion carinatum	*Stichopus monotuberculatus* (H)	Internal	1 ♂ copepod ejected from host upon collection	NW Indian Ocean (Ethiopia: Dahlak Archipelago)	Stock (1968b), Humes (1980a)
Calypsarion leprum	*Actinopyga mauritania, Actinopyga miliaris, Actinopyga lecanora* (H)	Internal	*A. miliaris* is most common host (76 copepods – 43 ♂ and 33 ♀ – collected from 726 hosts)	SW Indian Ocean (Madagascar: Nossy-Bé)	Humes and Ho (1969), Humes (1980a)
Calypsarion sentosum	*Bohadschia marmorata* (H)	Internal	8 copepods (5 ♂, 3 ♀) collected from 8 hosts	SW Indian Ocean (Madagascar: Nossy-Bé)	Humes and Ho (1969), Humes (1980a)
Calypsina changeuxi	*Holothuria tubulosa, Holothuria stellati* (H)	Esophagus and gonoduct	–	Mediterranean (Banyuls)	Changeux (1961), Stock and Kleeton (1963), Humes (1980a)
Cancerilla tubulata	*Amphipholis squamata* (O)	Attached to outer body surface	Infestation rate ca 10 % (Giard)	NE Atlantic (Brittany; North Sea)	Giard (1887), Carton (1968)
Chauliolobion bulbosum	*Actinopyga echinites, Actinopyga obesa* (H)	Internal	*A. echinites*: 11 copepods (4 ♀, 7 ♂) from 72 hosts; *A. obesa*: 21 copepods (4 ♀, 17 ♂) from 1 host	Tropical W Pacific (New Caledonia: Nouméa)	Humes (1975, 1980a)

Table 5-9 (continued)

Copepod	Host	Location in host	Remarks	Geographical area	Source
II. Cyclopoida (mostly Siphonostomata) (continued)					
Chauliolobion foliaceum	*Holothuria atra* (H)	Coelomic cavity	11 copepods (5 ♂, 6 ♀) collected	N Indian Ocean (Gulf of Mannar)	Ummerkutty (1970)
Chordeumium obesum	*Asteronyx loveni* (O)	Cysts within coelomic cavity (attached cysts)	Almost 100 % infestation; no more than 1 copepod gall^{-1}	North Sea (Skagerrak)	Mortensen (1912), Jungersen (1912, 1914), Boxshall (1988)
Clavisodalis dilatatus	*Diadema setosum* (E)	Esophagus	43 copepods collected from 65 echinoids	New Caledonia	Dojiri and Humes (1982)
Clavisodalis salmacidis	*Salmacis belli* (E)	Mouth and esophagus	4 copepods	Moreton Bay (Queensland)	Humes (1980b), Dojiri and Humes (1982)
Clavisodalis sentifer	*Diadema setosum* (E)	Esophagus	4 copepods collected from 17 echinoids	Moreton Bay (Queensland), Ambon	Dojiri and Humes (1982)
Codoba discovery	*Ophiura meridionalis* (O)	Calcified cysts inside bursa	3 infested ophiuroids were found	Antarctic Sea (South Georgia)	Heegard (1951)
Collocheres elegans	*Ophiocomina nigra* (O)	Attached to arm base	Up to 5 copepods ophiuroid^{-1} (infestation increases with host size)	Firth of Clyde (Scotland)	Gorzula (1978)
Collocherides astroboae	*Astroboa nuda*, *Astroboa albatrossi* (O)	Stomach	*A. nuda*: 2 infested individuals with respectively 29 (23 ♀, 6 ♂) and more than 100 copepods; *A. albatrossi*: 1 infested individual with 22 copepods (16 ♀, 6 ♂)	Red Sea (Eilat; Dahlate Archipelago); SE Indian Ocean (Java Sea)	Stock (1971)
Cucumariocola notabilis	*Cucumaria frauenfeldi* (H)	Coelomic cavity (free cysts lying in the cavity)	Of 337 investigated holothuroids, 63 were infested by 1 to 4 cysts	SW Indian Ocean (Cape Town Province)	Paterson (1958)
Dichelina phormosomae	*Phormosoma bursarium*, *Phormosoma verticillatum*, *Paraphormosoma alternans* (E)	Digestive tract	2 copepods (1 ♂, 1 ♀) taken from 1 individual of each host species	NW Pacific (Philippines: off Mindanao)	Stephensen (1933, 1935b)

Table 5-9 (continued)

Copepod	Host	Location in host	Remarks	Geographical area	Source
II. Cyclopoida (mostly Siphonostomata) (continued)					
Dichelina seticauda	*Hygrosoma hoplacantha* (E)	Internal	5 ♀ copepods taken from 1 echinoid	NE Indian Ocean (Celebes: off Menado)	Stock (1968c)
Diogenella deichmannae	*Holothuria arenicola* (H)	Internal	43 copepods (4 ♂, 39 ♀) collected from 88 individuals	Tropical Atlantic (Barbados)	Humes and Ho (1970), Humes (1980a)
Diogenella seticauda	*Holothuria surinamensis, Holothuria impatiens, Holothuria arenicola* (H)	Internal	23 copepods from 2 holothuroids (Stock 1968); 5 copepods (1 ♂, 4 ♀) from 47 holothuroids	Tropical Atlantic (Puerto Rico)	Stock (1968b), Humes and Ho (1970), Humes (1980a)
Diogenella spinicauda	*Holothuria mexicana, Actinopyga agassizi* (H)	Internal (ejected from *H. mexicana*; Stock)	2 copepods (1 ♂, 1 ♀) from *H. mexicana* (Stock); 46 copepods (22 ♂, 24 ♀) from 119 *H. mexicana*; 50 copepods (32 ♂, 18 ♀) from 62 *A. agassizi* (Humes and Ho)	Tropical Atlantic (Curaçao, Bahamas, Jamaica, Puerto Rico)	Stock (1968b), Humes and Ho (1970), Humes (1980a)
Diogenidium deforme	*Holothuria glaberrima, Holothuria arenicola, Holothuria mexicana* (H)	Internal (ejected from *H. glaberrima*; Stock)	2 copepods (1 ♂, 1 ♀) from 1 *H. glaberrima* (Stock); 7 copepods (1 ♂, 6 ♀) from 87 *H. arenicola* and 5 copepods (2 ♂, 3 ♀) from 24 *H. mexicana*	Tropical Atlantic (Puerto Rico, Barbados, Bahamas)	Stock (1968b), Humes and Ho (1971), Humes (1980a)
Diogenidium nasutum	*Actinopyga agassizi, Holothuria mexicana, Holothuria grisea* (H)	Coelomic cavity	9 copepods (5 ♂, 4 ♀) from 3 *H. mexicana* (Stock); 9 copepods (4 ♂, 5 ♀) from 69 *A. agassizi*; 10 copepods (4 ♂, 6 ♀) from 29 *H. mexicana*, and 6 copepods (4 ♂, 2 ♀) from 11 *H. grisea* (Humes and Ho)	Tropical Atlantic (Bahamas, Curaçao, Jamaica, Puerto Rico)	Edwards (1891), Stock (1968b), Humes and Ho (1971), Humes (1980a)

Table 5-9 (continued)

Copepod	Host	Location in host	Remarks	Geographical area	Source
II. Cyclopoida (mostly Siphonostomata) (continued)					
Diogenidium spinulosum	*Isostichopus badionotus* (H)	Internal (ejected from host; Stock)	1 ♂ copepod from 1 holothuroid (Stock); 4 copepods (2 ♂, 2 ♀) from 17 holothuroids (Humes)	Tropical Atlantic (Puerto Rico, Jamaica)	Stock (1968b), Humes and Ho (1971), Humes (1980a)
Diogenidium tectum	*Actinopyga agassizi* (H)	Internal	3 ♂ copepods from 64 holothuroids	Tropical Atlantic (Jamaica, Bahamas)	Humes and Ho (1971), Humes (1980a)
Echinosocius gulicolus	*Diadema setosum* (E)	Esophagus	85 copepods collected from 65 echinoids	New Caledonia	Dojiri and Humes (1982)
Enterognathus comatulae	*Antedon mediterranea, Antedon bifida* (C)	Digestive tract, rarely in coelomic cavity	11 copepods (2 ♂, 9 ♀) from 12 *A. mediterranea* (Changeux & Delamare-Deboutteville)	Mediterranean Sea (Banyuls, Naples); NE Atlantic (Dublin, Plymouth)	Giesbrecht (1900), Grainger (1950), Changeux and Delamare-Deboutteville (1956), Stock (1959)
Enterognathus lateripes	*Decametra chadwicki, Heterometra savignyi, Oligometra serripinna* (C)	Digestive tract	Not more than 4 copepods found	Indian Ocean (Red Sea)	Stock (1966)
Lecanurius intestinalis	*Actinopyga lecanora* (H)	Digestive tract	–	NW Pacific, (Philippines; Bohol Island)	Kossmann (1877, quoted by Stock 1968b)
Lecanurius kossmannianus	*Actinopyga lecanora, Actinopyga mulleri* (H)	Internal	14 copepods (7 ♂, 7 ♀) from 11 *A. lecanora* and 8 copepods (4 ♂, 4 ♀) from 205 *A. mulleri*	SW Indian Ocean (Madagascar: Nossy-Bé)	Humes (1968)
Lecanurius planifrontalis	*Actinopyga echinites, Actinopyga miliaris* (H)	Digestive tract (posterior part)	3 copepods (2 ♂, 1 ♀) from 3 *A. echinites*	Tropical W Pacific (New Caledonia, Great Barrier Reef)	Humes (1980a)
Lernaeosaccus ophiacanthae	*Ophiacantha disjuncta* (O)	Coelomic cavity (presumably)	Only 1 individual found	Antarctic seas (Palmer Archipelago)	Heegard (1951), Boxshall (1988)

Table 5-9 (continued)

Copepod	Host	Location in host	Remarks	Geographical area	Source
II. Cyclopoida (mostly Siphonostomata) (continued)					
Lichothuria mandibularis	*Holothuria atra, Holothuria scabra, Holothuria nobilis, Holothuria fuscopunctata* (H)	Internal (ejected from host; Stock)	4 copepods (2 ♂, 2 ♀) from 12 *H. atra* (Stock); 188 copepods (54 ♂, 134 ♀) from 470 *H. scabra*; 19 copepods (6 ♂, 13 ♀) from 24 *H. nobilis*; 139 copepods (34 ♂, 105 ♀) from 345 *H. atra*, and 1 ♂ copepod from *H. fuscopunctata* (Humes and Ho)	W Indian Ocean (Red Sea: Eilat; Madagascar Nossy-Bé)	Stock (1968b), Humes and Ho (1969), Humes (1980a)
Ophioicodes asymmetrica	*Ophiacantha imago* (O)	Bursae, forming slight swelling of host disc	Each of the 2 infested ophiuroids investigated had a pair (♂, ♀) of copepods encysted in 1 of their bursae	Antarctic seas	Pyefinch (1940), Boxshall (1988)
Ophioika appendiculata	*Ophiomitrella calvigera, Ophiacantha bidentata* (O)	Bursae, forming swelling of host's tissue	1 to 3 copepods host^{-1} (no more than 1 bursa infested host^{-1})	N Atlantic (S of Greenland, S of Iceland, W of Hebrides)	Stephensen (1935b, 1940)
Ophioika ophiacanthae	*Ophiacantha severa* (O)	Bursae, forming swelling of host disc	Only 1 ♀ copepod found	SE Indian Ocean (Bali Sea)	Stephensen (1933), Boxshall (1988)
Ophioika tenuibrachia	*Ophiacantha vivipara, Ophiacantha disjuncta* (O)	Bursae (presumably)	8 copepods found	Antarctic seas (South Georgia; Adelaide Land)	Heegard (1951)
Ophiopsyllus reductus	*Ophiocomella ophiactoides* (O)	Oral and lateral parts of arm base	40 to 65 % of host populations parasitized depending on period of year. Usually 1 to 3 copepods ophiuroid^{-1}	Jamaica	Emson and co-authors (1985), Emson and Mladenov (1987)

Table 5-9 (continued)

Copepod	Host	Location in host	Remarks	Geographical area	Source
II. Cyclopoida (mostly Siphonostomata) (continued)					
Parachordeumium amphiurae[1]	*Amphipholis squamata* (O)	Bursae	From 10 to 30 % infested ophiuroids according to season; up to 5 copepods per bursa (Emson and co-authors)	NE Atlantic (Roscoff; Devon coast); North Sea (Bergen); Mediterranean (Villefranche/mer; Adriatic Sea); NW Atlantic (Woods Hole region)	Fewkes (1887, 1888), Hérouard (1906), Le Calvez (1938), Bocquet (1952), Zavodnik (1960), Masson (1965); Goudey-Perrière (1979, 1980), Boxshall (1988), Emson and co-authors (1988)
Parachordeumium bocqueti	*Amphipholis squamata* (O)	Bursae	–	NE Atlantic	Goudey-Perrière (1980)
Parachordeumium hendleri	*Amphipholis squamata* (O)	Bursae	–	NW Atlantic (US coast)	Goudey-Perrière (1980)
Parachordeumium humesi	*Amphipholis squamata* (O)	Bursae	–	NW Atlantic (US coast)	Goudey-Perrière (1980)
Pinnodesmotes phormosomae	*Hygrosoma petersi*[2] (E)	Intracoelomic galls	1 or 2 (♂, ♀) copepods gall^{-1}	NE Atlantic (Azores)	Koehler (1898), Bonnier (1898)
Scottomyzon gibberum	*Asterias rubens* (A)	Galls in rosettes of pedicellariae	Most asteroids are infested; 4 to 175 copepods asteroid^{-1}	North Sea (off Helgoland; Kattegat)	Röttger (1969, 1971), see also Barel and Kramers (1977)
Synapticola teres	*Polyplectana kefersteini*, *Synapta maculata* (H)	Coelomic cavity, digestive tract (posterior part)	5 copepods (2 ♂, 3 ♀) free in coelomic cavity (Voigt)	Indian Ocean (Amboina); W Pacific (Queensland)	Voigt (1892), Humes (1979)
III. Monstrilloida					
Thespesiopsyllus paradoxus	*Ophiothrix fragilis*, *Ophiopholis aculeata* (O)	Stomach folds	About 50 % of *O. aculeata* infested	North Sea (Gullmarfjord)	Bresciani and Lützen (1962)

[1] Identified *Amphiurophilus amphiurae* by most authors (see Boxshall, 1988).
[2] Identified *Phormosoma uranus* by Koehler (1898).

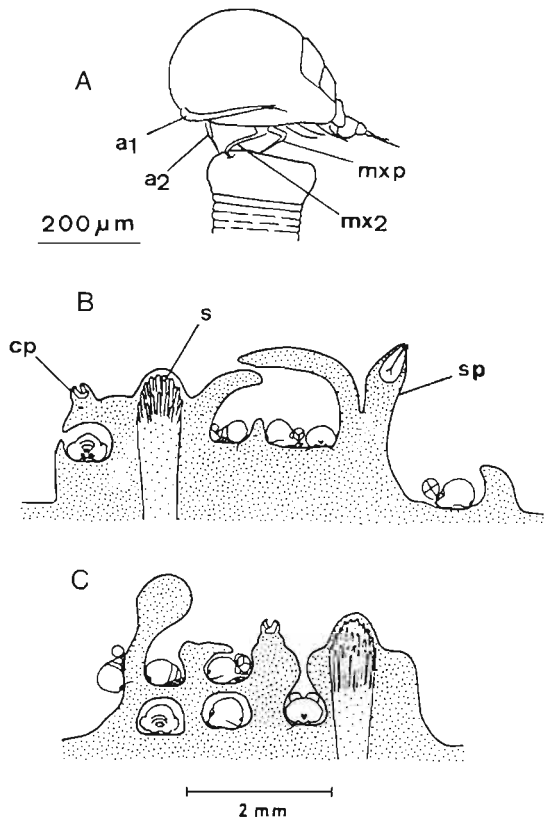


Fig. 5–24: *Scottomyzon gibberum,* an ectoparasitic copepod of the asteroid *Asterias rubens.* A: Female on tube-foot tip. B and C: Copepods embedded in dermal tissue of a pedicellarial rosette. (a1 and a2) Antennae; (cp) crossed pedicellaria; (mxp) maxilliped; (mx2) maxilla; (s) spine; (sp) straight pedicellaria. (After Röttger, 1969.)

Copepods have been found to induce gall formation in the spines of echinothurid echinoids by Hansen (1902), Stephensen (1935b) and Stock (1968a). According to Stock, the galls consist of loose calcareous material and always occur in the middle part of the spines. Galls contain a pigmented ampula in which lives a single copepod. Another gallicole copepod, *Pionodesmotes phormosomae,* occurs inside the echinoid *Hygrosoma petersi* (Bonnier, 1898; Koehler, 1898; see also Mortensen, 1935) (Fig. 5-25). *P. phormosomae* lives in conspicuous inner galls located in the oral hemisphere of the host's coelomic cavity. The spherical, calcified galls correspond morphologically to intracoelomic outgrowths of the echinoid body wall. Each gall opens on the host's outer body surface by a slit measuring 1 to 2 mm in length. The slit is protected by the spines of the echinoid. At least 1 large female copepod was found in each gall. According to Bonnier (1898), the copepod does not prey on host tissues and obtains its food mostly from the outside through the slit in the gall. Mortensen (1935) reported that empty galls progressively disappear: the slit enlarges, then the gall wall rightens, and finally new outer appendages develop.

The way in which intracoelomic copepods infest their host has been considered only for holothuroids and asteroids. According to Paterson (1958) and Changeux (1961),

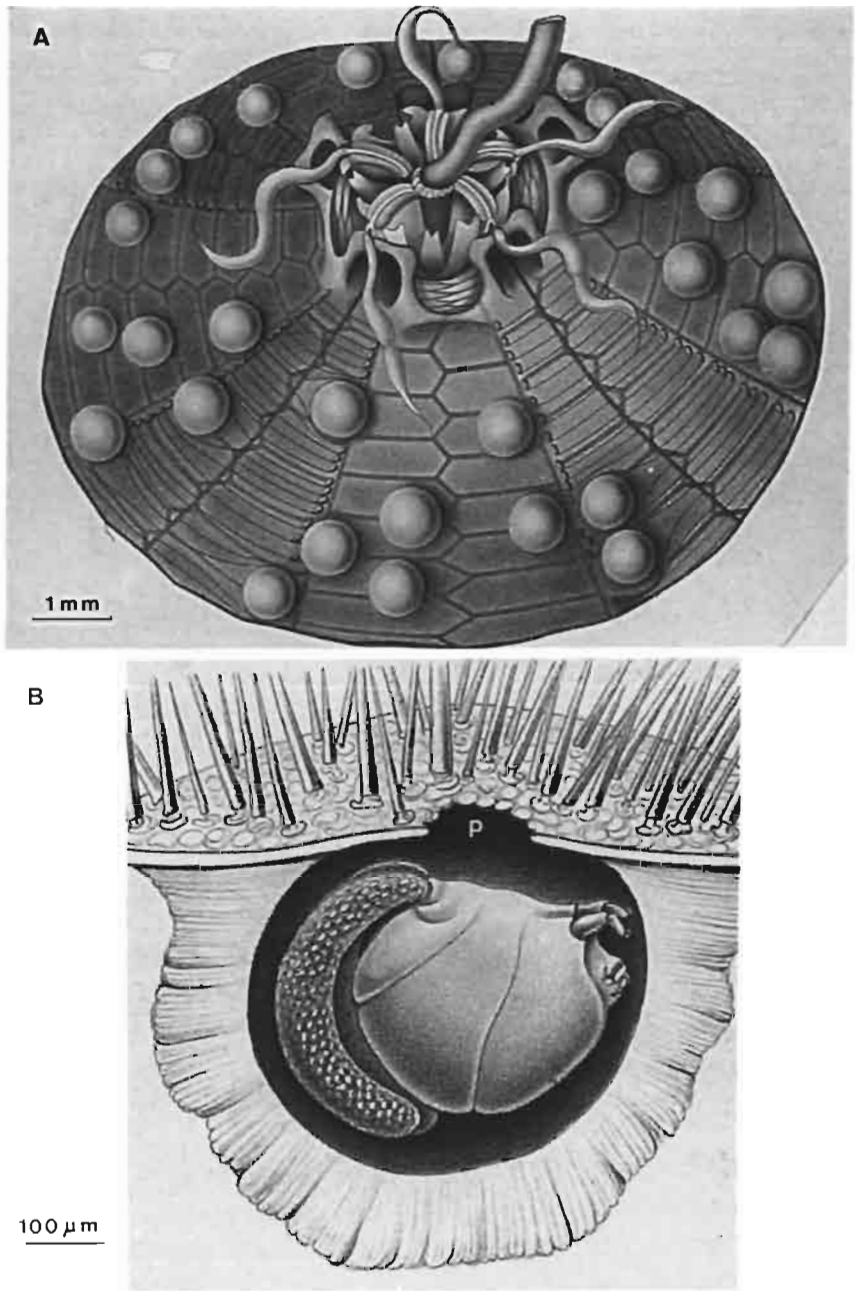


Fig. 5–25: *Pionodesmotes phormosomae*, a gallicole copepode parasite of the echinoid *Hygrosoma petersi*. A: Inner view of the oral part of a dissected echinoid showing the location of calcified galls. B: Section through gall containing an ovigerous female; (p) outer pore of the gall. (A after Koehler, 1898; B after Bonnier, 1898.)

holothuroids are infested by larval copepods which cross the digestive wall either at the level of the esophagus or at the level of the cloaca and respiratory trees. In asteroids, Carton (1974) showed that male *Botulosoma endoarrhenum* penetrate the coelom of *Echinaster purpureus* through the respiratory papulae. Female *B. endoarrhenum* actually settle and grow in papulae, living in a kind of integumental cavity.

The effects of parasitic copepods on the biology of echinoderms have been considered practically only for *Parachordeumium amphiurae* which parasitizes the genital bursae of the brooding ophiuroid *Amphipholis squamata* (e.g., Goudey-Perrière, 1979, 1980; Emson and co-authors, 1988). Goudey-Perrière reported that the occurrence of the parasite does not affect the host's gonads but decreases the host's fecundity by inhibiting the development of embryos incubated in the ophiuroid bursae. According to Emson and co-authors (1988), *A. amphiurae* causes the ophiuroid from a reduction of its brooding capacity to a complete loss of its reproductive capacity, depending on the number of copepods it harbours. They reported also that parasitization reduces the regeneration capacity of the host, and lowers its tolerance to environmental stress. Gonad destruction may occur in an unidentified copepod parasitizing the gonad of the South African ophiuroid *Ophiomitrella corynephora* (see Mortensen 1933b). According to Emson and co-authors (1985) occurrence of parasitic copepods on the ophiuroid *Ophiocomella ophiactoides* could decrease the tendency of the host to undergo fission (cross-disc division).

Agents: Crustacea Cirripedia

Thoracid cirripeds (i.e., barnacles) sometimes attach to the outer body surface of some species of regular echinoids (*Lytechinus anamesus, Tetrapygus niger, Stomopneustes variolaris, Strongylocentrotus* spp.) and clypeastroids *(Dendraster excentricus, Rotula orbiculus)*. Either of 2 situations may occur: (1) the barnacle attaches only loosely to the host's body wall overlaping an intact epidermis (Moore and McPherson, 1963); (2) the barnacle is strongly attached to the echinoid test, the epidermis being destroyed (Giltay, 1934; Dartevelle, 1940; Boolootian, 1958, 1964; Strachan, 1970; Ganapati and Sastry, 1972; Houk and Duffy, 1972; Hurley, 1973; Bay-Schmith and Jana, 1977). Barnacles *Balanus* spp. have been noticed on echinoids, mainly by casual observations, except for the association between *D. excentricus* and *Balanus concavus pacificus* detailed by Boolootian (1964). There appears to be general agreement that the activity of pedicellariae and spines would avoid settlement of barnacle cyprids on the echinoid body wall (e.g., Campbell and Rainbow, 1977). However, the reviewer believes that the failure of cyprids to settle results basically from the occurrence of an epidermis which prevents larval fixation. Presumably cyprid settlement can occur only when the echinoderm body wall is wounded, or when the epidermis is eroded for some mechanical or pathological reasons (such as the bald-sea-urchin disease; see p. 441). Barnacles of the genera *Pachylasma, Scalpellum,* and *Verruca* have been noticed firmly attached to the stem or cirri of several species of stalked crinoids (Clark, 1921).

Ascothoracida are 'naked barnacles'. They consist of about 70 species of parasites of either anthozoans or echinoderms, except holothuroids (Grygier, 1987a). They have been found either as ectoparasites on crinoids and ophiuroids (genera *Ascothorax, Parascothorax, Waginella*), or as endoparasites in the coelomic cavity of asteroids and spatangoid echinoids (genera, *Bifurgaster, Ctenosculum, Dendrogaster, Endaster, Gongylophy-*

sema, Paramedius, Ulophysema) (Table 5-10). *Waginella metacrinicola* lives attached to the crinoid calyx where it forms a conspicuous depression. It feeds on the 'fluids' of the host by piercing the body wall with its oral appendages (Okada, 1926). Ophiuroid-associated ascothoracids infest the bursae producing marked swellings of part of the host disc (Fig. 5-26). According to Wagin (1946) *Ascothorax ophioctenis* feeds on the cells of the bursal wall and probably also on the coelomic fluid of the host. As for intracoelomic ascothoracids, Brattström (1947) concluded that *Ulophysema oeresundense* directly absorbs nutrients from the echinoid's coelomic fluid, while Wagin (1976) believed that coelomocytes form the bulk of the diet of *Dendrogaster* spp. (Fig. 5-27).

Echinoderms react against intracoelomic ascothoracids in forming an host envelope that surrounds the mantle of the parasite. According to Wagin (1946), this envelope consists of coelomocytes and covers even the mantle opening which consequently is constantly cleaned and smoothed by the ascothoracid anterior appendages; he claimed that this appendage activity actually corresponds to the normal way of obtaining food by intracoelomic *Dendrogaster* species. However, investigations by Bresciani and Jespersen (1985) on *Ulophysema oeresundense,* parasitic in the coelom of *Echinocardium cordatum,* showed that the envelope is made of choanocyte-like cells and that consequently it should originate from host mesothelium. A conspicuous host reaction also occurs in *Parascothorax sinagodoides* which lives attached in the bursal cavity near the bursal slit of *Ophiura quadrispina* (Wagin, 1964, 1976). The ophiuroid first encysts the parasite with its own tissues; then both cyst and parasite grow into the bursal cavity. The cyst is gradually ejected from the cavity through the bursal slit. According to Wagin, *P. sinagodoides* apparently has adapted its life cycle to the reaction of its host, as rejected cysts as a rule include infesting ascothoracid larvae. Investigation by Grygier (1988) suggested, however, that cysts are not rejected but simply break before to release infesting larvae. Some asteroid-associated ascothoracids live in conspicuous inner galls located in the arm coelom of their host (Grygier, 1985b, 1987b, 1988; Stone and Moyse, 1985). Galls of the species *Endaster hamatosculum* are calcified and correspond morphologically to intracoelomic outgrowths of the asteroid body wall; each gall opens on the host's outer body surface by a slit-like hole (Grygier, 1985b).

Ascothoracids may castrate their host. Thus *Ascothorax ophioctenis* causes complete castration of *Ophiocten sericerum,* even though it does not feed on the host's gonads. Wagin (1946) found a single juvenile *A. ophioctenis* to inhibit the activity of the host's germinal epithelium leading to a regression of the whole gonad. Similar castration occurs in spatangoids infested with large-sized *Ulophysema oeresundense.* According to Brattström (1947), castration results from competition for food between gonads and the ascothoracid. Casual observations of host castration have been made also by Yosii (1928b), Fisher (1930) and Korschelt (1933). Heavy infestation of the ophiuroid *Ophionotus victoriae* by *Ascothorax gigas* (up to 7 bursae infested) causes occlusion of the ophiuroid mouth opening and reduces the development of the gonads (Grygier and Fratt, 1984). The gonads do not completely regress, however. The stomach volume is reduced, but there is no demonstrable reduction in the volume of stomach contents.

According to Brattström (1947) infesting larvae of *Ulophysema oeresundense* usually penetrate their host through the genital apertures and also through the ambulacral pores. Infesting larvae thus reach either the gonadal lumen or the axial sinus. They must cross the wall of the gonad or of the axial sinus in order to reach the general body cavity where they

Table 5-10

Parasitic ascothoracid cirripeds from echinoderms. Ascothoracid species names according to Wagin (1976) and Grygier (1981 to 1983). Hosts: A, asteroids; C, crinoids, E, echinoids; O, ophiuroids. Unidentified species of ascothoracids were reported from asteroids by Fisher (1940; *Diplasterias meridionalis*) and from ophiuroids by Bartsch (1982; *Ophiurolepis inornata*) (Compiled from the sources indicated)

Ascothoracid	Host	Location on/in host	Remarks	Geographical area	Source
Ascothorax bulbosus	*Amphiura belgicae, Amphiura microplax* (O)	Bursae	1 ascothoracid host^{-1}	Southern Atlantic (off South Georgia)	Heegard (1951)
Ascothorax gigas	*Ophinotus victoriae* (O)	Bursae	Up to 27 parasites host^{-1} (Grygier and Fratt)	South Sandwich Islands	Wagin (1968), Grygier (1983c), Grygier and Fratt (1984)
Ascothorax mortenseni	*Amphiura microplax* (O)	Bursae	–	South Georgia	Grygier (1983c)
*Ascothorax ophiocteni*s	*Ophiocten sericeum* (O)	Bursae	Infestation rate: 0.7 to 3.4 % (Wagin)	Barents Sea; Kara Sea; North Atlantic (around Iceland and Faroë Islands)	Djakonov (1914), Stephensen (1935a), Wagin (1946)
Ascothorax pilocaudatus	*Ophiosphalma armigerum* (O)	Bursae	–	NE Atlantic	Grygier (1983c)
Bifurgaster freyellae	*Freyella spinosa* (A)	Intracoelomic galls	–	Central Atlantic	Stone and Moyse (1985)
Bifurgaster kermadeca	*Freyella* sp. (A)	Intracoelomic galls	–	SW Pacific	Stone and Moyse (1985)
Ctenosculum hawaiiense[1]	*Brisinga evermanni* Unidentified brisingids (A)	Coelomic cavity	–	Tropical W Pacific (Hawaii); W Australia	Heath (1910), Warén (1981c), Grygier (1988)
Dendrogaster antarctica	*Acodontaster conspicuus, Odontaster validus* (A)	Coelomic cavity	Infestation level ca 2 %	Antarctic seas	Fisher (1930), Grygier (1981, 1987b)
Dendrogaster arborescens	*Dipsacaster sladeni, Coscinasterias calamaria* (A)	Coelomic cavity	1 to 3 ascothoracids host^{-1} (infestation not uncommon) (Okada)	S Indian Ocean (off Cape Town); Misaki (Japan)	Le Roi (1905, 1907), Okada (1925)

Table 5-10 (continued)

Ascothoracid	Host	Location on/in host	Remarks	Geographical area	Source
Dendrogaster arbusculus	*Hippasteria californica* (A)	Coelomic cavity	1 observation	NE Pacific (off California)	Fisher (1911), Grygier (1982)
Dendrogaster arctica	*Leptasterias groenlandica* (A)	Coelomic cavity	–	N Pacific (Bering Sea)	Fisher (1930), Korschelt (1933), Wagin (1950), Grygier (1986)
Dendrogaster argentinensis	*Anasterias minuta* (A)	Coelomic cavity	5 asteroids infested (326 investigated)	SW Atlantic	Grygier and Salvat (1984)
Dendrogaster astericola	*Henricia sanguinolenta* (A)	Coelomic cavity	3 infested *H. sanguinolenta* (500 investigated)	Barents Sea (White Sea)	Knipowitsch (1891)
Dendrogaster asterinae	*Asterina burtoni* (A)	Coelomic cavity	1 to 10 ascothoracids asteroid^{-1}	Red Sea (Gulf of Aqaba)	Achituv (1971)
Dendrogaster astropectinis	*Astropecten scoparius* (A)	Coelomic cavity	5 infested asteroids (250 investigated)	Misaki (Japan)	Yosii (1928b)
Dendrogaster beringensis	*Eremicaster tenebrarius*, *Hyphalaster inermis* (A)	Coelomic cavity	–	Bering Sea	Wagin (1957), Madsen (1961), Grygier (1985a)
Dendrogaster dichotomus	*Crossaster papposus* (A)	Coelomic cavity	3 infested asteroids (13 investigated)	NE Pacific	Wagin (1950)
Dendorgaster dogieli	*Pteraster obscurus* (A)	Coelomic cavity	–	Bering Sea	Wagin (1950)
Dendrogaster elegans	*Leptasterias polaris* (A)	Coelomic cavity	–	Bering Sea: Gulf of St Lawrence	Wagin (1950), Grygier (1986)
Dendrogaster fisheri	*Pedicellaster magister megalabis* (A)	Coelomic cavity	–	NE Pacific (off California)	Fisher (1928), Grygier (1982)
Dendrogaster hymenasteri	*Hymenaster membranaceus* (A)	Coelomic cavity	Infestation rate: 10.5 %; up to 4 parasites asteroid^{-1}	NE Atlantic	Stone (1987b)
Dendrogaster iwanowi	*Leptasterias fisheri* (A)	Coelomic cavity	–	Bering Sea	Wagin (1950)

Table 5-10 (continued)

Ascothoracid	Host	Location on/in host	Remarks	Geographical area	Source
Dendrogaster leptasteriae	*Leptasterias fisheri* (A)	Coelomic cavity	–	Bering Sea	Wagin (1950)
Dendrogaster ludwigi	*Echinaster luzonicus, Echinaster stereosomus, Certonardoa semiregularis, Nepanthia belcheri* (A)	Coelomic cavity, sometimes in a coelomic outpouching	–	W. Pacific (Philippines, Japan Sea, Tasmania); Australia (Northern Territory)	Le Roi (1905, 1907), Yosii (1928b), Kenny (1959), Grygier (1988)
Dendrogaster murmanensis	*Crossaster papposus, Solaster endeca* (A)	Coelomic cavity	–	Barents Sea; N Pacific (Okhotsk Sea)	Korschelt (1933), Wagin (1950)
Dendrogaster okadai	*Coscinasterias calamaria* (A)	Coelomic cavity	–	Misaki (Japan)	Yosii (1928b)
Dendrogaster orientalis	*Leptasterias polaris* (A)	Coelomic cavity	–	Bering Sea	Wagin (1950)
Dendrogaster pontasteri	*Pontaster tenuispinis* (A)	Coelomic cavity	–	NE Atlantic	Stone (1987b)
Dendrogaster psilasteri	*Psilaster andromeda* (A)	Coelomic cavity	–	NE Atlantic	Stone (1987b)
Dendrogaster punctata	*Poraniopsis inflata* (A)	Coelomic cavity	–	NE Pacific (off California)	Grygier (1982)
Dendrogaster ramosus	*Leptasterias fisheri* (A)	Coelomic cavity	–	Bering Sea	Wagin (1950)
Dendrogaster rimskykorsakowi	*Ctenodiscus crispatus, Hippasterias leiopelta* (A)	Coelomic cavity	–	NW Pacific (Okhotsk Sea)	Wagin (1950)
Dendrogaster sagittaria	*Sidonaster vaneyi* (A)	Coelomic cavity	2 individuals in a single asteroid	Philippine seas	Fisher (1919), Grygier (1985a)
Dendrogaster tasmaniae	*Allostichaster polyplax* (A)	Coelomic cavity	1 ascothoracid asteroid^{-1} (26 infested/116 investigated)	Around Tasmania	Hickman (1959)

Table 5-10 (continued)

Ascothoracid	Host	Location on/in host	Remarks	Geographical area	Source
Dendrogaster usarporum	*Poronia antarctica glabra* (A)	Coelomic cavity	–	Antarctic seas	Grygier (1987b)
Dendrogaster zoroasteri	*Zoroaster fulgens* (A)	Coelomic cavity	Infestation rate: 4 %	NE Atlantic	Stone (1987b)
Dendrogaster sp.	*Novodinia antillensis* (A)	–	–	Central Atlantic (off Bahamas)	Grygier (1988)
Endaster hamatosculum	*Zoroaster carinatus Pholidaster distinctus* (A)	Intracoelomic galls	1 to 2 ascothoracids asteroid^{-1}	Philippines Sea, Indonesia, W Australia	Grygier (1985b, 1988)
Gongylophysema asetosum	*Odontaster validus* (A)	Intracoelomic galls	Up to 9 parasites asteroid^{-1}	Antarctic seas	Grygier (1987b, 1988)
Paramedius californica	*Freyella microplax* (A)	Endoparasite	–	NE Pacific (California)	Stone (1987a)
Parascothorax synagodoides	*Ophiura quadrispina* (O)	In cysts in bursae	1 to 9 ascothoracids ophiuroid^{-1}	Okhotsk Sea	Wagin (1964)
Parascothorax aff. *P. synagodoides*	*Ophiophthalmus normani* (O)	Cysts in bursae	Infestation rate: 5 %	NE Pacific (California)	Grygier (1988)
Ulophysema oeresundense	*Echinocardium cordatum, Echinocardium flavescens, Brissopsis lyrifera* (E)	Coelomic cavity; more rarely gonads, ambulacral ampullae or axial sinus	1 to 9 (mostly 1) ascothoracids echinoid^{-1} (all stations together: 814 infested/42 874 investigated) (Brattström 1947); infestation rate: 20 to 25 % (Bresciani and Jespersen)	North Sea (Scandinavian coast)	Brattström (1936, 1938, 1946, 1947), Bresciani and Jespersen (1985)
Ulophysema pourtalesiae	*Pourtalesia jeffreysi* (E)	Coelomic cavity	1 or 2 ascothoracids echinoid^{-1}	N Atlantic (between Norway, Spitzbergen, Greenland and the Faroës)	Brattström (1937)
Waginella axotremata	*Metacrinus acutus, Metacrinus angulatus, Metacrinus cingulatus* (C)	On cirri	–	Indonesia; China Sea	Grygier (1983b)

Table 5-10 (continued)

Ascothoracid	Host	Location on/in host	Remarks	Geographical area	Source
Waginella metacrinicola	*Metacrinus rotundus* (C)	On stalk	–	Sea of Japan	Okada (1926, 1938), Grygier (1983b)
Waginella sp.	*Crinometra brevipinna* (C)	Attached to basal part of arms	–	Central Atlantic (off Trinidad)	Grygier (1988)

[1] Warén (1981c) showed that *Ctenosculum hawaiiense* (Heath, 1910) is better interpreted as an ascothoracid than as a mollusc; Grygier (1983a) confirmed this opinion from direct examination of *C. hawaiiense*.

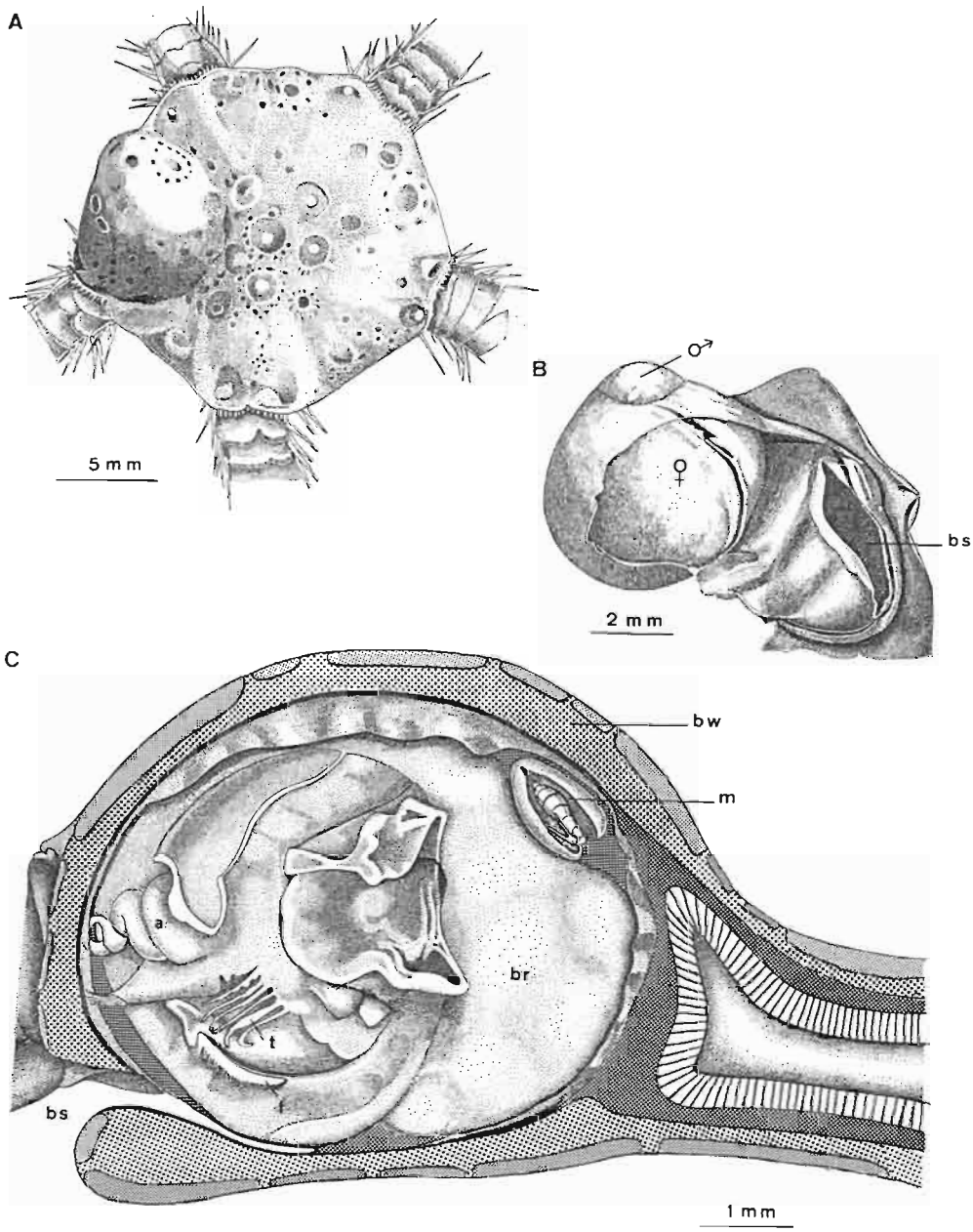


Fig. 5–26: *Ascothorax ophioctenis*, an ascothoracid parasite of the genital bursae of the ophiuroid *Ophiocten sericeum*. A: Aboral view of disc of an infested ophiuroid. B: Dissected genital bursa containing a pair of *A. ophioctenis*. C: Diagramatic drawing of parasites *in situ*. (a) Antennule; (br) brood pouch; (bs) bursal slit; (bw) ophiuroid body-wall; (f) furca; (m) dwarf male; (t) thoracic limbs. (Redrawn from Wagin, 1946; slightly modified.)

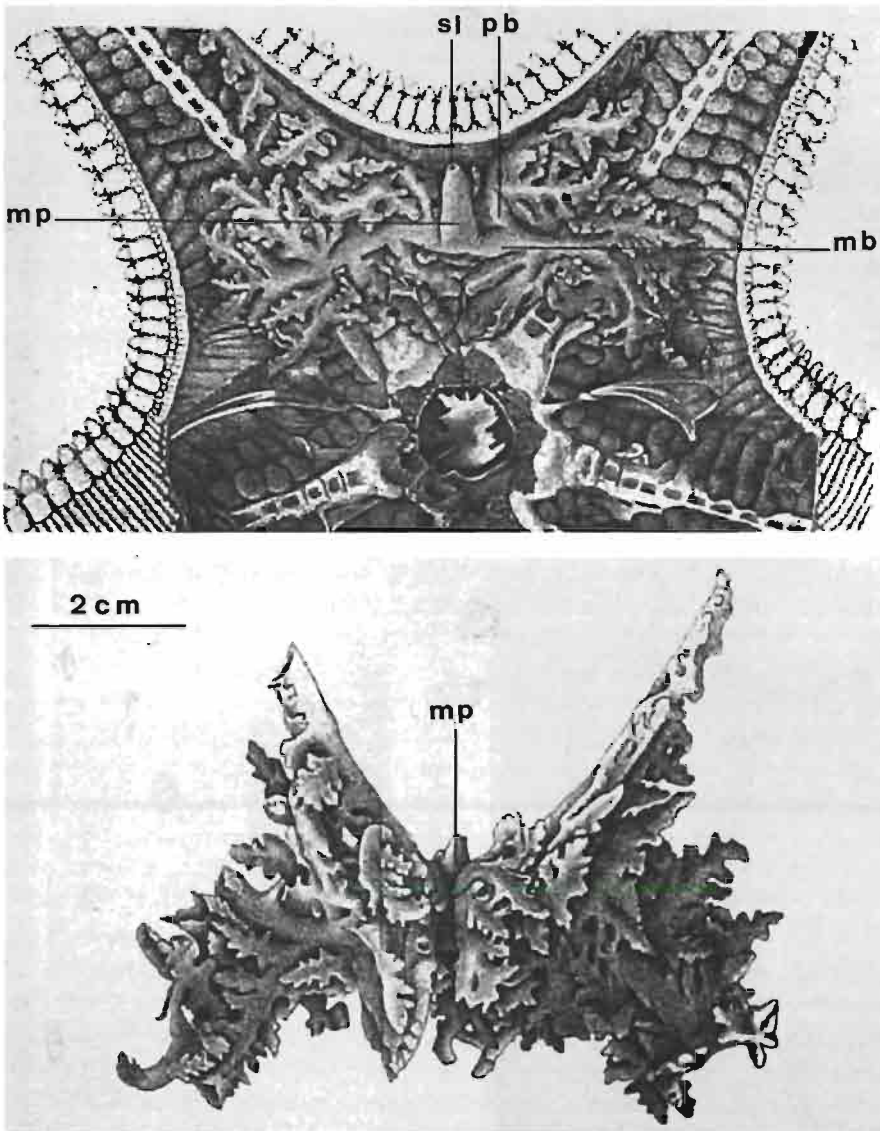


Fig. 5–27: *Dendrogaster arborescens,* an ascothoracid parasite of the coelomic cavity of the asteroid *Dipsacaster sladeni.* (mb) Main branch; (mp) middle piece; (pb) primary branch; (sl) distal slit. (After Le Roi, 1907.)

normally grow and reproduce. When mature, *Ulophysema* spp. most frequently attach to the apical part of the host's body wall in which they bore holes up to 1 mm in diameter. These holes are used by the larvae to escape from the host. Grygier (1981) suggested that the release of larvae from a female *Dendrogaster antarcticus* into the asteroid coelom is produced by rupturing of the mantle of the female. Larvae of *D. tasmaniensis* penetrate the asteroid *Allostichaster polyplax* through respiratory papulae; they infest the coelom

and escape by crossing the asteroid's stomach wall and passing to the outside through its mouth (Hickman, 1959).

Agents: Crustacea Malacostraca

Associations between amphipods and echinoderms were reviewed by Vader (1978). A parasitic relation has been inferred by Shoemaker (1919) for *Laphystiopsis iridometra* which lives embedded in the calyx of the crinoid *Iridometra melpomene*. Typical ectoparasitism occurs with the North Sea species *Epimeria parasitica* which feeds on the outer tissues of the holothuroid *Stichopus tremulus* and the asteroid *Porania pulvillus* (Vader, 1978). According to Ruffo (1957), the amphipods *Jassia ocia* and *Aristias neglectus* occur at Banyuls (France, Mediterranean Sea) in the coelomic cavity of the holothuroid *Stichopus regalis* and the crinoid *Antedon mediterranea*, respectively.

An unexpected association between the tanaidacean *Exspina typica* and 3 species of deep-sea holothuroids has been reported by Thurston and co-authors (1987). *E. typica* should be considered a facultative parasite; it occurs either in the intestine or body cavity of its host.

Crabs associated with and potentially harmful to echinoderms belong to 3 distinct families: Pinnotheridae (genera *Dissodactylus, Ophisthopus, Pinnaxodes, Pinnixa, Pinnotheres*), Parthenopidae (genera *Echinoecus, Zebrida*), and Portunidae (genus *Lissocarcinus*) (Table 5-11). Feeding habits of associated species of *Ophisthopus, Pinnixa, Pinnotheres* and *Lissocarcinus* have not been reported. Presumably these forms do not feed on host tissues, nor cause any other detrimental effects, except to slightly wound the wall of the respiratory trees or of the cloaca (Tao, 1930; Jones and Mahadevan, 1965; VandenSpiegel and Jangoux, 1989). Species inhabiting the posterior part of the echinoid digestive tract *(Echinoecus convictor, E. pentagonus, E. rathbunae* and *Pinnaxodes chilensis)* are generally said to feed on host fecal pellets (e.g., Miyake, 1939; Fenucci, 1967). Trophic relations between *E. pentagonus* and the echinoid *Echinothrix calamaris* were studied carefully by Castro (1971) (see also Suzuki and Takeda, 1974). Males and small immature females inhabit the peristomeal region where they feed on epidermal tissue and tube feet, damage being in equilibrium with the regenerating capacities of the host. Large mature females live in the rectum and ingest material from fecal pellets and aggregates of pigmented coelomocytes that migrate across the rectal wall.

According to Mortensen (1943a) and Suzuki and Takeda (1974) an obvious parasitic relation occurs between the parthenopid crab *Zebrida adamsi* and several echinoid species (Table 5-11): the crab feeds on appendages, skin and peripheral dermal tissue of echinoids, producing conspicuous naked test areas. Similar parasitic behavior also occurs with some species of *Dissodactylus* living on clypeasteroid and spatangoid echinoids (Dexter, 1977; Telford, 1982; Bell and Stancyk, 1983). Telford (1982) reported that, depending on the species studied, the associated *Dissodactylus* take 50 to 100% of their diet from host tissues. According to him, differences in feeding habits can be attributed partly to the structure of host spines, viz. larger test areas are denuded if the spines of the echinoid host are more porous (Fig. 5-28).

Gut-inhabiting species may produce deformations that are sometimes very conspicuous. Verrill (1867) reported that *Pinnotheres chilensis* considerably distends the rectal wall of the echinoid *Coenocentrotus gibbosus* to form a membranous cyst. The cyst attaches aborally to the echinoid body wall and extends near the mouth into the coelomic cavity.

Table 5-11
Parasitic decapods (Reptantia) of echinoderms. Hosts: E, echinoid; H, holothuroid (Compiled from the sources indicated)

Reptantia	Host	Location on/in host	Remarks	Geographical area	Source
Dissodactylus calmani	*Clypeaster rosaceus* (E)	Outer body surface (oral surface)	Infestation level 25 to 50 %	Tropical W Atlantic (Florida, Jamaica)	Telford (1982)
Dissodactylus crinitichelis	*Mellita sexiesperforata* (E)	Outer body surface	Infestation level 30 to 60 %	Tropical W Atlantic (Barbados)	Telford (1978, 1982)
Dissodactylus encopei	*Encope emarginata* (E)	Outer body surface	–	Tropical W Atlantic (Florida to N Brazil)	Rathbun (1918)
Dissodactylus glasselli	*Mellita longifissa* (E)	Outer body surface	–	Tropical E Pacific (Mexican coast)	Rioja (1944)
Dissodactylus lockingtoni	*Mellita longifissa, Encope micropora, Encope grandis, Encope californica* (E)	Outer body surface (proximal portion of posterior interambulacral lunule)	–	Tropical E Pacific (Northern Mexico)	Glassell (1935)
Dissodactylus mellitae	*Mellita quinquiesperforata, Echinarachnius parma* (E)	Outer body surface	13 crabs collected from 50 *M. quinquiesperforata* (Pearse); almost 100 % infestation (Johnson)	NW Atlantic (Massachusetts to Florida)	Rathbun (1918), Pearse (1947), Johnson (1952), Gray and co-authors (1968), Telford (1982)
Dissodactylus nitidus	*Encope stokesi, Encope micropora* (E)	Outer body surface (near lunule or marginal slits)	1 to 2 crabs host^{-1}; infest mostly large echinoids (diameter > 30 mm)	Tropical E Pacific (Panama)	Dexter (1977)
Dissodactylus primitivus	*Meoma ventricosa, Plagiobrissus grandis* (E)	Outer body surface (mostly on oral surface)	Infestation level 79 to 100 %	Tropical W Atlantic (Barbados, Jamaica)	Telford (1978, 1982)
Dissodactylus smithi	*Mellita longifissa* (E)	Outer body surface	–	Tropical E Pacific (Mexican coast)	Rioja (1944)
Dissodactylus xantusi	*Encope stokesi, Encope micropora* (E)	Outer body surface (near lunule or marginal slits)	More often on *E. micropora*	Tropical E Pacific (Panama)	Dexter (1977)

Table 5-11 (continued)

Reptant	Host	Location on/in host	Remarks	Geographical area	Source
Echinoecus convictor	*Echinothrix diadema* (E)	Internal swelling of anal tube	–	Tropical Indo-W Pacific (Gambier Island; Mauritius)	Bouvier and Seurat (1905), Mortensen (1940)
Echinoecus pentagonus	*Pseudocentrotus depressus, Anthocidaris crassispina, Echinothrix calamaris, Echinothrix diadema* (E)	Outer body surface (near peristome)	10 crabs collected on 48 *P. depressus* (Suzuki and Takeda)	Tropical Indo-W Pacific (Andaman, Hawaii, Japan: coast of Uchida)	Castro (1971, 1978), Suzuki and Takeda (1974), Sastry (1977, 1981)
Echinoecus rathbunae	*Echinothrix calamaris, Phyllacanthus dubius* (E)	Internal swelling of the anal tubes	–	NW Pacific (Ogasawara Islands)	Miyake (1939)
Lissocarcinus orbiculatus	*Bohadschia argus* (H)	Mainly respiratory trees, also cloaca	11 holothuroids infested (61 investigated)	NW Pacific (Philippines: Puerto Galera)	Trott and Garth (1970)
Lissocarcinus ornatus	*Holothuria* sp. (H)	Cloaca	–	N Indian Ocean (Andaman Sea)	Chopra (1931)
Ophisthopus transversus	*Parastichopus californicus* (H)	Cloaca	Occur also in other invertebrates (e.g., bivalve molluscs)	NE Pacific (California)	Rathbun (1918)
Pinnaxodes chilensis[1]	*Loxechinus albus, Coenocentrotus gibbosus* (E)	Partly embedded in rectum wall	1 to 2 crabs echinoid^{-1}; infestation generally very high (90 to 100 %) (Verrill, Clark, Rathbun, Schwabe)	SE Pacific (Ecuador to Patagonia)	Verrill (1867), Ortmann (1894), Meissner (1896), Clark (1910), Rathbun (1918), Schwabe (1936), Mortensen (1943b), Fenucci (1967)
Pinnixa barnharti	*Molpadia arenicola* (H)	Cloaca	–	NE Pacific (California)	Rathbun (1918)
Pinnixa timida	*Caudina chilensis* (H)	Cloaca	Infestation level 75 %	NW Pacific (Japan: Mutsu Bay)	Tao (1930)

Table 5-11 (continued)

Reptant	Host	Location on/in host	Remarks	Geographical area	Source
Pinnotheres decanus	*Holothuria scabra* (H)	Cloaca and respiratory trees; rarely in the coelomic cavity	10 holothuroids infested (25 investigated)	N Indian Ocean (Indian coast)	Chopra (1931), Jones and Mahadevan (1965), Adithiya (1969)
Pinnotheres setnai	Unidentified holothuroid	Respiratory trees	–	NE Indian Ocean (Andaman Sea)	Chopra (1931)
Pinnotheres villosissimus	*Actinopyga mauritiana, Actinopyga lecanora* (H)	Cloaca, also posterior intestine	Infestation rate 58% (Van den Spiegel and Jangoux)	NE Indian Ocean (Andaman Sea; west coast of Sumatra); NW Pacific (Papua New Guinea)	Doflein (1904), Chopra (1931), Van den Spiegel and Jangoux (1989)
Zebrida adamsi	*Diadema setosum, Tripneustes gratilla, Toxopneustes pileolus, Toxopneustes elegans, Asthenosoma ijimai, Salmacis sphaeroides* (E)	Outer body surface (near ambitus)	As a rule 1 crab echinoid^{-1} (11 crabs collected) (Suzuki and Takeda)	NW Pacific (Japan: Suruga Bay; coast of Thailand)	Rathbun (1910), Mortensen (1943a), Suzuki and Takeda (1974), Daniel and Krishnan (1978)

[1] Often identified *Fabia chilensis*.

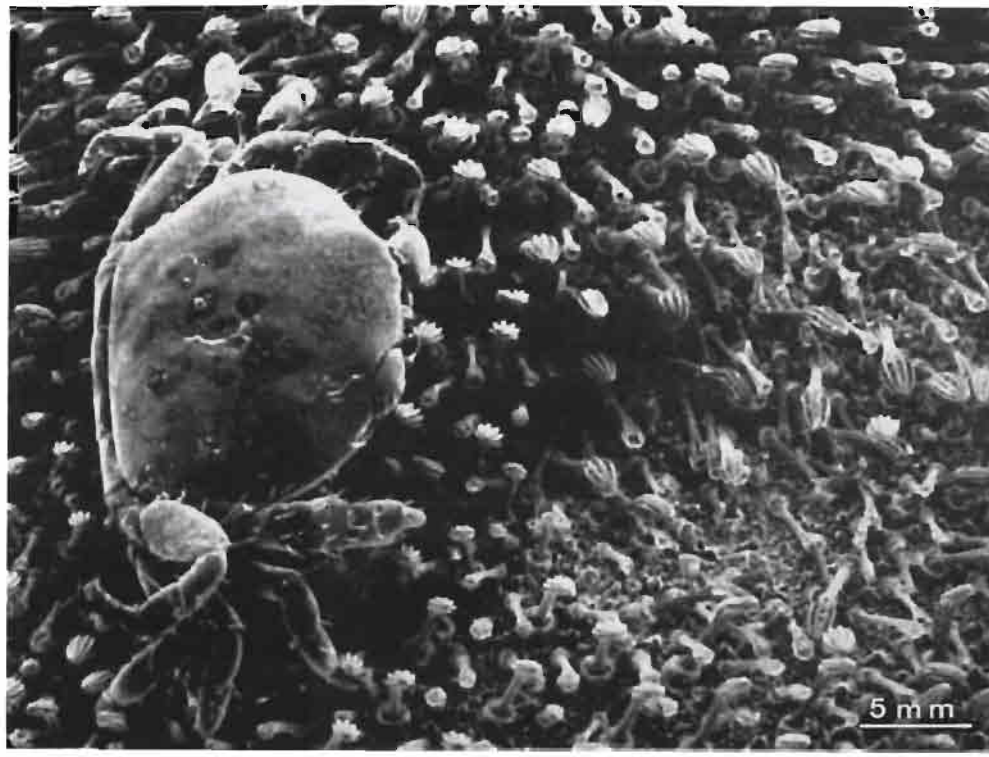


Fig. 5–28: *Dissodactylus crinitichelis,* an ectoparasitic crab of the clypeastroid echinoid *Mellita sexiesperforata.* The crab ist seen beside the devastated area where it had been feeding. (After Telford, 1982.)

The anal area of the host is depressed and distorded, and the echinoid test is usually swollen on the side over the cyst (Fenucci, 1967). *P. chilensis* parasitizes both *C. gibbosus* and *Loxechinus albus.* According to Mortensen (1943b) the crab does not induce major test deformation when infesting *L. albus.* Similar but less conspicuous deformations are caused also by *Echinoecus convictor, E. pentagonus* and *E. rathbunae* (respectively Bouvier and Seurat, 1905; Castro, 1971; Miyake 1939), all of them producing swellings of anal tube and periproct of their host.

According to Suzuki and Takeda (1974) and Dexter (1977), infestations by ectoparasitic crabs often exert major effects and may kill echinoids. The crabs select a particular site on the host's body surface and clear away the spines of the region (see also Telford, 1982). This produces an obvious decrease in movements and spine activities of infested regular echinoids (Suzuki and Takeda, 1974). Dexter (1977) observed a reduction in spine activity which decreased the ability of clypeasteroids to feed and to move about in aquaria, resulting in a substantial increase in mortality.

Agents: Arachnida; Pycnogonida; Insecta

The only arachnid reported to be an echinoderm parasite is the acarian *Enterohalacarus minutipalpus* found in the gut of the echinoid *Plesiodiadema indicum*

(Viets, 1939). Three specimens have been collected in echinoid material dredged by the Albatross Expedition off North Moluccas. Nothing is known about host-associate relations.

Pycnogonid-echinoderm associations have been noticed by several authors who generally suggested an ectoparasitic relation. Associations with holothuroids were observed by Prell (1910; *Pycnogonum littorale* / *Cucumaria frondosa*) and Ohshima (1927; *Lecythrorhynchus hilgendorfi* / *Holothuria lubrica*). According to Ohshima, a single host may carry up to 30 pycnogonids which supposedly absorb the blood or 'body juice' of the holothuroid. An obvious pycnogonid-ophiuroid association occurs at Aldabra (Indian Ocean) with 3 species of *Ophioderma* and the sea-spider, *Anoplodactylus ophiurophilus* (Sloan, 1979; Sloan and co-authors, 1979; Stock, 1979). Sloan reported various infestation levels – from 3.6 to 83.7 % — depending on the host species. He presented evidence that *A. ophiurophilus* has the potential to feed on the host's tube feet. The occurrence of pycnogonids on echinoids and asteroids has also been reported casually (Stock, 1981).

An unexpected association between the marine trichopter *Philanisus plebeius* and the asteroids *Patiriella exigua* and *Patiriella regularis* was reported by Anderson and co-authors (1976) and Winterbourn and Anderson (1980). Caddisfly eggs were found in the asteroid coelomic cavity. Singly or in small clumps, eggs occur loose within the coelom close to the peristome or enveloped in stomach folds. The authors presumed that eggs are deposited through the respiratory papulae and that newly hatched intracoelomic larvae leave the host either via the same route or through the stomach wall. According to Winterbourn and Anderson, eggs are found in the coelom most of the year, the infestation level being about 10 %.

Agents: Bryozoa

Bryozoans may be found firmly attached to the body surface of comatulid crinoids, mostly to their arms or cirri. They were recorded by Mortensen (1910) on *Poliometra prolixa*, and by Gautier (1959) on *Leptometra phalangium*. According to Gautier, about 25 % of the crinoid population was infested (6 different species of bryozoans were associated with *L. phalangium*). Moyano and Wendt (1981) report that up to 4 different species of Bryozoa have been seen attached to the outer body surface of the Antarctic holothuroid *Psolus charcoti*.

Agents: Pisces

Associations between echinoderms and carapid fishes (pearlfishes) are of particular interest as they concern — at least in a few cases — typical parasitic relations between an invertebrate host and a vertebrate associate. The systematics, general biology, and ecology of pearlfishes are documented satisfactorally, and some species have been studied in detail (e.g., Zankert, 1940; Arnold, 1956; Trott, 1970; Van Meter and Ache, 1974; Smith and co-authors, 1981; Trott, 1981). Pearlfish species which infest holothuroids and asteroids are listed in Table 5-12.

Most authors report that the stomach contents of *Carapus* spp. from echinoderm coelomic cavities do not show remains of host tissue (e.g., Trott, 1970; Smith and co-authors, 1981). *Carapus* spp. are predators. Basically, they use the host's coelom as shelter (Fig. 5-29). They leave it in order to catch prey, feeding primarily on crustaceans. Casual observations of feeding by *Carapus* spp. on the host's gonad have been reported, however

Table 5-12
Fishes endosymbiotic in echinoderms. Hosts: A, asteroid; H, holothuroid (Compiled from the sources indicated)

Fish	Host	Location in host	Remarks	Geographical area	Source
Carapus acus	*Holothuria tubulosa*, *Stichopus regalis* (H)	Mostly coelomic cavity; sometimes respiratory trees or cloaca	29 infested holothuroids (89 investigated) (Arnold)	Mediterranean Sea (mostly western part)	Emery (1880), Zankert (1940), Arnold (1953, 1956), Gustato (1977)
Carapus bermudensis	*Actinopyga agassizi* (usual host); also *Astichopus multifidus*, *Holothuria glaberrima*, *Holothuria lentiginosa*, *Holothuria princeps*, *Isostichopus badionotus*, *Thyone* sp. (H)	Coelomic cavity; sometimes respiratory trees	1 to 10 fishes holothuroid^{-1} (mostly 1); infestation level may reach 50 % (Smith and co-authors)	W Atlantic from Brazil to North to Bermuda	Linton (1907), Parker (1926), Ancona-Lopez (1956), Smith and Tyler (1969), Trott (1970), Dawson (1971), Koster and Caycedo (1979), Smith and co-authors (1981)
Carapus dubius	Unidentified holothuroid	Internal	–	Tropical W Atlantic (Caribbean)	Putnam (1874)
Carapus homei	*Stichopus chloronotus* and *Bohadschia argus* (usual hosts); also in *Actinopyga mauritiana*, *Holothuria atra*, *Thelonota ananas*, *Stichopus tropicalis* (H)	Mostly coelomic cavity; also respiratory trees	Infestation level 16 to 88 % according to period of year (Smith)	Tropical Indo-W Pacific	Bedford (1899), Fisher (1907), Sivickis and Domantay (1928), Mukerji (1932), Smith (1964), Hipeau-Jacquotte (1967), Trott (1970, 1981), Trott and Trott (1972)

Table 5-12 (continued)

Fish	Host	Location in host	Remarks	Geographical area	Source
Carapus mourlani[1]	*Culcita schmideliana* and *Culcita novaeguineae* (usual hosts); also *Acanthaster planci, Choriaster granulosus, Protoreaster lincki, Thromidia seychellensis*[2] (A) *Bohadschia argus* (H)	Coelomic cavity of asteroids; coelomic cavity and respiratory trees of holothuroids	1 to 2 fishes asteroid^{-1}; infestation level may reach about 100 % (Mortensen; Trott 1970). A single *B. argus* contained 15 fishes (Meyer-Rochow 1977)	Tropical Indo-W Pacific	Putnam (1874), Simpson and Brown (1910), Mortensen (1923), Mukerji (1932), Strasburg (1961), Smith (1964), Hipeau-Jacquotte (1967), Trott (1970), Trott and Trott (1972), Cheney (1973), Jangoux (1974), Meyer-Rochow (1977, 1979)
Carapus parvipinnis	*Bohadschia argus, Thelonota ananas* (H)	Coelomic cavity (?)	–	Tropical Indo-W Pacific	Smith (1964), Trott (1981)
Jordanicus gracilis	*Bohadschia argus* (usual host); also *Holothuria atra, Holothuria scabra, Stichopus chloronotus, Thelonota ananas* (H) *Culcita novaeguineae, Acanthaster planci* (A)	Coelomic cavity	Infestation level may reach 30 % (Trott and Trott)	Tropical Indo-W Pacific	Doleschall (1861), Arnold (1956), Strasburg (1961), Smith (1964), Trott (1970), Trott and Trott (1972), Cheney (1973)
Jordanicus sagamianus	*Holothuria monacaria* (H) *Certonardoa semiregularis* (A)	Intestine (?) of holothuroid; coelomic cavity of asteroid	Infestation common in *H. monacaria* (Tanaka)	NW Pacific (Japan: Sagami, Misaki)	Tanaka (1908), Yosii (1928a)

[1] Identified *Carapus homei* by many authors.
[2] Previously identified *Mithrodia fisheri* (Jangoux 1974).

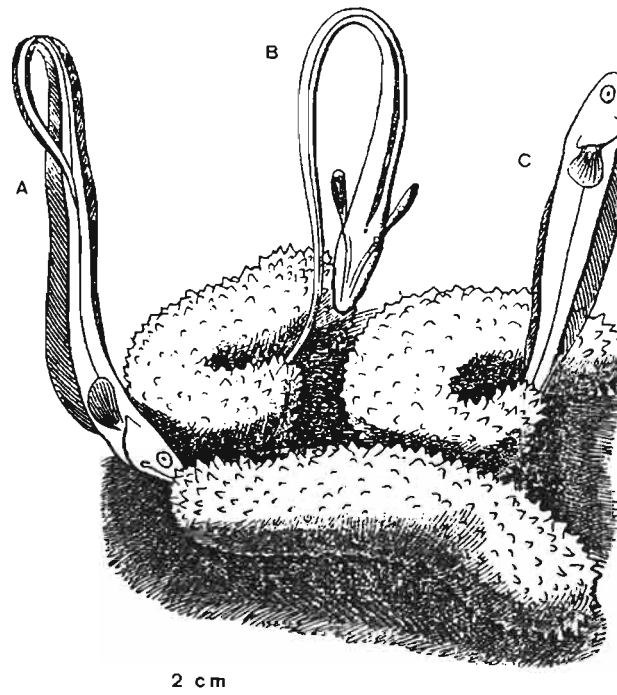


Fig. 5–29: *Carapus acus* penetrating the holothuroid *Holothuria tubulosa*. (A) Recognition; (B) twisting; (C) penetration. (After Emery, 1880.)

Hipeau-Jacquotte (1967) and some authors suggested that juvenile individuals could depend on their host for food (Jangoux, 1974; Meyer-Rochow, 1979). In contrast, *Encheliophis* spp. are considered true parasites which stay permanently in the echinoderm coelom feeding on host viscera (Strasburg, 1961; Smith, 1964).

Effects of pearlfish infestations are said to be practically non-existent except for *Encheliophis* spp. which presumably cause castration by consuming the host's gonads. *Carapus* spp. cause only slight injury by piercing the host's digestive wall when entering or leaving the coelomic cavity. At low infestation levels the effect on the host should be negligible as pearlfishes do not always infest the same host, but shelter in the nearest suitable host found. However, in a higher infested echinoderm population (high infestation levels were, for example, recorded by Mortensen, 1923 and Trott, 1970 for *Carapus mourlani* inhabiting *Culcita novaeguineae*) chances increase that a given host is infested regularly; hence repeated loss of coelomic fluid and successive wound repairs have to be considered.

GENERAL CONSIDERATIONS ON BIOTIC DISEASES

Animal agents associated with echinoderms are summarized in Table 5-13. More than one-third of these agents live on or in holothuroids (Fig. 5-30). Crinoids, regular echinoids and asteroids harbor a rather similar number of harmful associates. As for irregular echinoids, agents mostly infest spatangoids (18 species). Gastropods, turbellarians and

Table 5-13
Number of species of animal agents living with echinoderms. Only identified and documented agents are considered (detrimental effects demonstrated or probable) (Original)

Hosts	Agents																					
	Sporozoa	Protozoa[1]	Porifera	Cnidaria	Mesozoa	Turbellaria	Trematoda	Nematoda	Entoprocta	Polychaeta	Myzostomida	Gastropoda	Bivalvia	Tardigrada	Copepoda	Thoracida	Ascothoracida	Malacostraca	Arthropoda[2]	Bryozoa	Pisces	Total
Crinoidea	1	1	–	4	–	6	2	–	1	–	20	7	–	–	2	10	3	2	–	6	–	65
Holothuroidea	15	–	1	1	–	39	3	–	1	2	–	34	3	1	22	–	–	10	2	4	7	145
Echinoidea (Regularia)	–	4	–	1	–	14	7	3	–	–	–	24	–	–	11	6	–	5	2	–	–	77
Echinoidea (Irregularia)	7	–	–	–	–	6	–	–	–	–	–	–	1	1	–	2	2	10	–	–	–	29
Asteroidea	–	1	–	1	–	8	–	2	–	–	6	19	–	–	3	–	36	1	2	–	3	82
Ophiuroidea	–	–	1	1	1	1	4	1	1	1	3	6	–	–	19	–	7	–	1	–	–	47
Total	23	6	2	8	1	74	16	6	3	3	29	90	4	2	57	18	48	28	7	10	10	

[1] Non-sporozoan protozoans.
[2] Miscellaneous arthropod groups (acarians, pycnogonida, insects).

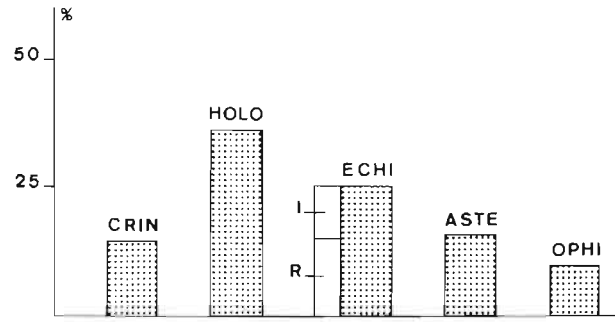


Fig. 5–30: Relative percentage of animal agent species living in each of the echinoderm groups indicated. (CRIN) Crinoidea; (HOLO) Holothuroidea; (ECHI) Echinoidea (I: irregular; R: regular; (ASTE) Asteroidea; (OPHI) Ophiuroidea. (Original.)

copepods comprise the most numerous agents. However, myzostomids, ascothoracids and sporozoans are also well represented. Holothuroids appear to provide a most suitable substrate for parasites of many kinds, except for myzostomids and ascothoracids. These latter 2 groups infest primarily crinoids or asteroids.

Location and Effects of Animal Agents

A classification according to host sites of harmful associates (on or in echinoderms) is presented in Table 5-14 (see also Fig. 5-31). Agents mostly inhabit 3 different sites: body wall, digestive cavity, and coelomic cavity. This corresponds to an external (A), intradigestive (B) or internal (C) location.

External Agents (A-agents)

Among external agents (A1 to A3-agents; Table 5-14), A1-agents mostly occur on holothuroids and echinoids. They comprise strictly ectoparasitic species such as, for example, eulimid gastropods and pinnotherid crabs infesting the external body surface. A1-agents presumably include many more species than considered here. This is because most unattached associates, while often said to be ectoparasitic, have received only taxonomical attention.

A2-agents can firmly attach themselves to the host's external body surface in spite of the presence of an epidermis. Such associates have been reported only casually on representatives of most echinoderm classes. However, crinoids appear to be rather sensitive to A2-agents. Possibly, crinoid sensitivity to these agents results from a weak defensive capacity of their epidermal barrier.

A3-agents are by far the most common external echinoderm associates. Although they all live, in one way or another, within the echinoderms' body wall, they show conspicuous differences in their feeding habits. Several A3-agents feed independently of their host. They may be, for instance, suspension-feeders simply sheltering in galls or cysts (i.e., most of the harmful myzostomids). Independent feeding also occurs in a few gastropods and copepods causing galls in echinoid spines or body wall. This could also be the case in some organisms inhabiting ophiuroid bursae. However, most A3-agents feed at the expense of their host, either by ingesting body-wall tissues or by sucking up internal

Table 5-14
Classification of animal agents affecting echinoderms according to location (Original)

Hosts	Agents							
	External			Intradigestive		Internal		
	A1	A2	A3	B1	B2	C1	C2	C3
Crinoidea	–	24	29	8	1	7	1	1
Holothuroidea	8	7	10	43	11	53	12	2
Echinoidea (Regularia)	6	8	24	29	7	13	–	6
Echinoidea (Irregularia)	3	10	–	6	–	9	–	–
Asteroidea	3	5	21	6	1	46	–	1
Ophiuroidea	1	6	25	2	1	4	–	6
Total	21	60	109	96	21	132	13	16
	A agents: 290			B agents: 117		C agents: 161		

A1-agents live free or simply cling to outer host-body surface.
A2-agents attach to outer host-body surface (*viz.* epidermis-covered body surface).
A3-agents have processes that permanently penetrate or cross the body wall, or live in cysts or galls on or in the body wall (including spines), or live permanently in naturally-occurring ectodermal invaginations (*viz.* genital bursae of ophiuroids).
B1-agents live free in digestive cavity.
B2-agents attach to or bury in the digestive wall.
C1-agents inhabit coelomic cavity or ambulacral system (live free, or attached to coelomic wall, or embedded in mesenteries).
C2-agents live in hemal system.
C3-agents live in gonads.

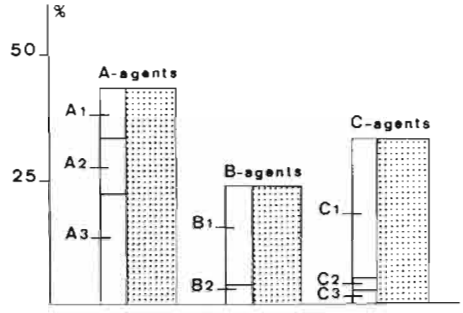


Fig. 5–31: Relative percentage of each category of echinoderm-infesting animal agents; for explanations see Table 5–14. (Original.)

fluids. Body-wall feeders are mainly gallicole gastropods, such as asteroid-associated *Stilifer* spp. A few copepods also feed in this manner (e.g., *Scottomyzon gibberum*). External fluid-feeders are also mainly gastropod molluscs. Several species use their proboscis to penetrate the body wall in order to suck up coelomic, ambulacral, or hemal fluid (e.g., respectively *Echineulima* spp., *Thyca* spp., *Pisolamia brychius;* Table 5-7). It has been suggested that the proboscis of parasitic eulimid gastropods would secrete particular material that brings about a rapid loosening of connective tissue of the echinoderm body wall, thereby facilitating penetration into the host's integument (Smith, 1984). Ectoparasitic gastropods sucking up hemal fluid occur mostly in holothuroids whose

hemal fluid has an high energy content. Possibly, a similar feeding habit has been developed by other ectoparasitic gastropods infesting crinoids (e.g., *Balcis devians* and *Melanella comatulicola*) and asteroids (*Thyca* spp.). The proboscis of these gastropods have been seen to be inserted into the host's perihemal or radial canals which are closely associated with a well-developed hemal lacuna. Coelomic-fluid-feeders appear to be a rather paradoxical adaptation, since the coelomic fluid is not particularly energy-rich. Such a feeding habit implies that the agents feed on coelomocytes or 'browse' on internal tissues, however this is poorly documented for A3-agents. Can ingestion of coelomocytes alone meet the energy requirements of the parasites? If so, such infestation should be rather harmless since echinoderms produce new coelomocytes almost continuously. Finally, many A3-agents of ophiuroids, especially crustaceans, inhabit the bursae of their host. Bursae are easily accessible and well sheltered. Moreover, gonads open into them which means a potential supply of energy-rich material. Yet this does not imply that bursal-inhabiting associates necessarily feed on the host's gonads.

Detrimental effects of external agents are not restricted to their feeding. Some induce conspicuous modifications of the host's skeleton. There are 3 kinds of such modifications: (1) Hypertrophy of skeletal ossicles (caused for example by gallicole myzostomids and spine-inhabiting gastropods or copepods); (2) development of supernumerary skeletal ossicles (e.g., around subcutaneous cysts of some crinoid-infesting myzostomids; around cysts of some copepods living in ophiuroid bursae); (3) inhibition of the development of skeletal ossicles (such as that caused by the asteroid-associated gastropod *Parvioris equestris*). Skeletal modifications have also been reported for diseases caused by microorganisms, i.e. the bald-sea-urchin disease (see p. 441). A3-agents may also cause dermal outgrowths, mostly in gastropods and copepods.

Echinoderm ectoparasites do not castrate their host, except for some forms which live in the bursae of ophiuroids. These, however, do not cause typical castration as none of them is said to feed on gonadal tissues. They are considered to inhibit both gonadal growth and germinal development. The bursal-inhabiting copepod *Parachordeumium amphiurae* which infests the brooding ophiuroid *Amphipholis squamata,* presumably suppresses incubating embryos by diverting part of their food supply without affecting the host's gonads.

Intradigestive Agents (B-agents)

Agents inhabiting the digestive system of echinoderms (B1 and B2-agents; see Table 5-14) mainly infest holothuroids and regular echinoids. While most B1-agents are turbellarian worms, a few gastropods and copepods also live freely in the digestive cavity. Presumed detrimental effects of these associates are poorly documented. However, they exhibit a typical parasitic behavior by feeding on host's digestive epithelium. In contrast, B2-agents, while living within the digestive wall and producing conspicuous deformations, seem to be rather independent from host tissues as far as feeding is concerned. Thus gut-associated crabs of echinoids ingest primarily the food pellets of the host. Similarly, bivalve molluscs inhabiting gut outgrowths are basically suspension-feeders. A detrimental effect on host nutrition might of course occur. This depends greatly on the location of the associates, those living in the holothuroid cloaca or echinoid anal tube being supposedly harmless. B2-agents feeding at the expense of host structures are almost exclusively gastropod molluscs. They generally do not feed on digestive tissues; in most species the proboscis penetrates hemal lacunae, gonads or the body wall.

Internal Agents (C-agents)

Internal agents (C1 to C3-agents; Table 5-14) live either within the coelom, the hemal system or the gonads of the host. Echinoderms harbor a highly diverse intracoelomic fauna (C1-agents): sporozoans, turbellarians, aberrant gastropods, copepods, ascothoracids and fishes are not uncommon in their body cavities. Turbellarians and copepods are the main free-living C1-agents. Their feeding habits require special attention by researchers. Only Changeux (1961) reported intracoelomic copepods 'browsing' on holothuroid mesothelium. The assumption of Jennings and Mettrick (1968) that intracoelomic turbellarians feed essentially on intracoelomic ciliates, although not impossible, appears improbable (so far, intracoelomic turbellarians have been recorded much more often than intracoelomic ciliates). Other free-living C1-agents are fishes and motile stages of sporozoans, viz. trophozoites and gamonts. These latter are mainly found in the coelom of spatangoid echinoids. (Trophozoites and gamonts of sporozoans of holothuroids inhabit primarily hemal lacunae.) Presumably they feed via direct intramembranous absorption of nutrients from the coelomic fluid. Fishes either behave as strict parasites in ingesting the host's gonads or respiratory trees, or they only shelter in the echinoderm coelom and prey upon shrimp and other free living crustaceans they catch outside.

A second category of C1-agents are simply floating or deposited in the host's body cavity, i.e., sporozoan cysts and most ascothoracids. Intrategumentary absorption of coelomic nutrients has usually been suggested as feeding method of intracoelomic ascothoracids. Wagin (1976), however, concluded that these feed mostly on coelomocytes.

The last category of C1-agents includes organisms attached to the coelomic wall and hanging into the body cavity: spatangoid ascothoracids and aberrant gastropods from holothuroids (*Enteroxenos* and allied genera). The body wall of the latter is always surrounded by a host-produced envelope consisting of an inner connective tissue layer and an outer mesothelial layer. As already indicated (see p. 497) hemal lacunae within the host's envelope would facilitate parasite nutrition. In the absence of hemal lacunae — an absence wich would be worth demonstrating — intrategumentary nutrition from the coelomic fluid must be assumed. The only intracoelomic gastropod that is definitely a hemal-fluid-feeder, *Gasterosiphon deimatis*, has been recorded in the body cavity of a deep-sea holothuroid.

C2-agents spend most of their life cycle in the host's hemal lacunae. So far, this group comprises only sporozoans, mostly holothuroid-infesting sporozoans.

Animal agents parasitizing echinoderm gonads (C3-agents) are not very numerous. They include species of protozoans, trematodes, nematodes and myzostomids. Most of them feed directly on gonadal tissues.

While internal agents often castrate their host, the method of castration may differ considerably. Classical castration occurs with agents feeding directly on germinal tissues, e.g., gonad-infesting ciliates of asteroids and gonad-infesting myzostomids of ophiuroids. Less drastic castration may be caused by encysted organisms invading the gonad and supposedly blocking the passage of hormonal substances needed by echinoderms for gametogenesis (Pearse and Timm, 1971). The 'passive castration' (Wagin, 1946) induced by some intracoelomic and intrabursal associates, is of great interest. It should be more properly termed 'competitive castration' as it appears to result from nutritional competition between associate(s) and host's gonads. It causes either slight or total regression of the gonads, depending on the number of agents, and is presumably reversible when the agents

are dislodged from their host. Competitive castration may be inferred for Mesozoa and some intracoelomic gastropods inhabiting holothuroids (e.g., *Entocelax schwanwitschi* and *Paedophorus dicoelobius*) as well as for intracoelomic ascothoracids (*Ulophysema* spp.).

Infestation Routes, Host Reactions and Disease Virulence

Infestation of echinoderms by internal agents takes place mainly through body openings (i.e., mouth, anus, gonopores; also bursal slits of ophiuroids), or through thin areas of the body-wall (e.g., tube feet, respiratory papulae). According to Changeux (1961) intracoelomic copepods of *Holothuria* spp. enter the host's body cavity by penetrating the anterior part of its gut. A similar behavior prevails in most sporozoans inhabiting deposit-feeding echinoderms. Cloaca and respiratory trees of holothuroids also allow the passage of infesting stages of internal agents (i.e., some sporozoans, turbellarians, small crabs and fishes). Larvae of most intracoelomic gastropods of holothuroids (*Enteroxenos* and allied genera) enter the host's body cavity through the digestive wall or, more rarely, through the body wall. These gastropods, however, live never totally free in the coelom; they are always surrounded by an envelope produced by the host (this envelope is continuous with the inner tissues of the host's digestive tract or body wall). Infesting larvae of asteroid-associated ascothoracids may enter their host through respiratory papulae, as do some intracoelomic copepods. Larvae of ascothoracids living in spatangoids enter the host through the genital pores. No report has come to the reviewer's attention that refer to the routes taken by nematodes and digenic trematodes which enter the internal tissues or body cavities of echinoderms.

There are 3 kinds of host reactions counteracting invading organisms: (1) inflammatory-like reactions; (2) connective-tissue reactions; (3) coelomocyte reactions. Inflammatory-like reactions occur mostly in diseases caused by microorganisms and algae or upon cutaneous wound repair (e.g., Menton and Eisen, 1973). Basically this reaction consists of a migration of red spherule cells and phagocytic cells towards the site of infection. These cells are of coelomic origin. Spherule cells produce antibacterial and antifungal substances. Their presence in or close to a wounded or infested area prevents penetration or settlement of unwanted organisms. Red spherule cells of holothuroids and echinoids always occur in every tissue or organ of an individual, although they are present generally in rather low densities. Spherule cells have not been reported in pathologically altered areas of asteroids or ophiuroids.

Connective tissue reactions counteract organisms which tend to stay within the connective tissue layer. A thick fibrous sheet is formed which surrounds and thus isolates the foreign organisms from the host's tissue. Such reactions are not an uncommon defense against sporozoan cysts, trematode metacercariae or invading nematodes. They also counteract some animal agents infesting either the genital bursae of ophiuroids or the coelomic cavity of holothuroids.

Coelomocyte reactions counteract agents entering the host's coelomic cavity. Generally, they are inconspicuous reactions resulting in phagocytizing or completely walling off the foreign organism. A massive and very particular coelomocyte reaction is initiated by motile stages of spatangoid intracoelomic gregarines: each participating coelomocyte becomes pointed, the parasite taking on the appearance of a minute pin cushion.

It was generally not yet possible to evaluate agent virulence, except in the case of

microorganisms or algae. They produce conspicuous pathological alterations which often kill the host. All known mass mortalities of echinoderms due to biotic disease agents are caused by microorganisms. So far, no animal agent has been reported to kill the host, except for a few small ectoparasitic crabs living on some echinoids. Diseases caused by animal agents become obvious only if they result in particular changes in the echinoderm's body shape. As a rule, internal damage is not detectable from the outside, and echinoderms always appear perfectly healthly even when massively infested (e.g., by some sporozoans or mesozoans).

Relative Sensitivity of Echinoderms to Animal Pathogens

The 'pathogenic index' for each echinoderm group has been tentatively estimated (Table 5-15). Although rather approximate, the indexes reveal that Echinozoa and Asterozoa exhibit a very different degree of sensitivity to animal pathogens. It is not

Table 5-15
Pathogenic indexes of echinoderm classes (Original)

Hosts	Estimated no. of recent host species	No. of species of animal agents	Pathogenic index*
Crinozoa	700	65	9.3
Echinozoa	2000	251	12.6
Holothuroidea	1100	145	13.2
Echinoidea	900	106	11.8
Asterozoa	3800	129	3.4
Asteroidea	1800	82	4.6
Ophiuroidea	2000	47	2.4

* (No. of species of animal agents/no. of echinoderm species) × 100

possible to explain this difference on the grounds of morphological, ethological or ecological considerations. The reviewer supposes that it results basically from physiological properties and implies that the asterozoans' defensive mechanisms are more efficient than those of echinozoans.

STRUCTURAL ABNORMALITIES AND NEOPLASIA

Test abnormalities were reported for many regular and irregular echinoids, especially by Koehler (1922, 1924), Chadwick (1924), Jackson (1927), Brattström (1946), Chesher (1969), Moore (1974), Hinegardner (1975) and Allain (1978). These abnormalities involve mainly non-pentamerous individuals or those with a pinched or bifurcated ambulacrum, a depressed or distorted test, a depressed apex, or a depigmented epiderm (for illustrations see, e.g., Koehler, 1922; Moore, 1974). Test abnormalities may have different causes: external injuries, genetic malformations, critical environmental conditions, biotic or nutritional diseases. That test abnormalities may be caused by malnutrition was emphasized by Koehler (1922). While Moore (1974) assumes that they result mainly from

metabolic upset, Hinegardner (1975) attributes the loss of pentamerous symmetry in laboratory-reared echinoids to as yet undetermined genetic factors. Whatever the cause, that these abnormalities are not uncommon indicates a great plasticity of the echinoids. Conspicuous test deformations in echinoids living in polluted areas (Dafni, 1980, 1983) suggest that many of the abnormalities reported may be induced by particular environmental conditions (see also Allain, 1978). Régis and Thomassin (1983) describe structural abnormalities of the spines of the echinoid *Heterocentrotus mammillatus* maintained under aquarium conditions: the spines partly decalcified and the stereomic arrangement of their skeleton became disorganized. They suggest these malformations either to be due to insufficient calcification rates in the spines or to result from exogenous factors.

Body shape abnormalities (e.g., abnormal arm numbers in normally pentamerous species) were reported also for asteroids. According to Hotchkiss (1979) these abnormalities appear to be the consequence of regeneration following predator injury. Watts and co-authors (1983), however, present evidence that ray-number abnormalities in asteroids can be caused by high salinities during early development.

A review of the early literature by Wellings (1969) does not reveal any definite cases of neoplasia among echinoderms. According to Sparks (1972) the only possible neoplasms recorded in echinoderms are the tumor-like epidermal lesions reported by Fontaine (1969) in the ophiuroid *Ophiocoma nigra*. Ophiuroid tumors consist of densely packed cells (mostly melanocytes and spherulocytes with light-brown granules); they lack connective tissue elements. These tumors may grow. Late-stage lesions are roughly divided into cortical and medullary regions, the latter having undifferentiated and fibroblast-like cells. According to Sparks (1972), tumors of *O. nigra* are apparent neoplasms and present evidence of metastases. However, similar kinds of 'tumors', i.e., clots of densely packed cells with brownish pigmented granules, frequently occur within echinoderm tissues. These clots are often located either in the hemal system or within epithelial tissues. Possibly, they correspond to unwanted material, mostly degenerating coelomocytes, in the process of being eliminated. As for the intestinal tumor observed by Smith and co-authors (1973) in the intestine of *Holothuria leucospilota,* the reviewer believes it to be simply an unusual outgrowth of the ventral hemal vessel of the holothuroid gut. It should be noted here that echinoderms naturally synthesize antineoplastic agents (Pettit and co-authors, 1981).

ECOLOGICAL CONSEQUENCES OF ECHINODERM DISEASES

Echinodermata constitute a large and highly distinctive group of marine invertebrates found from the shoreline to the deepest ocean trenches. World-wide they have colonized all marine benthic biotopes where they, characteristically, tend to occur in dense populations. They have developed a wide variety of feeding mechanisms, with representatives in every trophic category (except the parasitic) and display a broad spectrum of metamorphic development (from a typical planctonic larva to almost direct development). Clearly, the ecological radiation of the Echinodermata has been considerable and they can be considered a major macrobenthic animal group. Many littoral echinoderms greatly affect the bioeconomics of both hard- and soft-bottom communities, and some species have tentatively been classified as 'key species', viz species which can functionally dominate a community. Moreover, echinoids and ophiuroids from part of the diet of many fishes and macroinvertebrates, such as crustaceans. Although the ecological consequences of

echinoderm diseases have not been studied nor considered — except for a few cases of spectacular and virulent diseases caused by microorganisms — these diseases should have prominent effects on the biological environment.

The ultimate ecological consequence of diseases caused by microorganisms in echinoderm populations is a reduction or even elimination of the populations concerned. In contrast, most diseases caused by animal agents do not appear to result in major consequences for the echinoderm concerned and are well tolerated. One may consider each echinoderm, especially the holothuroids, as an animal substrate on/in which various other organisms live, either permanently or temporarily. Together, the echinoderm and its associates form a usually well-balanced biological complex. Some animal agents, however, may castrate echinoderms and consequently affect the renewal and long-term stability of the host populations. Quantitative estimations of the effect of castrating agents have never been made. Of course, the effects will depend on infestation rates. Ecological effects of other non-castrating agents are almost impossible to assess at the population level. We can only imagine that, when numerous, agents such as arm-infesting myzostomids, bursal inhabiting copepods or coelom-sheltered fishes would 'weaken' their host population.

Effects of echinoderm diseases on echinoderms' predators mostly concern echinoderms affected by biotic communicable diseases, i.e. those caused by digenic trematodes or by nematodes. Since numerous echinoids and ophiuroids represent a prominent part of the diet of many fish, the role of echinoderms as vectors of fish diseases requires investigations.

Littoral echinoderms are frequently top predators in their community (many paxillosid and forcipulatid asteroids; for review see Menge, 1982), or controlling agents of seagrass or kelp beds (numerous regular echinoids; for review see Lawrence and Sammarco, 1982; Harrold and Pearse, 1987). Experimental or natural removal of these predators or controlling agents produces major environmental changes (e.g., Paine, 1971; Estes and co-authors, 1978; respectively). Catastrophic decline of predatory echinoderms caused by disease was noted only by Dungan and co-authors (1982) in the asteroid *Heliaster kubinji;* these authors, however, did not consider impacts on the biological environment. Disease-related mass mortalities of the echinoid *Strongylocentrotus franciscanus* were followed by rapid expansion of 4 species of brown algae (Pearse and Hines, 1979). Subsequent competition among algal species was severe, and within 1 year only 1 algal species inhabited the area. Another lethal disease that affects the echinoid *Paracentrotus lividus* was studied by Boudouresque and co-authors (1980, 1981) who established that the decrease in echinoid density promoted an explosive growth of epithytes on leaves of the seagrass *Posidonia oceanica.*

Widespread disease-related mass mortalities of *Strongylocentrotus droebachiensis* occurred between 1980 and 1983 along the coast of Nova Scotia, Canada (Miller and Collodey, 1983; Scheibling, 1984). *S. droebachiensis* is the dominant herbivore of the kelp beds of Nova Scotia (e.g., Mann, 1982); mass mortalities of the echinoids was expected to results in colonization by subtidal macroalgae followed by an increase of benthic primary production (Miller and Colodey, 1983). Moore and Miller (1983) reported that in areas where *S. droebachiensis* was absent for 1 year the percentage algal cover was 2 to 14 times higher than in areas with echinoids. In the absence of echinoids, macroalgae expanded to deeper and more sheltered locations: the kelp *Laminaria longicirrus* gained a dominant status and fleshy seaweeds developed in the subtidal (Moore and Miller, 1983; Miller,

1985a; Johnson and Mann, 1986). According to Scheibling (1984) and Scheibling and Stephenson (1984), mass mortalities of *S. droebachiensis* would play a key role in determining the structure and stability of the rocky subtidal ecosystem off Nova Scotia, by controlling the abundance of the dominant herbivore. Thus the potential impact of controlling echinoid populations via the pathogen is considerable (Miller, 1985b; Scheibling, 1988).

The echinoid *Diadema antillarum* — the principal herbivore and the most effective bioeroder of the Carribbean region (e.g., Bak and co-authors, 1984; Liddel and Ohlhorst, 1986) — suffered a widespread and conspicuous mass mortality in 1983–1984. This resulted in a drastic reduction of the population densities to 1 to 6 % of their previous level in Panama (Lessios and co-authors, 1983), to 0.6 % in Curaçao (Bak and co-authors, 1984), to 1 % in Jamaica (Hughes and co-authors, 1985), and to 7 % in Barbados (Hunte and co-authors, 1986). Mass mortality was caused by a virulent biotic disease (see p. 458). Elimination of *D. antillarum* from most Caribbean reefs resulted in a significant increase of fleshy and filamentous algae as well as of some macroalgae (de Ruyter van Steveninck and Bak, 1986; Hughes and co-authors, 1987; de Ruyther van Steveninck and Breeman, 1987). This increase was achieved at the expense of other benthic organisms such as corals, crustose corallines and clionid sponges whose settlement has been considerably reduced (de Ruyter van Steveninck and Bak, 1986; Liddel and Ohlhorst, 1986). Above-cited investigators generally believed the situation will fundamentally affect the Caribbean reef ecosystem unless the populations of *D. antillarum* are restored or those of other herbivores take over the role of the diadematids. Increase of grazing by herbivores fishes after echinoid mass-mortality was reported by Carpenter (1988) who emphasized, moreover, the role of *D. antillarum* in structuring both the producer and consumer components of the ecosystem (see also Lewin, 1988; Morrison, 1988). Levitan (1988) demonstrated that postmortality echinoids show a considerable increase in body size compared to premortality echinoids, and concluded (p. 177) that "although premortality densities may not be reached (if indeed they ever return) for several decades, the ability of *Diadema* to grow indeterminately will cause urchin biomass and *Diadema*'s grazing potential to be restored much sooner".

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6. DISEASES OF UROCHORDATA

C. MONNIOT

Urochordata command restricted economical interest. Only some ascidian species are utilized as sea-food. Generally, the ascidians on sale are obtained via fishing natural populations. Few sea-farms grow ascidians: in Japan, *Halocynthia roretzi,* in Korea, *Styela clava.* Ascidian-farming does not seem to bring particular sanitary problems except the presence of parasitic copepods.

No true diseases causing mortality have ever been reported. However the large size of the branchial and cloacal cavities in tunicates, and the vital importance of the water current through them, constitute a wind-fall for many commensals or parasites.

Among the Urochordata, Ascidiacea are benthic, living settled or lying on the sea bottom, while the Thaliacea (Pyrosomata, Salpacea, Doliolidea) and Perenichordata (Appendicularia) are pelagic. Their respective parasites generally do not belong to the same taxonomic groups.

Organisms associated with Urochordata belong to many taxa, ranging from bacteria to fish. In some cases, and more so in the plankton, the host is only used as a shelter. The intruder may feed on its host and utilize the empty tunic as a home (cephalopods, amphipods, fishes); and sometimes it even modifies it, as the phronimes. Members of other groups dig into the ascidian tunic or lay their eggs in a hollow; this is the case with amphipods and molluscs.

Mainly in the ascidians, many phoronts, commensals or parasites use the natural cavities of the host — pharyngeal or cloacal cavities, channels in the colonies — where they simply shelter, and often benefit from nutritive particles assembled by the ascidian. This is true for nemertines, amphipods, and decapods. Many such species are not host specific but may occur in other invertebrates. Copepods are more intimately associated with ascidians; after the free-living juvenile phase, some males and all females must find a host to achieve their development. Copepods are particularly diversified; next to the fishes, ascidians entertain the largest number of copepod taxa. In some cases their parasitic way of life is very elaborated featuring spectacular adaptations. Copepods are totally missing in pelagic tunicates.

Commensal or parasitic protistans occur in all Prochordata. Our pertinent knowledge is very poor. Gregarines and ciliates are the only protistans recorded in both benthic and pelagic tunicates. Flagellates have been found mostly in appendicularians; rhizopods, in ascidians. Coccidians and haplosporidians are known from ascidians. An enigmatic group, the Nephromyces, are restricted to the renal sac in members of the ascidian family Molgulidae. Symbiotic algae were seen only in ascidians. Only one group of Urochordata has never been cited as hosts: the doliolids, probably because it has not been sufficiently investigated.

We have never found records of neoplasia, neither in the literature, nor in the large number of ascidians examined by us. However, structural abnormalities are rather

common. For the most part they can be related to regeneration following injury. They have been reported particularly by authors who apparently are not aware of the fact that ascidians have pronounced regenerative capacities. Experimental teratology was the subject of several studies, mostly linked to the search of blastomer behaviour.

We know very little about benthic or pelagic urochordates. The few intensive studies available were undertaken in restricted geographical areas, mostly in Europe or North America.

DISEASES CAUSED BY MICROORGANISMS

Agents: Virales

We have not found any literature about viruses identified in tunicates. However, some viruses — such as those responsible for human hepatites — may be filtered by ascidians in polluted areas, retained and concentrated in their branchial sac. Possibly this may be dangerous for people consuming some of these edible ascidians, as well as shell-fish.

Agents: Bacteria

It is not uncommon to find decomposing ascidians along the shore, and it is difficult to know whether the bacteria present have originally killed the ascidian or whether they developed on the cadaver. The presence of bacteria in living ascidians was first noticed by Metchnikoff (1892) in Botrylles in the form of 'phagocytes' in the common test. Harant (1931) confirms this observation and extends it to some Didemnidae and Polycitoridae. While Metchnikoff assumes the presence of bacteria to be natural, Harant considered it as the beginning of a bacterial overgrowth due to tunic lesions in the specimens collected. Aquarium-held ascidians are often attacked by bacteria following injury or decay of epibionts. In these cases, bacterial sheets enter through the siphon and finally kill the ascidian. In nature, dead epibionts are quickly eliminated by scavengers.

According to Gaver and Stephan (1907a), bacteria occur sometimes in high abundancy, in the 'corps péricardique' in *Ciona intestinalis*. Inside the blood sinuses of *Distaplia stelligera* Caullery (1927) found compact spherical masses, 80 μm in diameter, consisting of small rods, resembling bacteria. Saffo (1987) identified intracellular bacteria inside the symbiotic protist *Nephromyces* inhabiting the renal sac of Molgulidae.

In countries where ascidians are sold as sea-food, there are no particular sanitary controls different from those applied to sea-shells for health protection.

The association between *Pyrosoma* species and luminous bacteria is obligate. The bacteria are packed in luminous organs consisting of 2 cellular masses placed on each side of the pharynx under the peripharyngeal band of '*Pyrosoma ambulata*', and on each side of the cloacal siphon of '*P. fixata*'. Julin (1912) and Pierantoni (1921) have investigated the complex mechanism facilitating the transmission of luminous bacteria from generation to generation of the host. Bacteria living in special cells, form endospores (Fig. 6-1a) which are liberated into the blood stream and finally infest follicular cells of the *Pyrosoma* egg. Upon development of the latter, the follicle cells penetrate between the blastomeres (Fig. 6-1b). When the cyatozooid buds, its stolon produces the 4 primordial blastozooids; follicle cells holding the bacteria spores share out amongst the buds and constitute the luminous organs of the tetrazooide. Then the follicle cells disappear, liberating the spores which

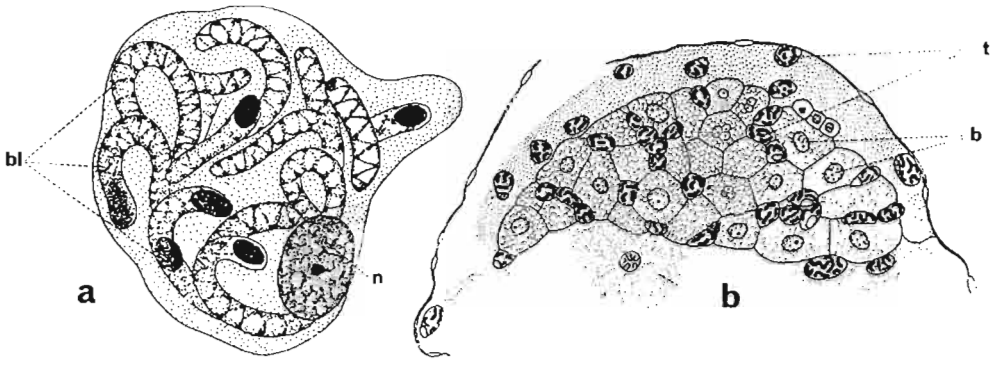

Fig. 6-1: Pyrosoma – bacteria association. a: Pyrosoma luminous cells; (bl) luminous bacteria, (n) cell nucleus; (after Pierantoni, 1921). b: Disk with 128 blastomers of pyrosoma egg with perivitellin cells; (b) blastomers, (t) perivitellin cells with bacterian spores. (After Julin, 1912.)

infest mesenchymatous cells, and, via these cells, the luminous bacteria reach all luminous organs of the colony.

DISEASES CAUSED BY PROTISTANS

Agents: Sarcomastigophora (Flagellata, Trichomonadina and Rhizopoda)

Flagellata

Chatton (1912) recognized as peridinians some enigmatic organisms found on appendicularians or salps: *Gymnodinium pulvisculus* Pouchet, 1885, *Gromia appendiculariae* Brooks and Kellner, 1908, and *Salpicola amylacea* Bargoni, 1894. He created for them the genus *Oodinium*.

Dinoflagellata linked to Urochordata are only known from pelagic tunicates, primarily appendicularians. Cachon and Cachon (1987), and Théodoridès (1989) present pertinent lists. The relations between the dinoflagellates and their hosts vary from phoresis to endoparasitism (Table 6-1). *Filodinium hovassei* Cachon and Cachon, 1968 (Fig. 6-2a), attached by its rhizoids to the cuticule of *Oikopleura* sp., feeds on particles filtered by its host. The Apodinidae redescribed by Cachon and Cachon (1973) live attached to appendicularians. They insert a rhizoid into the host's tissues, which is used at the same time as a fixation and absorption system (Fig. 6-2b). The Oodinidae have a 'sole absorbante' (Fig. 6-2c), and they may be osmotrophic (Cachon and Cachon, 1971). The apodinians show some specialization of their anchory point on the host, and multiple infestation is possible.

An internal peridinian parasitising gonads of *Fritillaria* species was noted for the first time by Neresheimer (1903). This protistan, with a metameric aspect, was redescribed by Cachon and Cachon-Enjumet (1964) and Cachon and Cachon (1966) under the name of *Neresheimeria catenata* (Fig. 6-2d). They clearly demonstrated that this organism belongs to the dinoflagellates. *N. catenata* was often considered causing host castration. Cachon and Cachon-Enjumet (1964) have shown that this happens only when the parasite infests a host having not yet reached sexual maturity. *N. catenata* has been brought close to the genus *Sphaeripara* parasitising other planktonic groups and is now cited under the name *S. catenaria*.

Table 6-1
Sarcomastigophora from Urochordata (Original; compilated from the sources indicated)

Host	Parasite/Symbiote	Source	Synonymy
Larvacea			
Appendicularia sicula	*Apodinium chattoni*	Cachon (1964)	*Parapodinium chattoni*
Appendicularia sicula	*Apodinium rhizophorum*	Cachon (1964)	*Parapodinium catenaria*
Appendicularia sicula	*Haplozoon inerme*	Cachon (1964)	
Fritillaria cophocerca	*Apodinium rhizophorum*	Chatton (1912)	
Fritillaria cophocerca	*Apodinium* sp.	Cachon and Cachon (1973)	
Fritillaria formica	*Sphaeripara catenata*	Fenaux (1963)	*Neresheimeria catenata*
Fritillaria formica	*Apodinium* sp.	Fenaux (1963)	*Parapodinium stylipes*
Fritillaria pellucida	*Apodinium mycetoides*	Chatton (1907)	
Fritillaria pellucida	*Apodinium chattoni*	Cachon and Cachon (1973)	
Fritillaria pellucida	*Apodinium zygorhizum*	Cachon and Cachon (1973)	
Fritillaria pellucida	*Oodinium fritillariae*	Chatton (1912)	
Fritillaria pellucida	*Sphaeripara catenata*	Fenaux (1963)	*Neresheimeria catenata*
Oikopleura albicans	'Sphères jaune orange'	Fenaux (1963)	
Oikopleura albicans	*Syndinium oikopleurae*	Hollande (1974)	
Oikopleura dioica	*Oodinium poucheti*	Fenaux (1963)	
Oikopleura dioica	*Filodinium hovassei*	Cachon and Cachon (1968)	
Oikopleura fusiformis	*Apodinium chattoni*	Fenaux (1963)	
Oikopleura sp.	*Oodinium poucheti*	Pouchet (1885)	*Gymnodinium pulvisculus*
Oikopleura tortugensis	*Oodinium poucheti*	Broocks and Kellner (1908)	*Gromia appendiculariae*
Unknown species	*Sphaeripara paradoxa*	Loeblich (1976)	
Salpida			
Unknown species	*Oodinium poucheti*	Bargoni (1894)	*Salpicola amylacea*
Ascidiacea			
Ciona intestinalis	*Trimastix cionae*	Parona Corrado (1886)	*Elvirea cionae*
Clavelina lepadiformis	*Amoeba clavellinae*	Huxley (1920)	
Phallusia mammillata	*Entamoeba phallusiae*	Mackinnon and Ray (1931)	

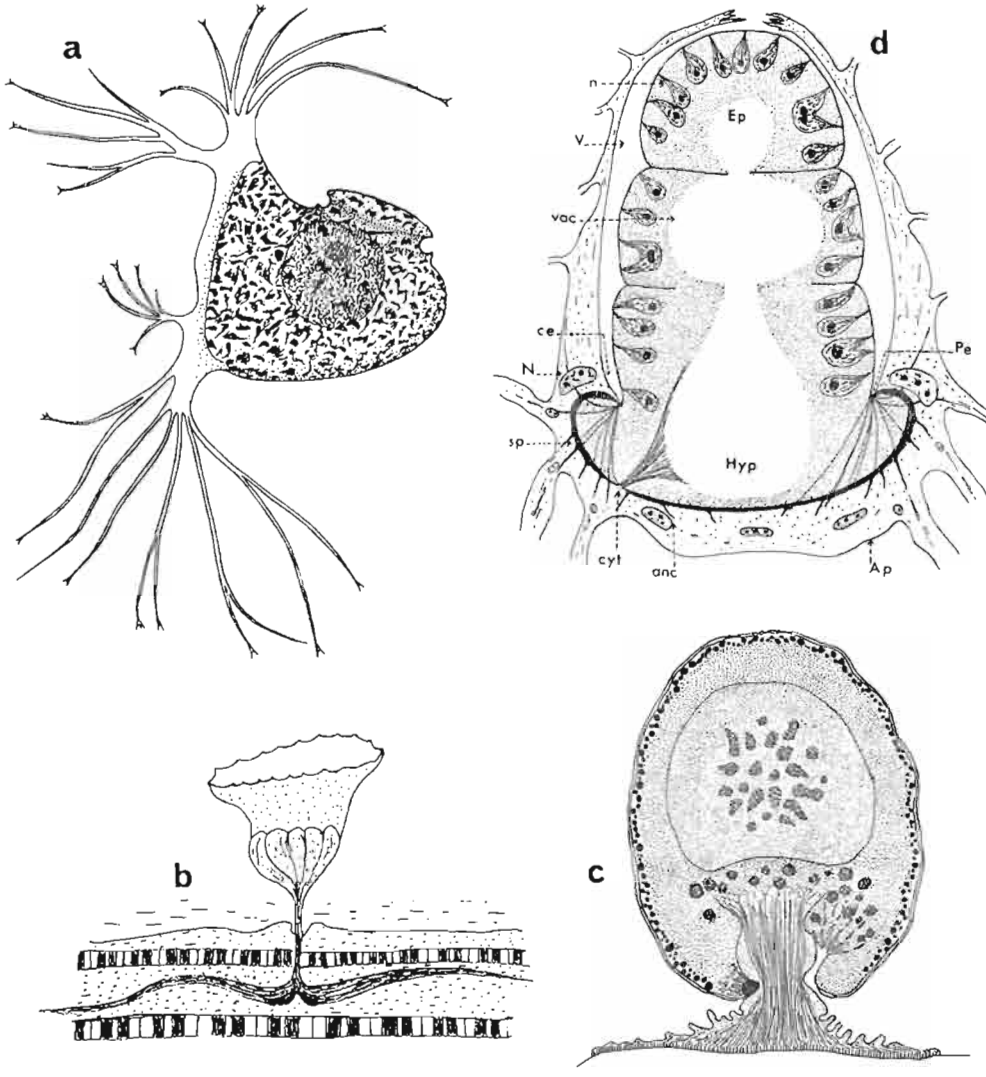

Fig. 6-2: Peridinians parasitising appendicularians. a: *Filodinium hovassei* (after Cachon and Cachon, 1968). b: *Oodinium fritillariae* (after Cachon and Cachon, 1971). c: *Apodinium chattoni* (after Cachon and Cachon, 1973). d: *Neresheimeria catenata* trophont at the beginning of sporozoit segmentation: (Ap) appendicularian; (N) nuclei of appendicularian: (n) parasite nucleus; (V) mucus vacuole of glandular cell; (vac) parasite axial vacuole; (ce) belt; (Pe) perinema; (sp) pedial sole: (cyt) cytosoma: (anc) anchoring fibrils; (Hyp) hyposoma: (Ep) episoma. (After Cachon and Cachon, 1966.)

In *Oikopleura albicans* Fenaux (1963) found orange-yellow spheres inside the general cavity which gave rise to peridinian sporozooids. Absence of early infestation stages has not allowed to place it in a previously known genus. This parasite was studied again by Hollande (1974) who illustrates sections of a peridinian Syndinidae parasite of *O. albicans*, but these are generally radiolarian parasites. The species was not described following the nomenclature rules, and has simply been cited as n. sp. In the figure legend it is referred to

as '*Syndinium oikopleurae* Hollande et Fenaux'. No information was given about its biology, and this species was not mentioned by Cachon and Cachon (1987).

Trichomonadina

According to Grassé (1952) the curious flagellate found in the digestive tract of *Ciona intestinalis*, briefly described by Parona Corrado (1886) as *Elvirea cionae*, belongs to the genus *Trimastix* (Fig. 6-3).

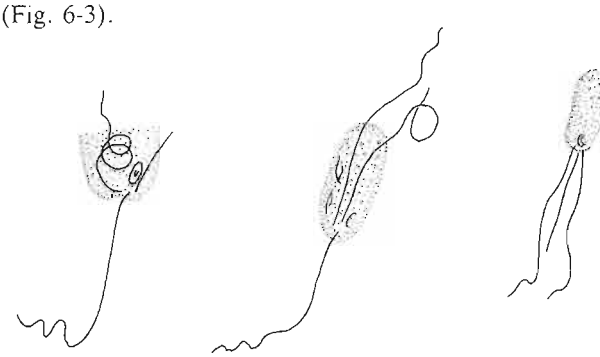

Fig. 6-3: *Trimastix cionae (Elvirea cionae)*. (After Parona Corrado, 1886.)

Rhizopoda

Rhizopods were noted on 2 occasions in ascidians. Huxley (1920) found *Amoeba clavellinae* in the stomach of *Clavelina lepadiformis* at Naples, Italy. The amoebae may completely fill the host stomach. Mackinnon and Ray (1931) described *Entamoeba phallusiae* (Fig. 6-4) from *Phallusia mammillata* near Plymouth, England. Both species have a 'sole' of glassy ectoplam and do not contain a retractile vacuole. Mackinnon and Ray supposed that the absence of a vacuole indicates the possibility of feeding by imbibition of liquid through the entire surface area, but this of course is insufficient for characterising the amoebae as true parasites.

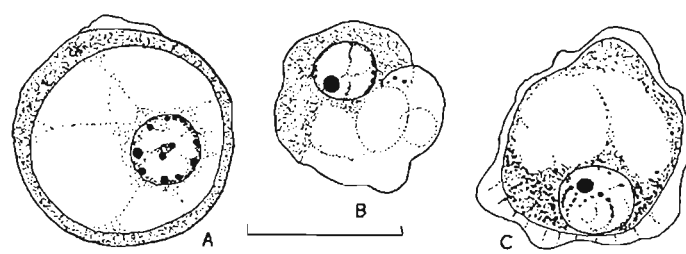

Fig. 6-4: Three *Entamoeba phallusiae* showing retraction of ectoplasm and tendency for a large vacuole to appear in the endoplasm, scale bar = 100 μm. (After Mackinnon and Ray, 1931.)

Agents: Apicomplexa (Gregarina, Coccidia and Enigmatic Parasites)

Gregarina

Gregarines are regularly present in Urochordata and at least half of the species examined turned out to be infested (Levine, 1981) (Table 6-2).

Gregarines of ascidians have first been reported by Kolliker (1848), but his descrip-

tions are so vague that it is not even sure whether these organisms are true gregarines. The first definitively identified gregarine was described by Lankester (1872), on the basis of vegetative stages, from the digestive tract of *Ciona intestinalis*, using the name *Gregarina (Monocystis) ascidiae*. It is now named *Lankesteria ascidiae* after the establishment of this genus, specific of ascidians, by Mingazzini (1891). In this species, Siedlecki (1899) described gregarine gametogenesis for the first time.

In the course of its life cycle (Fig. 6-5), the young gregarine cell penetrates into a cell of the intestinal epithelium, distending it, before it enters the gut cavity. There it attaches to another cell by its mucron (Siedlecki, 1901). Copulation occurs inside the host. Most often gregarines are discharged via the host's faeces in the form of cysts containing both sexes. Subsequent development takes place in sea water (Ormières, 1965). The full cycle can be achieved within one and the same host comprising all intermediate stages. Ormières (1965) refers to *Lankesteria diazonae* where cysts beginning development can be found in the host. Cysts at the stage of progamic mitosis were found in *L. perophoropsis*. In *L.*

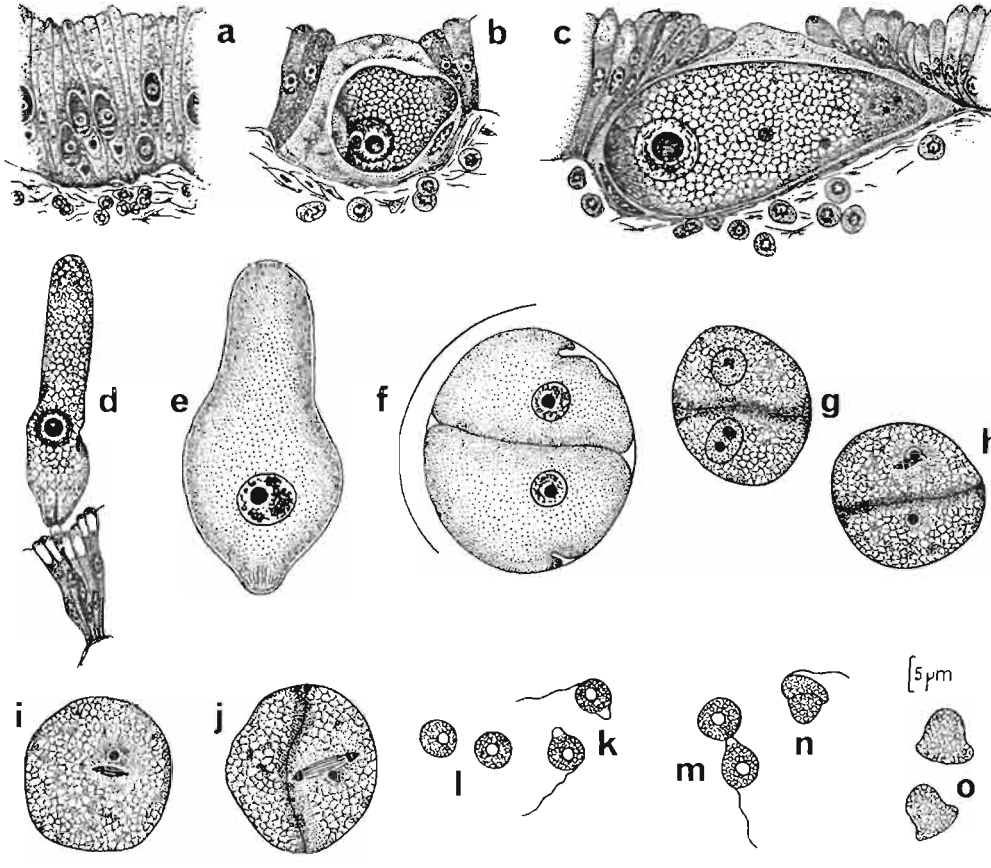

Fig. 6-5: *Lankesteria ascidiae*. (a) Intestinal epithelium of *Ciona intestinalis* at beginning of infestation; (b) host cell hypertrophy with nuclear atrophy; (c) maximum hypertrophy; (d) extracellular gregarine fixed to an epithelial cell; (after Siedlecki, 1901). (e) Typical shape of a free stage; (f to h) first mitosis; (i to j) anaphase; (k) male gametes; (l) female gametes; (m) beginning of gamete fusion; (n) fusion; (o) copulae. (After Ormières, 1965.)

parascidiae, the whole cycle is completed inside the host. Caullery (1927, 1929) describes the cycle of an unnamed gregarine (that Ormières, 1965, assumes to be *L. distapliae).* In the host, a colonial ascidian of the genus *Distaplia (D. stelligera),* the zooids are in permanent renewal. The oldest zooids, after closing their siphons, are lysing into the tunic. The gregarine, which infests most of the gut with motile stages, becomes liberated in the common test where it accomplishes sporogenesis. Ormières (1965) and Levine (1981) could not verify this phenomenon, not even in ascidian species which also have cyclic stages of disorganizing zooids (Polycitoridae, Polyclinidae or *Botryllus*).

The host's gut is the normal place for gregarine development. In some cases the intracellular stage becomes very large and induces real cysts protruding outside the digestive tract. Motile stages may then penetrate into the ascidian tissues but it seems that these gregarines cannot complete their life cycle (Ormières, 1965). *Lankesteria morchellii,* parasitising *Aplidium argus (Morchellium)* are commonly found in the heart and epicard of the host.

The digestive tract of ascidians has diverticules which may be invaded by gregarines, optionally for the hepatic gland or pyloric caecum of the stolidobranchiate ascidians. *Lankesteria parascidiae* (Fig. 6-6) lives obligatorily in the pyloric gland of *Aplidium elegans (Sidnyum* or *Parascidia elegans).* Another gregarine, *L. amaroucii* var. *magna*, also occurs exclusively in the gut of the same host; owing to this double parasitism, *L. parascidiae* completes its whole development in the ascidian (Ormières, 1965).

The presence of gregarines does not seem to exert any negative influence on the host, although Ormières (1965) considers that gregarines occur more often in colonial ascidians which seem less healthy.

Ascidian gregarines seem rather host specific. Harant (1931) provided a complete list of gregarines and their hosts, showing that there may be several hosts for the same parasite. Ormières (1965) confirmed parasite host specificity every time he was able to examine host and parasite together, except for *Lankesteria ascidiellae* occurring in *Ascidiella aspersa* and *A. scabra,* which many authors consider to be 2 different forms of one and the same species. Ormières estimates that the parasite's host specificity may assist ascidian taxonomists. However, he neglects that *L. siedlichii* lives in *Ascidia conchilega* and *A. mentula,* and *L. botrylli* in *Botryllus schlosseri* and *Botrylloides leachi* (Table 6-2). Host specificity has been ascertained by Levine (1981) for gregarines of ascidians in California (USA). The gregarines seem to be the same over the whole distributional area of the host: *L. ascidiae* is known in Europe and California (USA) from *Ciona intestinalis. L. ascidiae*, in turn, is parasitised by the microsporidian *Perezia lankesteriae* (Léger and Duboscq, 1909).

The only precise indications on infestation rates have been provided by Levine (1981) who has examined 361 ascidian specimens belonging to the 20 commonest species of the Californian coast (USA). Fourteen ascidian species were parasitized at infestation rates varying according to season, and reaching 10 to 100 % according to species.

Gregarines in pelagic tunicates have been known since the 19th century in the Mediterranean Sea to occur in salps (Leuckart, 1854). Frenzel (1885) placed them into the genera *Gregarina* and *Lankesteria*; Ormières (1965) created the genus *Thalicola*, and Théodoridès and Desportes (1968) set up the family Thalicolidae. These parasites are known to live only inside the gut. Some species exhibit a peculiar structure of their nuclear membrane (Desportes and Théodoridès, 1969) (Fig. 6-6b) which is, however not particular

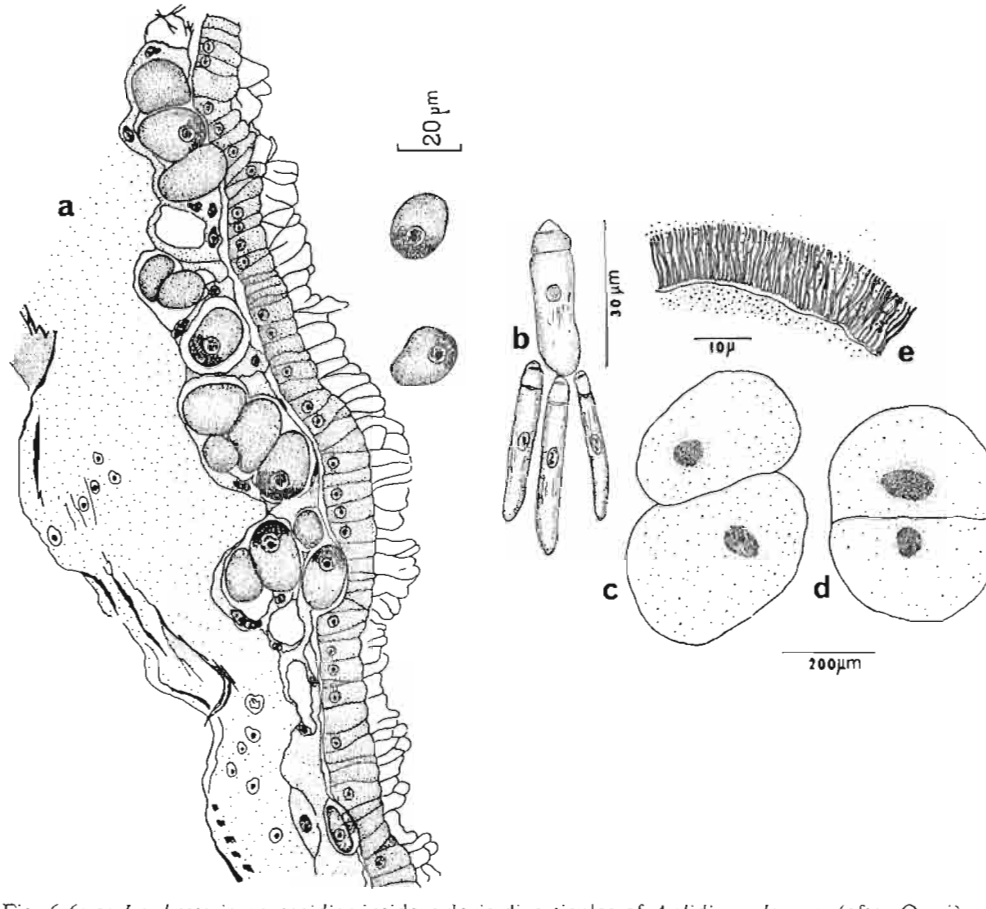

Fig. 6-6: a: *Lankesteria parascidiae* inside pyloric diverticules of *Aplidium elegans;* (after Ormières, 1965). b to e: *Thalicola salpae;* (a) salp gregarin; (b) multiple association; (d) syzygy; (d) cyst; (e) nuclear membrane detail. (After Théodoridès and Desportes, 1968.)

to the family. An unidentified gregarine was reported from a pyrosoma by Trégouboff and Rose (1957). Fenaux (1963) reports a gregarine Dicytidae with a development of the same kind as *Stenophora* in the appendicularian *Oikopleura dioica*.

Coccidia

The only coccidian parasitising ascidians, *Grasseella microcosmi*, has been reported from *Microcosmus sabatieri (sulcatus)* by Tuzet and Ormières (1960) and was redescribed by Ormières (1965) (Fig. 6-7). The parasite develops in the hepatic gland epithelium of its host, more rarely in its intestine, up to the micro- and macrogamete stages. Then it is expelled together with the host faeces. Fertilization occurs either in the faeces, or in sea water, and subsequent stages live in sea water.

Ormières (1965) has neither observed schizogonia in the host nor in sea water. He believes that schizogonia does not exist but he does not exclude their occurrence in an intermediate host, e. g., a crustacean. Parasite development leads to the destruction of the host's hepatic tissue. Ormières assumes that massive infestation, which he has never

Table 6-2
Apicomplexa and Acetospora from Urochordata (Original; compiled from the sources indicated)

Host	Parasite/Symbiote	Source
Larvacea		
Oikopleura albicans	Gregarine	Fenaux (1963)
Pyrosomatida		
Pyrosoma sp.		Trégouboff and Rose (1957)
Salpida		
Pegea confoederata	*Thalicola flava*	Ormières (1965)
Salpa fusiformis	Gregarine	Ormières (1965)
Salpa fusiformis	Simple cysts	Ormières (1965)
Salpa fusiformis	Cysts with cramps	Ormières (1965)
Salpa maxima	*Thalicola salpae*	Ormières (1965)
Salpa maxima	Cysts with cramps	Ormières (1965)
Salpa maxima	*Thalicola salpae*	Théodoridès and Desportes (1968)
Thalia democratica	*Thalicola ensiformis*	Ormières (1965)
Thalia democratica	Simple cysts	Ormières (1965)
Ascidiacea		
Aplidium argus	*Lankesteria morchellii*	Ormières (1965)
Aplidium argus	*Minchinia* sp.	in Ormières (1965)
Aplidium brementi	*Lankesteria* aff. *amaroucii*	in Ormières (1965)
Aplidium densum	*Lankesteria* aff. *amaroucii*	in Ormières (1965)
Aplidium elegans	*Lankesteria amaroucii magna*	Ormières (1965)
Aplidium elegans	*Lankesteria parascidiae*	Ormières (1965)
Aplidium elegans	*Minchinia ascidiarum*	in Ormières (1965)
Aplidium fuscum	*Lankesteria* aff. *amaroucii*	in Ormières (1965)
Aplidium nordmanni	*Lankesteria tuzetae*	Ormières (1965)
Aplidium nordmanni	*Lankesteria striata*	Ormières (1965)
Aplidium nordmanni	*Minchinia ascidiarum*	Ormières (1965)
Aplidium pallidum	*Lankesteria striata*?	in Ormières (1965)
Aplidium proliferum	*Minchinia ascidiarum*	Ormières (1965)
Aplidium proliferum	*Lankesteria* aff. *amaroucii*	in Ormières (1965)
Aplidium punctum	*Lankesteria amaroucii*	Ormières (1965)
Aplidium solidum	*Lankesteria aplidii*	Levine (1981)
Aplidium sp.	*Selysina incerta*?	Ormières (1965)
Aplidium turbinatum	*Lankesteria* sp.	Ormières (1965)
Ascidia ceratodes	*Lankesteria pittendrighi*	Levine (1981)
Ascidia conchilega	*Lankesteria siedlickii*	Ormières (1965)
Ascidia mentula	*Lankesteria siedlickii*	Ormières (1965)
Ascidiella aspersa	*Lankesteria ascidiellae*	Ormières (1965)
Ascidiella aspersa	*Lankesteria acutissima*	Ormières (1965)
Botrylloides leachi	*Lankesteria botrylli*	Ormières (1965)
Botryllus schlosseri	*Lankesteria botrylli*	Ormières (1965)
Botryllus sp.	*Lankesteria* sp.	Levine (1981)
Ciona intestinalis	*Cardiosporidium cionae*	Gaver and Stephan (1907b)
Ciona intestinalis	*Lankesteria ascidiae*	Ormières (1965)
Clavelina huntsmani	*Lankesteria abbotti*	Levine (1981)
Clavelina lepadiformis	*Lankesteria clavellinae*	Ormières (1965)
Dendrodoa grossularia	*Lankesteria zonata*	Ormières (1965)
Diazona violacea	*Lankesteria diazonae*	Ormières (1965)
Diazona violacea	*Lankesteria monstrosa*	Ormières (1965)
Diplosoma listerianum	*Lankesteria* aff. *amaroucii*	in Ormières (1965)

Table 6-2 (continued)

Host	Parasite/Symbiote	Source
Distaplia magnilarva	*Lankesteria distapliae*	Ormières (1965)
Distaplia rosea	*Lankesteria distapliae*	in Ormières (1965)
Distaplia stelligera	*Lankesteria distapliae*	in Ormières (1965)
Distomus violaceus	*Lankesteria globosa*	Ormières (1965)
Ecteinascidia herdmani	*Lankesteria perophoropsis*	Ormières (1965)
Eudistoma diaphanes	*Lankesteria diaphanis*	Levine (1981)
Eudistoma molle	*Lankesteria montereyensis*	Levine (1981)
Eudistoma psammion	*Lankesteria psammion*	Levine (1981)
Eudistoma ritteri	*Lankesteria ritteri*	Levine (1981)
Euherdmania claviformis	*Lankesteria euhermaniae*	Levine (1981)
Microcosmus sabatieri	*Grasseella microcosmi*	Ormières (1965)
Molgula appendiculata	*Lankesteria molgulidarum*	Ormières (1965)
Phallusia mammillata	*Lankesteria butschlii*	Ormières (1965)
Polycarpa gracilis	*Lankesteria gracilis*	Ormières (1965)
Polycarpa pomaria	*Lankesteria maculata*	Ormières (1965)
Polycarpa pomaria	Coccidian	Ormières (1965)
Polycarpa pomaria	*Selysina duboscqui*?	Ormières (1965)
Polyclinum aurantium	*Lankesteria* sp.	Ormières (1965)
Polyclinum aurantium	*Lankesteria tethyi*	in Ormières (1965)
Polysyncraton lacazei	*Lankesteria* aff. *amaroucii*	in Ormières (1965)
Pyura microcosmus	*Lankesteria gigantea*	Ormières (1965)
Pyura microcosmus	Coccidian	Ormières (1965)
Rhopalaea neapolitana	*Lankesteria gyriniformis*	Ormières (1965)
Ritterella pulchra	*Lankesteria ritterellae*	Levine (1981)
Ritterella rubra	*Lankesteria pescaderoensis*	Levine (1981)
Stolonica socialis	*Selysina perforans*	Ormières (1965)
Styela partita	*Lankesteria duboscqui*	Ormières (1965)
Synoicum parfustis	*Lankesteria synoici*	Levine (1981)

observed, would cause heavy injuries to the host. Under unfavourable conditions (e. g., reduced salinities), the size of expelled parasites decreases and the host eventually gets rid of its parasites.

Enigmatic Parasites (Cysts and Selysina)

Ormières (1965) reviews the cysts found in tunicates. In salps occur simple cysts which may be coccidian stages or gregarines, as well as cysts with cramps close to those of gregarines. Ormières believes that these cysts may represent growing or waiting stages. In many ascidians Ormières found what he named 'kystes durables', containing bushes of sporozoans.

Van Gaver and Stephan (1907a, b) describe, but do not illustrate, the enigmatic sporozoan *Cardiosporidium cionae* which lives in the pericardiac body of *Ciona intestinalis*. The pericardiac body is commonly found in *C. intestinalis* and many other phlebobranchs, located in the pericardiac cavity; it is composed of ascidian cells (myocard, blood cells) and varied debris often invaded by bacteria (Van Gaver and Stephan, 1907a). After describing different aspects of the parasite, Ormières (1965) concludes that it is at present impossible to construct the parasite's life cycle and to define its systematic position. Millar (1953) does not believe in the existence of this parasite at all.

Selysina perforans Duboscq, 1917 forms other types of cysts encountered only in

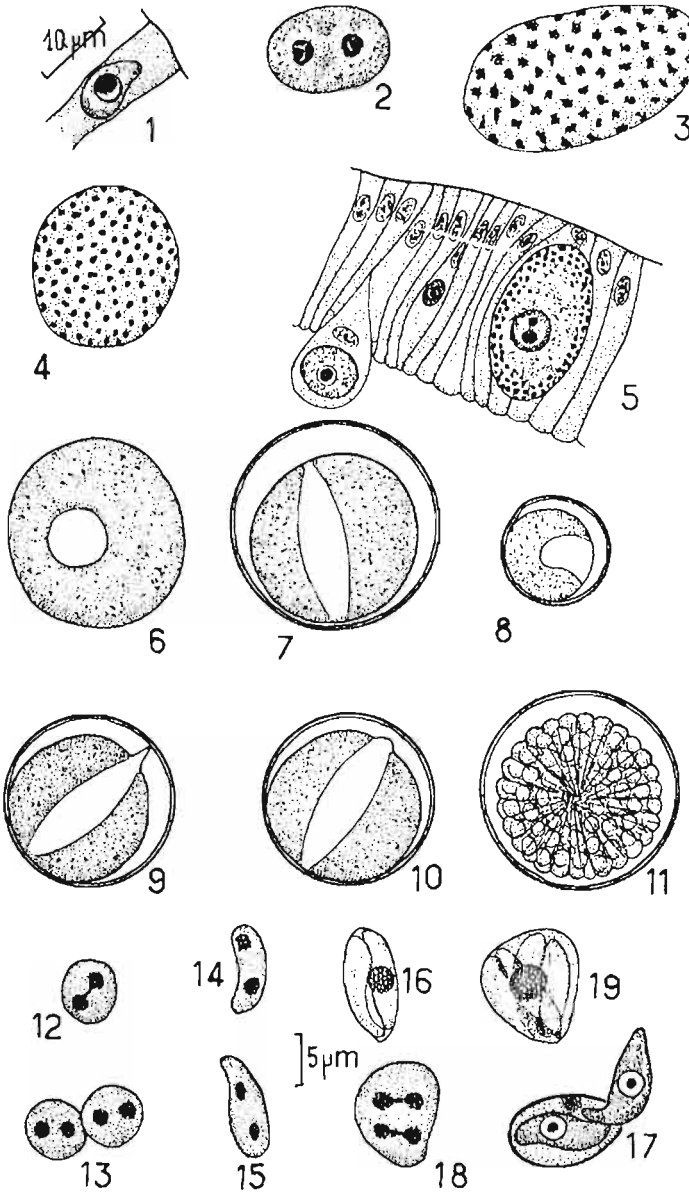

Fig. 6-7: *Grasseella microcosmi*. (1) Young non-differenciated coccidia; (2) beginning of microgamete evolution; (3 to 4) microgametocyt formation; (5) macrogamont evolution; (6 to 10) macrogamont evolution; (11) prelude to division in sporoblasts; (12 to 16) sporoblast evolution and spore formation; (17) dehiscence; (18 to 19) formation of abnormal spore with 4 nuclei. (After Ormières, 1965.)

Stolonica socialis. Ormières (1965) describes the different aspects of the parasite (Fig. 6-8). The cysts inside the ascidian tissues can be ejected to the outside passing through the tunic.

Harant's (1943) hypothesis is that these cysts represent 'xenoparasitic' complexes of sporozoans ingested by the ascidians and coming to a dead-lock. Ormières (1965) accepts

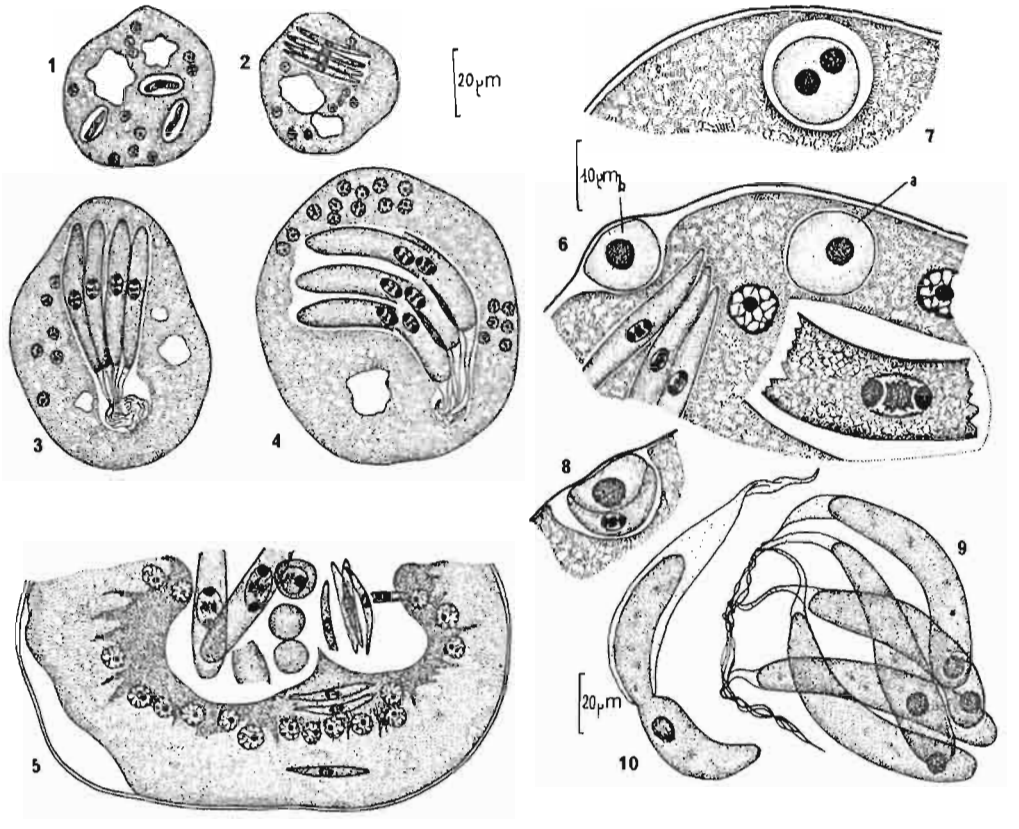

Fig. 6-8: *Selysina perforans*. (1) Young plasmode containing monozoic spores and vacuoles; (2) young plasmode with a bundle of sporozoites; (3 to 4) medium plasmodes with large sporozoans; (5) large cysts with 2 protoplasmic areas containing large sporozoans inside the vacuole and small sporozoans in cytoplasm and vacuole; (6) section of a 100 µm cyst with 1 large sporozoan, a bundle of small ones and uninucleated rounded elements; (7) same but binucleated; (8) uninucleated element with a small sporozoan; (9) bundle of large sporozoans liberated from cyst; (10) spontaneous emission of a large sporozoan. (After Ormières, 1965.)

the existence of 'durable cysts' but with much reservation in case of *Selysina perforans*, taking into account the specific relation with the host *Stolonica socialis*. Duboscq and Harant (1923) and Harant (1931) established 2 other *Selysina* species from *Aplidium (Parascidia)* sp.: *Styela canopus (partita)*, and *Polycarpa pomaria*, but few stages have been described, and Ormières did not find them again.

Ormières (1965) considers that, in some cases, these enigmatic parasites may correspond to stages of other sporozoans, using tunicates as intermediate hosts. Attempts of infesting fishes, crabs, and shrimps with such cysts from ascidians have never been successful.

Agents: Acetospora (Haplosporidia)

Duboscq and Harant (1923) noted for the first time an haplosporidian in a Polyclinidae. Ormières (1965) redescribed the agent concerned under the name *Haplosporidium ascidiarum* (Fig. 6-9). Taking into acount the taxonomic revision of haplospori-

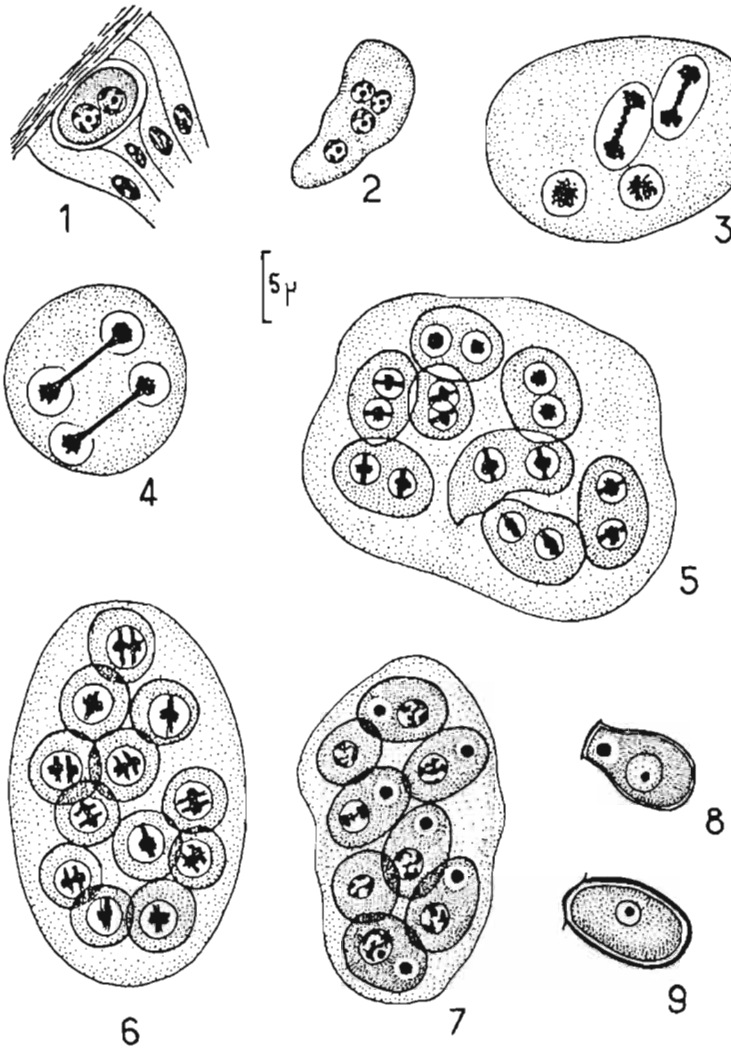

Fig. 6-9: *Minchinia ascidiarum*. (1) Youngest binucleated stage observed; (2) stage with 4 nuclei; (3) mitosis with a remaining nuclear membrane; (4) synchronous mitoses; (5) binucleated elements with parallel bundle fibers remaining; (6) copulae?; (7) sporoblast formation; (8) isolated sporoblast in its shell; (9) spore. (After Ormières, 1965.)

dian by Sprague (1963) he indicated that this species must be included in the genus *Minchinia*.

The whole life cycle can take place in the intestinal epithelium of the host, but the parasite may invade all ascidian tissues. Infestation can be so heavy that it reveals itself to the naked eye due to the dark coloration caused by the brown spores of the parasite.

Agents: Ciliophora

Commensal and parasitic ciliates closely associated with urochordates (Table 6-3) belong to several taxonomic groups. Parona Corrado (1886) noted and roughly illustrated

Table 6-3
Ciliata from Urochordata (Original; compiled from the sources indicated)

Host	Parasite/Symbiote	Source	Remarks
Pyrosomatida			
Pyrosoma elegans	*Trichophrya salparum pyrosomae*	Trégouboff (1916)	
Pyrosoma elegans	*Tunicophrya sessilis*	Théodoridès (1989)	
Pyrosoma giganteum	*Conchophrys davidoffi*	Chatton (1911)	
Salpida			
Thalia democratica	*Trichophrya salparum*	Entz (1884)	
Thalia democratica	*Heterocoma hyperparasitica*	Chatton and Lwoff (1939)	on *T. ascidiarum*
Ascidiacea			
Aplidium argus	*Trichophrya morchellii*	Trégouboff (1916)	
Ascidia callosa	*Trichophrya salparum*	Burreson (1973)	
Ascidia callosa	*Euplotaspis cionaecola*	Burreson (1973)	
Ascidia callosa	*Parahypocoma rhamphisokarya*	Burreson (1973)	
Ascidia conchilega	?	Harant (1931)	
Ascidia mentula	?	Harant (1931)	
Ascidia mentula	*Parahypocoma collini*	Chatton and Lwoff (1939)	
Ascidia paratropa	*Trichophrya salparum*	Burreson (1973)	
Ascidia paratropa	*Euplotaspis cionaecola*	Burreson (1973)	
Ascidiella aspersa	*Trichophrya salparum*	Collin (1912)	= *T. ascidiarum*
Ascidiella aspersa	*Hypocoma ascidiarum*	Harant (1931)	
Boltenia villosa	*Trichophrya salparum*	Burreson (1973)	
Boltenia villosa	*Parahypocoma rhamphisokarya*	Burreson (1973)	
Botryllus sp.	*Trichophrya salparum*	Collin (1912)	= *T. ascidiarum*
Botryllus sp.	*Hypocoma ascidiarum*	Collin (1912)	
Botryllus spp.	*Hypocoma ascidiarum*	Harant (1931)	
Botryllus spp.	*Hypocoma ascidiarium*	Chatton and Ségéla (1936)	
Ciona intestinalis	*Trichophrya salparum*	Collin (1912)	= *T. ascidiarum*
Ciona intestinalis	?	Harant (1931)	
Ciona intestinalis	*Amphileptus cionaecola*	Chatton and Ségéla (1936)	
Ciona intestinalis	*Parahypocoma collini*	Chatton and Lwoff (1939)	
Clavelina lepadiformis	*Parahypocoma collini*	Chatton and Lwoff (1939)	
Clavelina nana	*Hypocoma ascidiarum*	Harant (1931)	
Microcosmus sabatieri	?	Harant (1931)	
Molgula manhattensis	*Trichophrya salparum*	Calkins (1902)	
Perophora listeri	*Hypocoma ascidiarum*	Harant (1931)	
Polyclinum sp.	*Trichophrya ascidiarum*	Lachmann (1859)	Nomen nudum
Pyura haustor	*Trichophrya salparum*	Burreson (1973)	
Pyura haustor	*Euplotaspis cionaecola*	Burreson (1973)	
Pyura haustor	*Parahypocoma rhamphisokarya*	Burreson (1973)	

an unidentified holotrich ciliate, in the branchial sac of *Ciona intestinalis*. Chatton (1911) described *Conchophrys davidoffi* (Fig. 6-10a). This commensal ciliate of *Pyrosoma gigantea* actively moved about in the buccal apertures of all specimens observed in Villefranche (France). This species has never yet been redescribed.

The suctorian *Trichophrya ascidiarium* was named but not described by Lachmann (1859) in a *Polyclinum* species. Later *T. salparum* (Fig. 6-10b) was recorded in *Thalia democratica* at Naples, Italy (Entz, 1884) and in the Red Sea (Sewell, 1953). This species was found again in several ascidians at Woods Hole (USA), (Calkins, 1902), in the Mediterranean Sea (Collin, 1912) and in the North-East Pacific Ocean (Burreson, 1973).

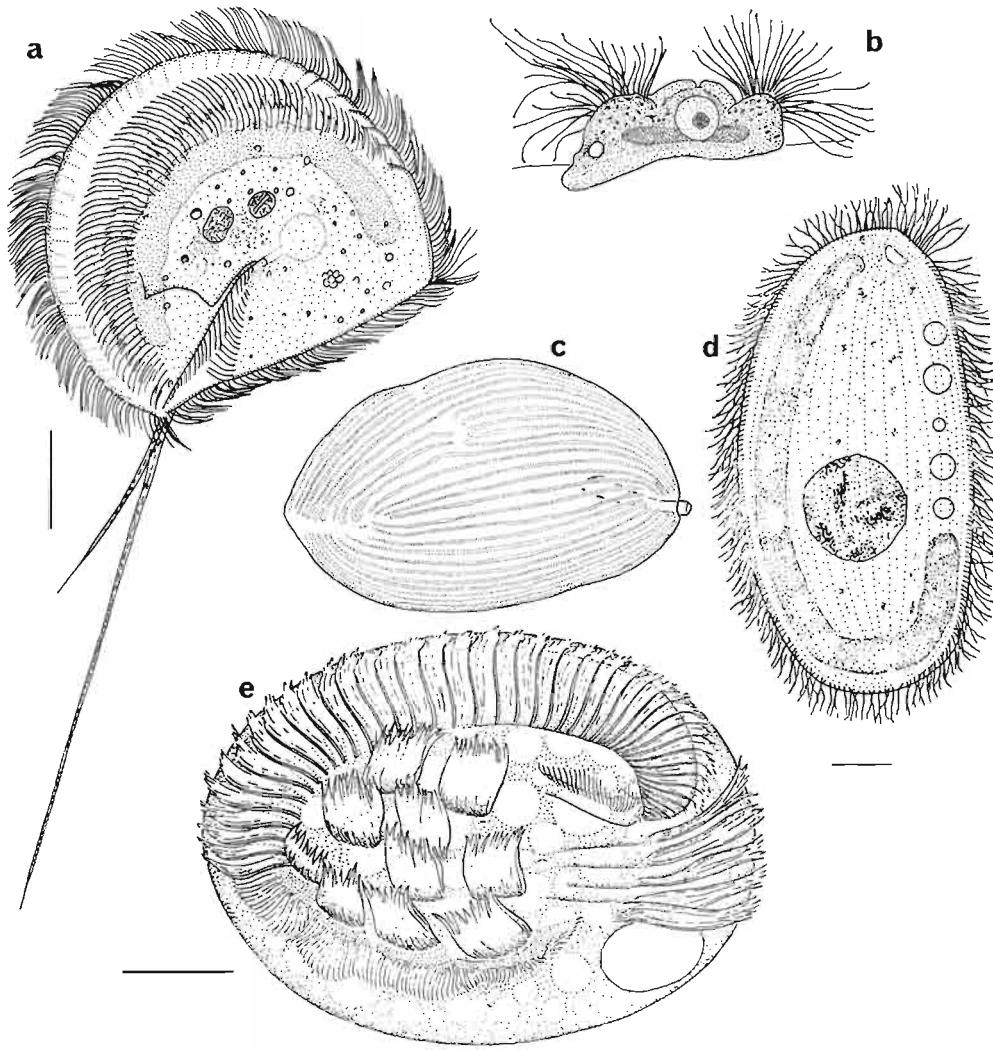

Fig. 6-10: Ciliata parasiting tunicates. (a) *Conchophrys davidoffi* (after Chatton, 1911; scale bar = 20 µm; (b) *Trichophrya salparum* (after Sewell, 1953); (c) *Parhypocoma collini* (after Chatton and Lwoff, 1939; (d) *Parhypocoma rhampisokarya;* scale bar = 20 µm; (after Burreson, 1973). (e) *Euplotaspis cionaecola;* scale bar = 10 µm. (After Chatton and Ségéla, 1936.)

Trégouboff (1916) has studied *Trichophrya* species occurring in ascidians, salps, and pyrosomes. He concluded that the parasites of salps and pyrosomes were quite similar, and he described *T. salparum pyrosomae*. This species is now known as *Tunicophrya sessilis* (Théodoridès, 1989). According to Trégouboff the ascidian parasite was clearly different; in *Aplidium (Morchellium) argus* he described *Trichophrya morchelli*. Chatton and Lwoff (1939) do not refer to this species and use the name *T. ascidiarum* for all *Trichophrya* species found by them in ascidians, while Burreson (1973) uses the name *T. salparum* for all *Trichophrya* recorded in a given host. Théodoridès (1989) uses the name *Actinobranchion salparum* instead of *T. salparum*. According to Burreson (1973) *T. salparum* may be, as Calkins (1902) thought, a parasite feeding directly on host tissue fluids, or an endocommensal, as these ciliates were always attached with their tentacles directed away from the host. Chatton and Lwoff (1939) described an other ciliate, *Heterocoma hyperparasitica*, living on *T. salparum*.

Hypotriches, which act as commensals, are only known from *Euplotaspis cionaecola* Chatton and Ségéla, 1936 (Fig. 6-10e) recorded later by Burreson (1973). *Hypocoma ascidiarum* Collin, 1912, cited by Harant (1931) is considered by Chatton and Lwoff (1950) as too badly described to be classified. They doubt that this species belongs to the genus *Hypocoma*.

Agents: Protista *incertae sedis (Nephromyces)*

Lacaze-Duthiers (1874) was the first to recognize these parasitic organisms in the renal sacs of members of the ascidian family Molgulidae, and erroneously assumed that they were gregarines. A pronounced polymorphism of the parasite was described. Giard (1888) studied living parasites and has described the principal stages of their development. Giard established 3 species in the genus *Nephromyces*, infesting the 3 Molgulidae species studied. He placed the genus in the fungus family Chytridinae. Harant (1931) redescribed the parasite in *Molgula appendiculata (Ctenicella)*, and for him it is undeniably a member of the Chitridinae.

The problem was reconsidered by Saffo (1981, 1982, 1988, 1989), Saffo and Davis (1982), and Saffo and Nelson (1983); these authors have shown that *Nephromyces* species were present in all adult specimens examined (more than 300) belonging to 6 species of Molgulidae off North America, as well as along the Atlantic and Pacific coasts. Saffo (1982) and Saffo and Nelson (1983) described and illustrated different aspects of *Nephromyces* sp. occurring together in the renal sac. Saffo and Nelson (1983) demonstrated that the different types of cells (Fig. 6-11) did not belong to the host. Very young individuals of *Molgula manhattensis*, grown in the laboratory, are devoid of parasites, but as soon as they get in contact with adults or a renal sac content, they become infested by the *Nephromyces* sp.

Saffo (1981) regroups the different forms of *Nephromyces* in a putative cycle completed by the discovery of the infesting stage (Saffo and Nelson, 1983) (Fig. 6-12). Saffo (1981) and Saffo and Fultz (1986, p. 1309) state that "*Nephromyces* shows vague similarities to several protistan groups, but compelling identity with none". The exact systematic position of *Nephromyces* sp. remains unknown. Saffo and Fultz (1986) found chitin in *Nephromyces* sp. suggesting fungal affinities; however, this is in contradiction with the morphological characters which suggest affinities to non-fungal taxa (mitochondrial ultrastructure). More over, "the swarmer cell of *Nephromyces* poses a particularly

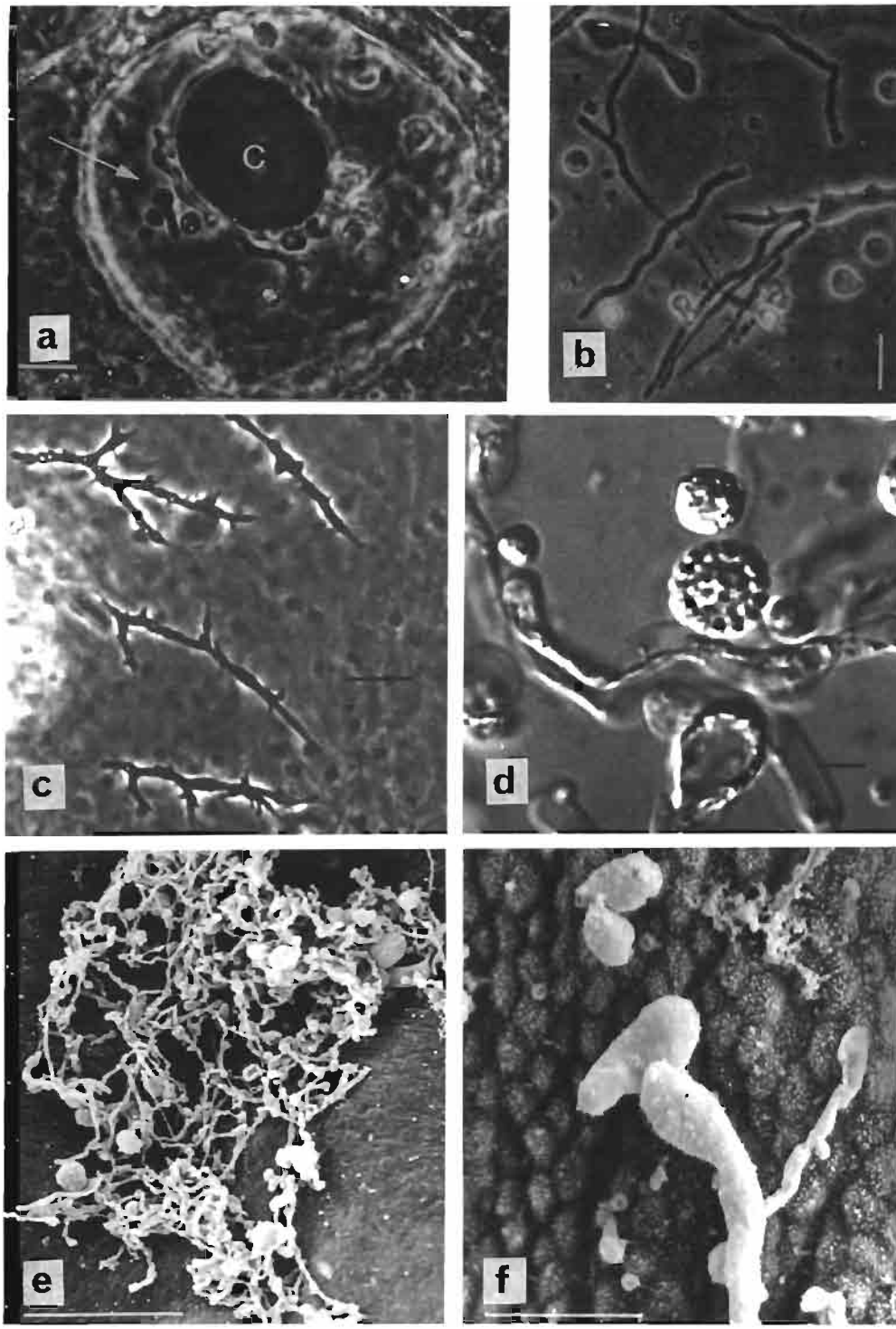

Fig. 6-11: *Nephromyces*. (a) Early round cells and filamentous cells; scale bar = 10 μm; (b) filamentous cells and slender filaments; scale bar = 20 μm (after Saffo and Nelson,1983); (c) irregular filaments on the inner sac wall; scale bar = 15 μm; (d) vacuolate filaments and sporangium; scale bar = 15 μm (after Saffo, 1982); (e and f) filaments from *Molgula manhattensis* of Arcachon (France); scale bar = 40 and 10 μm. (Original.)

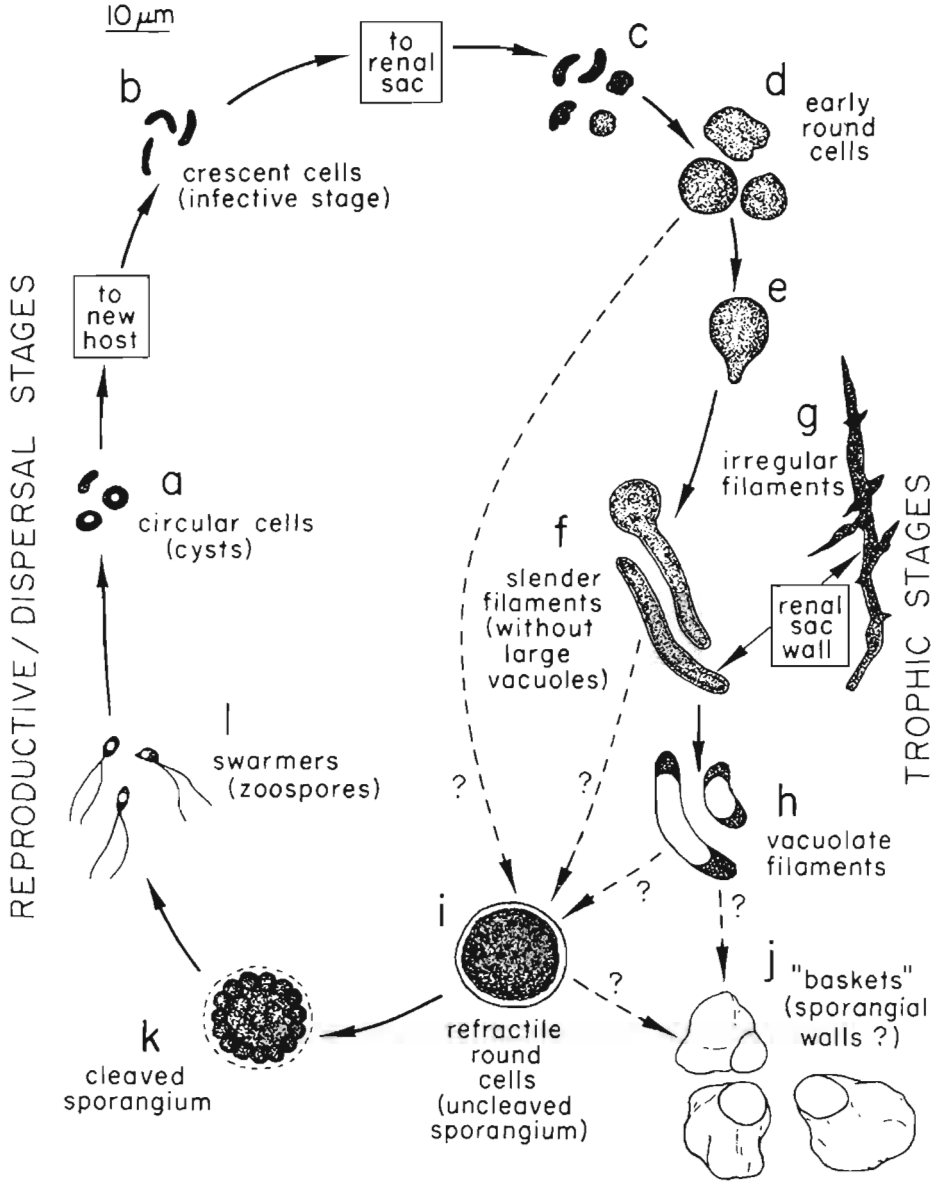

Fig. 6-12: Putative life cycle of *Nephromyces* from *Molgula manhattensis*. (After Saffo and Nelson, 1983.)

explicit taxonomic puzzle, as its possession of two posterior whiplash flagella distinguishes it from typical flagellated stages of both fungal and nonfungal protistan phyla" (Saffo and Fultz, 1986, p. 1309).

Nephromyces sp. may be infected by an endosymbiotic bacterium (Saffo, 1987) which is particularly abundant in the trophic (filamentous) stage of the host. This association was disclosed in all samples studied. Saffo (1988) further studied urate degradation in the

molgulid renal sac in the presence of *Nephromyces* sp. and has seen an intensive uricolysis in *Nephromyces* cells. She thinks that renal sac urates may constitute a source of carbon and nitrogen for the parasite. Such association would be beneficial to the host (Saffo and Fultz, 1986, 1989), the deposit of uric acid should not be permanent and by the intermediate for the *Nephromyces*-bacteria complex the renal content may be used as nitrogen source for the molgulid. Saffo considers a co-evolution likely between *Nephromyces* sp. and ascidians of the family Molgulidae. Similar organisms have been searched for in renal vesicles of Ascidiidae but without success (Harant, 1931; Saffo, 1988).

In the pyloric glands of *Styela* and *Polycarpa* spp. Harant (1931) described cells similar to *Pseudoklossia* sp. found in bivalves and described *Pseudoklossia legeri*. Later, with a more abundant material, Harant (1936) placed the parasite into the Mycochytrinidae under the name of *Nephrococcidioides legeri*. The quality of Harant's description and illustrations does not allow a new interpretation.

Agents: Protophyta (Algae)

The presence of unicellular algae in ascidians was suspected for a long time. The first such alga described was *Protococcus* sp. (Maurice, 1888); then followed reports on algae in *Perophora listeri* and *Ciona intestinalis* (Seeliger, 1907); these, however, turned out to be blood cells with green or yellow pigments. Ascidian taxonomists who found algal cells in tropical Didemnidae have simply noted their presence (Herdman, 1906; Michaelsen, 1920; Hastings, 1931). Smith (1935) described the first true case of an alga living in the cloacal cavities of 4 tropical Didemnidae. He considered the alga to be close to the zoochlorellas inhabiting corals, and assumed that ascidians do not take any advantage of this association. In the following 40 years this question was not considered further.

Examining algae associated with didemnids at the Australian Great Barrier Reef, Newcomb and Pugh (1975) identified these as bluegreens. Lewin (1975) described, from *Didemnum* sp. of Baja California (Mexico), *Synechococcus didemni* and placed it among the chroococales (Cyanophyta). Schulz-Baldes and Lewin (1976) studied the ultrastructure and pigments of this algae, and Lewin (1976) proposed a new algae taxon: the Prochlorophyta and created the new genus *Prochloron* (Lewin, 1977) (Fig. 6-13a). *Prochloron* members are characterized by the presence of chlorophyll *a* and *b*, and by the absence of phycobiliproteins.

From the Guadeloupean *Tridemnum cyanophorum*, Lafargue and Duclaux (1979) described *Synechocystis trididemni*, a true cyanophyte (Fig. 6-13b). Since then, the number of papers devoted to these unicellular algae have increased to more than 50. The *Prochloron* taxonomy awaits clarification. The only completely described species is *Prochloron didemni* (Lewin, 1975). Newcomb and Pugh (1975) assume that 4 distinct species exist, representing the 4 associations they studied. Cox (1986) compares different *Prochloron* populations; he admits specific differences, and describes 3 'typological groups' but is now waiting for more biochemical data. *P. didemni sensu stricto*, lives on the ascidian surface only (Cox, 1986). *S. trididemni* is a cyanophyte containing a chlorophyll only, with a structure so close to *Prochloron* that it 'seems likely to be related to the cyanophytan ancestor of the Prochlorophyta' (Cox, 1986, p. 440).

Cox (1986) lists 3 types of relation between algae and ascidians (Table 6-4) considering them as evolutionarily advanced. *Prochloron didemni* lives on the surface of numerous

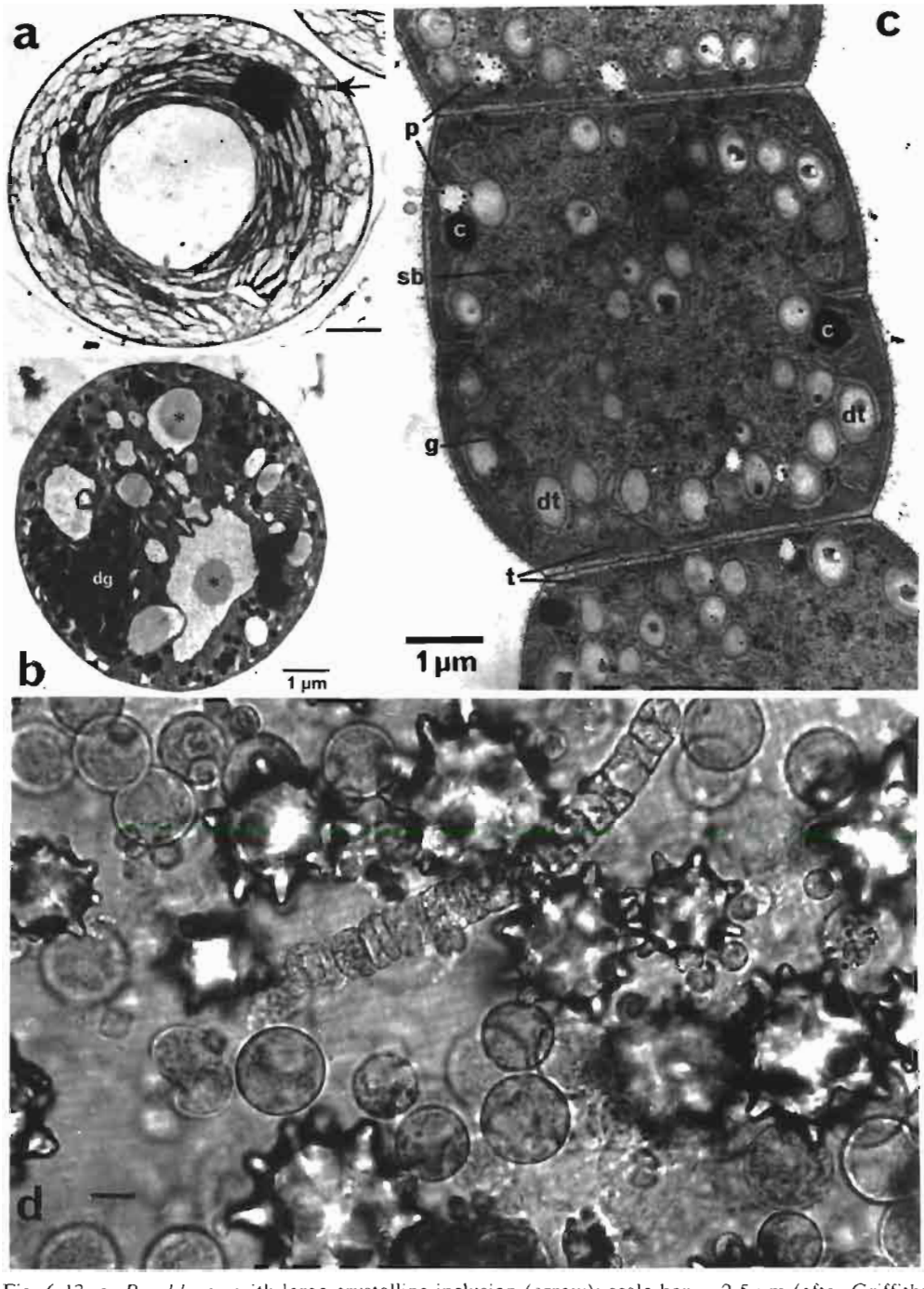

Fig. 6-13: a: *Prochloron* with large crystalline inclusion (arrow); scale bar = 2.5 µm (after Griffiths and co-authors, 1984). b: *Synechocystis trididemni* containing many dense granules (dg) and a reduced number of peripheral tylacoides (after Cox and co-authors, 1985). c: *Ocitorella* sp. from *Trididemnum miniatum* (after Larkum and co-authors, 1987). d: Test inclusion of *Trididemnum clinides* including spicules, *Prochloron* and cyanophytes; scale bar = 10 µm. (After Kott, 1982.)

Table 6-4
Protophyta from Ascidiacea (After Cox, 1986; modified)

Host	Algae	Localization	Source
Aplidium spp. (4)	Prochloron	Surface	Cox (1986)
Botryllus schlosseri	Prochloron	Surface	Cox (1986)
Ciona intestinalis	Cyanophyta	Test	DeLeo and Patricolo (1980)
Didemnum digestum	Prochloron	Surface	Cox (1986)
Didemnum etiolum	Prochloron	Test	Cox (1986)
Didemnum fulgens	Prochloron	Surface	Müller and co-authors (1984)
Didemnum menbranaceum	Prochloron	Surface	Cox (1986)
Didemnum molle	Prochloron	Cloacal cavity	Cox (1986)
Didemnum moseleyi	Prochloron	Surface	Cox (1986)
Didemnum proliferum	Prochloron	Surface	Cox (1986)
Didemnum psammathodes	Prochloron	Surface	Cox (1986)
Didemnum sp.	Prochloron	Surface	Cox (1986)
Didemnum viride	*Synechocystis trididemni*	Test	Cox (1986)
Didemnum viride	Prochloron	Cloacal cavity	Kott (1980)
Diplosoma handi	Prochloron	Cloacal cavity	Cox (1986)
Diplosoma mindori	Prochloron	Cloacal cavity	Cox (1986)
Diplosoma multipapillata	Prochloron	Cloacal cavity	Cox (1986)
Diplosoma pavonia	Prochloron	Cloacal cavity	Monniot and Monniot (1987)
Diplosoma similis	Prochloron	Cloacal cavity	Cox (1986)
Diplosoma translucidum	Prochloron	Surface	Cox (1986)
Diplosoma virens	Prochloron	Cloacal cavity	Cox (1986)
Echinoclinum triangulum	Prochloron	Test	Cox (1986)
Eudistoma amplus	Prochloron	Surface	Cox (1986)
Eudistoma sp.	Prochloron	Surface	Cox (1986)
Leptoclinides dubius	Prochloron	Surface	Cox (1986)
Leptoclinides lissus	Prochloron	Surface	Cox (1986)
Lissoclinum bistratum	Prochloron	Cloacal cavity	Cox (1986)
Lissoclinum fragile hospes	?	Test	Monniot F. (1984)
Lissoclinum patella	Prochloron	Cloacal cavity	Cox (1986)
Lissoclinum punctatum	Prochloron	Cloacal cavity	Cox (1986)
Lissoclinum punctatum	Prochloron	Test	Cox (1986)
Lissoclinum voeltzkovi	Prochloron	Cloacal cavity	Cox (1986)

Table 6-4 (continued)

Host	Algae	Localization	Source
Polysyncraton bilobatum	Prochloron ?	Surface	Lafargue and co-authors (1986)
Trididemnum cerebriforme	Prochloron	Surface	Cox (1986)
Trididemnum clinides	Prochloron	Test	Cox (1986)
Trididemnum clinides	*Synechocystis trididemni*	Test	Cox (1986)
Trididemnum clinides	*Oscillatoria* sp.	Test	Larkum and co-authors (1987)
Trididemnum cyanophorum	*Synechocystis trididemni*	Test	Cox (1986)
Trididemnum cyclops	Prochloron	Cloacal cavity	Cox (1986)
Trididemnum miniatum	Prochloron	Test	Cox (1986)
Trididemnum nubilum	?	?	Kott (1980)
Trididemnum palmae	?	Test	Monniot F. (1984)
Trididemnum paraclinides	?	Test	Kott (1982)
Trididemnum paracyclops	Prochloron	Cloacal cavity	Cox (1986)
Trididemnum solidum	*Synechocystis trididemni*	Test	Cox (1986)
Trididemnum strigosum	Prochloron	Test	Cox (1986)
Trididemnum tegulum	*Synechocystis trididemni*	Test	Cox (1986)
Unidentified ascidians	*Phormidium* (?) *ectocarpi*	Surface	Lewin and Cheng (1975)
Unidentified ascidians	*Phormidium spongeliae*	Surface	Lewin and Cheng (1975)
Unidentified ascidians	Diatoms	Surface	Edsbagge (1968)

ascidian species in all tropical shores worldwide, and has also been recorded in the Mediterranean Sea (Müller and co-authors, 1984; Lafargue and co-authors, 1986). This association seems obligatory for the algae only. It is probably more widely distributed than the literature indicates; it can only be observed *in situ*, collection of the host causes dispersion of the algal cells (own obs.). There is no visible reaction of the host at the tunic level.

Other *Prochloron* species and *Synechocystis trididemni* live inside the tunic of some tropical didemnids (Fig. 6-13d). Some *Prochloron* sp. invade the cloacal cavities and branchial sac of tropical Didemnidae. In the 2 last-mentioned cases, the association seems obligatory for algae and ascidian*; a mode of transport of algal cells by ascidian larvae was recorded for the first time by Eldredge (1967) and later described by the term 'rastrum' by Kott (1980) (Fig.6-14). Müller and co-authors (1984) assume that the alga-ascidian

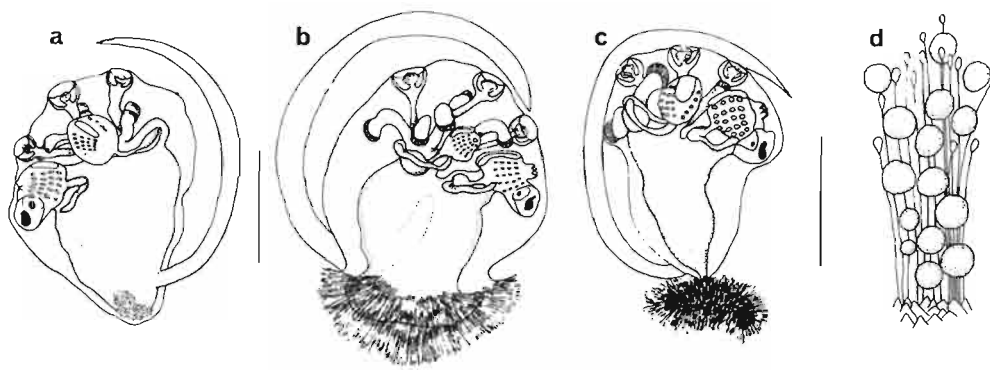

Fig. 6-14: *Diplosoma papillata*. (a to c) Rastrum development in larvae; (d) rastral hairs entangling algal cells; scale bars = 0.5 mm a to c, 0.05 mm d. (After Kott, 1980.)

symbiosis may be linked to the presence, in the ascidian host, of a large quantity of lecithin which is essential to the growth in cultures of *Prochloron* sp. *Didemnum molle* colonies without algae should not contain lecithin.

Lewin and Pardy (1981) and Pardy and Lewin (1981), have shown a transfer of metabolites from the algae to the ascidian by translocation; this was confirmed by Paerl (1984) who assumes that the presence of symbiotic algae benefits the ascidian, allowing it to survive under very unfavourable conditions. Cox (1983) demonstrated that ascidian cells can phagocyte algae. Monniot and Monniot (1987) studied Central Pacific Didemnidae and suppose that the presence of Didemnidae with algae in well-lightened substrates, allows these ascidians to colonize areas normally avoided by them, thus contributing to a diversification of these animals in coral reefs. However, the presence of *Prochloron didemni* on the colony surface does not seem to modify the ecological distribution of host species concerned. In natural populations, only a small portion of the colonies are carrying algae.

Cox (1986) assumes that co-evolution occurred in *Prochloron* sp. and members of the Didemnidae. The problem is probably more complex as *Prochloron* sp. living in cloacal

* Müller and co-authors (1984) noted the absence of algae in very small colonies of *Didemnumn molle* at 25 m water depth. This observation has to be ascertained.

ascidian cavities, are not known from in the Atlantic Ocean, and because the symbiosis is limited to only a few species in 4 different Didemnidae genera which entertain no particular phylogenetic relations (author's own opinion).

Other symbiotic algae have been described. A filamentous cyanophyte lives in symbiosis with a *Prochloron* sp. inside the tunic of *Tridemnum clinides* (Kott, 1982) (Fig. 6-13d), and was described by Larkum and co-authors (1987) under the name *Oscillatoria* sp. (Fig. 6-13c). De Leo and Patricolo (1980) found at Palermo (Italy) a cyanophyte making cysts in the tunic of *Ciona intestinalis* but they have not been able to identify the algae. Lewin and Cheng (1975) recorded other algae on ascidian colonies in Baja California (Mexico), a pink filamentous cyanophyte *Phormidium* (?) *ectocarpi,* and other pink cyanophytes. *P. spongeliae* also lives on ascidians.

Colonial diatoms with square cells in zig-zag filaments, probably members of *Striatella,* cause tan-coloured patches on white didemnidae. Presumably non-specific associations with diatoms have been noted along the Swedish coast (*Striatella unipunctata* and *Grammatophora* spp.) by Edsbagge (1968).

DISEASES CAUSED BY METAZOANS

Agents: Cnidaria

In a solitary ascidian collected in Washington Sound (USA), Fraser (1937) found a thriving colony of the hydroid *Entocrypta huntsmani.* One of the polyps contained the completely ingested body of an oviferous female notodelphyd copepod (Illg, 1958). In deep-sea ascidians of the family Agnesiidae the reviewer has found gymnoblastic hydrozoans living attached inside the cloacal cavity.

Agents: Ctenaria

Korotneff (1888) described *Gastrodes parasiticum* forming a cyst in the tunic of *Salpa fusiformis.* This associate possesses fertile gonocystes and is interpreted as a 'dissogone' Ctenaria larva, of the genus *Lampetia* in its last developmental stage (Trégouboff and Rose, 1957). This situation seems exceptional and has never been recorded again.

Agents: Turbellaria

Planaria schlosseri linked to *Botryllus schlosseri,* was recorded by Giard (1873); however, it turned out to be only a colour variation of a free-living planarian. Several authors (e.g.; Jennings, 1957; Lambert, 1968; Ching, 1977) reported turbellarians feeding on ascidians.

Agents: Annelida

The presence of annelids in the branchial or cloacal cavities of ascidians is not frequent and seems to be accidental. However, Illg (1958) notes the presence of 5 individuals of polychaetes in the branchial and cloacal cavities of 20 ascidians. The reviewers has found once 10 Syllidae in the branchial sac of *Microcosmus anchylodeirus* from the West Indies.

Agents: Bryozoa

Ectoproct bryozoans occurred in 2 specimens of the bathyal ascidian *Culeolus herdmani* living on the slope of New-Caledonia between 300 and 1000 m water depth. The bryozoans were attached between the oral tentacles and the peripharyngeal grove of the ascidians, without apparently causing damage to the host (reviewer's own observation).

Agents: Nemertina

Nemertines inhabit the cloacal cavities of large solitary ascidians. In Europa 2 species are linked to ascidians. The first species, *Tetrastemma vittatum,* inhabits *Phallusia mammillata, Ascidia mentula* and *Ciona intestinalis;* it was also recorded by Harant (1931) between body wall and tunic of *Microcosmus sabatieri (sulcatus), Molgula occidentalis (impura),* and *M. appendiculata (Ctenicella)* where it digs galleries. The second species, *T. flavidum,* was recorded — in the Mediterranean Sea — in *Phallusia mammillata* and *Microcosmus sabatieri.*

Monniot (1965b) studied at Roscoff (France) the behaviour of *Tetrastemma vittatum* in *Ciona intestinalis* and *Ascidia mentula.* The nemertine inhabits the insides of both the branchial and cloacal cavity. Passing from one to the other, it pierces the branchial sac of its host, but in *A. mentula* it penetrates the branchial button hole.

In species of *Microcosmus,* in the Mediterranean Sea, *Tetrastemma flavidum* may spend its whole life cycle in the cloacal cavity. Normally, nemertines live inside the branchial folds; juveniles succeed in penetrating the branchial cavity by gliding through the stigmata. Monniot (1961a) provided evidence in support of an antagonism between nemertines and ascidicolous copepods. While there are on average of 5.4 nemertines in a *Microcosmus* sp. in the absence of copepods, this average decreases to 2.9 in the presence of *Notodelphys acanthomela,* and to 1.0 in the presence of adult *Doropygus pulex.* Juvenile copepods seem less effective, allowing an average nemertine number of 4.2. No juvenile nemertines were found in the presence of adult copepods.

Gononemertes australiensis which lives in Australian ascidians has been studied particularly by Gibson (1974), Gibson and Egan (1976), Egan and Anderson (1979), and Egan (1984a). *G. australiensis* has morphologically adapted to commensalism resulting in the loss or reduction of some sensory organs, and gonadal hypertrophy (Gibson, 1974). The nemertine infested 100 % of the shallow-water population of *Pyura pachydermatina.* While infestation by a single nemertine is common, multiple infestation is possible. Egan (1984a) has shown that the host has an annual life cycle, and that there are 3 adaptations of the nemertine cycle allowing quick invasion of young ascidians. The nemertine possesses permanent fecundity, with a maximum during ascidian reproduction. Juvenile nemertines, living in young ascidians acquire precocious maturity, and this allows fast infestation among aggregated populations of *Pyura pachydermatina.* Egan (1984b) studied simultaneously the nemertine's life cycle and that of the copepod *Pachypygus australis* which coexist in ascidians. He noted no antagonism between these 2 commensals. *G. autraliensis* inhabits the cloacal cavity, *P. australis* the branchial cavity. Young nemertines hid between the ascidian's hepatic lobes and hence never came into contact with the copepods.

Agents: Gastropoda

Two families of gastropods are linked to ascidians: the Cypraeidae and Lamellidoridae. In both cases, the agents live on the tunic of colonial ascidians, where they lay eggs and feed on the ascidians (Fretter and Graham, 1962; Gulliksen, 1975). A species close to the Lamellidoridae, *Pseudosacculus okai* (Hirase, 1927, 1928), forms gall-like swellings as do the *Modiolaria* in the test of *Ascidia prunum* and *Boltenia ovifera* in the North-East Pacific. The tunic hole communicates with the outside by a narrow slit.

At Naples (Italy), Diehl (1970) observed how the prosobranch gastropod *Fusinus* sp. laid egg capsules inside the tunic of several ascidian species. The presence of gastropods in the branchial and cloacal cavities of ascidians has rarely been observed and seems hazardous.

Agents: Bivalvia

In all seas of the world, species of Mytilidae are associated with numerous species. The mytilids belong to the genera *Modiolus, Modiolaria, Crenella,* and *Musculus.* Such associations have been studied particularly in the Mediterranean Sea by Bourdillon (1950, 1955). The bivalve becomes totally enclosed in a hollow of the tunic wich opens by 2 lips linked by byssus filaments. The siphons are the only parts of the bivalve which protrude to the outside. This association is not obligatory but sometimes the bivalve agents are so numerous and so consistently present that the name of the ascidian was given in reference to that association: *Polycarpa mytiligera.*

Bourdillon (1950) hypothesized that the bivalve may be attracted by chemical components of the tunic. In 1955 he established that the ascidian's water current attracts bivalves.

Bertrand (1971) studied the association between *Musculus lateralis* and *Molgula occidentalis* along the east coast of the United States and describes the building of the 'nest'. Inclusion of the bivalve by the tunic is purely mechanical: tension of the byssus filament progressively drills a hole into the tunic, the bivalve's burrowing speed depends on tunic consistency. Bertrand (1971) confirms that the bivalves are attracted by the water current and that they settle close to the ascidian's siphons. The mollusc holds its position by exerting pressure on the tunic. When the bivalve dies, the tunic displays a tendency to regain its original shape and thus to expell the shell.

Agents: Cephalopoda

The male of *Orythoe tuberculata,* a planktonic octopod, is small-sized (3 cm), while the female reaches 30 cm. The octopod seems to live permanently in diverse floating shelters, and so it has been considered as the 'owner' of the argonaute shell. *O. tuberculata* has been recorded in *Salpa tilesii* by Jatta (1896) at Naples (Italy) and its behaviour inside *Tethys vagina* was studied under *in situ* conditions employing SCUBA diving (Hardwich, 1970) off the Californian coast. An octopod inhabited the cavity of a dead salp in which the diagonal septum was destroyed. The cephalopod leaves and reenters its shelter; frightened by a diver, it swam away into the open sea. Banas and co-authors (1982) conducted similar observations in young *Argonauta* sp. living in chains of *Pegea socia,* but in this case, no damage seemed to occur to the salp. Only one argonaute was found in each salp chain.

Agents: Copepoda

Ascidians, with their large branchial cavities, are attractive to copepods; next to fish they entertain the most numerous associations with copepods. Relations between ascidians and copepods are very diverse and range from simple presence in the branchial cavity to internal parasitism inducing host defence; however, the ascidians never seem severely hurt by the copepods.

Associations between ascidians and copepods are a very general phenomenon encountered in all seas, ranging from the Arctic to the Antarctic and down to maximum water depths. A large number of copepods remains undescribed (the reviewer keeps a collection of non-studied copepods containing more than 900 samples). There are large sea areas in which parasitic copepods have never been studied, in particular the tropical Indian and Pacific Oceans, where ascidians are most numerous and diversified. Based on the reviewer's own experience, at least some 50% of the ascidian species are estimated to shelter parasitic copepods.

The first work about copepods associated with ascidians (Thorell, 1859a, b) was of an extraordinary high quality, all species described are still taxonomically accepted to this day. Thorell dissected, described, and figured all mouth-parts and created the main systematic divisions based on their structure. He determined 2 essential characteristics of ascidian-copepod associations: (1) several species of copepods can be found in 1 individual ascidian; (2) a given copepod species can live in several ascidian species occupying the same geographical area (non-specific commensalism).

Ascidicolous copepods mostly belong to the Cyclopoida and Gnathostoma. The suborder Notodelphyoidea Sars (1903) is no longer accepted. The ascidicolous gnathostoms belong to 3 families: Archinotodelphyidae Lang (1949b), Notodelphyidae Thorell (1859a), and Ascidicolidae Thorell (1859a) *sensu* Illg and Dudley (1980). Inside ascidians are also found some representatives of the large Lichomolgidae family, recently revised by Humes and Stock (1973), which belong to the Cyclopoida Poecilostoma. Two very modified genera, *Gonophysema* and *Capistrum,* still lack a defined systematic position. Izawa (1987) assumes that *Gonophysema* may belong to the Poecilostoma based on nauplius setation.

Archinotodelphyidae are typical cyclopoid copepods with no reductions of their appendices compared to their free-living counterparts, and they have external ovisacs (Fig. 6-15a). All Notodelphyidae (Fig. 6-15b) possess a dorsal brood sac opening at the junction of the somites with the 4th and 5th pedigerous somite. The Ascidicolidae differ from the Notodelphyidae by the structure of their mouth pieces; especially the maxilliped is reduced in the Notodelphyidae but forms a strong hook in the Ascidicolidae. Except in the Buprorinae, egg strings are external (Fig. 6-15c). The Lichomolgidae are much smaller and have 2 external ovisacs and the typical mouth pieces of poecilostoms (Fig. 6-15d).

The Notodelphyidae show the greatest number of morphological adaptations to their life inside ascidians, and the most pronounced dependancies on the host. Members of the less modified genus *Notodelphys* are able to swim actively and to leave the ascidian when it dies. The most modified genera are almost completely devoid of setae and live in the common cloacal cavities of the Didemnidae. In copepods adaptation to parasitic life is primarily expressed by setae reduction, loss of mobility, reduction and loss of appendages, and loss of superficial metamery. This evolutionary trend can be followed genus after

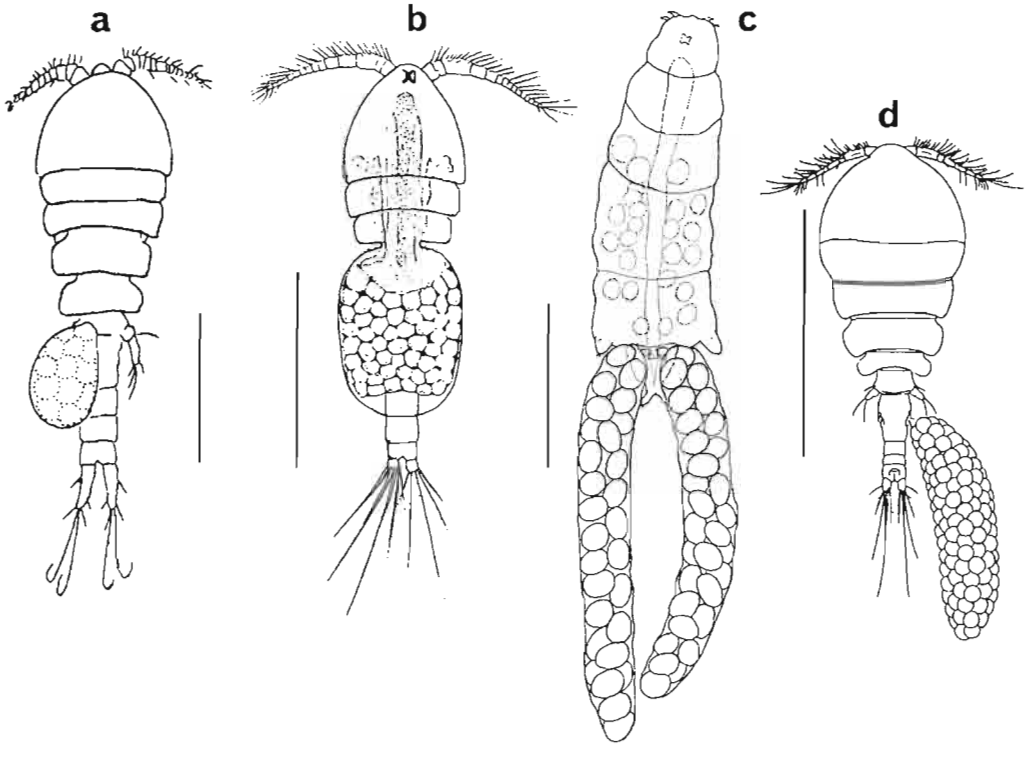

Fig. 6-15: Families of ascidicolous copepods. (a) Archinotodelphyidae *Pararchinotodelphys gurneyi* (after Illg, 1955, modified from Lang, 1949); (b) Notodelphyidae *Notodelphys tenera* (after Thorell, 1859 a, b); (c) Ascidicolidae *Haplostoma dentatum* (after Ooishi and Illg, 1977); (d) Lichomolgidae *Ascidioxynus bermudensis*. Scale bars = 1 mm. (After Humes and Stock, 1973.)

genus in the Notodelphyidae and can be related to the location of the copepods inside the host as well as the morphology of the ascidian's cavities.

It is in the large Phlebobranchiata and Stolidobranchiata that copepods were first described in Scandinavia by Thorell (1859a, b), Aurivillius (1882a, b, 1883, 1885), Sars (1921), and Lang (1948, 1949a); in the Mediterranean Sea by Buchholtz (1869), Gerstaecker (1863), and Giesbrecht (1892); in Great Britain waters by Brady (1878), and Scott (1907); and in the British Channel by Canu (1886, 1891a, b, 1892), Chatton (1909), Chatton and Brément (1911). Ascidicolidae copepods have been studied mostly in the British Channel and Mediterranean Sea by Canu (1892), Brément (1909, 1911), Chatton and Brément (1909a, b, c, 1910, 1915a, b, c), Chatton and Harant (1922a, b, c, 1924a, b, c, d, e). The copepods collected outside Europe by Hartmeyer have been described by Schellenberg (1922). North American species were studied by Blake (1929, 1933), Gray (1933, 1938), Pearse (1947, 1952), and later by Illg (1955, 1958), Dudley and Solomon (1966), Ooishi and Illg (1977), Dudley and Solomon (1966), Illg and Dudley (1980). Papers published since Illg's monographies (Illg, 1958; Illg and Dudley, 1980) are listed in Table 6-5.

Table 6-5
Publications refering to agent-host relations of ascidians and copepods, published after the monographies of Illg (1958)
(Original; compiled from the sources indicated)

Authors	Sea area	Authors	Sea area
Gotto (1959)	British Islands	Monniot (1968a)	Channel
Stock (1959)	Mediterranean Sea	Lafargue and Laubier (1968b)	Mediterranean Sea
Bocquet and Stock (1960)	Channel	Monniot (1968b)	Channel
Bresciani and Lützen (1960)	Sweden	Gotto (1970)	Australia
Gotto (1960)	British Islands	Stock (1970)	Caribbean
Bocquet and Stock (1961)	Channel	Illg (1970a)	Japan
Bresciani and Lützen (1961)	Sweden	Illg (1970a)	New Caledonia
Gotto (1961)	British Islands	Ooishi (1972)	Japan
Illg and Dudley (1961)	Mediterranean Sea	Hamond (1973)	British Islands
Ooishi (1961a)	Japan	Ooishi and Illg (1973)	Japan
Monniot (1961b)	Mediterranean Sea	Gotto and Logan (1974)	British Islands
Ooishi (1961b)	Japan	Laubier and Lafargue (1974)	Mediterranean Sea
Monniot (1961c)	Mediterranean Sea	Ooishi and Illg (1974)	Japan
Bresciani and Lützen (1962)	Sweden	Jones (1974)	New Zealand
Monniot (1962)	Mediterranean Sea	Gotto (1975a)	Australia
Gotto (1962a)	British Islands	Lafargue and Laubier (1977)	Mer Rouge
Ooishi (1962)	Japan	Lafargue and Laubier (1978a)	Singapore
Monniot (1963)	Mediterranean Sea	Lafargue and Laubier (1978b)	Mediterranean Sea
Roland (1963)	Channel	Jones (1979)	New Zealand
Ooishi (1963a)	Japan	Gotto and Threadgold (1980)	British Islands
Ooishi (1963b)	Japan	Jones and Montez-Moreno (1981)	Caribbean
Illg and Dudley (1965)	Mediterranean Sea	Monniot (1982a)	Channel
Gotto (1966)	British Islands	Monniot (1982b)	Channel
Stock (1967a)	Red Sea	Monniot (1983)	Caribbean
Stock (1967b)	Red Sea	Ho (1984)	Japan
Lützen (1968)	Sweden	Ooishi and Illg (1986a)	Japan
Lafargue and Laubier (1968a)	Mediterranean Sea	Ooishi and Illg (1986b)	Japan

Relations Between Ascidians and Copepods

Copepods ordinarily inhabit the natural cavities of their host —most often branchial sac, cloacal cavity or gut; they do not seem to cause inconvenience or damage to their ascidian host. Gotto (1975b) found 123 individuals of *Doropygus flexus* in the branchial sac of 1 *Pyura praeputialis* which did not seem disturbed. Most copepods found several times or represented by several individuals inhabit different host species and belong sometimes to different families or orders. Copepods seem more linked to the shape of ascidian cavities than to the ascidian itself. However, recent studies (see below) show that the ascidian plays a role in the copepod's sex-determination and morphological expression. For the Notodelphyidae some relations can be established, at the generic level, between the morphologies of ascidians and copepods (Table 6-6). Of special significance is the relation between size and structure of the branchial sac and the copepod's mobility.

Members of the Archinotodelphyidae and the Notodelphyidae genera *Notodelphys* (Fig. 6-16a), *Paranotodelphys* (Fig. 6-16b), *Notodelphyopsis, Notopterophoroides,* and *Pygodelphys*, are all able to leave their host; some can swim and all can move about actively. They indifferently live in the branchial sac of members of all solitarian ascidian families (Phlebobranchiata and Stolidobranchiata); among the Aplousobranchiata they occur only in species of *Clavelina* which have large branchial sacs. These copepods are more numerous in phlebobranchs than in stolidobranchs which have a branchial sac with folds. According to Gotto (1979), preference for the genera *Ascidia, Phallusia* and *Ciona* may be related to branchial structures. All 3 genera possess papillae above the longitudinal bars making the internal branchial surface rugose rather than smooth. Gotto assumes that this could be related to "the biomechanism of feeding in species of *Notopterophorus*" (p. 40), thus *Notopterophorus* species are absent in *Ascidiella* which have no papillae. However, *N. elongatus* is known to occur in *Ascidiella* sp. and *Clavelina lepadiformis* and this diminishes the value of Gotto's argument.

'Creeping genera', i.e., those with more reduced mobility, and with a furca no longer natatory — *Doropygus* (Fig. 6-16c), *Doropygella,* and females of *Pachypygus* (Fig. 6-16d) — live more often in stolidobranchs with branchial folds, but also in phlebobranchs. Inside the stolidobranchs they are always found at the bottom of the folds or inside the crease between the branchial basket and peripharyngeal grove. The 'creeping genera' with aliform expansions — such as *Notopterophorus* (Fig. 6-16e), and *Doropygella* — exclusively inhabit phlebobranchs and live in *Clavelina* species.

Several small-sized genera, with restricted mobility – *Demoixys* (Fig. 6-17b), *Doroixys* (Fig. 6-17a), *Mesoixys* (Fig. 6-17c), and *Ooneides* (Fig. 6-17h) — live in the small branchial cavity of aplousobranchs and species of the small colonial phlebobranch genera *Diazona* and *Perophora*. Species of *Botachus* (Fig. 6-16f), and *Lonchidiopsis* (Fig. 6-16g, h), as well as 2 monospecific genera — *Microra* (Fig. 6-16j) and *Remex* (Fig. 6-16k) — are only known from phlebobranchs. The adult (in *Lonchidiopsis* males and juveniles) lives between the branchial epithelium and the web of the blood sinuses characteristic of the order.

'Creeping' copepods with a brood sac occupying 3 thoracic segments comprise *Bonnierilla* (Fig. 6-16i) and 3 allied genera defined by Stock (1967b) — *Periproctia, Sesir,* and *Thoracodelphys* — comprise species able to live in members of the 3 ascidian orders; however, *Bonnierilla arcuata* was found only in Didemnidae.

Table 6-6
Distribution of ascidicolous copepod genera among ascidian families (Original)

Copepod	Species number	Ascidian families										Special genus
		Polycitoridae	Didemnidae	Polyclinidae	Cionidae	Perophoridae	Corellidae	Ascidiidae	Styelidae	Pyuridae	Molgulidae	
Notodelphyidae												
Achelidelphys	5		7									*Didemnum*
Agnathaner	2	1							2			
Anoplodelphys	1		1									
Apodelphys	1							1				
Bonnierilla	9	1	6		1			4	4	6	1	
Botachus	1				1			4				
Brementia	2		4									
Campopera	1										1	
Cephalodelphys	1		1									
Cochlodelphys	1		1									
Demoixys	5	1	3	1								
Doroixys	3	15	1	3	1				*			* *Botryllus*
Doropygella	7						1	6	1	2		
Doropygopsis	2					1		5	1	4	1	
Doropygus	29	1			2			8	25	25	8	
Dysgenopsyllus	1					3	1					Unknown
Goniodelphys	2				1			1				
Gunenotophorus	2							4	8	2	1	
Haplostatus	1			1								
Kystodelphys	1									1		
Lobodelphys	1									1		
Lonchidiopsis	3											
Mesoixys	1	1				1	3					
Microra	1							1				
Notodelphyopsis	2				1							
Notodelphys	31		?	1	11		3	41	4	6	5	
Notopterophoroides	2					3		3				
Notopterophorus	6			1	5			14				
Ooneides	1		3									
Ophiodelphys	1											Unknown
Ophioseides	3		1						5	1		
(Scolecimorpha)	2								2	2		= ? *Ophioseides*
Pachypygus	5			1	3			8	7	6	3	
Paranotodelphys	9							4	1	2		
Periproctia	2		1			1			1			
Pholeterides	1	1										
Pomphopygus	1							1				
Prophioseides	6	2	3					1				
(Sphaerothylacus)	1								1			= ? *Prophioseides*
Pygodelphys	5							1	4	5	4	
Pythodelphys	2	1		1		1						
Remex	1							1				

Table 6-6 (continued)

Copepod	Species number	Polycitoridae	Didemnidae	Polyclinidae	Cionidae	Perophoridae	Corellidae	Ascidiidae	Styelidae	Pyuridae	Molgulidae	Special genus
	Ascidian families											
Scolecodes	1								3	2		
gen. aff.	1									1		
Sesir	1								1			
Sicyodelphys	1		1									
Syndelphys	1		1									
Thoracodelphys	1		1						1			
Ustina	1											Unknown
Archinotodelphyidae												
Archinotodelphys	3					1		1	1	2		
Pararchynotodelphys	2						1	1				
Ascidicolidae												
Ascidicola	1	1			2	3		8	4	9	1	
Botryllophilus	9	2	1	1					7		1	
Buprorus	3	2			1			3	1			
Enterocola	17	20	5	6					2			
Enterocolides	1		1	1								
Enteropsis	9				1			2	7	5	1	
Haplosaccus	2	3	3									
Haplostoma	12	12	3	3	1				1			
Haplostomella	10	6	3						1	1		
Haplostomides	6	6		1								
Lequerea	2								2			
Mycophilus	2								3			*Botryllus*
Paulillgia	1								1			
Schizoproctus	2							3		1	1	
Styelicola	2				1				2	1		
Lichomolgidae												
Ascidioxynus	2						1	1				
Debruma	1			1								
Henicoxiphium	1								2			
Hermannella	1							1				
Heteranthesius	1									1		
Lichomolgides	1		1									
Lichomolgidium	2								1	2		
Lichomolgus	7			1	7	3	1	10	3		5	
Macrochiron	1		2									
Zygomolgus	3	2	2									
Family ?												
Capistrum	1											*Gasterascidia lyra*
Gonophysema	1							1	1	1		

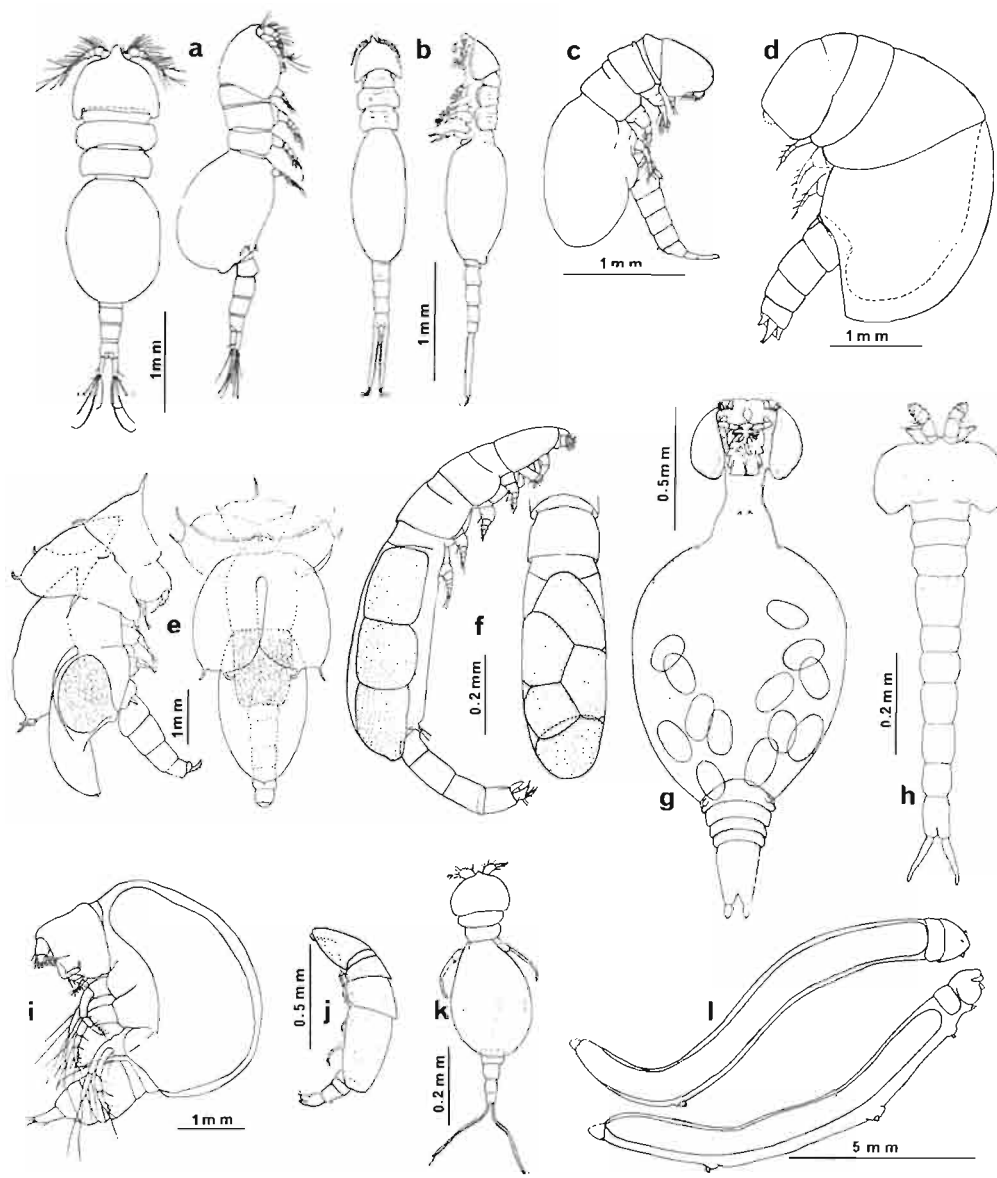

Fig. 6-16: Notodelphyid copepods. (a) *Notodelphys acanthomela* (after Illg and Dudley, 1961); (b) *Paranotodelphys villosus* (after Ooishi, 1963b); (c) *Doropygus pulex* (after Illg and Dudley, 1961); (d) *Pachypygus gibber*; (e) *Notopterophorus elongatus*; (f) *Botachus cylindratus* (after Illg and Dudley, 1965); (g) *Lonchidiopsis setosus* female (after Jones and Montez-Moreno, 1981); (h) *Lonchidiopsis hartmeyeri* male (after Ooishi and Illg, 1986); (i) *Microra angulata;* (j) *Remex obesus* (after Monniot, 1983); (k) *Bonnierilla armata*; (l) *Ophioseides cardiacephalus*. (After Illg and Dudley, 1961.)

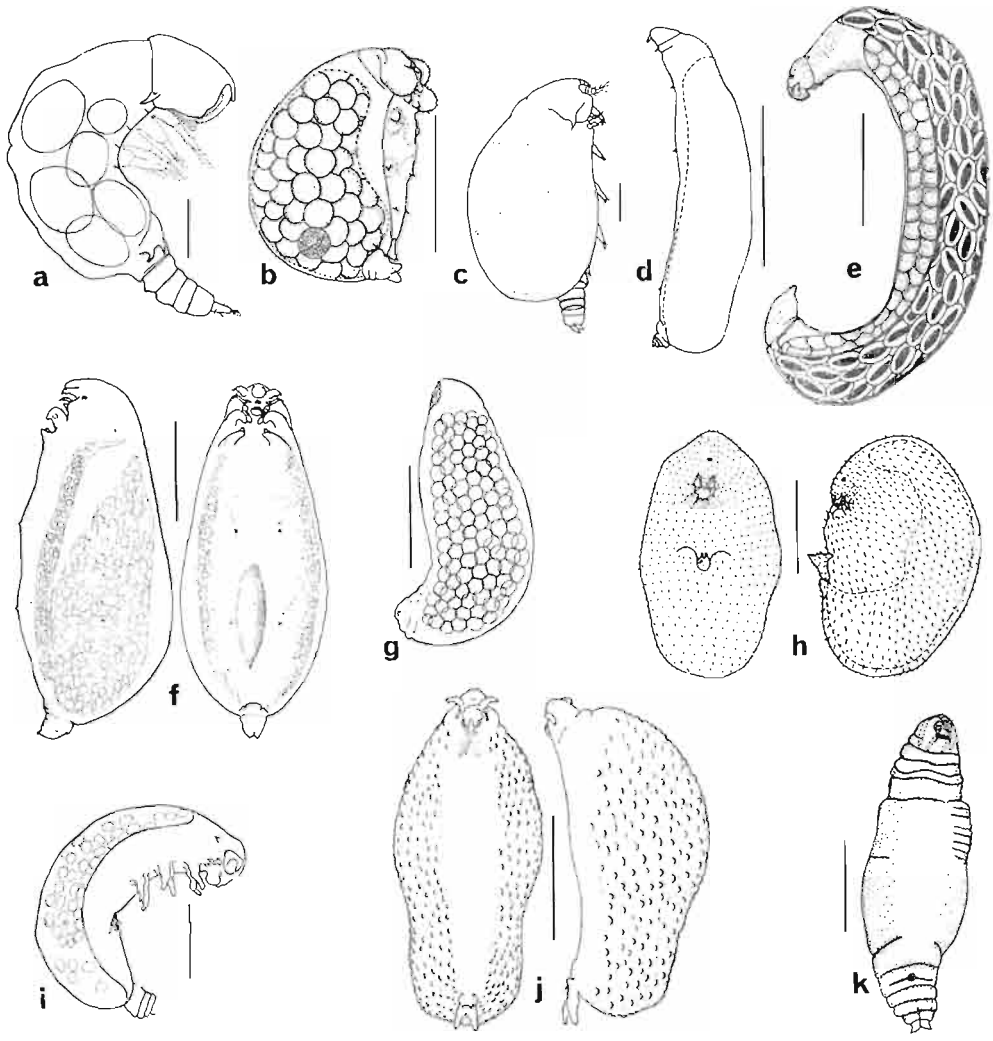

Fig. 6-17: Notodelphyid copepods from compound ascidians. (a) *Doroixys minuta* (after Stock, 1970); (b) *Demoixys chattoni* (after Illg and Dudley, 1961); (c) *Mesoixys otaria* (after Illg and Dudley, 1965); (d) *Prophioseides abdominalis* (after Dudley and Solomon, 1966); (e) *Sicyodelphys bocqueti* (after Lafargue and Laubier, 1968b); (f) *Ophiodelphys illgi* (after Bocquet and Stock, 1961); (h) *Ooneides amela* (after Illg and Dudley, 1961); (i) *Pholeterides furtiva* (after Illg, 1958); (j) *Cochlodelphys delamarei* (after Lafargue and Laubier, 1968a); (k) *Haplostatus incubatrix* (after Illg and Dudley, 1961). Scale bars = 1 mm, except for a and d = 0.1 mm

A series of copepod genera ('ophioseimorphes', Bocquet and Stock, 1961) acquired a worm shape and do not live inside branchial cavities but in the tunic. *Ophioseides* (Fig. 6-16l) digs galleries into the tunic of Pyuridae, Styelidae, Didemnidae and Polyclinidae. *Prophioseides* lives in Didemnidae, and among the Polyclinidae, one species digs into the tunic of *Phallusia nigra*, and another lives in cysts made into the branchial tissue of *Polycarpa cryptocarpa*. Members of the genus *Haplostatus* (Fig. 6-17k) live in *Cystodytes* (Polycitoridae), of *Pholeterides* (Fig. 6-17i) in Polyclinidae. A series of genera (Fig. 6-17,

18), morphologically more and more regressed (Lafargue and Laubier, 1977), have adapted to life in Didemnidae (Table 6-7). The 3 last genera listed in the table, with distributions limited to Asian coasts ranging from the Red Sea to Singapore (Lafargue and Laubier, 1978a, b) and south India (own obs.), are star-shaped and live associated with Didemnidae (cloacal cavity). Lafargue and Laubier (1977) hypothesized co-evolution of copepods and Didemnidae.

Encysted copepods

Five cases of Notodelphyidae living inside ascidian-made cysts are known, but only 2 have been studied. The better known case involves *Scolecodes hunstmani* (Henderson,

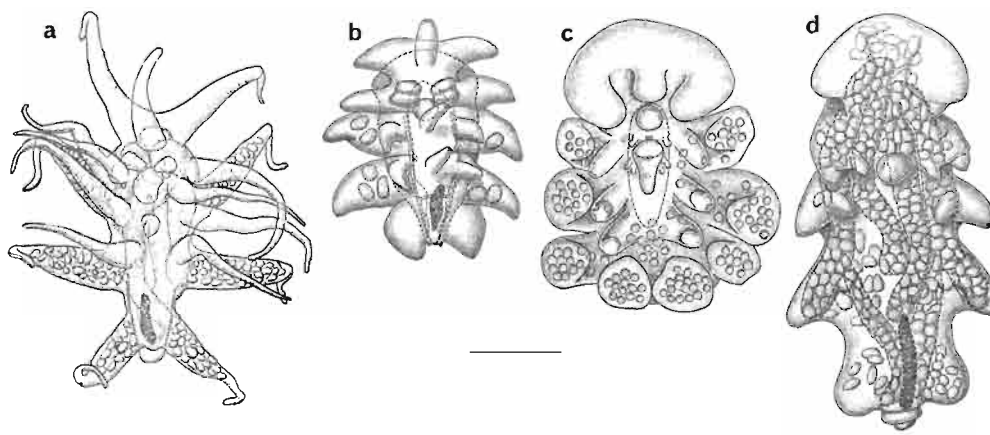

Fig. 6-18: Notodelphyid copepods from cloacal cavities of Didemnidae. (a) *Achelidelphys drachi* (after Lafargue and Laubier, 1978a); (b) *Achelidelphys steinitzi;* (c) *Cephalodelphys stellata;* (d) *Syndelphys reducta* (after Lafargue and Laubier, 1977). Scale bar = 0.5 mm

Table 6-7
Notodelphyidae living in colonial ascidians; classified according to regressive adaptation levels (After Lafargue and Laubier, 1977; modified)

Copepoda	Didemnidae	Polyclinidae	Polycitoridae	Other families
Demoixys	x	x	x	
Mesoixys		x		
Prophioseides	x	x		Ascidiidae Styelidae
Pythodelphys		x	x	
Brementia balneolensis	x			
Sicyodelphys	x			
Ophiodelphys				Host unknown
Ooneides	x			
Pholeterides		x		
Brementia illgi	x			
Anoplodelphys	x			
Cochlodelphys	x			
Haplostatus			x	
Achelidelphys	x			
Cephalodelphys	x			
Syndelphys	x			

1931) studied by Dudley (1968). The copepod lives in the blood sinus (under the endostyle) of 4 ascidian species in British Colombia (Canada), and in 1 species in Japanese waters (Illg, 1970). The copepod is embedded in a cyst compound of ascidian cells, hanging inside the subendostylar sinus (Fig. 6-19a). The cyst of the female opens into the cloacal cavity by a ciliated funnel (Fig. 6-19b) probably facilitating male entrance and expulsion of the young stages.

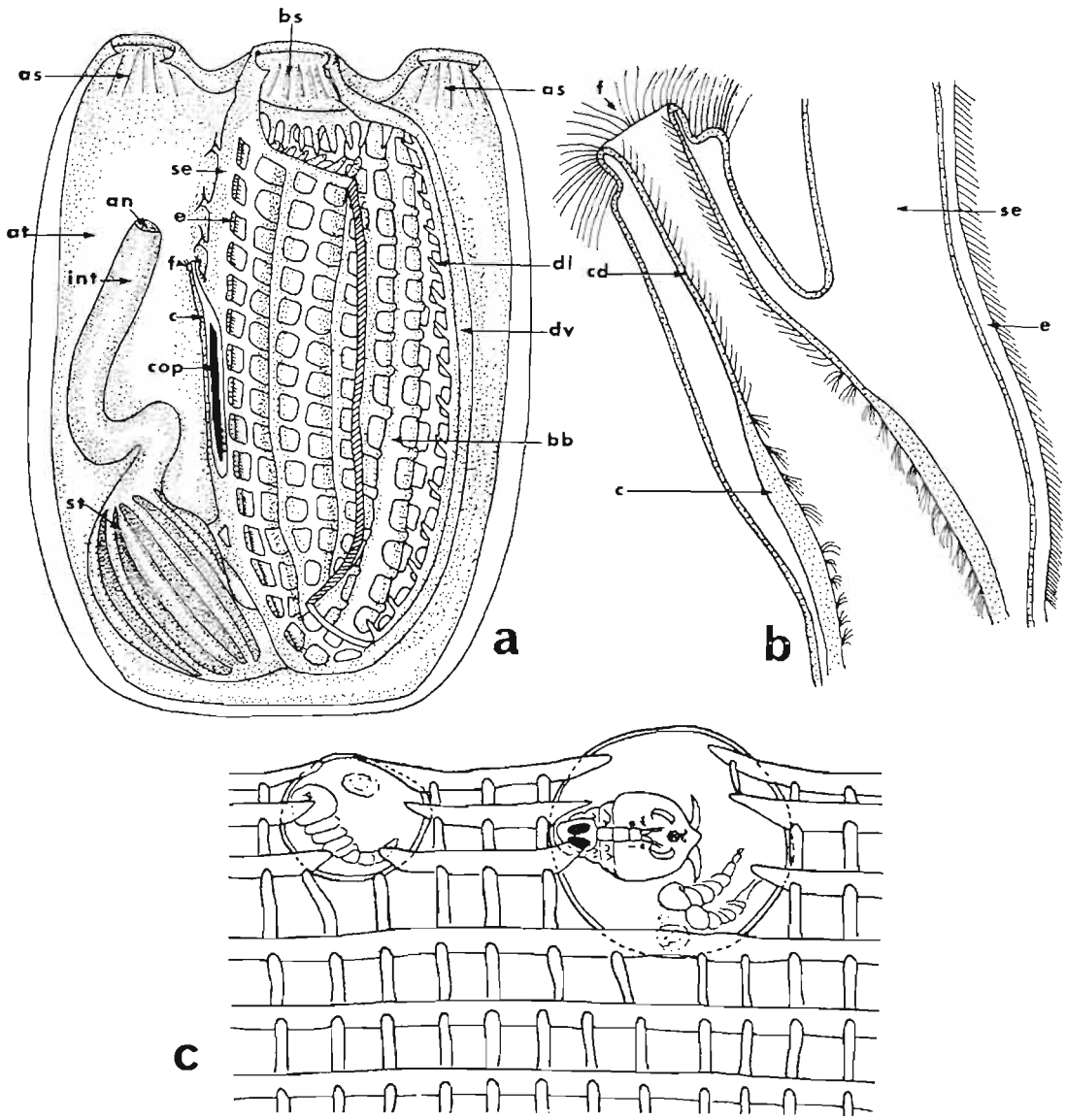

Fig. 6–19: Copepods in cysts. a, b: Diagram showing copepod female *in situ* in the ascidian; (cop) *Scolecodes huntsmani;* (c) cyst; (sc) subendostylar blood vessel; (cd) ciliated duct; (f) funnel. (After Dudley, 1968). c: *Kystodelphys drachi* inside branchial bars of *Microcosmus savignyi.* (After Monniot, 1963.)

Both sexes develop in the ascidian. The mode of penetration of Copepodite 2 into the subendostylar sinus is not known; it is surrounded by cells which, according to Dudley (1968), are 'totipotent lymphocytes' of the host, and form a cyst of 0.14 mm in diameter where the young develops until Copepodite 5. If a male, the copepod probably leaves the host, because when experimentally liberated in sea water it actively swims, while a similarly treated young female is unable to do so and dies. The female's cyst lengthens and reaches the cloacal cavity, thus establishing communication with the outside. The cells composing the cyst are not attached to a basal lamina; they are columnar epithelial cells, held together by complex interdigitations at their basal ends. They are ciliated (Fig. 6-20c). Inside the cyst, female exuviae are present and sometimes dead males surrounded by phagocytes and globular cells serving as food for the copepods. Dudley (1968) assumes that these cells originate from the cyst wall and do not represent ascidian blood cells passed through the cyst wall via diapedism. Species of *Scolecodes* exhibit adaptations unique among the Notodelphyidae with all leg setae transformed into short paddles.

Members of a genus closely allied to *Scolecodes,* but with more reduced mouth pieces,

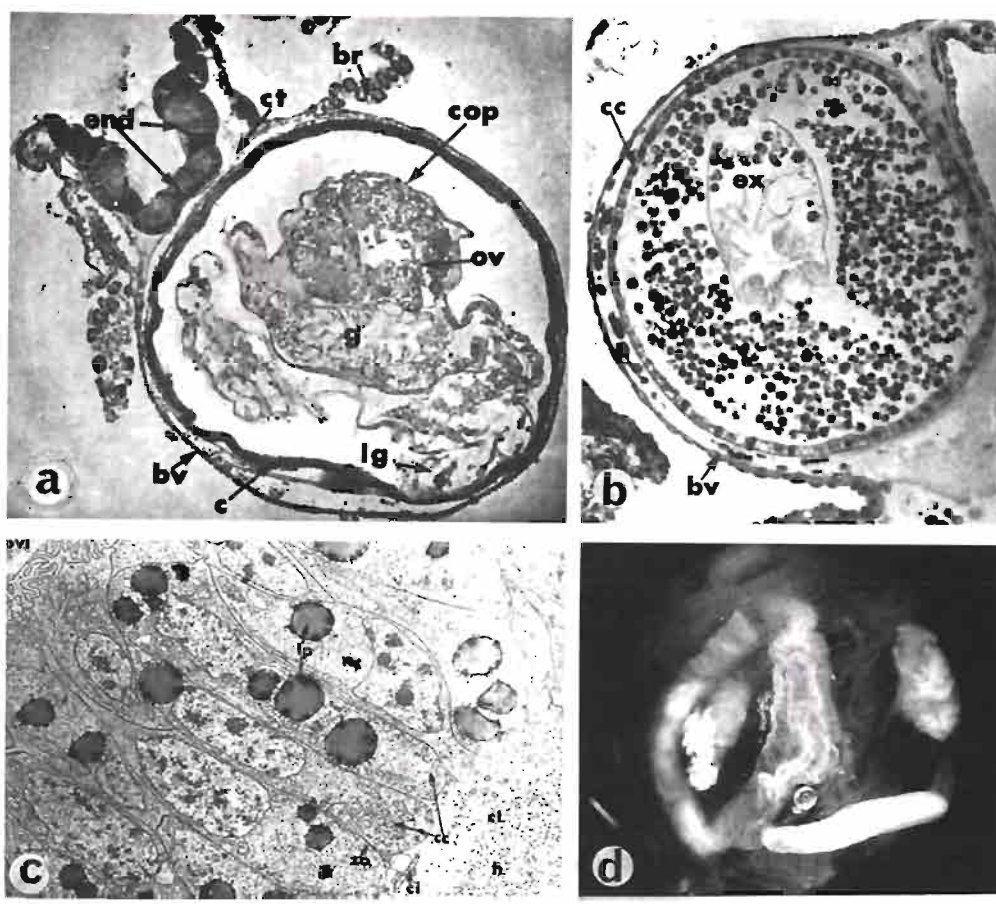

Fig. 6–20: Copepods in cysts. a: *Scolecodes hunstmani* inside subendostylar blood vessel; b: male exuvium; c: cells of cyst. (After Dudley, 1968). d: *Boltenia hirta* with, in the heart, a copepod allied with *Scolecodes*. (After Monniot and Monniot, 1977.)

have been found in the heart of the abyssal ascidian *Boltenia hirta* in the South Indian Ocean (Monniot and Monniot, 1977) (Fig. 6-20d).

Kystodelphys drachi Monniot, 1963 produces a cyst in the branchial bars of *Microcosmus savignyi* (Fig. 6-19c). The male is atypic and the female is unknown. In this case, too, the cyst is made of ascidian cells but these are less high than those decribed by Dudley (1968). We have re-examined this material but no cyst-opening was found.

Two other Notodelphyidae have females living in cysts made in branchial bars: (1) *Sphaerothylacus polycarpae* Sluiter, 1884, described as a cirriped but apparently belonging to the genus *Prophioseides* (Ooishi, pers. comm.), and (2) an unidentified species living in an Antarctic *Molgula pedunculata* (own obs.). The cyst represents an ascidian reaction to the copepod presence in its blood system. As the copepods belong to several genera, there is not co-evolution in this case.

Among the family Ascidicolidae links may be established between different genera and their localization in the host. Genera inhabiting the branchial sac — such as *Botryllophilus* (Fig. 6-21a), *Buprorus* (Fig. 6-21b), *Enteropsis* (Fig. 6-21c), *Schizoproctus*, and *Styelicola* (Fig. 6-21d) — are generally present in all 3 ascidian orders, but more frequently in phlebobranchs and stolidobranchs. Only 1 genus, *Mychophilus* (Fig. 6-21h), is exclusively known from *Botryllus* species.

Genera linked to the digestive tract — *Enterocola* (Fig. 6-21e), *Enterocolides*, *Haplostoma* (Fig. 6-21f), *Haplosaccus* (Fig. 6-21g), *Haplostomella*, and *Haplostomides* — are found more often in members of the 3 aplousobranchs families, but with a preference for Polyclinidae and Polycitoridae. Some very large-size species live in *Ciona intestinalis* or a Stolidobranch. Ascidicolidae sometimes obstruct the gut thus preventing zooid feeding. The position of the copepod in the host gut has been studied by Brément (1911) (Fig. 6-22). Infested zooids generally survive and develop gonads, though Brément noted testis degeneration in zooids of *Diplosoma listerianum* parasitized by *Enterocola pterophora*, this beeing due to the colonial common blood system. Very often, when the old zooid degenerates, the copepod remains alive in the common test.

The genus *Ascidicola* comprises only 1 species *A. rosea*, with a wide distribution — Europa, North America, Antarctic — inhabiting most large phlebobranchs and stolidobranchs. It was found once in an *Aplidium*. *A. rosea* lives on the alimentary cord of the ascidian. Gotto (1957) described detail behaviour and morphological adaptations of the copepod (Fig. 6-23). *A. rosea* possesses very long spiny setae on its leg endopodites used for anchoring and creeping along the mucus cord, and a spinous pad on the penultimate segment allowing to make a U turn in the oesophagus. *A. rosea* enters the oesophagus head first and then turns back to accomodate itself on the mucus cord, creeping on it as it sinks into the gut.

The large family Lichomolgidae has been subdivided into 5 groups by Humes and Stock (1973). The ascidicolous species belong to 2 of these new families: Sabelliphilidae with 2 genera living exclusively inside ascidians: *Henicoxiphidium* and *Lichomolgidium*, and Lichomolgidae *sensu stricto* in which 3 genera live exclusively in ascidians: *Ascidioxynus*, *Debruma*, and *Lichomolgides*, and 3 genera in several invertebrates — *Lichomolgus* inside molluscs and ascidians, *Macrochiron* associated with algae, hydrozoans, echinids, and ascidians, and *Zygomolgus* living on algae, and inside holoturians and ascidians. All lichomolgides inhabit ascidian cloacal cavities, swim actively and may leave their host. None of these species shows any morphological adaptations to its ascidian host.

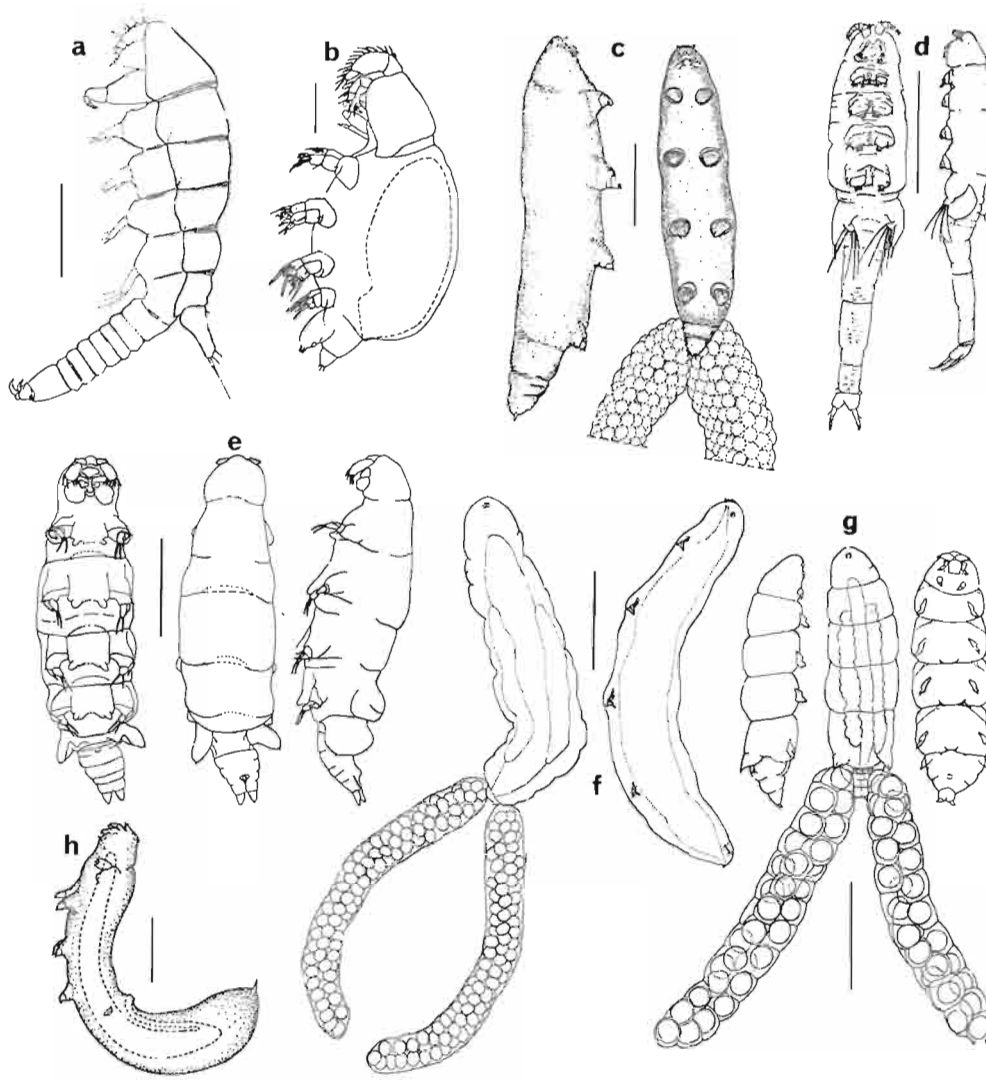

Fig. 6-21: Copepods Ascidicolidae. (a) *Botryllophilus randalli* (after Stock, 1970); (b) *Buprorus caudatus;* (c) *Enteropsis roscoffensis; Styelicola lighti; Enterocola fertilis* (after Illg and Dudley, 1980); (f) *Haplostoma minutum; (g) Haplosaccus elongatus* (after Ooishi and Illg, 1977); (h) *Mychophilus roseus* (after Illg and Dudley, 1980). Scale bars = 0.1 mm (b); 0.25 mm (a, e, h); 0.5 mm (g); 1 mm (c, f, d)

Gonophysema gullmarensis

Gonophysema gullmarensis was described in Sweden from *Ascidiella aspersa* as a very modified copepod impossible to place in a given family (Bresciani and Lützen, 1960, 1961). The copepod is partially included in the host tissues, its ovisacs hanging into the cloacal cavity (Fig. 6-24a). It is totally devoid of a digestive tract and a large gonad occupies its whole body. Up to 7 males, extremely reduced, live in the seminal vesicle of a female. Bresciani and Lützen (1961) described the development (Fig. 6-25) with a naupliar stage

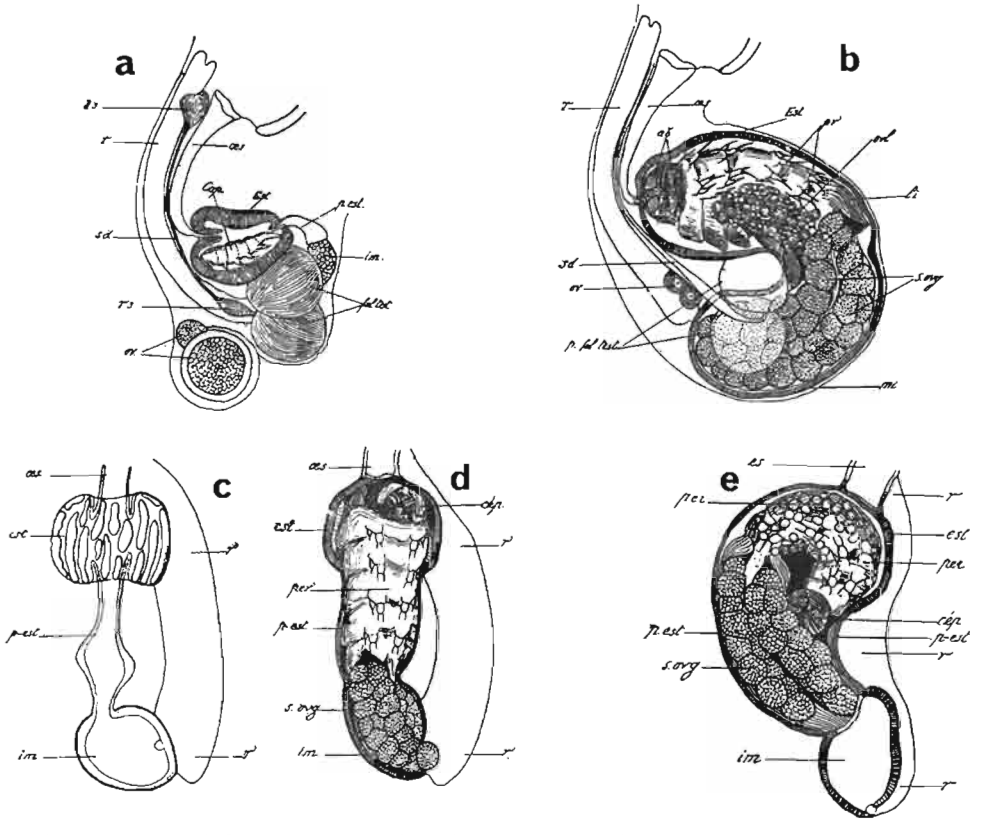

Fig. 6-22: Position of *Enterocola pterophora* in the duct. a: Young specimen in didemnid gut. b: Adult. c: Non-infested gut of a Polyclinidae. d and e: Two copepod positions inside stomach. (After Brément, 1911.)

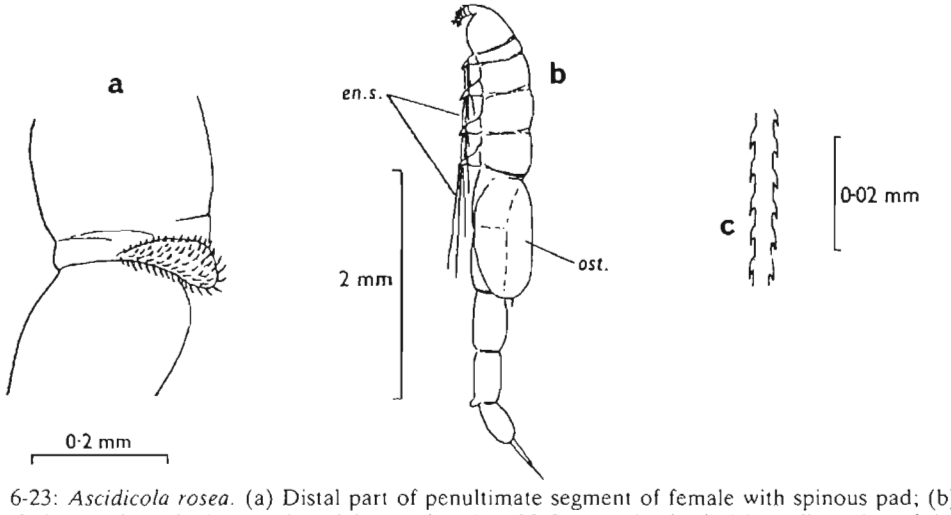

Fig. 6-23: *Ascidicola rosea*. (a) Distal part of penultimate segment of female with spinous pad; (b) lateral view to show the long endopodal setae (en.s) and left oostegite (ost); (c) small portion of the endopodal seta. (After Gotto, 1957.)

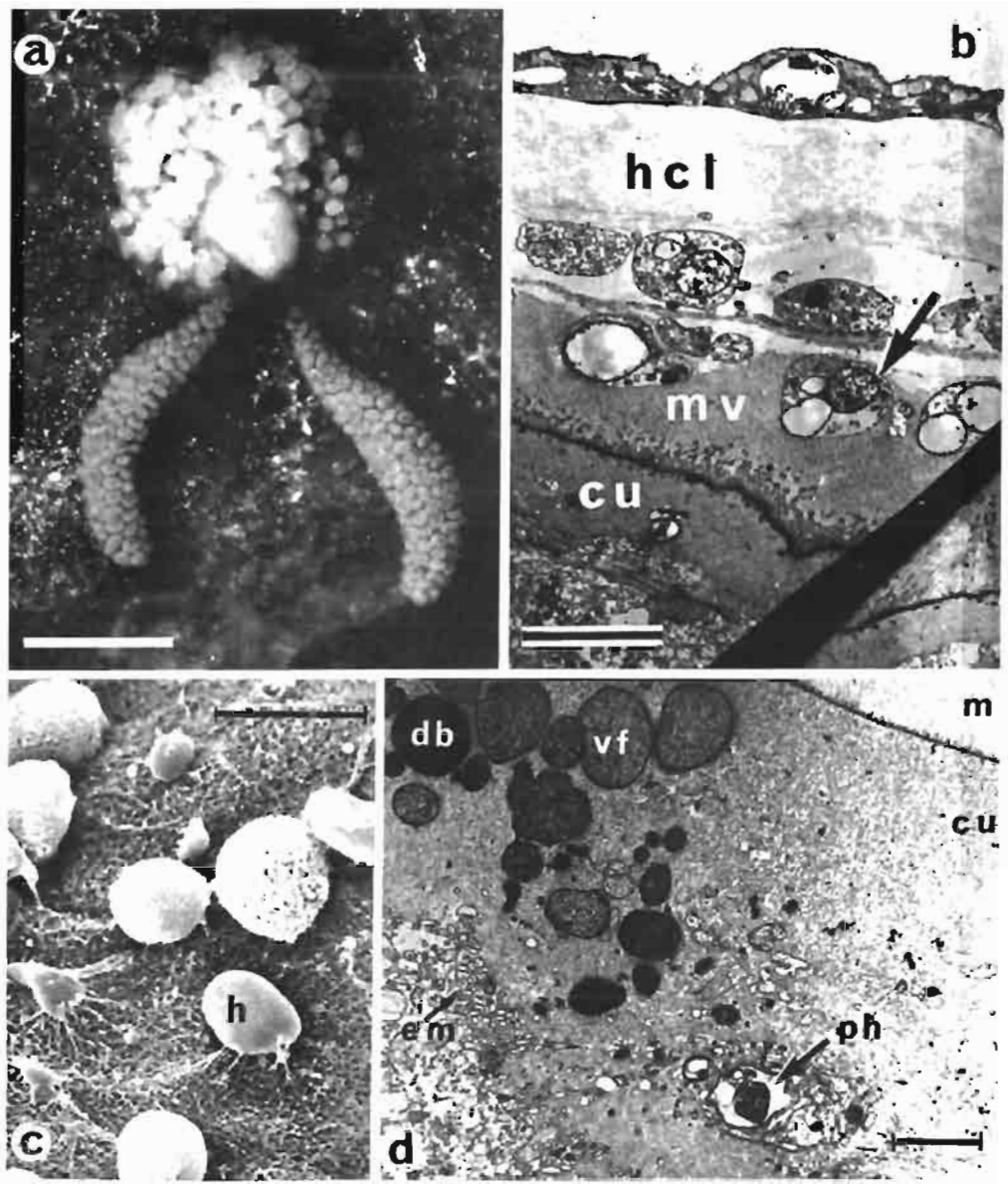

Fig. 6-24: *Gonophysema gullmarensis*. a: Female *in situ* in the wall of the *Ascidiella aspersa* peribranchial cavity. b: TEM micrograph, transverse section through host tissue and parasite integument; (hcl) host tissue; (mv) microvillosities; (cu) cuticule; note blood cells from host trapped by villosities (arrow); scale bar = μm. c: SEM micrograph of copepod surface free from host tissue; note host cells about to be trapped by microvillosities (h); scale bar = 10 μm. d: TEM micrograph, transverse section of cuticule; (em) apical portions of epithelial cells; (mv) microvillosities; (cu) cuticule; (db) dense bodies; (vf) vacuoles with fibrillar contents; (ph) phagosome; scale bar = 2 μm. (After Bresciani, 1986.)

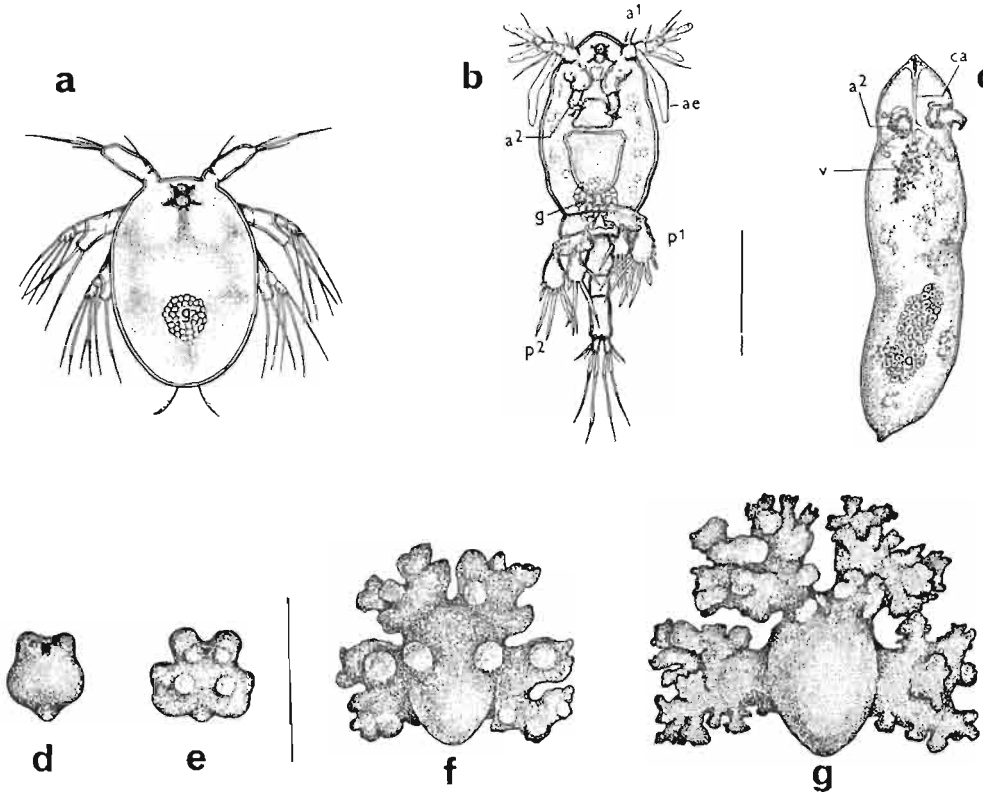

Fig. 6-25: *Gonophysema gullmarensis* development. (a) Newly hatched nauplius; (b) copepodite; (c) onychopodite; (d to g) successive stages viewed from the dorsal side. (After Bresciani and Lützen, 1961.)

and a normal copepodite with setae. The last stage turns into an 'onychopodite' infesting stage, entering ascidian tissues. It is carried by the blood before settling and turning into an adult. According to Bresciani and Lützen, sex determination may be environmentally induced: the onychopodite develops into a female when fixed to the ascidian, but near a differentiated female an onychopodite must penetrate into the seminal vesicle and then develop into a male. Multiple infestations are common. Bresciani and Lützen (1960) studied localization of the parasite in the ascidian considering 160 copepods inhabiting 50 ascidians.

Without a gut, *Gonophysema gullmarensis* must take up food through its cuticle. Bresciani and Lützen (1960) describe giant-cell complexes (Fig. 6-26) regularly distributed over the epiderm. They never saw "a or b cells penetrate all the 3 layers of the chitin" (p. 364) but they conclude that the whole organization indicates a passage of substances through the cell complex. However, they could not decide on the passage direction, i.e., whether absorption or excretion, is involved, even though the histological structures point to excretion.

Bresciani (1986), studying the fine structure of the *Gonophysema gullmarensis* integument, found a thin cuticule (5 to 10 μm) covered by a dense layer of microvilli (20 to 25 μm) which trap host blood cells (Fig. 6-24b, c). The cuticule includes also some

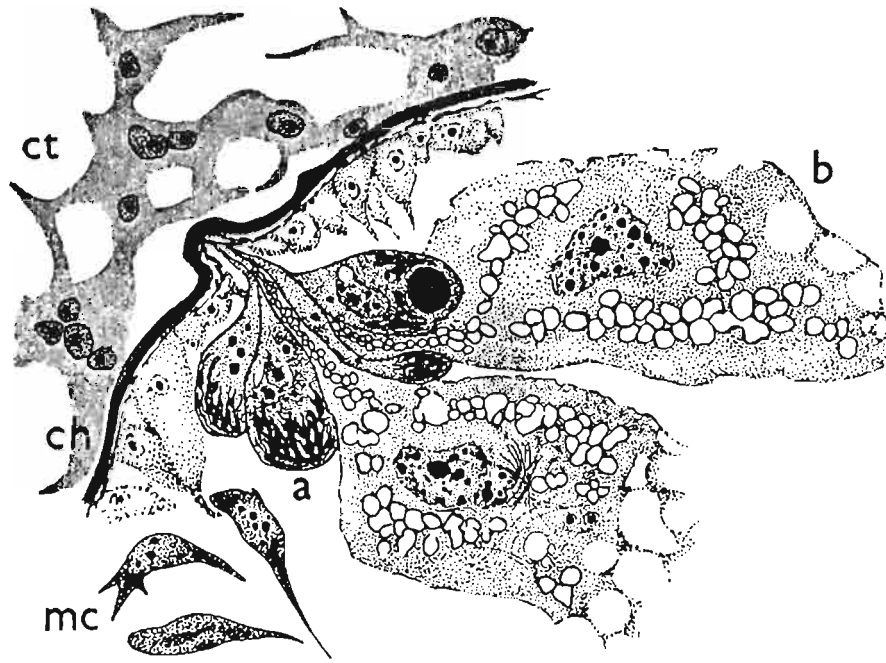

Fig. 6-26: *Gonophysema gullmarensis,* giant-cell complex; (a) a-cells; (b) b-cells; (mc) mesenchym-cells; (ch) vacuolized layer of chitin. (After Bresciani and Lützen, 1960.)

phagosome-like structures and dense bodies (Fig. 6-22d) whose role is uncertain. Phagosome-like structures are known also from other parasitic copepods, e.g., in Lamippidae, but the dense bodies were present only in *G. gullmarensis* (Bresciani, 1986). How *G. gullmarensis* obtains food remains unknown.

Gonophysema gullmarensis has been recorded by Bresciani and co-authors (1970) also in *Distomus variolosus* from the Mediterranean Sea and in *Heterostigma reptans* from Norway. The reviewer found *G. gullmarensis* in its type station in Sweden in *Ascidia obliqua,* and a *Gonophysema* sp. in an *Ascidia* sp. on the coast of South Brittany (France).

Copepods like *Gonophysema* live in the abyssal ascidians *Bathystyeloides enderbyanus* and *Minipera papillosa*, but they have not been described. Near the Kerguelen Islands too, several Polyclinidae species harbour a copepod similar to *Gonophysema* but with dwarf males fastened on the outside of the female.

Capistrum sorberae (Fig. 6-27), described by Monniot (1985) in the abyssal Sorberacea *Gasterascidia lyra,* is the most simplified copepod known. The female, devoid of a digestive tract, lives in the host's esophageal mesenchyme, accompanied by several males (up to 5). The ovisacs are located in the host's cloacal cavity. The female gonad only comprises 2 ovaries and a unique cementary gland. There is no seminal vesicle, and fertilization probably takes place outside.

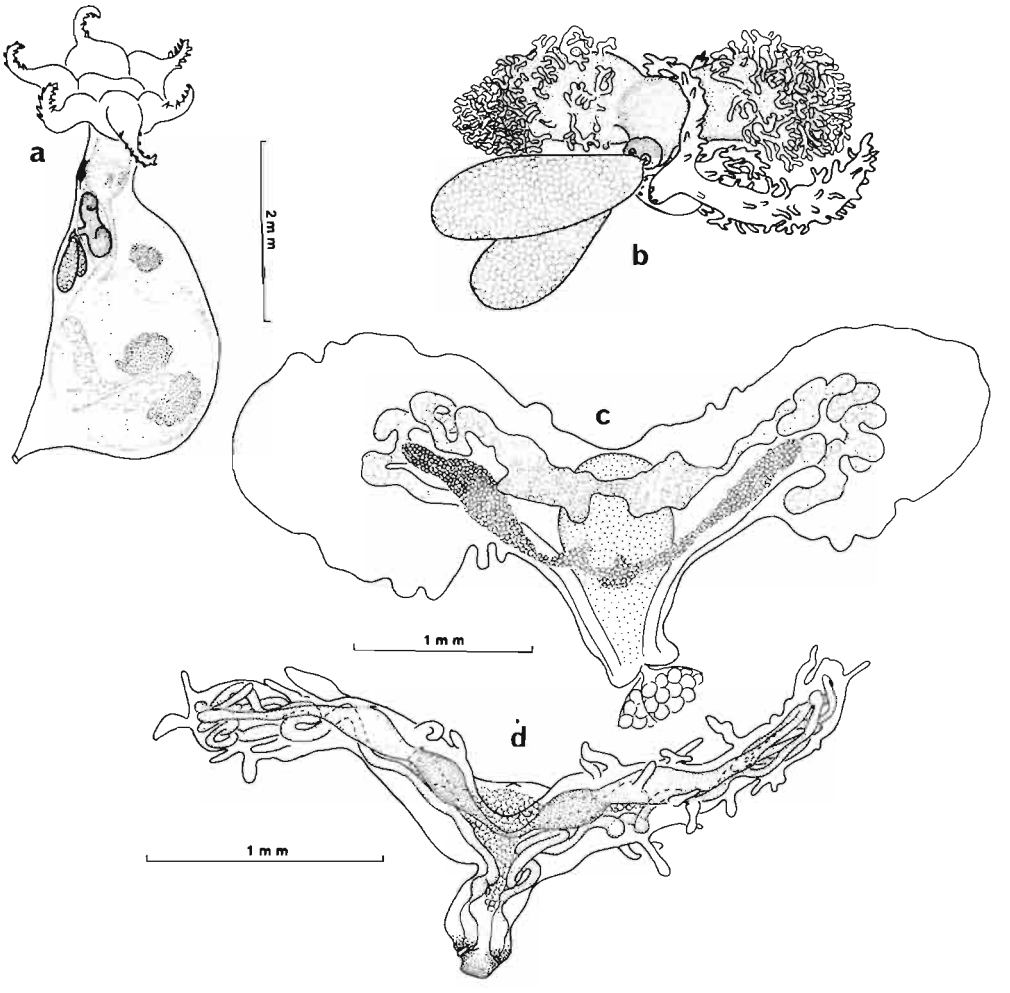

Fig. 6-27: *Capistrum sorberae.* (a) *In-situ* copepod in *Gasterascidia lyra;* (b) female accompanied with 2 males (3 males which were on the right side have been removed); (c) female anatomy; (d) male. (After Monniot, 1985.)

Host Influence on Copepods

Influence on sex determination

According to the age of the host *Ciona intestinalis,* the Copepodite 2, infesting stage of *Pachypygus gibber,* develops into a female, a normal male, or an atypical male (Hipeau-Jacquotte, 1978a, b, 1980a, b). When the Copepodite 2 penetrates a very young *Ciona intestinalis* (Stage 6 protostigmata), it transforms into an atypical male; beyond the 6 protostigmata stage it transforms into a female; if other copepodites enter a *C. intestinalis* already harbouring a female, they develop into typical males Hipeau-Jacquotte (1982) and Heussner (1983). In old *C. intestinalis* several parasite females can be present together. Transformation into an atypical male can be provoked *in vitro* in the presence of squashed young *C. intestinalis.* Accordingly, Hipeau-Jacquotte (1982) suggests the exist-

ence of a 'young host factor'. Transformation into an atypical male also occurs in young *Ascidiella aspersa* and *Clavelina lepadiformis*.

The atypical male of *Pachypygus gibber* was found in *Polyclinum aurantium* by Canu (1891a, 1892) and described under the name *Agnathaner minutus*. Synonymy was established by Hipeau-Jacquotte (1980b). Atypical males of other species have been described but the females remain unknown: *Agnathaner typicus* in *Dendrodoa grossularia* described by Canu (1891a) at Wimereux in France and in Norway in *Styela rustica* (Sars, 1921); *A. freemani* (Hamond, 1968) in a British unidentified ascidian, and *Kystodelphys drachi* producing cysts in the branchial sinus of *Microcosmus savignyi* in the Mediterranean Sea (Monniot, 1963).

The American species *Pachypygus macer* also possesses atypical males found in Bermuda in *Perophora viridis* and *Symplegma brakenhielmi* (own obs.). Dudley (1966) found 2 types of males in *Doropygus seclusus*. Ooishi and Illg (1977) noted the presence of 2 types of males, swimming and creeping, in several species of *Haplostoma*. Transformation into an atypical male can be induced experimentally in young ascidians. In natural conditions the phenomenon occurs in members of the 3 orders and in most ascidian families in adults of small-sized species (Monniot, 1986). He suggested that the hematophage diet of atypical males, noted by Hipeau-Jacquotte (1982, 1984) and Heussner (1983) and also relevant for *Kystodelphys drachi*, might play a role in atypical male transformation.

Among the morphological characteristics of atypical males, must be mentioned the pronounced development of the sensory system (Hipeau-Jacquotte, 1986, 1987) and the formation of a cephalic organ composed of numerous ciliated holes (Fig. 6-28, 6-29). The same kind of structure has been described by Gotto and co-authors (1984) in the male of *Mychophilus roseus* (Family Ascidicolidae). While the *Mychophilus roseus* female lives in *Botryllus schlosseri* zooids, the male is free-living, and was caught during light-fishing. Gotto and authors assume that the peculiar sensory equipment plays an important role in the searching for the female. The sensory equipment of the atypical male may allow it to leave its small-sized host in which it has developed and where a female cannot live, in order to search for a partner (Hipeau-Jacquotte, 1986). Hipeau-Jacquotte (1984) postulates that the phenomenon may be more general, and "the development of atypic males would not come to an evolutionary deadlock" (p. 63).

Males of other copepods, such as *Ascidicola rosea,* were described as free-living copepods (Sars, 1921; Monniot, 1965a). In many species of ascidicolous copepods males are unknown, and it is tempting to suppose that they are free-living. Sex determination could be linked to substrate effects. The infesting stage would become a female or a typical male; specimens not finding a host would become an atypical or a typical male depending on the parasite species (reviewer's own opinion).

Host influence on copepod females (parasite specificity)

Most parasite species living in large ascidians are not host specific (Thorell, 1859a, b). He separated the species from each other by ratios of different parts of their body and by their setation. Later authors, with less precision, have identified Thorell's species in various ascidians everywhere in European waters. Illg (1958) and Illg and Dudley (1961) did not completely succeed in resolving the taxonomic problems concerning *Notodelphys* genera in Europe; they determined the species by their body proportions and arrange-

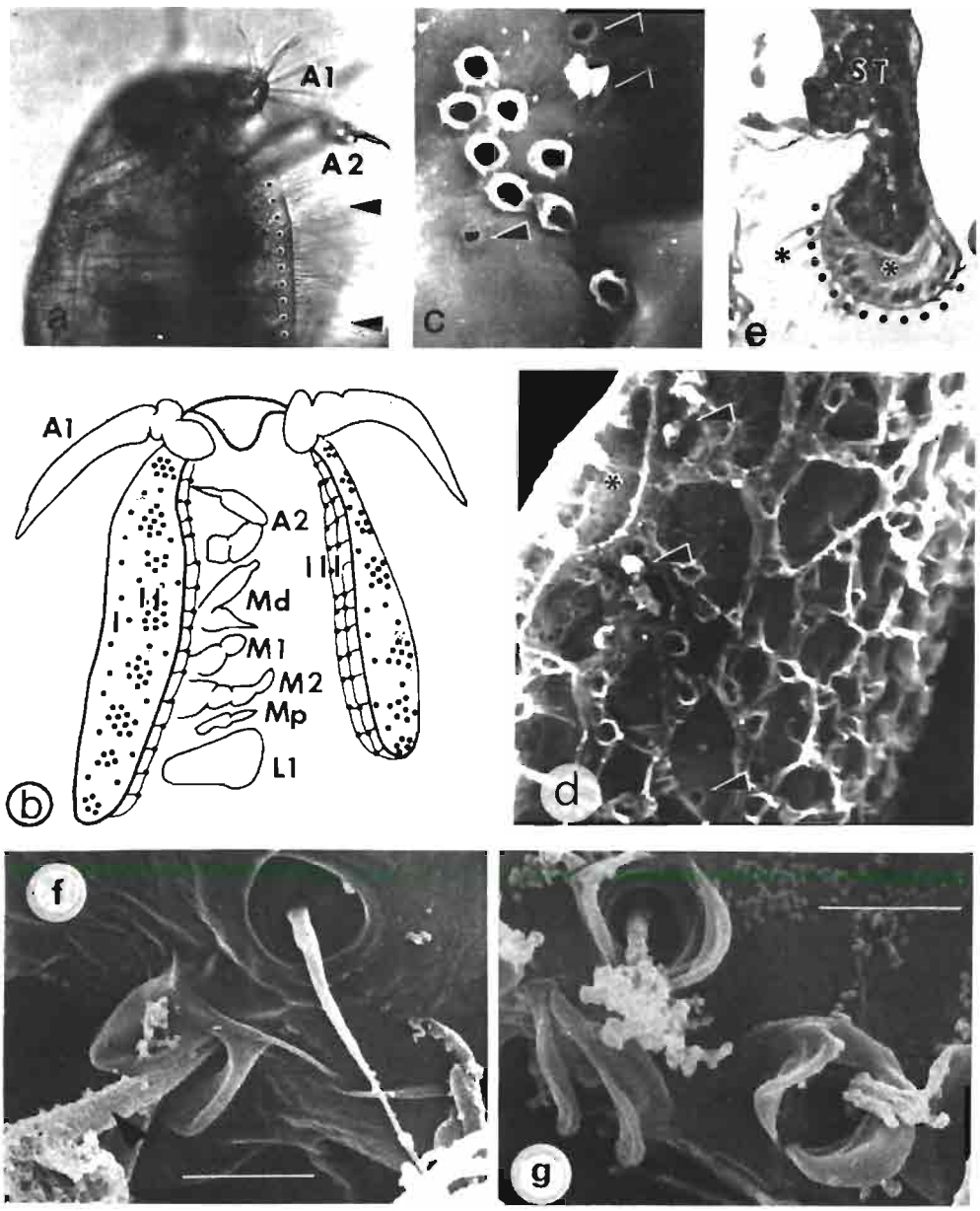

Fig. 6-28: Sensory pores in the cuticule of *Pachypygus gibber* atypical male pleural borders. a: Head of living copepod, lateral view; dots: pleural edge, arrows: sensory filaments. b: ventral view (diagram) of head showing distribution of sensory pores. c: SEM of group of 8 sensory pores with raised rims (II) and 3 glandular pores (I) (arrows); filaments destroyed in critical-point treatment. d: SEM frontal view of pleural ventral edge with pores of Type III. e: Semithin section of pleuron showing sensory tissue (ST) from wich protrude curved filaments (*) going through cuticular edge (after Hipeau-Jacquotte, 1986). f: *Mychophilus roseus*. Pore and emergent filament (arrow), right: plain seta. g: Two pores with emergent filaments, their broken ends apparently unravelling; scale bar = 1 µm. (After Gotto and co-authors, 1984.)

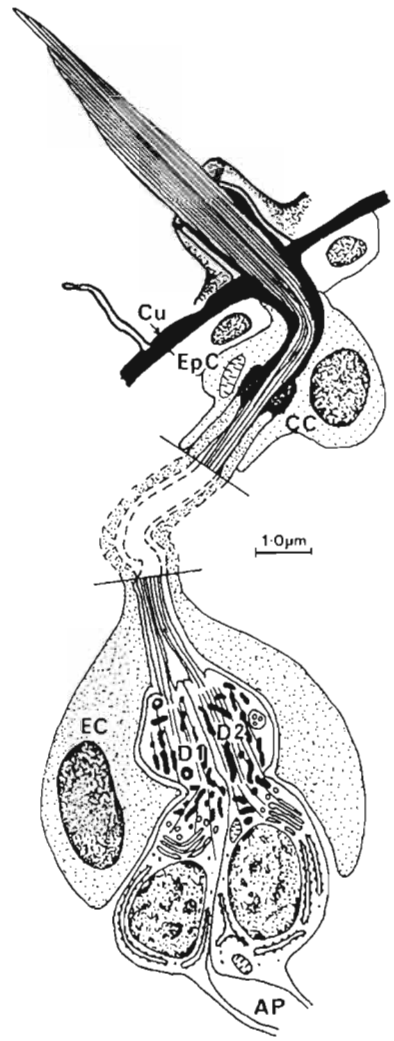

Fig. 6-29: *Pachypygus gibber.* Atypical male sensory organ. Schematic representation in longitudinal section of a functional unit formed by 2 sensory cells (Dendrites D1, D2), 1 basal enveloping cell (EC) and 1 apical canal cell (cc). Anoxic processes of 1 and 2 (AP). Epithelial cell (EpC) and cuticle (Cu). (After Hipeau-Jacquotte, 1986.)

ments of setae on the furca. In the Mediterranean Sea they established a series of *Notodelphys* species, specific for a given ascidian host. In Naples (Italy), Illg and Dudley (1965) were facing the same problem with regard to the genus *Notopterophorus* characterized by wing-shaped extensions of the thoracic segments. In other genera, they demonstrated that there is only 1 European species, *Doropygus pulex,* infesting all large ascidians (Illg and Dudley, 1961, 1965; Gotto, 1975b). Bocquet and Stock (1960) had difficulties to separate some *Notodelphys* species in Roscoff (France).

Attempting to precisely re-describe the Scandinavian *Notodelphys*, Lejeune and Monniot (1964) realized that differences based on presence-absence of some setae, were

not significant. Many previous records turned out to be based on inacurate observation or vestigial setae. The furca ratio varies according to the ascidian host or even different populations of the same ascidian species. *Mychophilus roseus* in *Botryllus schlosseri* have not the same outline as in *Botrylloides leachi* (Gotto, 1954). The body proportions of *Pygodelphys aquilonaris* vary remarkably in different hosts (Illg, 1958). Setation variability of *Notodelphys agilis* in *Ciona intestinalis* and *Corella parallelogramma* was first studied by Monniot (1982c). He showed that very few specimens can be considered perfect, and that abnormalities were numerous (absence, doubling, reduction of setae, and appendage malformations).

Analysis of copepod variability in 2 particular cases demonstrated host influence leading to modified setation. Such differences would be considered sufficient for describing new species in independent populations. In *Pachypygus macer,* imported less than 3 years ago in a Guadeloupean harbour and living in *Styela canopus* (*plicata*) and *Microcosmus exasperatus,* differences by 3 setae occurred (Monniot, 1986). *Archinotodelphys polynesiensis* living in 3 hosts on 3 islands in French Polynesia, revealed host influence on the frequency of mouth-piece abnormalities, as well as influence of geographic isolation on the abnormalities (Monniot, 1987a). In an accidental host all appendages of a specimen differed from the norm, while in the same 3 hosts a *Bonnierilla* sp. did not show any variability (reviewer's own unpubl. obs.).

Previous species identifications for some genera, such as *Notodelphys,* require experimental verification. In fact experimentation may lead to re-definitions of species boundaries for ascidicolous copepods.

No such studies have been undertaken in Ascidicolidae. However female *Botryllophilus* species show a marked asymmetry in pereiopod structure, a phenomenon which seems unique among copepods (Stock, 1970). Since the sub-family Botryllophilinae is the only one not recently revised, asymmetry remains without explanation. Lang (1948) assumes a 'genetically controlled reduction which has not attained stability' (p. 2).

Multiple Infestations and Mutual Copepod Influences

Copepod-copepod influences have been reported but were studied in detail only in *Microcosmus sabatieri* (Monniot, 1961a), where 5 copepod species can cohabit with a nemertine in a single host individual. The different species of copepods did not exactly occupy the same places in their host. Lichomolgidae dwell in the cloacal cavity together with nemertines; *Ascidicola rosea* was located in the oesophagus; *Ophioseides cardiacephalus* between body wall and tunic; *Notodelphys acanthomela* and *Doropygus pulex* occupied the branchial sac (*N. acanthomela* was located close to the endostyle while *D. pulex* prefered the dorsal lamina area). Such differential distribution does not seem to witness antagonism among the parasites: they retain their respective distributions also in the absence of one or the other neighbour. However, in *Microcosmus vulgaris, N. acanthomela* was never present, while *D. pulex* was distributed over all parts of the branchial sac.

Presence of one of the symbiotes in the branchial sac of *Microcosmus sabatieri* favours settlement of the others, and the rate of co-infestation is at least double that of simple infestations of one or the other species (Monniot, 1961a).

Copepod Life Cycle in the Host

Development of the copepod families Notodelphyidae and Ascidicolidae has been studied first by Canu (1890, 1892) but is now well known for Notodelphyidae (Dudley, 1957, 1966) and Ascidicolidae (Anderson and Rossiter, 1969; Ooishi, 1980). These morphological papers pay little attention to host-copepod relations. The female copepod oviposits in the ascidian branchial cavity. At this time the nauplius is still encased by membranes; eclosion normally occurs immediately after the embryos were expelled from the brood pouch. The nauplii swim immediately toward the light. However, their travelling out through the ascidian oral siphon has not yet been observed under natural conditions. At any rate, the nauplii leave the ascidians and it is at the Copepodite 2 stage that the copepods re-enter a new host (Dudley, 1966; Hipeau-Jacquotte, 1980a).

Ascidicola rosea penetrates the stomach to lay eggs and its young are expelled through the digestive tract together with host faeces (Gotto, 1957). Adult gravid *Doropygus* females were placed by Dudley (1966) into the transparent ascidian *Corella willmeriana* (which is not the usual host). Many nauplii were trapped in the mucus of the pharynx and then digested by the ascidian. In one case, the expelled eggs did not hatch immediately and passed undamaged through the gut. Dudley (1966) considers 'it is quite possible that the hatching of copepods in the pharynx happened to be simultaneous with normal back flushing reaction of the tunicata' (p. 18); however, this problem is not yet adequately solved.

There are some particular cases; *Mychophilus roseus* does not wear 2 external ovisacs but lays its eggs in small clusters distributed in the common test of *Botryllus* species (Gotto, 1954). The eggs of *Enterocolides ecaudatus* are placed in the superficial layer of the host colony, though adult females live in deeper layers (Chatton and Harant, 1922c). Bresciani and Lützen (1962) observed that the network of tunnels burrowed by *Ophioseides* sp. has a gallery ending close to a siphon, and when the female is engaged into this gallery, its anal part is near the siphon. The authors suppose that the test might be perforated to allow the nauplii to escape.

Some workers (e.g., Gage, 1966; Egan, 1984b) noted a relation between copepod and sexual maturity of the host, with young copepodites present when the young ascidian population appears. Gage (1966) assumes that in *Ascidicola rosea* copepod life lasts as long as host life.

Diet

Several kinds of diets for copepods parasitic in invertebrates have been listed by Gotto (1979). According to him, the Lichomolgidae and some Notodelphyidae like *Ooneides* living in the cloacal cavity of ascidians, may be 'debris feeders'. Species of *Notodelphys* and *Pachypygus* inhabiting the host's branchial cavities are considered 'larder feeders' taking their food from the host's mucous film. The most specialized forms such as species of *Ascidicola* and *Styelicola*, feeding on the ascidian's alimentary cord when it enters the oesophagus or is inside the stomach, as in *Enterocola,* belong to the 'larder feeders'.

Copepods in cysts and *Mychophilus* species are considered 'blood feeders' by Gotto (1979). Regarding copepods in cysts, Dudley (1968) observed that *Scolecodes* species did not ingest blood cells but cyst cells whose origin is to be looked for among the host's blood and mesenchyme cells. *M. roseus* is often recorded from the common blood system of *Botryllus* species, but it does not make a cyst. The presence of this copepod in the common

test is a consequence of the cycle of host zooids. A *Botryllus* zooid is functional for several days before desorganizing. The copepod introduced to it is then released into the tunic. The case of *M. roseus* is not singular: numerous Notodelphyidae and Ascidicolidae living in colonial ascidians remain in the tunic and obligatorily become 'blood feeders'. It is not impossible that copepods, known only from colonial ascidian tests, came from dead and decayed zooids.

Hipeau-Jacquotte (1982) estimated that the atypical male of *Pachypygus gibber* is at least partially a 'blood-feeder'; this would explain that transformation into an atypical male can be obtained *in vitro* in the presence of a squashed young *Ciona*. Gotto (1979) assumes that *Ophioseides cardiacephalus* (*Scolecimorpha joubini*) digging galleries in the host tunic, ingests at the same time blood cells and 'semi-fluid material resulting from the tunician break-down' (p. 79). The mechamism used by the copepod to cause tunic lysis is unknown.

Sensory organs of nauplii and copepodites have been studied only by Dudley (1969, 1972) who found several adaptations. The eye anatomy of young *Doropygus seclusus* revealed that 'during the first four naupliar stages, neither rhabdomers nor axons are apparent, but begin to form during the fifth naupliar stage' (p. 39). The infesting stage possesses an eye identical to the one of the adult, but smaller. At this time a change occurs from positive to negative phototaxis.

In nauplii and copepodites of *Doropygus seclusus* Dudley (1972) studied the structure of a cephalic organ. The bilateral organ is located in the enterodorsal head region. It consists of 2 parts; the 'end organ A' is made of a dendrite bundle ending by encapsulated cilia; the 'end organ B' has the same structure with dendrites and setae but crosses the supporting cells and reaches the cephalic cuticle. The 'end organ A' shows maximum development at the Copepodite 2 stage (infestating stage) and then regresses, without disappearing in the adult. The 'end organ B' disappears in the subsequent stages. The function of these organs is unknown but Dudley (1972) supposes that they may assist in host recognition. Neither Hipeau-Jacquotte (1986, 1987) nor Gotto and co-authors (1984) have conducted comparisons with latero-ventral organs of free-living or atypical males.

Agents: Amphipoda

Amphipods are often found inside the branchial and cloacal cavities of large ascidians. *Aristias neglectus, A. tumidus,* and *Leucothoe spinicarpa* were reported from several solitarian ascidians (Harant, 1931). None of these species is specific of the ascidian, where they only find shelter (phoresis; see Volume I, p. 19). However, some amphipods may damage the branchial tissue of the host.

In all seas of the world, amphipods live inside the common test of colonial ascidians, with only their antennae protruding to the outside. The association between *Aplidium* spp. (*Amaroucium*) and *Polycheria osborni* has been described in detail by Skogsberg and Vansell (1928). The amphipod (Fig. 6-30) is lying on its back, embedded in a hollow of the host's tunic, holding the edges with Pereiopods 1, 2, 4, and 5 while the 3rd pereiopods are used to remain in the bottom cavity. The amphipod is able to leave its chamber and to make another one. Skogsberg and Vansell describe the chamber construction as follows: 'In making its burrow, the animal does not dig in the strict sense of this word. It lies on its back, grasps the surface of the ascidian test with its first, second, fourth, and fifth pereiopods and then begins to pull slowly. The test slowly yields. In this way the back of

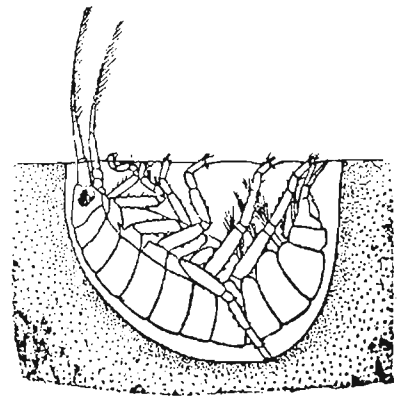

Fig. 6–30: *Polycheria osborni* in its burrow. (After Skogsberg and Vansell, 1928.)

the animal is gradually pushed into the test; and finally it is so deep down that the edges of the cavity thus formed can be pulled over the animals. This operation required, when observed in an aquarium, several hours' (p. 294). According to Skogsberg and Vansell, amphipods seem to prefer ascidian colonies with many dead zooids.

Hyperiid amphipods are allied to littoral gammarids and form a specialized group with gelatinous zooplankters (see review by Laval, 1980). The reality of this association has been clearly demonstrated in underwater observations by Madin and Harbison (1977). We must differentiate between associations in which the amphipods simply live inside a tunicate, and those in which they destroy the tunicates using the tunic to make a barrel, as is the case with members of the family Phronimidae.

The first type of association prevails in the family Vibiliidae, members of which live in salps; representatives of the Hyperiidae and Oxycephalidae, in salps; and Lycaeidae, in salps or pyrosomes. Laval (1965), Harbison (1976), and Madin and Harbison (1977) have shown that juvenile and female amphipods of the genera *Lycaea,* and *Vibilia* entertain an obligate symbiotic relation with various salp species. In part, the salps are used as a source of food. Madin and Harbison (1977) state that 'as commensal and parasite on nearly all species of salps, amphipods may influence the growth and reproductive rate of their host' (p. 460). This interaction remains to be quantified.

The Phronimidae live in a transparent barrel. Owing to superficial similarity between barrel shape and dolioles, Delle Chiaje (1841) erroneously described as *Doliolum* 3 'species' of barrels in the Mediterranean Sea, and even now some authors still believe that doliolids act as host's for phronimes. The barrel has been subject of numerous studies concerning histology (Pagenstecher, 1861), morphology (Claus, 1862, 1872; Mayer, 1879), or chemistry (Dudich, 1926), and it was demonstrated that most barrel 'species' relate to salps or pyrosomes, others to siphonophores or ctenophores. Laval (1978), employing a multivariable analysis (Fig. 6-31) demonstrated that in the Mediterranean Sea, origin of barrels can be identified for *Phronima sedentaria.* A-group (Fig. 6-32a, b) stems from *Salpa fusiformis;* F-group, from oozooids and blastozooids of *Thalia democratica* and from blastozooids of *Thalia democratica* and *Ihlea punctata*; other barrel types stem from *Pyrosoma atlantica* (Fig. 6-32c, d, e).

Barrel formation begins with the destruction of tunicate tissues, easy to endure for

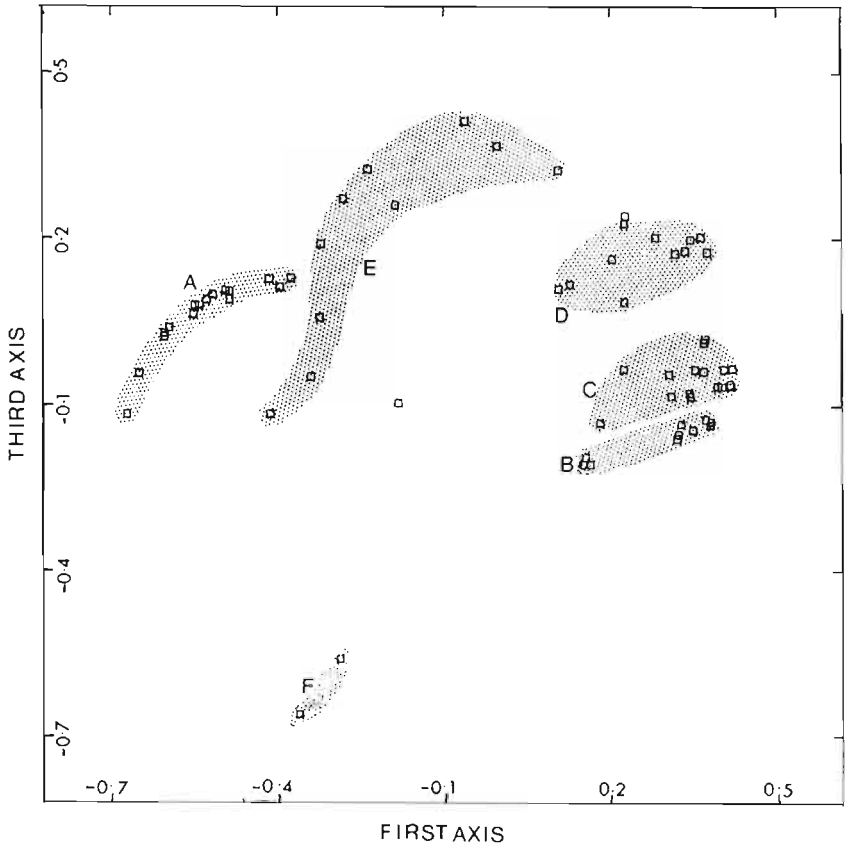

Fig. 6-31: Phronim barrels: principal coordonate analysis of quantitative and qualitative characters of the barrels. (After Laval, 1978.)

salps, but much less so for pyrosomes which carry many zooids inside their tunic. At least in pyrosomes the tunic remains alive as it contains many mesenchymatous cells. These cells rebuild the tunic, fill the cavities which had contained zooids, and close siphonal openings (Laval, 1978). The finger-like superficial processes retract with time. The tunic also modifies its internal side in contact with the Phronime and becomes harder. Possibly, tegument glands of the amphipods play a role in this process, but this remains to be demonstrated (Laval, 1978).

Male and female phronimes live isolated, each in its barrel. The females arrange their larvae (up to 600) along the internal side of the barrel and exhibit a maternal protection behaviour which has not yet been studied in detail.

Agents: Isopoda

The only known case of a commensal isopode residing inside an ascidian is presently being studied; it involves an isopod population living in *Halocynthia hispida* collected on the slope of the Galapagos Islands at 750 m water depth (R. Brusca, pers. comm.).

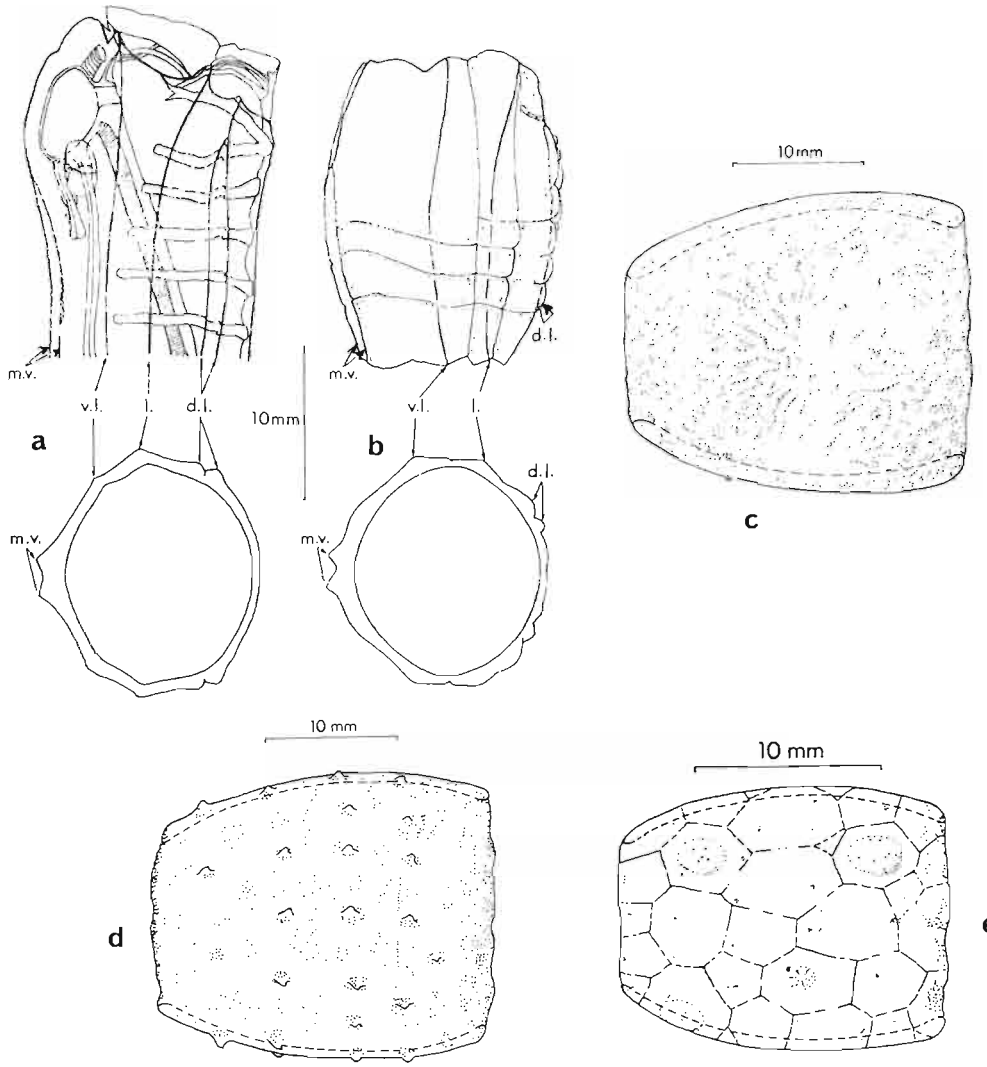

Fig. 6-32: Phronim barrels. a and b: Comparison of posterior region (right) of an oozooid of *Salpa fusiformis;* (a) with an 'A' group barrel. c and d: Barrels from *Pyrosoma atlantica;* (c) a typical 'B' group barrel with its ornementation of very small denticles. d: 'C' group barrel with tubercules. e: 'E' group barrel with polygonal network. (After Laval, 1978.)

Agents: Cirripedia

A parasitic crustacean, *Sphaerothylacus polycarpae,* attributed to the cirripedia, lives in cysts of the branchial tissue of *Polycarpa cryptocarpa* in Indonesia (Sluiter, 1884). The reviewer has found in New-Caledonia, in the same host, the same cysts. However, the parasite presently being studied, is a notodelphyid copepod, belonging to the genus *Prophioseides* (Ooishi, pers. comm.).

In a polyclinid ascidian from the coast of Portugal the reviewer has found barnacles

devoid of calcified walls, completely included into the tunic, with only its appendages extending above the colony surface. This association has not yet been studied in more detail.

Agents: Decapoda Natantia (Shrimps)

Since the 19th century shrimps were recorded inside ascidian branchial sacs (Heller, 1864). That study showed that they belong to particular species, living only in ascidians. All species presently recorded (Table 6-8) belong to 3 genera: *Pontonia, Dasella,* and *Periclimenaeus* of the sub-family Pontoniinae.

A. J. Bruce (pers. comm.) assumes that all *Pontonia* sp. are closely connected with ascidians even when they were not described from ascidians, or when they were said to be associated with other groups as sponges or corals. In fact, in most cases, these shrimps

Table 6-8
Pontoniid shrimps from ascidians (Original compiled from the sources indicated)

Host	Commensal	Source
Ascidia conchilega	*Pontonia maculata*	Lagardère (1971)
Ascidia depressa	*Dasella ansoni*	Bruce (1983)
Ascidia empheres	*Pontonia ascidicola*	Holthuis (1952)
Ascidia mentula	*Pontonia maculata*	Heller (1861)
Ascidia paratropa	*Pontonia californiensis*	Standing (1981)
Ascidia sp.	*Pontonia okai*	Bruce (1981)
Ascidia sp.	*Pontonia sibogae*	Bruce (1977)
Ascidia sp.	*Pontonia* sp.	Bruce (1976)
Ascidia vermiformis	*Pontonia californiensis*	Holthuis (1951)
Ascidia willeyi	*Pontonia okai*	Kemp (1922)
Atapozoa deerata	*Periclimenaeus pachydentatus*	Bruce (1970)
Cnemidocarpa pedata	*Pontonia katoi*	Bruce (1981)
Cnemidocarpa whiteleggei	*Pontonia sibogae*	Bruce (1972)
Diazona violacea	*Pontonia maculata*	Heller (1864)
Diplosoma modestum	*Periclimenaeus hecate*	Bruce (1970)
Diplosoma rayneri?	*Periclimenaeus diplosomati*	Bruce (1980a)
Halocynthia ritteri	*Pontonia katoi*	Kubo (1940)
Lissoclinum fragile	*Periclimenaeus tridentatus*	Holthuis (1952)
Microcosmus hartmeyeri	*Pontonia katoi*	Kikuchi & Miyaki (1978)
Phallusia julinea	*Pontonia okai*	Bruce (1979)
Phallusia mammillata	*Pontonia maculata*	Heller (1864)
Polycarpa annandalei	*Pontonia anachoreta*	Kemp (1922)
Polycarpa aurata	*Pontonia katoi*	Bruce (1980b)
Polycarpa cryptocarpa	*Pontonia katoi*	Bruce (1976)
Polycarpa pedunculata	*Pontonia katoi*	Bruce (1983)
Polycarpa sp.	*Pontonia katoi*	Bruce (1981)
Pyura momus	*Pontonia katoi*	Bruce (1977)
Pyura momus	*Dasella herdmaniae*	Bruce (1983)
Rhopalaea birkelandi	*Pontonia spighti*	Fujino (1972)
Styela palinorsa	*Pontonia katoi*	Holthuis (1952)
Styela sp.	*Pontonia katoi*	Bruce (1980)
In ascidian	*Pontonia ascidicola*	Borradaile (1917)
in ascidian	*Periclimenaeus ascidiarum*	Holthuis (1951)
Probably from ascidian	*Periclimenaeus manihinei*	Bruce (1980a)
Probably from ascidian	*Periclimenaeus nobilii*	Bruce (1980a)

were found in the bottom of vials containing corals with dead parts covered with sponges and ascidians.

Member of the genera *Pontonia* and *Dasella* were recorded only in solitary ascidians and live inside the branchial sac. Only some species of the genus *Periclimenaeus* are associated with colonial ascidians. They inhabit the common cloacal cavities where they move actively. The shrimps do not seem to cause any damage to their hosts. When living, they are generally not visible through the ascidian test, except in some transparent colonies where their black eyes reveal their presence.

Agents: Decapoda Reptantia

The 'Sargassum crab' *Planes minutus* can live on salp chains (Madin and Harbison, 1977). Portunides and megalopes live associated with the aggregated form of *Pegea confederata* in the Gulf of California.

Some pinnotheres, which normally inhabit other invertebrates, may live in large solitarian ascidians. Harant (1931) reports *Pinnotheres veterum* from *Polycarpa pomaria,* and *P. pisum* from *Ascidia mentula* and *Microcosmus* spp. We have observed pinnotheres entering *Microcosmus sabatieri* in Banyuls (France). The crab climbs on the ascidian and attempts to enter through its oral aperture. The ascidian contracts and the crab stays outside with some of its legs inserted into the siphon. This situation can last more than 48 h until the ascidian is forced to open its siphon again and let the crab enter.

The reviewer has often noticed large torn parts of branchial ascidian tissue used as shelter by pinnothers but since the animals were preserved it is not possible to know whether the holes were made by crabs struggling in the fixative medium.

Some Dromiidae, commonly hidden under sponges or alcyonids, may dig a cavity into ascidians. In some cases a whole population of Dromiidae can live in one type of ascidian. This has been observed by Harant (1931) in *Aplidium densum* (Mediterranean Sea), by Michaelsen (1904) for *Pseudodromia latens* in *Gynandrocarpa domuncula* (Cape Town, South Africa), and the reviewer in *Botrylloides leachi* (Arcachon 'France'). The only case of a Dromiidae associated with a solitarian ascidian was observed in New Caledonia (Monniot, 1987b), the crab inhabiting a large tunic depression of *Polycarpa contecta*; the ascidian body occupied no more than ¼ of the tunic.

Ascidians very often cover crab shells. In most cases, pieces of colonies, taken by the crab, grow on their shell. The solitarian ascidian *Styela changa* (Monniot and Andrade, 1983) from Chile, is known only from the shell of *Lophorochina parabranchia* where it is abundant.

Agents: Pisces

The presence of fishes in cavities of salps and pyrosomes has been known for a long time (Chamisso, 1819; LoBianco, 1909; Fitch, 1949, 1951). However, originally this was considered accidental (Mansuetti, 1963). Direct observations employing SCUBA have clearly shown that, at least for members of the fish genus *Tetragonurus,* such associations are a "highly specific and stereotyped comportment" (Janssen and Harbison, 1981, p. 918). These authors found inside salps numerous specimens belonging to 3 species of the genus *Tetragonurus* in the Atlantic, Pacific, and Indian Oceans.

For the fish, the tunicate host serves both as shelter and food source (Fig. 6-33). The

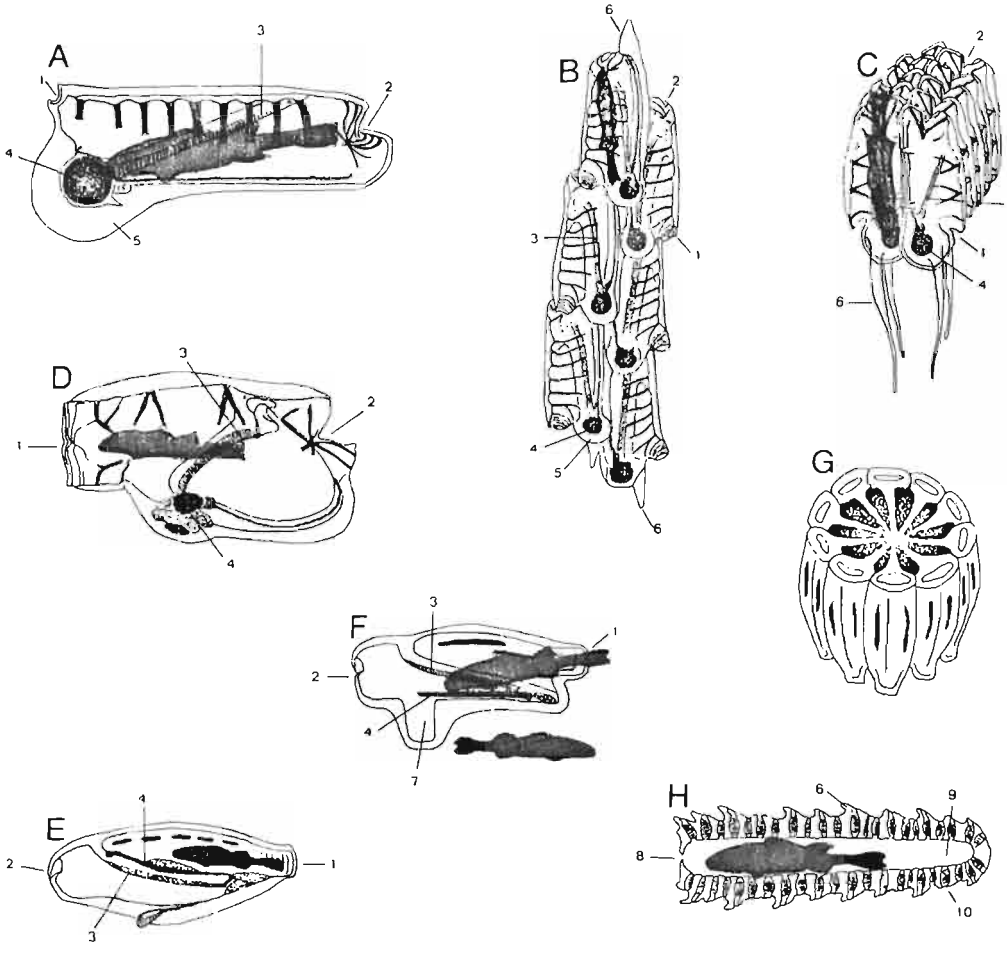

Fig. 6-33: Morphology of salps and pyrosomes and position of resting and feeding fishes. A: *Salpa maxima* solitary, fish in branchial chamber feeding on gut. B: *Salpa maxima* aggregate, fish resting in branchial chamber. C: *Pegea* sp. aggregate, fish in branchial chamber feeding on gut. D: *Pegea* sp. solitary, fish resting in branchial chamber. E: *Cyclosalpa pinnata* solitary, single fish resting in branchial chamber. F: *Cyclosalpa pinnata* aggregate one zooid, 1 fish in the branchial chamber feeding on gut, 1 fish resting among peduncles. G: *Cyclosalpa pinnata* aggregate. H: *Pyrosoma* sp. fish in colonial chamber. (After Janssen and Harbison, 1981.)

fish feed mainly on the salp's nucleus and branchial tissue. Only young fish were observed *in situ*, the adults are mesopelagic and live in deeper waters. Janssen and Harbison (1981) assume that adult fish have the same behaviour, as their stomach content is made up of salp nuclei and tissue pieces of pyrosomes. Fish also have been observed inside pyrosomes by Thompson (1948).

Other fishes, especially juveniles, have been observed *in situ* in pelagic tunicates (Madin and Harbison, 1977; Janssen and Harbison, 1981), but their presence does not constitute a true association as is the case for *Tetragonurus* spp.

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TAXONOMIC INDEX

(Abbreviations: S. N. = List of Scientific Names, C. N. = List of Common Names)

LIST OF SCIENTIFIC NAMES

(Including names of viruses)

LIST OF COMMON NAMES

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