A word from the editor

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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

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Biologische Anstalt Helgoland
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VOLUME II
Introduction, Bivalvia to Scaphopoda

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FOREWORD

Volume II of 'Diseases of Marine Animals' reviews comprehensively and critically all essential information published to date on the biotic diseases, proliferative disorders and structural abnormalities of marine Bivalvia, Amphineura and Scaphopoda. The treatment of the diseases of molluscs, which started with the Gastropoda at the end of Volume I, will be completed with the Cephalopoda in Volume III.

Volume II is subdivided into 4 sections:

Introduction to Volume II
Chapter 13: Diseases of Mollusca: Bivalvia
Chapter 14: Diseases of Mollusca: Amphineura
Chapter 15: Diseases of Mollusca: Scaphopoda

Conceptually, Volume II is based on the perspectives outlined in Volume I in the introduction to the treatise. Our original plan that Dr. G. Lauckner of the Biologische Anstalt Helgoland be the sole author of all forthcoming contributions to this treatise has been abandoned. The information available is so vast and the fields to be covered so divergent that a number of world-wide-acknowledged and highly regarded experts have been chosen to cover specific topics in Volume IV.

In order to make this important treatise available to as many interested libraries and individual scientists as possible, the treatise will — beginning with Volume II — be sold on a non-profit-making basis, i. e. at self cost price, and be published by the Biologische Anstalt Helgoland.

It is with great indebtedness that I acknowledge here the technical and bibliographical assistance by Martin Söhl and Ingrid Schritt as well as editorial help by Seetha Murthy and Helga Witt — all members of the Biologische Anstalt Helgoland. Hildegard Lauckner has typed most of the manuscript and assisted in matters of literature quotation.

O. KINNE

Hamburg, June 8, 1982

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INTRODUCTION
BIVALVIA TO SCAPHOPODA

INTRODUCTION TO VOLUME II

O. KINNE

GENERAL SCOPE

This book follows the scope of the four-volume treatise outlined in Chapter 1 of Volume I (pp. 1–2). It roots in the general framework of concepts, perspectives and terms developed in Chapter 2 of Volume I (pp. 13–64).

As its predecessor, the present tome concentrates on biotic diseases, proliferative disorders and structural abnormalities. Chapters 13 to 15 review the diseases of Bivalvia, Amphineura and Scaphopoda. Volume II, like Volume I, is organized around the two partners, or antagonists, involved in a biotic disease: host and agent.

Mirroring economic importance, amount of scientific literature available and relative size of the animal groups considered, the overwhelming portion of Volume II is devoted to the Bivalvia (ca. 96 %). Some 3 % deal with the Amphineura and ca. 1 % with the Scaphopoda.

Based almost exclusively on original-source information, Chapters 13 to 15 attempt to stress the significance of diseases in terms of the pathology and ecology of the organisms involved, and to point out, trace and correct misquotations, misnomers and misconcepts which have, over many decades, accumulated in reviews and original research papers.

As the preceding volume of this treatise, Volume II addresses itself to all those professionally concerned with life in oceans and coastal waters, especially researchers, university teachers and advanced students in the fields of pathology, parasitology, ecology, fisheries biology and aquaculture.

NEED FOR MORE RESEARCH

Hitherto largely the domain of parasitologists and pathologists, the diseases of marine organisms must also become a focal point of marine ecological research. This is an important prerequisite for consolidating the body of knowledge emerging on life in oceans and coastal waters, for determining safe limits of stress endurance, and for developing sound measures for controlling and correcting man's growing impact on the marine environment*.

Though facts of considerable interest have been disclosed on the etiology of a few diseases of Bivalvia, Amphineura and Scaphopoda, such cases have largely remained limited to very few species of immediate commercial interest. The disease as such has rarely been the main theme of research. Most papers presently available concentrate on the description of parasites, their taxonomy, life history, distribution and abundance.

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* Kinne, O. (Ed.) (in press). *Marine Ecology*, Vol. V, Ocean Management, Parts 3 and 4: Pollution and Protection of the Seas. Wiley, Chichester.

Structural abnormalities have been described and evaluated primarily by taxonomists and morphologists. The ecological significance of such deviations from the normal state (health), analysis of the underlying causes, as well as interpretative, critical syntheses of the biological phenomena involved require more attention. Only in rather recent work on microbial infections and proliferative disorders (tumours) has the emphasis been on disease *sensu stricto*.

Consequently, large portions of the chapters in this volume had to be devoted to bringing together and to evaluating the tremendous body of widely scattered, descriptive information on symbioses (Vol. I, p. 18) between marine Bivalvia, Amphineura and Scaphopoda on the one hand and a large variety of intimately associated marine organisms on the other. Only in a limited number of cases did the information at hand allow to attach definite labels to such symbioses in terms of phoresis, commensalism, parasitism or mutualism (Vol. I, pp. 18–21); to consider etiological aspects; and to evaluate the resulting phenomena with a view of consequences for ecological dynamics.

Much more research is necessary before definite statements can be made with regard to causes and development of diseases, agent effects and virulence, host predisposition and defense, and — for cultivation and experimentation — of disease prevention and therapy.

The pressing need to define limits of tolerance to man-made environmental deformation cannot be met without taking into account the health-status of the organisms involved. Diseased organisms tend to exhibit modified, usually reduced capacities for tolerating environmental pollution and for coping with extreme intensities of natural environmental factors.

As defined in Volume I (p. 14), 'disease' denotes a demonstrable, negative deviation from the normal state (health) of a living organism. In this definition, 'negative' implies an impairment, quantifiable in terms of a reduction in the ecological potential. The degree of impairment is measurable, for example, in terms of rates of survival, growth, reproduction, energy procurement, stress tolerance or competitive capacity. The negative deviation may be functional or structural or both; it may be due to a single cause or to several causes acting in concert. This definition characterizes disease as an ecological phenomenon. A biotic disease may not only affect the relationship between agent and host, but also influence coexisting species, such as the host's prey, predators or competitors and, at the level of epizootics, may modify functions and structures of an ecosystem.

In order to achieve progress in ecological research and to build a sound fundament for environmental protection, we must learn how to measure and evaluate the total amount of stress encountered by a living system — natural-environmental, disease-caused plus man-made. All three groups of potential stress may affect — mostly synergistically — functional and structural properties, such as metabolic rate and pathway; growth, development, reproduction and behaviour; morphological expressions at the cellular, organ and organism level; as well as presence, abundance and performance of species in a given habitat. For ecological success or failure of an organism, the total amount of stress encountered and the long-term capacity for adjustment (non-genetic and genetic adaptation) are essential parameters.

Thus far, experimental ecologists, physiologists and biochemists have sadly neglected to determine the health-status of their test organisms prior to experimentation and to study potential effects of disease-causing entities on metabolic, reproductive and competitive performances. Consequently, we must now question the ecological validity of a large body

of information which has accumulated for many decades in the scientific literature on organismic responses to natural and man-made stress.

In order to have ecological significance, in terms of organismic performance under environmental change, future research must be conducted under defined, adequate conditions of environment and nutriment, using stabilized* organisms of known health status**.

Where information presented in Volume II was obtained under ecologically inadequate conditions — and this was apparently the case in much of the experimental work — scepticism must prevail whether the information gained on the agents involved, the degree and type of pathogenicity and the potential of the diseased organisms to counteract agent virulence is indeed extrapolatable to *in situ* conditions. We know that the balance between the capacity of a potentially disease-causing entity and the disease-counteracting capacity of a target organism may be delicate and subject to change as a function of environmental and nutritional conditions, as well as to the physiological states of the organisms involved (Vol. I, p. 14 and pp. 17–27). Organisms that coexist without demonstrable negative consequences under one set of conditions may turn into 'enemies' under another. A bacterial population that would not normally harm a bivalve, may turn into a lethal 'disease agent' as soon as the bivalve's physiological state becomes severely impaired due to environmental deterioration, nutritional deficiency, injury, severe competition, senility or negative deviations caused by other simultaneously effective disease-causing entities.

In the relatively few cases of molluscan diseases recognized, described and analyzed, measures for disease prevention and disease therapy have remained largely unconsidered and uninvestigated. Here lie wide virgin and fertile fields for marine parasitologists, pathologists, aquafarmers and experimental ecologists.

Of course, in marine animals effective disease therapy is in essence restricted to conditions controllable by man, especially to situations where the animals concerned are grown in experiments or commercial culture projects. Nevertheless, studies of the diseases of marine animals in general should not be limited to disease etiology, but also include considerations regarding potential measures of disease prevention and therapy. Such knowledge may, in context with the increasing deformation of the marine environment due to man-produced impact, become an important issue in the future for protecting life in the seas and for compensating for damage already incurred.

SUMMARIES OF CHAPTER CONTENTS

Comments on Chapter 13: Diseases of Mollusca: Bivalvia

Marine bivalves are of considerable economic importance. Not least because of this, the amount of information available on bivalve diseases is extraordinarily large and comprehensive. In fact, we now know more about the diseases of bivalves than of the diseases of all other marine invertebrate groups together. While early investigators turned

* Stabilized = 'new steady state' attainment after environmental change: Kinne, O. (1970). Temperature: Animals. Invertebrates. In O. Kinne (Ed.) *Marine Ecology*, Vol. I, Environmental Factors, Part 1. Wiley, London. p. 474.

** Especially with regard to degree and type of parasitization, infection, injury and structural disorders.

their attention primarily to the disease-causing capacities and life histories of metazoan parasites, modern researchers focus on — and sometimes tend to overemphasize — the significance of microbial and protozoal disease agents.

Agents Capable of Causing Diseases in Bivalves

Major disease-causing agents of marine bivalves are viruses, bacteria, fungi, Labyrinthomorpha, Apicomplexa, Ascetospora (*Marteilia*, 'haplosporidians'), Ciliophora, Trematoda Digenea, Annelida and Copepoda.

Although viruses are known, or expected, to cause severe negative deviations from the normal state, the etiology of viral diseases in marine bivalves remains to be studied in more detail.

Most marine bacteria examined are not detrimental to adult bivalves, unless present in cell densities far above those normally attained under *in situ* conditions. In experiments, bivalve larvae turned out to be more vulnerable to bacteria than their adult counterparts.

Fungi may biodegrade bivalve shells (shell disease) and cause mycotic infections in the soft parts of larvae and adults. In general, fungi appear somewhat less dangerous to bivalve health than assumed by early investigators.

Members of the Labyrinthomorpha ('fungus-like protistans') may inflict severe diseases and heavy losses. Most of the potential agents have neither been clearly identified nor adequately described.

The newly established phylum Apicomplexa (*'Dermocystidium'*, gregarines, coccidians) contains disease agents with highly destructive potentials (e.g., *Perkinsus marinus*, the agent of 'Dermocystidium disease'), as well as harmless or moderately pathogenic forms, such as gregarines and coccidians. Many gregarines use marine bivalves as intermediate hosts.

Ascetospora (also a newly created phylum) can cause serious diseases and high mortalities, especially species of the genera *Marteilia*, *Haplosporidium* and *Urosporidium*. *Marteilia refringens* is the causative agent of 'Aber disease'; haplosporidians are dangerous cytozoic and histozoic parasites; urosporidians comprise species which are hyperparasites of larval trematodes and of a nematode parasitizing marine bivalves.

While numerous Ciliophora associate with marine bivalves, most do not seem to act as definite disease agents. The ciliates associate as (i) filter-feeding commensals utilizing food particles collected in the bivalves' ciliary currents; (ii) particle feeders on gills or mantle epithelium; (iii) parasites consuming the contents of epithelial gill cells. The latter are represented mainly by Rhynchodida which suck nutrients from the host's cells in gills and palps. Damage to the host manifests itself in the formation of keratin fibres inside affected cells and accumulation of lipid inclusions — possibly indicators of disturbed host-cell metabolism. When the ciliate leaves an open wound after detachment, such physical injuries may facilitate entry for microbial invaders. In the gills, heavy infestations may interfere with respiration. *Trichodina* sp. is thought to inflict large-scale mortalities among recently settled *Cardium edule*, particularly in hot summers.

Digenetic Trematoda are the most frequent and most important metazoan parasites of bivalves. Larval flukes are known from almost every marine bivalve species.

Unfortunately, many parasitologists have focussed their attention primarily or exclusively on taxonomical detail, host-parasite lists, life-cycle sequences and distributional

locality records. Ecological, physiological and biochemical aspects await thorough attention. In spite of numerous taxonomic descriptions, natural systematic relationships and evolutionary trends have remained insufficiently analyzed. In order to focus the picture and to straighten out the often winding, if not irritating, paths of misidentifications, excessive synonymities and incorrect quotations, the reviewer had to invest a tremendous amount of time, work and patience.

Agent effects produced by rediae or sporocysts in the primary gastropod host have been documented in Volume I (Chapter 12). Contrary to wide-spread opinion, trematode metacercariae are capable of causing a vast array of — often detrimental — physiological, biochemical, morphological and behavioural consequences in their respective hosts.

Among the Annelida, spionid polychaetes of the genus *Polydora* may cause substantial mortalities. Particularly their damage to oysters and mussels of commercial value has created considerable concern among aquaculturists. *Polydora* species cause 'mud blisters' and excavate U-shaped tunnels in molluscan shells. The magnitude of shell damage depends on agent and host species, as well as on environmental circumstances. Worm penetration in the region of the adductor muscle produces yellowish pustules or abscesses, presumably due to mud entering the muscle tissue.

Numerous copepod parasites have been reported from marine bivalves. A famous example is *Mytilicola intestinalis*. Although this species has attracted unparalleled attention, its significance as a potential disease agent remains to be critically documented and detailed. While *M. intestinalis* causes temporary metaplastic changes in the gut epithelium of *Mytilus edulis*, the degree of its pathogenicity appears to be low. Few copepod parasites are host-specific, but some clearly prefer a definite host. Most of the copepod/bivalve associations reported in the literature are characterized by a deplorable lack of conclusive evidence regarding the roles of agent and host.

Considerable information has also accumulated on the disease-causing potentials of Rickettsiae, Chlamydiae and Mycoplasmas among the micro-organisms, as well as the Porifera, Nematoda, Decapoda and Pantopoda.

Members of the Rickettsiae and Chlamydiae are obligate intracellular pathogens; Mycoplasmas, the smallest known free-living organisms. Similar to bacteria, all three groups contain members which may be capable of causing diseases in bivalves. Among the Porifera, boring sponges — while not true parasites — can become serious pests of bivalves, particularly oysters. Boring into shells and occasionally also affecting the soft parts, clionid sponges have been blamed for significant losses on oyster beds. The severity of clionid damage varies with the bivalve species concerned and appears to depend on shell microstructure. Effective control measures against sponge damage have not yet been developed. Nematoda thus far encountered in marine bivalves were all larvae. As far as is known, the degree of their pathogenicity to bivalves is moderate. Among the Decapoda, numerous brachyuran crabs live — as phoronts, mutualists, commensals or parasites — in the bivalve's mantle cavity, especially in tropical and subtropical waters. While some cases may qualify as quasi-parasitism, the disease-causing potential seems, in general, rather limited. Diseases due to associated pinnotherid crabs include emaciation, reduced filtering rates and damage to gills, palps and mantle. Pantopoda/bivalve associations may not be as rare as the sparse literature suggests. Destruction of gill and gonad tissues and damage to visceral mass, foot and palps have been reported.

The following animal groups have either received rather little attention or are unlikely to cause overt disease phenomena in bivalves, or both: Sarcomastigophora, Microspora, Protista *incertae sedis* (a provisional grouping of heterogenous forms), Protophyta, Mesozoa, Cnidaria, Turbellaria, Nemertea, Cestoda, Gastropoda, Bivalvia, Cirripedia, Amphipoda and Isopoda.

While the Sarcomastigophora (flagellates and rhizopods) do not seem to comprise true bivalve pathogens, members of the Microspora associate as parasites with marine Bivalvia. The evaluation of possibly detrimental effects due to Protista *incertae sedis* suffers from taxonomical and etiological uncertainties. Associations between bivalves and Protophyta (unicellular algae) are numerous and range from mutualism to parasitism. The algae are found in shells and soft parts. Only heavy infestation appears to evoke severe symptoms. Of considerable significance are dinoflagellate blooms ('red tides'). Ingested, dinoflagellate toxin ('paralytic shellfish poison') is a potentially serious health hazard to man (p. 615–616). Mesozoa have been found as parasites in gonads, mantle and gills of jingle shells; heavy infestations have seriously damaged the gonads (host castration). While many Cnidaria associate with bivalves, they appear to qualify mostly as phoronts (inquilines) or mutualists, rather than parasites.

Among the Turbellaria, Rhabdocoela and Alloeocoela establish symbioses with bivalves — mostly as commensals — but with the possibility of attaining parasite status in special situations, especially in cases of heavy 'infestation'. Some species of 'oyster leeches' are regarded as pests. A few Nemertea (genus *Malacobdella*) live as phoronts in the mantle cavity of several bivalve species. Except for irritation, damage to the host has not been reported. Cestoda, although recognizable by the naked eye as opaque bodies in bivalve tissues do not seem to cause overt diseases.

Attached externally to the bivalve's shell, species of Gastropoda (Fam. Pyramidellidae) suck on the host's body fluids. In cases of high parasitization this may cause shell deformities and interference with growth and development. In oyster and mussel farms, oyster drills have caused significant losses. For measures of biological control of drills see p. 802. Specialized boring Bivalvia may cause damage, and some have been labelled parasites. Among the Cirripedia, several barnacle species attach to bivalve shells. They qualify as phoronts or commensals (in a few extreme cases as parasites). The Amphipoda and Isopoda reported to be associated with bivalves are all phoronts and/or commensals, except *Cardiophilus baeri* which may be intermediate between commensal and parasite.

Major Diseases of Bivalves

Of the diseases of bivalves which have received appreciable attention from investigators we mention here the following.

Gill disease (p. 481), presumably a virosis — although experimental transmission of the virus has not yet been achieved — manifests itself first in the appearance of yellow spots. Subsequently, the disease causes destruction of gill filaments, impairment of nutritional and respiratory functions, heavy weight loss, and finally death.

Focal necrosis (p. 492), attributable to bacteria, or — more likely — to physiological stress (possibly in combination with detrimental bacterial activities), results in emaciation, gaping, and — prior to death — pronounced inflammation as well as

degenerative processes in the digestive gland. Necrotic foci have been shown to contain colonies of Gram-positive, rarely Gram-negative, bacteria.

Bacillary necrosis (p. 498), the main disease in artificial propagation of commercially important marine bivalves, is caused by bacteria of the *Vibrio* group. The bacteria cause reduced motility, extensive tissue lysis, granular necrosis, and death within 8 to 18 h after inoculation in larvae. The disease can be treated effectively with streptomycin or wide-spectrum antibiotics. The ecological significance of bacillary necrosis in natural larval populations requires critical analysis. In fact, the present concept of bacterial pathogenicity in free-living bivalves is in need of reappraisal.

Mycelial disease (p. 500) of oysters appears to be caused by an actinomycete. 'Mycelia' develop on the host's epithelia; they interfere with feeding and ultimately cause mortalities, sometimes of epizootic dimensions.

Shell disease (p. 511) due to fungus infection (*Ostracoblabe implexa, Althornia crouchii*), is widespread in bivalves of European waters. Initial stages of small white pecks on the inner shell surface develop into greenish knobs and warts; the warts coalesce and eventually cover one or both valves. In advanced stages the adductor muscle is weakened or even detaches from the shell; the bivalve loses weight and finally dies.

The highly contagious Malpeque Bay disease (p. 526), presumably produced by 'fungus-like protistans' (Labyrinthomorpha), can destroy whole oyster populations. All ages are susceptible. Infected oysters rapidly lose weight, growing and fail to spawn. The tissues reveal haemocytic infiltration, increase in number of 'brown cells' and heavy collagen deposits.

Amber disease (p. 528) involves severe damage to the Leydig-cell system and appears to be caused by agents referable to the Labyrinthomorpha.

For the Winter disease (p. 531) — a main problem in the New South Wales oyster industry — the causative agent has still to be determined. Geographically confined to the New South Wales coast, the disease may attain epizootic proportions in autumn and winter. It is characterized by yellow spots on the body surface, abscesses in the stomach wall, ulceration on the epithelium of gills, palps, mantle and gonads, as well as heavy haemocytic infiltration.

Dermocystidium disease (p. 532) involves severe emaciation, gaping and pale appearance of the digestive gland. Advanced stages lead to damage in the shell-secretion mechanism, mantle shrinkage and cessation of shell growth. Finally, the causative agent, *Perkinsus marinus,* may completely obliterate the haemolymph sinuses and obstruct all but the largest blood vessels. Adult oysters are most susceptible immediately after spawning. Mortality rates increase with host age and size. *P. marinus* is transmitted directly from oyster to oyster. The environmental requirements of *P. marinus* have received appreciable attention (p. 538 ff.).

Abers disease or 'digestive-gland disease' (p. 553), caused by *Marteilia refringens* of the phylum Ascetospora, leads to progressive emaciation. The digestive gland turns brownish to pale yellow, the mantle becomes transparent and shell growth ceases. In heavily infested oysters the visceral mass appears shrunken and 'slimy'. First symptoms occur in autumn of the year of planting; subsequently, the condition tends to aggravate until death ensues in autumn of the following year. Significantly, *M. refringens* has also been recorded from apparently healthy individuals with normal gonads. The conditions governing agent virulence and host defense remain to be studied in depth.

Delaware Bay disease (p. 559) significantly affected the whole oyster industry in Chesapeake Bay (USA). Its causative agent was identified as *Haplosporidium nelsoni*. Unspecific gross symptoms include mantle recession, gaping, emaciation, pale digestive gland, and occasional pustule formation on the inner shell surface; microscopic symptoms include diapedesis, reduction in number of phagocytes, increase in number of hyaline haemocytes, phagocytosis, fibrosis, cellular infiltration, abscess formation, ulceration, excessive pigment-cell formation, mechanical disruption, pyknosis and necrosis. Terminal infestations cause massive pyknosis of nuclei and tissue necrosis prior to death. Environmental requirements of *H. nelsoni* have been subject to appreciable research (pp. 566).

Seaside disease (p. 571), caused by *Haplosporidium costale*, is characterized — in contrast to Delaware Bay disease (which kills oysters throughout the year) — by pronounced seasonality and timing. With the first symptoms beginning in February, mortalities peak in early June and cease in early July.

Denman Island disease (p. 603) was first observed in Baynes Sound on Denman Island (B. C., Canada). It usually develops in early spring at water temperatures above 8 °C and disappears about mid-July at 18 °C. The disease is highly contagious and resembles Malpeque Bay disease (pustule formation, occurrence of 'microcells'). Its agent belongs to the genus *Labyrinthula*.

Abnormalities and Neoplasia

Structural abnormalities reported from marine bivalves concern both soft parts and shell. Deviations from normal soft-part structures — such as malformations, hypertrophy, hyperplasia, metaplasia and neoplasia (for definition of terms consult Vol. I, Chapter 2) — have received considerable attention, but are often difficult to distinguish properly in molluscs. Damage to shells include mechanical injuries inflicted by parasites, commensals or other causes (e.g., blisters, excavations, holes) as well as malformations (e.g., valve asymmetry, shell deformation). Abnormal pigmentation, tissue discolouration, lesions, etc. have also been reported.

The most conspicuous structural abnormalities, tumours, were often interpreted to represent neoplasia, until histological examination revealed most of them to be inflammatory responses to microbial or parasitic infestations. Neoplasia in bivalves is in need of critical research.

Developed for vertebrates, tumour terminology cannot be applied to invertebrates without further qualification. While there is some indication that neoplasia in bivalves may be correlated to water pollution, particularly to the presence of carcinogenic substances, neoplastic disorders have also been reported from bivalves collected far away from polluted areas. Antineoplastic substances have been isolated from several bivalves and other marine invertebrates.

Comments on Chapter 14: Diseases of Mollusca: Amphineura

In contrast to oysters and mussels, the ca. 1,000 marine species of Amphineura are of little or no commercial interest. While an appreciable number of animals have been reported to associate intimately with these molluscs, the information available on their diseases is scarce. There appear to be only a few microbial and metazoan parasites which may cause true diseases, and only a small number of protozoans are suspected or have

been shown to act as disease agents. Nevertheless, it seems only fair to warn that the few instances of disease reported may reflect insufficient research, rather than absence of disease.

Thus far, the attention devoted to potential disease-causing entities and circumstances in Amphineura has been quite limited. It was directed mostly on taxonomy, life cycles and distributions of the associates. There is a general lack of in-depth investigations concerning inter-organismic dependencies, flow patterns of energy and matter between the partners involved, agent virulence, host defense, and disease etiology. Sound categorization of amphineuran associates into phoronts, commensals, parasites or mutualists (Vol. I, pp. 19–21) is mostly not yet possible.

Agents shown or suggested to be capable of causing disease in Amphineura are Apicomplexa, Ascetospora and Ciliophora among the Protistans, as well as Platyhelminthes, Gastropoda and Arthropoda among the metazoans.

Abnormalities observed in Amphineura cover relatively rare deviations from the normal number and structure of shell plates (e.g., hypomery).

Comments on Chapter 15: Diseases of Mollusca: Scaphopoda

Comprising some 300 marine species, the Scaphopoda have received even less attention than the Amphineura. Only a few protozoans and 1 trematode have been shown thus far to parasitize scaphopods, causing demonstrable, negative deviation from the normal state.

Among the protistans, 2 apicomplexans and 1 ascetosporan may cause cellular damage, such as nuclear and cytoplasmic hypertrophy; displacement of host cell nuclei; cell atrophy; and epithelial deformation. Metazoan parasites appear to be extremely rare. Among the trematodes, heavy *Cercaria prenanti* infestation causes complete castration. Future research may be expected to reveal more disease agents and more disease phenomena of scaphopods.

13. DISEASES OF MOLLUSCA: BIVALVIA

G. LAUCKNER

Among the marine invertebrates, bivalve molluscs constitute the group of major economic importance. Oysters, mussels, scallops, cockles and various species of clams, worth millions of dollars, are harvested each year in various parts of the world. Many species, particularly oysters, have been cultivated on a commercial scale for decades.

Both long-term fluctuations in abundance and sudden mass mortalities in bivalve populations have been witnessed. Epizootics in commercially exploited species have repeatedly struck the related industries, sometimes causing their virtual extinction.

Metazoan parasites of marine bivalves have been recognized for long time, but our knowledge on their microbial and protozoal diseases has primarily evolved during the past 2 or 3 decades. It clearly parallels the economic importance of the species studied. Literature on oyster diseases is most abundant, that on mussel and clam diseases far less voluminous (Sindermann, 1968b, 1970a, b, c, 1977). Little is known about the diseases of unexploited bivalves. However, many ot these are of indirect economic importance because they serve as fish food or occupy an otherwise relevant position in the marine food web. In total, our present knowledge of diseases of marine bivalves by far exceeds all available information on diseases of the other marine invertebrate groups together.

Focal points of research on shellfish diseases have changed in the course of the decades. Early investigators have mostly concentrated on the study of parasitic (metazoan) diseases of marine bivalves, whereas today microbial and protozoal diseases are being recognized as the more serious threats, and are studied more thoroughly. This apparent inbalance has led to numerous misconceptions not only in the past, but continues to obscure many field data and observations on longevity and recruitment of natural bivalve populations.

The early authors have frequently overemphasized the role of metazoan parasites in bivalve pathology. Sometimes, a deliquent to be accused of otherwise unexplainable molluscan mass mortalities was needed, and was promptly presented, by hasty, uncritical workers, in the shape of some more or less innocent macroscopic parasite. Such scientific superficiality has virtually prevented, in several documented cases, scrutinized search for the truly responsible microbial or protozoal agent. Today's shellfish biologists and ecologists, on the other hand — being confronted with spectacular bivalve epizootics and ever new protistan and microbial pathogens — generally tend to underrate the role of metazoan parasites as disease agents. It is well established that many macroscopic parasites are capable of considerably debilitating their hosts and causing constant (although mostly unspectacular and, hence, unnoticed) attrition to molluscan populations. The often repeated, seemingly logical phrase, 'It cannot be to the interest of a parasite to harm its host', is

nothing but claptrap, easily disproven by sound investigational work. Examples will be presented in this review.

Among the micro-organisms, viruses and fungi have been identified as serious pathogens of free-living, cultivated bivalves. Bacteria and fungi pose problems in bivalve hatcheries. Protistans — mainly members of the phyla Labyrinthomorpha, Apicomplexa and Ascetospora — have caused and continue to cause epizootics in commercially exploited lamellibranch species, causing tremendous financial losses.

The present review is primarily concerned with these 'biotic' diseases. There is increasing evidence, however, that environmental stress — and man-made water pollution in particular — may lead to debilitation and disease in numerous marine animals including bivalves (Fig. 13-1; Sparks, 1972; Gardner and co-authors, 1975; Fries and Tripp, 1976;

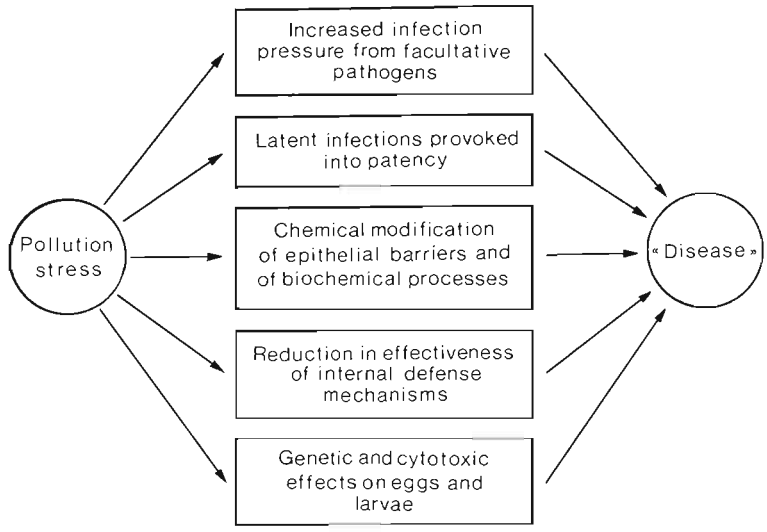

Fig. 13-1: Relationship between pollution stress and disease. (After Sindermann, 1980.)

Hodgins and co-authors, 1977; Sindermann, 1979a, b, 1980; Yevich and Barszcz, 1980; and others).

When considering the effects of pollution stress, one must take into account that bivalves are also subjected to biological (reproductive, competitional) stress and natural environmental stress (changes or marginal values of salinity, temperature, oxygen tension, etc.). Natural environmental stress evokes a variety of physiological responses (Bayne, 1972, 1973a, b, c, 1975, 1976; Bayne and Livingstone, 1977; Bayne and co-authors, 1978b).

Pollution stress and natural environmental stress act synergistically or, rarely, antagonistically, and largely determine an organism's response to disease-causing agents. The effect of stress on the parasite or disease agent is another factor to be considered (Esch and co-authors, 1975). Aside from the direct cytotoxic, mutagenic and teratogenic effects of pollutants on susceptibles, there are manifold interactions between pollution stress and the manifestation of disease. With recent studies, a picture arises that has been characterized by Sindermann (1980, p. 5) as follows:

"The overriding principle which emerges is that whether etiology is of infectious or non-infectious nature, pollution stress can be a major contributing factor in the occurrence of disease in degraded habitats."

DISEASES CAUSED BY MICRO-ORGANISMS

Agents: Virales

Viruses are among the most destructive pathogens of vertebrate and invertebrate animals. The discovery of viruses in marine molluscs is a fairly recent event. In an exhaustive review on viruses in invertebrates, edited by Gibbs (1973), a single paper referring to a viral disease of cephalopods was quoted (Vol. III). Meanwhile, several viruses or virus-like entities, pathogenic or non-pathogenic, have been reported from bivalves. Some of these agents are detectable only in molluscs that are suffering from another disease or from environmental stress, such as pollution. In such cases, the role played by the viruses is difficult to evaluate. At least in some instances, they appear to be 'stress parasites', becoming demonstrable only in hosts living under adverse conditions of physiological stress, as is the case in some crustacean viruses.

Some molluscan viruses are morphologically similar to oncogenic viruses found in homoeothermic animals. The range of infectivity of these agents has not yet been determined and, in particular, it is not known whether any of them are able to produce disease in homoeothermic animals including man (Rosenfield, 1976a, b). In the past, several apparently contagious molluscan diseases, which had caused high losses or had even wiped out entire bivalve populations, have been attributed to infection by unknown viruses until other organisms were identified as causative agents. Other bivalve diseases, attributed to readily identified invasive organisms, subsequently turned out to be of viral etiology (see below).

Farley (1978) has summarized the information pertaining to viruses and virus-like lesions in marine molluscs and has attempted to categorize them systematically into appropriate families. From their morphology and development, Virales occurring in bivalves may be grouped with the Pedoviridae, Papovaviridae, Herpetoviridae, Togaviridae, Retroviridae, Paramyxoviridae and Reoviridae. For general information on the classification, nomenclature and main characteristics of viruses, the reader is referred to Wildy (1971) and R. E. F. Matthews (1979).

During a study on the effects of elevated water temperature on growth and survival of American oysters *Crassostrea virginica* from Maine (USA), it was found that individuals kept at 28 to 30 °C suffered higher mortality than controls kept at 18 to 20 °C. Dead oysters had dilated digestive diverticula, cellular infiltrates in the vesicular connective tissue about the haemolymph sinuses, and, in advanced cases, massive cellular aggregates at these sites. Histological examination revealed intranuclear inclusion bodies within the cells around the haemolymph sinuses, comparable to Cowdry-Type A inclusions associated with herpesvirus infections in other animals. Electron microscopy demonstrated typical herpes-type virus particles — hexagonal in shape, 70 to 90 nm in capsid diameter, and with a single envelope — in the nuclei of oyster cells containing inclusions. Some particles contained a dense nucleoid, others were empty. Some were seen to have several fine filaments extending through the coat from a dense, eccentrically placed nucleoid,

which resulted in a flagellate appearance. The nuclear inclusions sometimes contained microtubules, 45 to 55 nm in diameter.

Two months after the start of the experiment, 31 of 60 oysters in the high-temperature set had died, as compared to 11 of 60 individuals in the low-temperature control group. In the absence of disease, temperatures of 28 to 30 °C have no adverse effect on oyster survival and growth. Thus, the higher mortality in the experimental set was probably attributable to the herpes-virus infection. The oysters had been taken from the Marsh River near Wiscasset, Maine, a locality to which they had previously been transplanted from a site in the Piscataqua River near Elliot, Maine. Subsequent gross inspection of oysters from the latter site revealed a pallor of the digestive gland in 1 of 200 individuals. Electron microscopy confirmed the presence of inclusion bodies and virus particles in the single grossly diseased oyster. Consecutive sampling of oysters from Marsh River indicated seasonal variation in the prevalence of overt virus infections. It was concluded that the agent is enzootic under ambient temperature conditions, and that elevated temperatures appear to favour spread of the infection or activation of the disease from an occult to an overt phase, or both (Farley and co-authors, 1972).

The temperature-influenced behaviour of the oyster virus was considered to be relevant to energy-related developmental programs and aquaculture projects in the middle U.S.-Atlantic and other coastal areas. It suggests that environmental modification, in this case elevated water temperature, may favour the spread, activation or enhancement of viral infection in aquatic animals (Rosenfield, 1976b).

The virosis described by Farley and co-authors (1972) is the first virus disease detected in bivalves and the second herpes-type virus infection recognized in any invertebrate species. Cowdry-Type A intranuclear inclusions, similar to those found in *Crassostrea virginica,* were seen in individuals of *C. gigas* imported into North Wales for cultivation. Their presence, in the oysters, was associated with an enzootic disease in summer (Alderman, 1980). The author's tentative diagnosis of a possible herpes-type virus being implicated with the disease has not yet been confirmed by electron microscopy or *in vitro* studies (Buchanan and Richards, 1982). Other possible herpes-type infections, based on the similarity of Cowdry-Type A intranuclear inclusions, have been seen in the mantle epithelium of juvenile and adult *Crassostrea virginica* (Meyers, 1981), in *Ostrea edulis* (Alderman, in Farley, 1978) and *Mercenaria mercenaria.* Observations on gonadal tumours in *M. mercenaria* indicate the presence of herpes-type intranuclear inclusions in affected cells (Barry and Yevich, 1972; Farley, 1978; Kern, in Farley, 1978).

A second viral entity, related to the Papovaviridae and most similar to *Papillomavirus,* has been identified as the causative agent of 'ovacystis' disease in *Crassostrea virginica.* The condition is characterized by massive hypertrophy of ova and gametocytes, which contain large Feulgen-positive (DNA) granular masses in the nucleus. While normal ova attain a maximum size of about 75 μm, hypertrophied cells are up to 500 μm in diameter. The amount of cytoplasm is extremely scanty. Electron microscopy reveals the presence of dense accumulations of virus-like particles within the nuclei of hypertrophied cells. The non-enveloped icosahedral virions are uniform in size (53 nm). Replication and assemblage of viral material is intranuclear and sometimes associated with the presence of intranuclear filaments and microtubules, 20 nm in diameter. Paranuclear and intracytoplasmic particles are evident in lysing cells. Particles are also found in extracellular spaces and adhering to the glycocalyx of developing ova (Farley, 1973, 1976a, 1978). Ovacystis

disease has a very low incidence in oysters from Long Island, New York, and appears to have little pathological significance (Meyers, 1981). Similar histological lesions have been observed in *C. gigas, C. commercialis (= Saccostrea cucullata), Ostrea lurida* and *O. edulis* (Bonami, Farley, Kern and Wolf in Farley, 1978). Feulgen-positive, intranuclear inclusions, suggestive of papovavirus infection, occur in the connective tissues, haemocytes and gill epithelium of *Mya arenaria*. Associated non-enveloped, icosahedral virions, 40 to 45 nm in size and most closely resembling *Polyomavirus,* replicate and assemble in the nucleus and cause some hypertrophy of affected cells (Farley, 1976b, 1978; Walker, in Rosenfield, 1976b). Inclusions, similar to the above-described but not associated with hypertrophy, have been observed in *Macoma baltica* and *Mya arenaria* haemocytes, and unidentified virus-like, Feulgen-positive inclusions occurred in *M. baltica* gametocytes (Farley, 1977, 1978; Harshbarger and co-authors, 1979).

'Maladie des branchies' ('gill disease') has been identified as the cause of recurrent mass mortalities of Portuguese oysters *Crassostrea angulata* in France since 1966. In some years, the disease affected over 70 % of the population, with resultant mortalities reaching up to 40 %. Maximum losses on oyster grounds in the Marennes-Oléron and Arcachon regions on the French Atlantic coast occurred in 1967 and subsequently decelerated. In 1968, survivors of the previous epizootics recovered from the disease, showing cicatrization of the old lesions. At present, the disease persists at enzootic levels in oysters on natural beds in Spain and Portugal (Deltreil, 1969, 1971; Franc and Arvy, 1970; Comps, 1972a, 1978).

Gross symptoms of the disease have been described in detail by Arvy and Franc (1968), Besse (1968) and Marteil (1968, 1969). Besse (1968) found diseased oysters to be concomitantly infested with *Trichodina* sp. (see section 'Agents: Ciliophora'). Although *Trichodina* is potentially capable of causing gill erosion, the lesions observed by Besse in oysters from Seudre are possibly not attributable to these ciliates. Alderman and Gras (1969) and Gras (1969) discussed the possible involvement, in the disease, of a 'fungus' related to or identical with *Dermocystidium marinum* (= *Perkinsus marinus*) (see section 'Agents: Apicomplexa'). Arvy and Franc (1968) and Franc and Arvy (1969, 1970) identified *Thanatostrea polymorpha,* a protistan parasite of uncertain systematic affinities (probably a labyrinthulid), as the presumptive etiological agent of gill disease. Caty (1969) observed membraneous proliferations of unknown nature, formed by oyster tissue incubated in thioglycollate medium, which Key and Alderman (1972), however, identified as lipids released by tissue breakdown. Finally, Comps and Duthoit (1976) demonstrated the presence of an iridovirus in the gill tissues of diseased *Crassostrea angulata.* It is now generally assumed that 'gill disease' is a virosis, although experimental transmission of the virus and production of the disease have not yet been achieved.

On the gills and palps, the first symptom is the appearance of one or several yellow spots. These increase in size and the tissues at the centre die, become brown and eventually leave a perforation (Figs 13-2 and 13-4, a), which enlarges to eventually cause a deep identation and, in advanced stages of the disease, total destruction of affected filaments (Figs 13-3 and 13-4, b). On the adductor muscle and on the mantle, yellow or green pustules develop and mantle perforations may occur as on the gills (Alderman and Gras, 1969; Comps, 1969, 1970a).

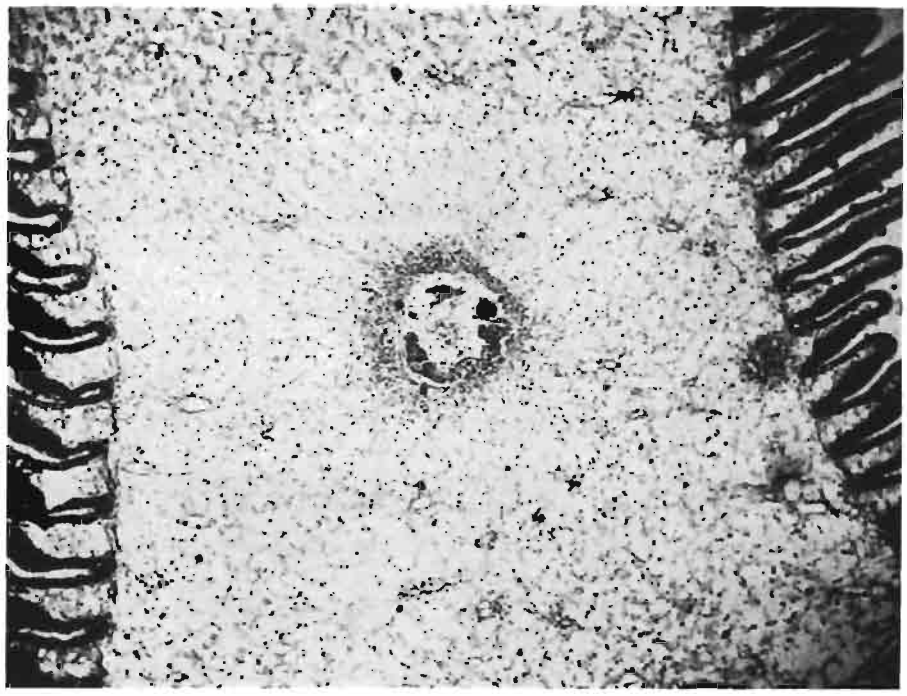

Fig. 13-2: *Crassostrea angulata*. Longitudinal section of palp showing initial stage of 'gill disease', × 46. (After Comps, 1969.)

Fig. 13-3: *Crassostrea angulata*. Cross section of gill filaments showing different areas affected by 'gill disease'. In the centre, filaments are entirely destroyed, × 44. (After Comps, 1969.)

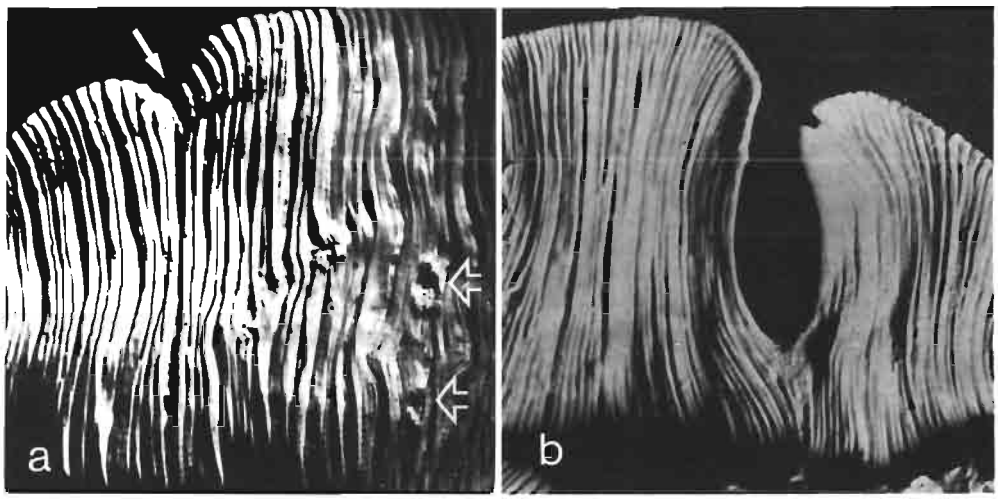

Fig. 13-4: *Crassostrea gigas*. 'Gilll disease'. (a) Perforations (halfway down lamella) and early stage of marginal indentation; (b) advanced indentation and necrosis. (After Comps, 1970a.)

For purposes of comparison, a classification scheme was proposed to describe the severeness of the affection (Marteil, 1968; His, 1969):

Stage I: Presence of an indentation or frayed contours on a single gill filament;
Stage II: Profound and numerous alterations on one or more filaments, which distinctly reduce the respiratory surface;
Stage III: Total or almost total degeneration of the entire gills.

In diseased *Crassostrea angulata*, necrotic tissue changes severely impair the particle-collecting and respiratory functions of the gills. Using a modification of the method proposed by Cole and Hepper (1954), His (1969) demonstrated massive depressions in the methylene blue clearance rates in affected oysters (Fig. 13-5). Reduced food uptake resulted in considerable weight loss, which amounted to 65.8 % in individuals with Stage III of gill disease (Table 13-1). Emaciated oysters eventually die.

Electron microscopy demonstrated that the large globular cells, 30 to 40 µm in diameter, which become apparent during the necrotic changes of the gill tissues and have previously been interpreted as stages in the life cycle of *Thanatostrea polymorpha*, are in fact hypertrophied host cells containing viral entities (Fig. 13-6). The virions are icosahedral in shape and have a diameter of about 300 nm. A central electron-dense core, 190 nm in diameter, is surrounded by an electron-light zone followed by another dense layer, 45 nm in thickness. Two unit membranes, separated by a clear zone, enclose the particle. Feulgen-positive staining reaction suggests the presence of DNA. The particles assemble in cytoplasmic inclusions by budding through *de novo* membranes at the periphery of the virogenic stroma. There is no evidence for nuclear involvement. The various properties, as well as its assemblage within the cytoplasm, characterize the entity as a member of the Iridoviridae (Comps and Duthoit, 1976, 1979; Comps, 1978). It bears some resemblance to the lymphocystis virus of fishes.

'Maladie des branchies' also affects *Crassostrea gigas*, although to a lesser extent (Fig. 13-4). The lesions never exceeded Stage II, which is suggestive of partial resistance. Pacific

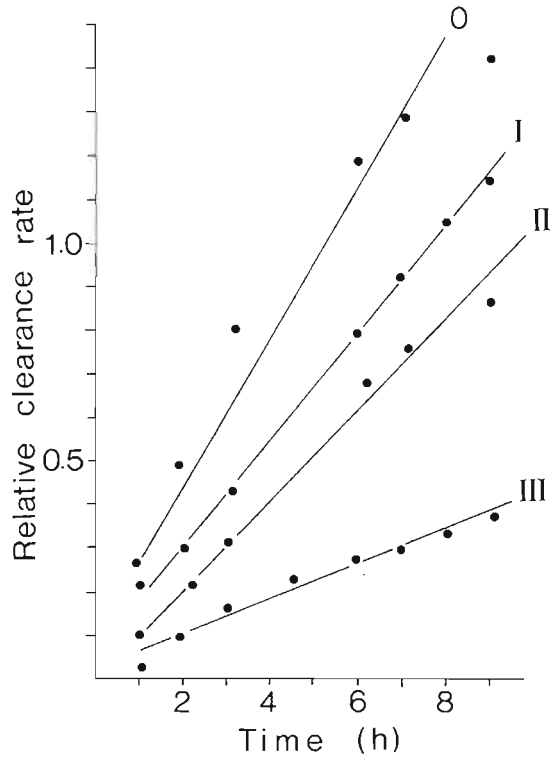

Fig. 13-5: *Crassostrea angulata.* Relative methylene blue clearance rates of healthy oysters (0) and individuals with Stages I to III of 'gill disease'. (Based on His, 1969.)

Table 13-1
Crassostrea angulata. Average body-weight loss as a consequence of 'gill disease' (n = 150 oysters)
(Recalculated from His, 1969)

	Healthy oysters	Oysters with 'gill disease' Stage I	Stage II	Stage III
Fresh wet weight (g), whole animal	29.6	28.1	26.6	25.7
Tissue-dry weight (g)	0.73	0.66	0.49	0.25
Loss of tissue-dry weight (%)		9.6	32.9	65.8

oysters, transplanted into French waters in 1968 from Japan, exhibited lesions in 27 % of the individuals, while of a lot obtained from Korea, 56 % acquired the disease. Interestingly, the incidence in *C. gigas* imported from British Columbia was only 14 % (Marteil, 1969; Comps, 1970a). It appears that *C. gigas* cultivated in France has subsequently acquired complete resistance to gill disease since from 1969 to 1971 no gill-diseased individuals of this species have been found, although mortalities among *C. angulata* run as high as 70 % (Comps, 1972a). It is likewise possible that the mortalities among *C. gigas* are, in fact, attributable to another virus, unknown at that time and discovered by Comps and Bonami in 1977 (see below). Comps (1980a) concluded that 'maladie des branchies', in its characteristic form, is a specific disease of *C. angulata.*

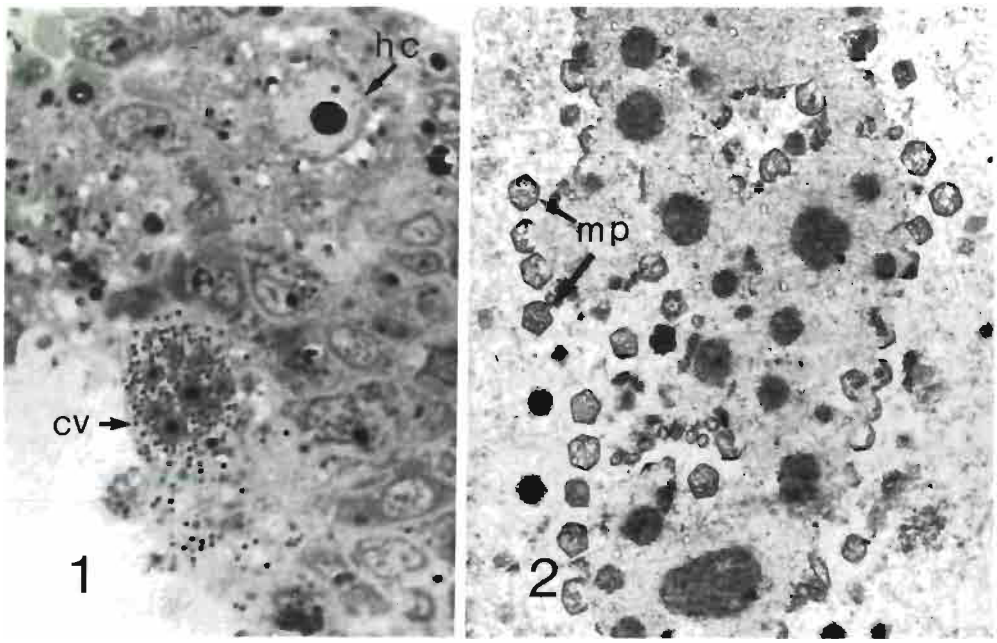

Fig. 13-6: *Crassostrea angulata*. Viral particles associated with 'gill disease'. (1) Hypertrophied cell (hc) and cell (cv) with virus-like particles, × 1,600; (2) section of hypertrophied cell showing particle formation and mature particles (mp), × 12,000. (After Comps, 1978.)

Gill disease has also been reported from *Ostrea edulis* and, although lesions were seldom severe and surpassing Stage II, recurrent mass mortalities of flat oysters have occurred in France in 1961, 1962, 1966, 1967, 1969 and 1970 (Gras, 1969; Lasserre, 1969; Gras and Herrbach, 1971; Herrbach, 1971a; Marin, 1971; Marteil, 1971b). Gras (1971), Herrbach (1971b) and Marteil (1971a) discussed the mortalities in relation to adverse environmental factors.

On gross inspection, 'l' affection branchiale' (Comps, 1970b) of *Ostrea edulis* appears to produce the symptoms described for 'maladie des branchies', but closer examination reveals major differences. Comps associated the disease with the presence of a protistan parasite in the gill epithelium and the digestive tract. The agent was later described as *Marteilia refringens* by Grizel and co-authors (1974a) (see section 'Agents: Ascetospora'), but its etiological relationship with the gill affection could not be established. 'Affection branchiale' may be due to a specifically unidentified haplosporidian, described from the gills of *Ostrea edulis* by Comps (1976b), and only known in its plasmodial stage. Lesions produced by the latter agent were essentially identical to those reported by Comps (1970b).

In 1970, epizootic *Crassostrea angulata* mortalities, caused by another iridovirus, occurred in the estuary of Marennes-Oléron on the French Atlantic coast and were paralleled by a similar event at Étel, Bretagne. By 1971, the disease also touched the Arcachon region. Vast mortalities led to an almost total extermination of *C. angulata* in French Atlantic waters in 1973. Electron microscopy revealed the presence of icosahedral viral particles, 350 nm in diameter and with an electron-dense core, 190 nm in diameter,

within the cytoplasm of connective-tissue cells (Comps and co-authors, 1976; Comps, 1978). The agent was in all respects very similar to the virus associated with 'maladie des branchies'. *C. gigas* proved to be entirely resistant to the disease and, after the extermination of *C. angulata*, has replaced the latter species in France (Marteil, 1976; Bonami, 1977).

It did not take long for *Crassostrea gigas* cultivated at Arcachon and Marennes-Oléron, to contract a viral disease 'of its own'. In 1977, a 15 % mortality was noticed in oysters kept in a purification plant at Arcachon. Similar to *C. angulata*, affected Pacific oysters exhibited virtually no external disease signs, except for a grayish discolouration of the visceral mass in some cases. Histological examination, however, revealed considerable degeneration of connective tissues and the presence of atypical cells.

Electron microscopy demonstrated the presence of icosahedral viral particles, 350 nm in diameter and with a 250 nm electron-dense core, in the cytoplasm of affected cells. With respect to its morphology and morphogenesis, the virus closely resembled the two former types (Comps and Bonami, 1977; Comps, 1978, 1980a; Comps and Duthoit, 1979).

Leibovitz and co-authors (1978) described a serious disease of hatchery-reared larval *Crassostrea gigas*, characterized by velar and mantle erosion. A mycotic etiology was initially suggested. Closer inspection of serial sections showed affected velar epithelial cells to be vacuolated, poorly defined, and containing intracytoplasmic inclusions detectable at the light microscope level. Electron microscopy revealed the presence of viral particles, predominantly hexagonal in profile and averaging 228 ± 7 nm in diameter. The particles were found only in the cytoplasm of velar epithelial cells. Affected cells were seen to undergo diapedesis, i.e., extrusion through the velar epithelium into various parts of the mantle cavity (Elston, 1979, 1980a). The virus was not classified but, as pointed out by Comps (1980a), bears close resemblance to the above-described agents from adult *C. angulata* and *C. gigas*.

This is the first report of a presumptive virus infection in larval bivalves, but its role in development of the disease of *Crassostrea gigas* veligers is not clear. It must be emphasized that the pathogenicity of none of the above Iridoviridae has been demonstrated conclusively by experimental transmission to healthy bivalves and artificial production of disease. In the case of the larval *C. gigas*, a 'fungus' of dubious affinities has been isolated from diseased individuals (Leibovitz and co-authors, 1978; Elston, 1980b). Thus, the virus may be acting secondarily to, or in concert with, other pathogens. Loosanoff (1954, 1974), however, suspected that viruses may be directly responsible for mass mortalities of bivalve larvae reared under laboratory conditions, as well as in nature. Hill and Alderman (1979) were able to produce overt disease signs in bivalves experimentally infected with a reovirus (see below).

A reovirus sharing immunological properties with the infectious pancreatic necrosis (IPN) virus of fish has been isolated from homogenates of the digestive gland of *Tellina tenuis*. The icosahedral, RNA containing particles have a diameter of approximately 59 nm. The exact location of the virus is not known but there is some evidence of infection of haemocytes. The virus was readily propagated in fish-cell lines at 14 °C and several of its *in vitro* properties were determined. Single inoculations of tissue-culture grown virus into the tank water provoked infections in individuals of *T. tenuis* and *Crassostrea gigas*. The virus was reisolated from experimentally infected hosts 3 months after inoculation. No mortalities of adult hosts could be related to the infection, but a steady increase in

specifically fluorescing cells in the digestive gland was observed. However, in one case viruses were isolated from a population of oysters used as brood stock at a hatchery where abnormally high mortalities were being experienced with larvae. The presence of the virus in the larvae was confirmed, but conclusive evidence of a correlation between infection and larval mortality is awaited (Hill, 1976a, b; Underwood and co-authors, 1977). An experiment testing the pathogenicity of two different strains of the virus under laboratory conditions was not quite conclusive. Viral uptake by juvenile *Ostrea edulis* was much higher at 8 to 10 °C than at 16 to 18 °C. Histopathological effects were readily detectable. They consisted of a general development of tissue edema, with the maximum effect being observed in the digestive gland where haemocytic infiltration and connective-tissue necrosis occurred. In both experiments there was a noticeable loss of meat quality and loss of digestive-gland pigmentation in the inoculated groups. The effects were more pronounced in individuals from the low-temperature experiment. Considerable deterioration of the tissues and mortalities also occurred in the control groups, possibly due to food shortage, and made the results difficult to interpret (Hill and Alderman, 1979).

It would be interesting to see whether the infection of molluscs with the reovirus parallels IPN disease in fish. If so, one may anticipate an asymptomatic carrier state and transovarial transmission, which are characteristic features of IPN disease.

Twelve further field isolates of the *Tellina* virus, representing 4 or 5 distinct serotypes, have been obtained from *T. tenuis, Crassostrea gigas, C. virginica, Ostrea edulis* and *Mercenaria mercenaria,* as well as from gastropods *Patella vulgata* and *Littorina littorea* (Hill, 1976b). *T. tenuis* may harbour, in the cytoplasm of digestive-gland secretory cells, another similar virus, which is distinguished from the former (59 nm) by its larger size (66 to 68 nm). This agent is, however, actually located in rickettsial pathogens infecting the clam's digestive gland, and may be a phage (Buchanan, 1973, 1978, 1979b; see section 'Agents: Rickettsiae, Chlamydiae and Mycoplasmas'). Another reo-like virus, about 79 nm in diameter, has been isolated from homogenized tissue of juvenile *C. virginica* obtained from a hatchery in Long Island Sound, New York. Virus replication occurred in various fish-cell cultures, with progressive cytopathic effects becoming apparent after successive passages (Meyers, 1979a). Molluscan tissue and organ culture, as described by Vago and Chastang (1960), Tripp (1963), Perkins and Menzel (1964), Jones (1966), Li and co-authors (1966), Tripp and co-authors (1966), Vago (1967), Bayne (1968), Cecil (1969), Cousserans and co-authors (1974), Cousserans (1979), and others, may become a promising tool in future studies on molluscan viruses. Graves (1980) described a method for the extraction of DNA from oysters, which could be used to grow viruses or produce lesions in hosts and thereby circumvent the need for tissue cultures.

'Viral particles', seen by Bonami and co-authors (1971) and Grizel and co-authors (1974a) in the 'tertiary cells' (spores) of *Marteilia refringens* infesting *Ostrea edulis* (see section 'Agents: Ascetospora') might be taken as suggestive of hyperparasitism. However, as pointed out by Perkins (1976c), these particles actually represent 'haplosporosomes', which are unique cytoplasmic organelles of unknown significance, found exclusively in Stellatosporea. However, 'true' virus-like particles were seen side by side with haplosporosomes in cells of a yet unnamed balanosporidan parasite of *O. edulis* (Vivarès and co-authors, 1982; Fig. 13–44).

Repeatedly, viruses have been implicated with molluscan tumour genesis. Viruses from oncogenic groups occur in oysters and other bivalves, but, with a single exception (see

below), have not yet been associated with neoplastic disorders in these invertebrates (Farley, 1977).

Particles resembling viruses have been found in the nucleus of germinoma cells in *Mya arenaria* from Searsport, Maine, but the suspected viral nature of these tumour-cell inclusions has not yet been confirmed. Non-enveloped viral particles, 55 nm in diameter and resembling papovavirus, filled the nucleus and marginated the chromatin of non-tumorous *M. arenaria* cells resembling atypical haemocytes (Harshbarger and co-authors, 1979). Another papovavirus, producing lytic disease in germinal tissues of *Crassostrea virginica* (Farley, 1973, 1976a, 1978), exhibited no relation to epizootic neoplasia displayed by these oysters. Some herpes-virus infections are known to be associated with lymphoproliferative disease in homoeothermic vertebrates. As discussed by Farley and co-authors (1972), a lytic disease, produced by a herpes–type virus in *C. virginica*, may also have a proliferative component, manifesting itself by the presence of cellular aggregates forming around haemolymph sinuses and in the vesicular tissue. The origin of these aggregates is not clear, but they appear to be derived from haemocytes. It is also not clear whether the cell aggregates represent a self-limited reactive response, or a neoplasm. However, there is little if any evidence to support this contention (Farley, 1976a).

Recently, Appeldoorn and Oprandy (1980) have demonstrated a causal relationship between the occurrence of haematopoietic neoplasia in *Mya arenaria* and the presence of an (initially uncharacterized) virus in affected clams. Infections and tumours were produced experimentally by injecting clams with purified viruses obtained from neoplastic cells. Viral entities were, in turn, reisolated from these experimentally infected clams. Most of the inoculated clams developed neoplasia within 2 months. Repeated passage of the virus was successful. Subsequently, the agent was found to resemble B-type retroviruses. It is enveloped, pleomorphic, and approximately 120 nm in size with an eccentric nucleoid, 60 to 70 nm in size. Particles with central nucleoids, averaging 80 nm in diameter, were also seen. *In vivo* bleeding techniques made possible the non-destructive diagnosis of haematopoietic neoplasia in *M. arenaria*, the accuracy of these methods depending on disease severity. There was a distinct positive correlation between the degree of tissue involvement and the number of circulating neoplastic haemocytes (Oprandy and co-authors, 1981; Cooper and co-authors, 1982).

Since retroviruses have been associated with haematopoietic neoplasms in mice, fowl and cats, Oprandy and co-authors (1981) concluded that comparable lesions observed in soft-shell clams could also be caused by a retrovirus. *Mya arenaria* tumours have previously been believed to be caused by oil pollution (see section 'Neoplasia'). The experiments conducted by Appeldoorn and Oprandy (1980) and Oprandy and co-authors (1981) represent the first successful transmission of neoplasia in a marine invertebrate species and the first reliable demonstration of involvement of a virus in the production of neoplasia in molluscs.

As evidenced by the available information, the role played by viruses in the development of disease in marine bivalves is not yet readily understood. Particularly, their role of being either primary pathogens or secondary stress parasites need to be investigated. Another point meriting further study is the question whether molluscan viruses are capable of replication and disease production in homoeothermic animals including man.

Considerable impact, on the other hand, results from the capability of bivalves to accumulate human enteroviruses from the water. Numerous studies have shown that

molluscs can serve as virus carriers and can accumulate high levels of particles (Liston and co-authors, 1968; Fries and Tripp, 1970; Fugate and co-authors, 1975; Pellegrino and co-authors, 1977; Sobsey and co-authors, 1978; Ellender and co-authors, 1980). Survival of viral particles may be higher in oyster tissues than in sea water (J. S. Feng, 1966; Liston and co-authors, 1968). There are several reports on outbreaks of infectious hepatitis epidemics traceable to the consumption of raw oysters (Mason and McLean, 1962; Schäfer and Witt, 1973).

Agents: Bacteria

The overwhelming majority of prokaryotes associating with marine Bivalvia are Gram-negative bacteria; Gram-positive (Eu-)bacteria are distinctly less frequent. In addition, a few presumed representatives of the Actinomycetales and Spirochaetales have been reported from bivalves. The prokaryotic Rickettsiaceae (Rickettsiae), Chlamydiae and Mycoplasmataceae (Mycoplasmas), which have fairly recently been discovered in bivalves, are treated in the subsequent section of this review. Actually being prokaryotes, not eukaryotes, the Cyanophyceae ('Cyanophyta') are normally grouped with the Bacteria. However, since in the classical literature the photosynthetic 'blue-greens' are included in the plant kingdom, they have been listed under the heading 'Agents: Protophyta'.

Due to their efficient filter-feeding mechanism, bivalves are capable of accumulating, from the surrounding sea water, large numbers of micro-organisms, and consequently harbour an exceptionally rich bacterial flora, including Gram-negative species of *Achromobacter* (= *Acinetobacter*), *Aeromonas*, *Alcaligenes*, *Flavobacterium*, *Pseudomonas* and *Vibrio* (= *Beneckea*), as well as Gram-positive representatives of *Bacillus*, *Corynebacterium* and *Micrococcus* (Eliot, 1926; Colwell and Liston, 1960a, 1961, 1962; Chakroun, 1967; Colwell and Sparks, 1967; Lovelace and co-authors, 1968; Murchelano and Brown, 1968; Murchelano and Bishop, 1969; and others).

Without themselves contracting a bacterial disease, filter-feeding molluscs can act as passive carriers of micro-organisms pathogenic to man (Johnstone, 1905c; Trawinski, 1929, 1933; Berner, 1939; Steiniger, 1956; Sakazaki and co-authors, 1963; Moussa, 1965; Brisou, 1974). *Vibrio parahaemolyticus*, previously known as *Pasteurella haemolytica*, is the major source for bacterial shellfish poisoning in various parts of the world, particularly in Japan. The halophilic agent is worldwide distributed in oceanic and coastal waters and has repeatedly been isolated from lamellibranchs. *V. parahaemolyticus* infections of the extremities may occur in persons cut or scratched by the sharp edges of clams or oysters embedded in the sand of marine shore areas (Takikawa, 1958; Sakazaki and co-authors, 1963; Sakazaki, 1965; Baross and Liston, 1968, 1970; Molenda and co-authors, 1972; Thomson and Thacker, 1972; Ayres and Barrow, 1973; Davis and co-authors, 1973; Fishbein and Wentz, 1973; Kaneko and Colwell, 1973, 1975; Lewis, 1973; Liston and Baross, 1973; Kristensen, 1974; Thompson and Vanderzant, 1976; Colwell and co-authors, 1973, 1977; Boccia and co-authors, 1978). As a consequence of uptake and accumulation of pathogenic bacteria, shellfish in Chesapeake Bay are more likely to be a source of *Vibrio cholerae* infection than water (Kaper and co-authors, 1979). Bacterial shellfish poisoning will be discussed in detail in Volume IV.

The role of bacteria as organisms causing disease in pelecypods is not always clear. Species of *Vibrio* and *Pseudomonas* are normal — and frequently the dominant — constituents of the natural bacterial flora of the digestive tract of clams (Brisou and co-

authors, 1962; Colwell and Liston, 1962; Beeson and Johnson, 1967; Chakroun, 1967; Colwell and Sparks, 1967; Prieur, 1976a, 1982a).

Vibrio parahaemolyticus has been incriminated directly of causing disease and mortality in marine bivalves. Thus, Lipovsky and Chew (1971) classified the organism as "a suspected marine molluscan pathogenic bacteria", but later (1972, 1973) were more cautious with respect to the specific diagnosis. According to Grischkowsky and Liston (1974), *V. parahaemolyticus* identification at that time was based on biochemical tests then available. New techniques, including DNA homology, have indicated that some earlier identifications of bacteria from diseased molluscs as *V. parahaemolyticus* may be in doubt (Anderson and Ordal, 1972; Vanderzant and Nickelson, 1973). Vibrios definitely (or at least facultatively) pathogenic to bivalves are *V. anguillarum* and *V. alginolyticus* (Jeffries, 1982).

Frequently, potentially pathogenic bacteria have been isolated from bivalves, but their proper location has not been determined. Preparation of inoculates from whole-animal homogenates does not permit to discriminate between bacteria adhering to the epithelial surfaces or present in the digestive tract and agents liberated from tissues or haemolymph. Pauley and Maulsby (1967) described a method facilitating the detection of bacteria in oyster tissue. Johnson (1968a) developed a new medium for the cultivation of marine bacteria used in infectivity tests.

Even healthy-appearing bivalves may have bacteria in their tissues. Of 39 *Macoma baltica* collected from Tred Avon River, Maryland (USA), and kept in the laboratory for 3 months, 23 % had Gram-negative colonies of bacteria in the connective tissue. Subsequent exposure to different concentrations of dieldrin did not result in a significant increase in bacterial numbers in live clams, and colony counts were even lower in moribund individuals (Farley, 1977). One may conclude that vibrios, pseudomonads and other Gram-negative forms frequently associated with mortalities of adult bivalves are secondary invaders rather than primary pathogens. Healthy adult bivalves possess efficient humoural and cellular defense mechanisms acting against, and destroying or eliminating, foreign material in the tissues (Tripp, 1958, 1963, 1969, 1970, 1974; S. Y. Feng, 1966, 1967; McDade and Tripp, 1967a, b; Foley and Cheng, 1972, 1975, 1977; Cheng and Cali, 1974; Cheng and Rodrick, 1974; Rodrick and Cheng, 1974; Cheng and Foley, 1975; Cheng and co-authors, 1975; Cheng, 1976a, b; Cheng and Rudo, 1976; Arimoto and Tripp, 1977; Cheng and Howland, 1979; McHenery and Birkbeck, 1979; McHenery and co-authors, 1979; Cheng and co-authors, 1981; Poder and co-authors, 1982; and others). Moreover, unidentified antimicrobial substances have been isolated from marine bivalves (Li, 1960; Bonelly de Calventi and co-authors, 1967).

In considering the equivocal reports on the effects of bacteria on marine bivalves, we must also evaluate these micro-organisms in terms of the 'infection versus disease' concept. In nature, bacteria-free pelecypods do not exist and, as far as the reviewer was able to determine, molluscs have not yet been grown axenically in the laboratory. Hence, normally bacterial *infections* exist in bivalves without causing bacterial *diseases*. Species of *Pseudomonas* and *Vibrio* are natural constituents of the bacterial flora of the molluscan digestive tract. On the other hand, species of these genera have been implicated in most bacterial diseases affecting this invertebrate group (Tubiash and co-authors, 1965, 1970, 1973; Colwell and Sparks, 1967; Lipovski and Chew, 1971, 1972, 1973; Tubiash, 1971; C. Brown, 1973; Grischkowsky and Liston, 1974; and others). Distinction between non-

pathogenic species (or strains) and true pathogens is often difficult, as is the analysis of factors determining the apparent switching of a bacterium from non-pathogenicity to pathogenicity. Moreover, there appears to be a *quantitative* aspect in the host–micro-organism relationship. In many experiments, bivalves which were challenged with high concentrations of Gram-negative bacteria, died. This led to the misconception that these organisms are true pathogens. Later it was shown that more 'reasonable' concentrations of the same organisms were tolerated by bivalves without producing disease (see below).

Many pelecypods, particularly mytilids and other species with fine-meshed gills, ingest and digest bacteria. In the laboratory, adult individuals of *Mytilus californianus* could be maintained for several months on an exclusive diet of bacteria, during which time they gained in size and weight when fed 10^8 to 10^9 ml^{-1} washed bacteria once a day (ZoBell and Feltham, 1938). The species used in the experiment were *Rhodococcus agilis*, a motile Gram-positive coccus, and *Bacillus marinus*, a Gram-positive rod.

Most marine bacteria are not injurious to adult bivalves, unless present in excessive numbers which are, however, rarely reached in nature. Under experimental conditions, adult bivalves can tolerate considerably higher bacterial concentrations in the medium than larvae. Thus, adult *Crassostrea virginica, Mercenaria mercenaria, Mytilus edulis* and *Mya arenaria,* exposed for 24 h in standing sea-water cultures to massive concentrations of *Aeromonas* sp. and *Vibrio* sp., showed no ill effects, although they ingested vast numbers of the test bacteria (Tubiash and co-authors, 1965). Much lower concentrations of the pathogens produced bacillary necrosis in spat, and concentrations of 10^6 cells ml^{-1} killed exposed bivalve larvae (Guillard, 1959). Similarly, Martin (1976) isolated a *V. anguillarum* strain, which proved to be highly pathogenic to larval *M. edulis* but was tolerated, even in high concentrations, by adult mussels.

During a parasitological survey of *Crassostrea virginica* from Chesapeake Bay, Tubiash and co-authors (1973) noted sporadic cases (0.04 %) of greatly enlarged and edematous pericardia. Examination of pericardial fluid showed heavy concentrations of Gram-negative rods morphologically and culturally compatible with *Vibrio anguillarum.* Except for pericardial enlargement, the oysters appeared to be grossly and histologically normal. Experimental production of 'cardiac vibriosis', as the condition was termed, proved unsuccessful. The authors concluded that the presence of potential pathogens need not necessarily be associated with overt pathology, and that in the edematous syndrome displayed by the oysters, the vibrios are probably opportunists, which may be eliciting a host response bordering on pathology.

Colwell and Sparks (1967) found that the bacterial flora of dead or dying *Crassostrea gigas* included a somewhat higher incidence of pseudomonads. One of these, *P. enalia,* appeared to be definitely pathogenic. Death of oysters occurred when body tissues were injected with viable bacterial cell suspensions. Histological examination of such oysters suggested bacterial tissue invasion.

Recurrent large-scale mortalities of one-year-old *Crassostrea gigas* occurred in Hiroshima Bay (Japan) and adjacent waters from 1946 onward. Histological examination of dead oysters revealed acute necrotic inflammatory tissue changes, particularly in the ovaries, digestive diverticula and intestine. The disease existed in an exudative and a tubercular phase, the former representing the early or acute stage. Both Gram-positive and negative bacteria were isolated from moribund and dead oysters and were most numerous in tubercles in the connective tissue surrounding the gut, suggesting the digestive

tract as entrance port (Fujita and co-authors, 1953, 1955). Takeuchi and co-authors (1955, 1956, 1957) implicated a Gram-negative, 1 to 3 µm motile bacterium, believed to be an *Achromobacter*. However, this is probably a misidentification since the classification and nomenclature of that genus are in a chaotic state, and organisms variously described as *Achromobacter, Moraxella, Mima* and *Herellea* have recently been included in the genus *Acinetobacter* (Davis and co-authors, 1973). Moreover, members of this group are non-motile, plump, paired rods ('diplobacilli'). Bacteria could be isolated from both diseased and healthy oysters. Three strains isolated from dead individuals were pathogenic to normal oysters and produced the same pathological changes as occurring in the field. The strains lost virulence in successive passages through artificial media but regained virulence to a certain degree by passage through living oysters. Entrance of the agent via the digestive tract was confirmed (Takeuchi and co-authors, 1955, 1956, 1957). Sindermann (1976) states that the evidence that a bacterial pathogen was responsible for the Hiroshima Bay oyster mortalities, is somewhat inconclusive, and prefers to categorize the mortalities as of 'unknown origin'.

'Focal necrosis' is a microbial disease of adult *Crassostrea gigas* from the North American Pacific coast and of imported Japanese seed oysters (Sindermann and Rosenfield, 1967; Lindsay, 1969; Sindermann, 1970a, 1976, 1977). Presumably, it is etiologically identical with a condition found in *C. gigas* in Japan, and termed 'multiple abscesses' by Japanese workers (Imai and co-authors, 1965, 1968; Tamate and co-authors, 1965). As a consequence of mass mortalities of *C. gigas*, which occurred annually in late summer in Matsushima Bay, Miyagi Prefecture (Japan), from 1961 to 1965 (Ogasawara and co-authors, 1962; Imai and co-authors, 1965; Kan-no and co-authors, 1965; Mori and co-authors, 1965a, b; Numachi and co-authors, 1965; Tamate and co-authors, 1965), an extensive histological study was conducted, paying particular attention to the occurrence of multiple abscesses (Imai and co-authors, 1968). These studies have made the Matsushima Bay mortalities the best documented, but least understood oyster mortalities in Japan.

Gross signs of the disease are emaciation, gaping and a pale appearance of the digestive gland. Histological examination discloses necrotic foci containing colonies of Gram-positive and — very rarely — Gram-negative bacteria (Fig. 13-7). Colonies occur most numerously in the mucous membranes (lamina propria mucosae) of the digestive tract and the surrounding connective tissues, indicating that entry of the pathogens is via the alimentary canal. Intense haemocytic infiltration surrounding the colonies is characteristic.

Interestingly, peak incidences of multiple abscesses — 20 % in October 1965, 30 % in January 1966 and 20 % in March 1966 — did not coincide with mortality rates, which were 23 % in August, 16 % in September and 8 % in October 1965, but zero during the remaining months. This casts some doubt on the bacterial infection being the primary cause of death. Judging from the fact that the occurrence of abscesses was preceded by the sudden onset of mortalities, one might speculate that the mortalities were caused by the active, fulminating phase of the pathogen, while the later-appearing necrotic foci merely represent the arrested, chronic disease state. Imai and co-authors (1968) observed pronounced inflammatory and degenerative processes in the digestive gland of *Crassostrea gigas* during the spawning season, i. e., in August and September. In accordance with Mori and co-authors (1965a, b), they were inclined to attribute the observed mass mortalities to physiological disorders and metabolic disturbances associated with spawning and adverse

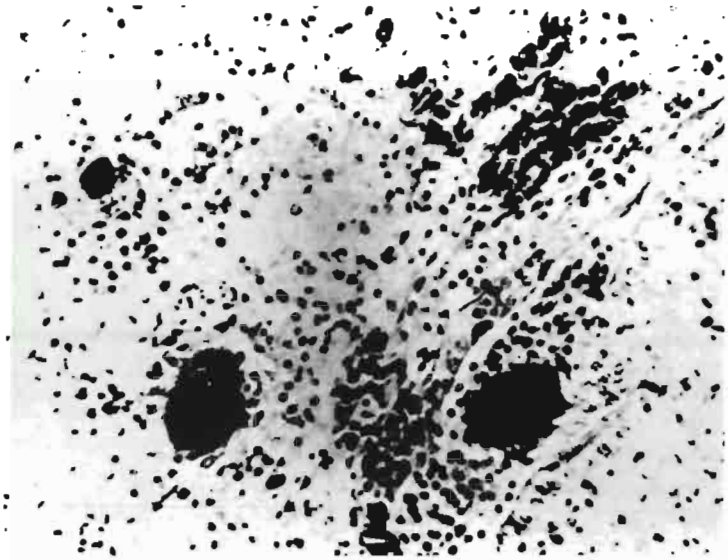

Fig. 13-7: *Crassostrea gigas.* Focal necrosis of connective tissue, surrounded by extensive haemocytic infiltration. (After Sindermann and Rosenfield, 1967.)

environmental conditions, such as high water temperature and eutrophication, rather than to bacterial infection.

Similarly, the association of 'focal necrosis' with repeated *Crassostrea gigas* mortalities observed during the 1960's on the U.S. Pacific coast is still unconfirmed. Oysters typically died during their second summer following planting, and fast-growing individuals were most seriously involved. The failure of pathological studies to detect a causative agent tends to strengthen the Japanese authors' physiological stress hypothesis.

Subsequently, however, Lipp and co-authors (1976) found 'summer disease' of *C. gigas* in Willapa Bay and Rocky Bay in central Puget Sound (Washington) to be associated with bacterial infection. Dead or moribund oysters were shown to contain vibrios in the heart blood. Apparently healthy oysters from the mortality areas also contained bacteria, although healthy individuals usually have sterile heart fluids. As revealed by inoculation experiments, vibrios from these oysters were virulent toward healthy *C. gigas* and could be recovered from their heart fluids. Oyster losses in late July and August, possibly attributable to high water temperature and high nutrient levels plus *Vibrio* infection, amounted to 15 to 20 %.

High-temperature stress is a major factor controlling the susceptibility of bivalves to fatal bacterial infections. Lipovsky and Chew (1971, 1972, 1973) showed 18 °C to be the apparent critical temperature for significant laboratory mortality of 2-year-old *Crassostrea gigas*, presumably resulting from infection with 'mesophilic vibrios' (probably *V. anguillarum* and *V. alginolyticus*, not *V. parahaemolyticus*). Over a period of 20 to 25 days, oysters kept at 21 °C suffered 100 % mortality, while individuals kept at 9 °C had zero mortality. Oysters periodically transferred between these temperature extremes exhibited intermediate death rates. Histologically, cellular responses, tissue pathology and bacterial numbers in the tissues increased progressively with the duration of high-temperature stress.

Soft-shell clams *Mya arenaria* were found to be susceptible to *Vibrio anguillarum, V. alginolyticus* and *Vibrio* spp. when inoculated into heart and siphonal tissues at 20 °C and 22 °C (Tubiash, 1971). S. Y. Feng (1966) showed that experimental bacterial (probably *Pseudomonas*) infections of *Crassostrea virginica* were successfully controlled at 9 °C and 16 °C, but were fatal at 23 °C. Baross and Liston (1968, 1970) and Liston and Baross (1973) found *V. anguillarum* and *V. alginolyticus* prevalences in healthy individuals of *C. gigas* from Puget Sound, Washington (USA), to be directly correlated with temperature in environmental ranges, and vibrios (particularly *V. anguillarum*) isolated from dying oysters produced mortalities when introduced into previously healthy individuals under high-temperature conditions by either active or passive inoculation (Lipp and co-authors, 1976). Sparks (1981) concluded that oyster-summer mortalities, as reported by the various authors, are probably of bacterial etiology, with the physiological stress of gonadal maturation and spawning of, at best, minor importance.

Heavy oyster mortalities, attributed to adverse environmental conditions, have also been reported from British Columbia (Canada). All these losses occurred in Boundary Bay during summers which were warmer than usual (Quayle, 1969). Mass mortalities have also struck oyster populations in Europe (Orton, 1923, 1924; Fischer, 1951; Korringa, 1952a), but their causes remain obscure. It would be tempting to speculate on the involvement, in these losses, of viral agents or protozoal pathogens. Recent evidence suggests that some of the documented epizootic oyster mortalities may be due to haematopoietic neoplasms (see section 'Neoplasia').

Mass mortalities due to unknown causes have also been reported from other marine pelecypods (Burkenroad, 1946; Coe, 1955, 1956). Johnson (1968b), in her investigations on the causes of such 'population crashes' in the Pacific bean clam *Donax gouldi*, isolated various species of apparently non-pathogenic bacteria and fungi from diseased clams, including *Pseudomonas* sp. and *Vibrio* sp. Addition of bacterial cultures to water containing healthy clams as test animals did not accelerate mortality, which was virtually as high in experimental as in control batches. The author concluded that, since members of a population are in a similar physiological state, they respond to environmental stress more as a unit than as individuals, and that some epizootics in other marine invertebrates may also be a manifestation of unit reaction.

An apparently non-lethal bacterial disease, reported by Mackin (1962), comprised the formation of watery cysts in the visceral mass, palps and mantle of *Crassostrea virginica* from Louisiana waters. The cysts were large conspicuous, bubble-like cavities, generally distended by internal pressure of accumulated fluid. Sections showed them to contain a central granular material deposited in concentric layers with embedded masses of small bacilli. Recovery from infection appears to occur through sloughing of the cysts, which rupture when fully formed, discharging their contents to the exterior.

Although there is little conclusive evidence of bacteria being primary pathogens of adult bivalves, their role as disease agents in larval lamellibranchs is well documented. However, bacterial infections can in no way be regarded as the sole mortality cause of cultivated bivalve larvae (Prieur, 1976b).

Unidentified rod-shaped, presumably non-pathogenic bacteria usually cover the outer surface of the gelatinous membrane of *Cardium edule* and *C. lamarcki* eggs when incubated in the absence of antibiotics (Fig. 13-8, a). The former species has planktotrophic, pelagic eggs and the latter has lecithotrophic, demersal eggs (Lauckner, 1972). Dense

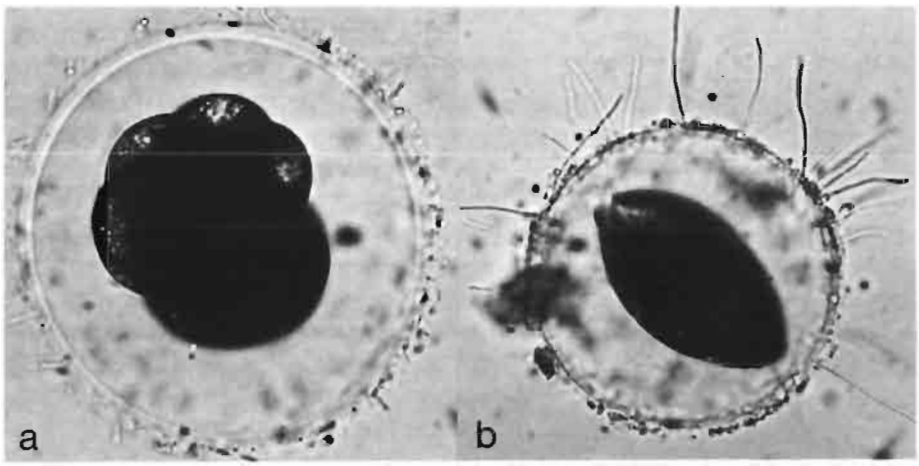

Fig. 13-8: *Cardium lamarcki*. Bacterial growths on surface of developing eggs. (a) 8-cell stage with rod-shaped organisms and short *Leucothrix mucor* (?) filaments; (b) egg with enclosed early veliger larva and dense overgrowth of long bacterial filaments. (After Lauckner, 1972.)

bacterial upgrowths sometimes kill *C. edule* ova, presumably by inhibition of proper oxygen diffusion rather than by excretion of toxic metabolites and/or exoenzymes. *C. lamarcki* eggs, which have a thicker membrane, are not affected, although the bacterial density on their surface may exceed that on *C. edule* ova. *C. lamarcki* eggs, which usually develop attached to the substrate in sheltered marine and estuarine habitats characterized by the frequent occurrence of reduced oxygen levels in near-bottom water layers, apparently have a lesser oxygen demand. Reduced diffusion rates due to bacterial upgrowth, therefore, do not seem to affect survival of the eggs of this species.

Leucothrix mucor, a unique non–pathogenic filamentous bacterium normally known as an epiphyte of tropical marine algae (Bland and Brock, 1973), may be responsible for large–scale mortalities of pelagic eggs and other life-cycle stages of various marine animals by causing them to sink below the surface (Anderson and Conroy, 1968; Johnson and co-authors, 1971). *L. mucor* was formerly believed to be a fungus (K. Wolf, 1958). There is some evidence to suggest that the bacterium derives at least part of its carbon, energy and nitrogen from the algae to which it attaches, although the filaments do not penetrate the plants' mucilaginous covering (Bland and Brock, 1973). A filamentous bacterium, similar to or identical with *L. mucor* (Fig. 13-8, b), caused considerable mortalities of the pelagic eggs of cockles *Cardium edule*, while even dense populations of this bacterium on the surface of the demersal *C. lamarcki* eggs produced no detectable adverse effects (Lauckner, pers. obs.).

Bacteria incriminated in mortalities among bivalve larvae are mostly *Pseudomonas* spp., *Vibrio anguillarum* and *V. alginolyticus*. The taxonomic status of these 2 genera is at present in considerable disorder and is poorly understood (Jeffries, 1982). Identification schemes and techniques for the differentiation of Gram-negative bacteria have been described by Shewan and co-authors (1954, 1960), Schubert (1962), Shewan (1963), Hentzschel (1981) and Rüger (1981). For comments on the taxonomy of the relevant groups see Colwell and Liston (1960b), Davis and Park (1962), Schubert (1967) and

Baumann and co-authors (1971); for general recent information consult Kandler and Schleifer (1980). On the basis of extensive physiological, nutritional, and morphological studies, Baumann and co-authors (1971) arrived at the conclusion that most of the forms previously placed in *Vibrio, Aeromonas* and *Pseudomonas* — including the well-known species *V. parahaemolyticus* and *V. alginolyticus* — should be transferred to the recently created genus *Beneckea*. Therefore, the strains isolated from marine bivalves should be restudied.

Some of the bacteria isolated from larval bivalves may be derived from the algal food. Stock algal cultures are frequently contaminated with a wide variety of bacteria, particularly with *Pseudomonas, Acinetobacter* and *Flavobacterium* or *Cytophaga* spp. (Berland and co-authors, 1969; Prieur, 1976a; Leibovitz, 1978a; Prieur and Carval, 1979).

Of 120 isolates obtained from 15 mass cultures of 6 different genera of food algae, 98.3 % were asporogenous, Gram-negative rods — types commonly found in sea water. The majority of these (81.6 %) belonged to the genus *Pseudomonas*. Gram-positive *Bacillus* and *Corynebacterium* were found in only 2 of the 120 isolates (Murchelano and Brown, 1969).

Vibrios and pseudomonads constituted 8.7 % and 54.4 %, respectively, of the total bacterial flora of healthy, 9-month-old and 10- to 15-mm-long laboratory-reared *Crassostrea virginica*. The percentage of vibrios in sea-water samples taken from the culture enclosures, on the other hand, was substantially higher (37.1 %), that of the pseudomonad concentration (38.7 %) significantly lower. Representatives of other genera present included *Achromobacter* (= *Acinetobacter*) sp., *Bacillus* sp., *Cytophaga* sp., *Flavobacterium* sp. and *Micrococcus* sp., as well as a low percentage of unidentified bacteria. *Cytophaga* sp. was found in the oysters, but not in the sea water, while *Micrococcus* occurred in the water alone. The generic composition of the bacterial flora of these laboratory-reared juvenile oysters was very similar to that of adults from Long Island Sound, USA (Murchelano and Brown, 1968; Murchelano and Bishop, 1969). In contrast, sick or dying oyster-larval cultures were found to be characterized by a sharp decrease in the percentages of *Pseudomonas, Flavobacterium* or *Cytophaga, Acinetobacter* and Enterobacteriaceae and a concomitant rise in the percentage of Gram-positive forms (Leibovitz, 1978a). One may speculate whether this increase of the latter may be due to the breakdown of the host–lysosomal system.

Metabolites released by algae may or may not interfere with bacterial growth in the cultures. In the studies of Murchelano and Brown (1969), *Pseudomonas, Flavobacterium* and *Vibrio* were isolated from *Phaeodactylum tricornutum* cultures. Their growth was apparently not inhibited. On the other hand, the acrylic acid produced by this alga exhibited an anticoliform effect. *Escherichia coli* counts decreased rapidly in sea water in the presence of *P. tricornutum*, as well as in oysters which had been fed this alga. *Pavlova lutheri* was ineffective against *E. coli* (R. K. Brown and co-authors, 1977). Introduction of pathogenic or potentially pathogenic bacteria with algal food must, therefore, be taken into consideration and presents a problem in bivalve hatcheries (Prieur and Le Roux, 1975; Prieur and Carval, 1979).

While adult lamellibranchs could be maintained on an exclusive bacterial diet (ZoBell and Feltham, 1938), none of 13 strains of marine bacteria tested by Davis (1953) were utilized as food by *Crassostrea virginica* larvae. In contrast, such a diet may retard growth and eventually kill the larvae. Bacterial toxins and/or exoenzymes were believed to

account for the mortalities observed by ZoBell and Feltham (1938), but Guillard (1959) maintained that active bacteria are necessary to destroy larvae and that metabolites in the bacterial inoculum are not harmful to bivalve larvae. Similarly, C. Brown (1973) found that neither culture filtrate nor heat-killed *Pseudomonas* spp. and *Vibrio* spp. (which, when viable, caused gross morphological abnormalities in developing *C. virginica*) were toxic to oyster larvae. Hidu and Tubiash (1966) even succeeded in raising larval *Mercenaria mercenaria* and *C. virginica* in antibiotic-treated sea water inoculated with a mixed flora of (specifically unidentified) marine bacteria. Larvae kept in this medium showed significant growth, while cultures that received the bacterial inoculum but no antibiotics showed little or no growth. Probably, these authors dealt with different species and/or different strains of bacteria. While some may produce toxic metabolites, others may not. A *Vibrio* sp., isolated from moribund *Crassostrea virginica* cultures, was found to produce a proteinaceous toxin capable of breaking down tissue structures and readily killing developing oyster larvae. High concentrations of this toxic substance appear to act as a teratogen, since up to 56.8 % of fertilized oyster eggs developed abnormally in increasing concentrations of the filtered vibrio broth culture (C. Brown and Losee, 1978). C. Brown (1981) found a red prodiginine pigment, produced by a species of *Pseudomonas*, to be toxic to embryos and larvae of *C. virginica*. A comparative study of a non-pigmented and 2 pigmented mutants of the red parental strain indicated that virulence was associated and varied with pigmentation, the 'pigment' extracted from the white mutant being non-toxic.

The pathogenicity of bacteria for bivalves may vary with the developmental stage of the mollusc and normally decreases with increasing age. Of 123 different isolates comprising *Pseudomonas* and *Vibrio* spp., 20 were found to affect at least one of 3 developmental stages of *Crassostrea virginica*, i.e., fertilized eggs, 48-h-old larvae, or 2-week-old larvae. Of these 20 bacteria, 19 adversely affected the embryonic stage, 13 affected 2-day-old larvae, while only 12 affected presetting larvae (C. Brown, 1973).

Different bivalve species may differ with respect to their susceptibility to infection by a given bacterial strain (Loosanoff, 1974). In rearing experiments involving two closely related species, *Cardium edule* and *C. lamarcki* (Lauckner, 1972), veligers and setting stages of the former species were much more susceptible to bacterial infection than the latter, even when kept in mixed cultures. On the other hand, larvae of one and the same species, but obtained under different conditions, may differ with respect to their sensitivity to bacterial inoculates (Prieur, 1976c).

Both virulence and pathogenicity may also vary considerably with the bacterial species involved. Thus, pathogenic strains isolated from *Crassostrea virginica* were neither as virulent nor as active as those from *Mercenaria mercenaria*, but each was interchangeably pathogenic for larvae of homologous or heterologous species of the above and other bivalves challenged (Tubiash and co-authors, 1965).

Of twelve strains of bacteria isolated from a single laboratory-held, moribund larval *Mercenaria mercenaria*, 10 other clones, and mixed bacteria from sea water, assayed by adding broth culture yielding 10^6 to 10^7 cells ml^{-1} to beaker cultures of healthy clam larvae, only the mixed bacterial culture from the moribund larva and 2 of the 12 strains isolated from it caused extensive mortality. One of the virulent clones was a species of *Vibrio*, the other was a *Pseudomonas* (Guillard, 1959).

Of 48 bacterial isolates comprising *Pseudomonas* and *Vibrio* spp., 17 appeared to impair development of fertilized *Crassostrea virginica* eggs to normal 2-day-old larvae.

Either the embryos developed abnormally, or the larvae died after reaching the veliger stage. The abnormalities consisted of incomplete shell formation or velum protrusion. Further experiments with these 17 suspected pathogens showed 6 of them to cause consistently abnormal embryonic development, while 3 killed veligers. In another set of experiments, 13 of 29 bacterial strains, tested on 48-h-old larvae, showed to cause mortality and/or decreased growth. Six of these pathogens caused increased mortality and decreased growth, one caused only mortality, while another caused only decreased growth (C. Brown, 1973).

The bacterial disease reported by the various authors from laboratory-reared larval and juvenile bivalves has been termed 'bacillary necrosis'. In the initial study, conducted by Tubiash and co-authors (1965), seven strains of *Vibrio*, some sharing physiological properties with *V. anguillarum* and *V. alginolyticus*, have been identified as etiological agents. Larvae of *Crassostrea virginica, Ostrea edulis, Mercenaria mercenaria, Argopecten irradians* and *Teredo navalis* were similarly affected. In experimentally infected cultures, the course of the disease is swift and dramatic. Within 4 to 5 h after seeding with bacterial suspensions containing approximately 5×10^8 cells ml^{-1}, prodromal signs are a reduction of motility and a tendency for many larvae to lie quiescent with either their rudimentary foot or velum extended. 'Swarms' of bacteria originating from discrete foci on the margins of scattered larvae appear simultaneously. The swarming of bacteria is a pathognomonic sign of bacillary necrosis although, at this point, the larvae seem otherwise normal. The swarming becomes progressively more dense and ubiquitous, and within 8 h after inoculation, death of larvae with granular necrosis is widespread. In heavily affected cultures, mortality is often complete within 18 h. After the culmination of the bacteria–induced epizootic, ciliates frequently appear as scavengers. They apparently play no part in the primary microbial infection. From the fact that malformed, non-feeding larvae are the last to show signs of infection, it was concluded that entry of the pathogen is via the alimentary tract (Tubiash and co-authors, 1965, 1970). In contrast, C. Brown and Losee (1978) suggested that the bacteria attach to, and penetrate, the velum, causing it to become deformed and sometimes to detach from the larvae. Spread of the pathogen to the tissues presumably occurs from this initial site.

Histological examination of affected larvae confirmed massive bacterial invasion and proliferation with extensive lysis and necrosis of tissues, supporting the designation of the disease as bacillary necrosis. On the other hand, adult *Mercenaria mercenaria, Crassostrea virginica, Mytilus edulis* and *Mya arenaria*, exposed to massive concentrations of all pathogenic serotypes for 24 h in standing sea water, showed no ill effects although they ingested vast numbers of these bacteria.

Of 27 marine bacterial cultures, isolated from presumably normal marine fauna, and with generally similar morphological and physiological characteristics, none had antigens in common with the larval pathogens, and none was pathogenic for bivalve larvae. It was concluded that the agents are enzootic, causing overt disease and mortality when critical infective levels are reached due to adverse environmental conditions, such as overcrowding and accumulation of waste products under laboratory conditions (Tubiash and co-authors, 1965).

Bacillary necrosis is the main disease problem in the artificial propagation of bay scallops *Argopecten irradians* in Virginia. The disease was effectively treated with streptomycin (50 mg l^{-1}) or with a wide-spectrum antibiotic, such as chloromycetin or polycillin.

Care must be taken, in such procedures, in estimating dosage, since antibiotics often cause the larvae to stop feeding for several days and overdoses cause mortalities (Castagna, 1975).

'Bacterial swarming' around the velar and mantle margins of bivalve larvae suffering from bacillary necrosis has also been observed by Guillard (1959), and infections with the same or similar vibrios and pseudomonads may have been responsible for larval-bivalve mortalities reported by Loosanoff (1954), Walne (1956, 1958, 1966), and others. With respect to the ubiquity of the etiological agents of bacillary necrosis it was assumed that the pathogens normally exist as widely distributed saprophytes or commensals of marine forms, since typable pathogens were frequently isolated from cultures maintained in unsanitary or otherwise unfavourable physical environments. It was also speculated that, during mid-summer, when ecological conditions favour both molluscan spawning and bacterial proliferation, natural epizootics of bacillary necrosis may limit the recruitment of commercially valuable bivalves (Tubiash and co-authors, 1965, 1970). In fact, there is evidence for vibriosis-associated increased mortalities among natural bivalve populations, particularly in *Crassostrea gigas* (Grischkowsky and Liston, 1974; Martin, 1976).

However, the possible impact of bacillary necrosis on natural populations of bivalve larvae need to be studied in more detail. It appears likely that the speculation of Tubiash and co-authors (1970) is somewhat premature. More sophisticated pathogenicity tests rather suggest that almost all bacterial isolates at concentrations $> 10^5$ ml^{-1} — as used by Guillard (1959) and Tubiash and co-authors (1965) — are pathogenic for shellfish, but that only 'true' pathogens kill at very high dilutions ($< 10^3$ ml^{-1}). This, in turn, suggests that these true pathogens require larvae for growth. It was also found that the presence of higher concentrations of even sterile nutritive broth produces a lethal effect —a fact not normally considered by previous workers. Accordingly, it appears that high food concentrations, dead or decaying algal cells or dead larvae may aggravate the pathogenic effect of both external (in the medium) and internal (in the mantle cavity or in the alimentary tract) bacterial concentration. With bacterial counts $\geq 10^7$ ml^{-1}, lethal effects develop rapidly, and it was found that diseased cultures of bivalve larvae are usually associated with concentrations of that order of magnitude (Leibovitz, 1978b).

At levels of 10^3 to 10^7 cells ml^{-1} of a *Vibrio* sp., isolated from a Long Island (New York) oyster hatchery, larval *Crassostrea virginica* mortalities ranged from 40 to 100 % within 5 days. First clinical signs — changes in the behaviour of the larvae, including decreased swimming activity, abnormal swimming patterns ('flip-flop' movements, reversed and spinning patterns) and cessation of feeding — were usually evident by 2 days post inoculation. Observations of live larvae, backed by ultrastructural examination, indicated that detached velar retractor muscles resulted in continuously extended vela. General visceral shrinkage was a prevalent response in older larvae. In straight-hinge individuals, nearly complete visceral degeneration could occur, while relatively intact vela enabled the larvae to continue swimming. Attachment of bacteria to the larval shells, prior to selective invasion of mantle tissue, apparently contributed to the virulence of the pathogen. Histological examination of diseased larvae revealed masses of bacteria in the viscera and mantle tissues. In all experiments, some degree of phagocytosis of whole bacteria by oyster haemocytes was observed, and a decrease of bacterial concentrations in the cultures suggested that oyster larvae selectively remove *Vibrio* organisms from the medium (Elston and Leibovitz, 1980; Leibovitz and Elston, 1980).

A 5-year study on the qualitative and quantitative bacterial flora of the incoming water and algal, as well as oyster larval cultures in the Long Island hatchery indicated that outbreaks of larval vibriosis with high oyster larval morbidity and mortality were distinctly correlated with peaks of *Vibrio* abundance and the amount of suspended organic material in the incoming bay water. Although the epizootics were initiated during peak concentrations of *Vibrio,* they persisted in the hatchery after the peak periods had passed and vibrios could no longer be demonstrated in the water supply. Careful monitoring of bacterial counts in the incoming water, as well as timed precautions can, therefore, contribute to successful production of viable oyster brood (Leibovitz, 1979).

There have been numerous attempts to overcome the problem of bacterial disease in cultivated larval and juvenile bivalves by more or less deliberate application of various antibiotics. From 1956 to 1976, more than 50 publications concerning this topic have appeared. An analysis of these papers indicates that such treatments or precautions have met with varying success (Le Pennec and Prieur, 1977). Elimination of certain species of bacteria may favour the apparition of others and reduce the molluscs' resistance to infection by pathogenic forms (Martin, 1977). As emphasized by Kinne (1977, p. 917),

> "the latter fact must be considered especially detrimental in cases where oyster spat are later to be transferred to *in situ* conditions. Since billions of oyster larvae can be produced without much difficulty, it would be worthwhile to investigate the possibilities of increasing their resistance to microbial activities via selection and immunity principles. Maximum larval survival rates are scarcely an achievement if followed by high rates of spat mortality."

A careful evaluation of the pros and cons favours the view that antibiotics should be used only exceptionally and in well-defined cases (Le Pennec and Prieur, 1972, 1977; Le Pennec and co-authors, 1973; Martin and Vicente, 1975a, b; Martin, 1976, 1977; Prieur, 1979). Studies conducted by Martin (1976) and Prieur (1982b) have shown that, contrary to previous experience, larval bivalves do not only tolerate appreciable concentrations of micro-organisms but grow even better on an algal diet supplemented by bacteria.

It appears, therefore, that our concept of the pathogenicity of bacteria for marine bivalves requires a reappraisal.

There are several inconclusive reports of organisms resembling actinomycetes in oysters. Eyre (1923, 1924) isolated a 'fungus' identified as *Cladothrix dichotoma* from *Ostrea edulis* examined during the mass mortalities of 1919–23 in western Europe. The organism was not pathogenic in experimental studies. Dollfus (1921a, b) believed that the organism described by Eyre was an actinomycete of the genus *Nocardia.* In fact, the fungus-like Actinomycetes — Gram-positive organisms that grow slowly as branching filaments — can easily be mistaken for true fungi. What appears to be the same organism, has been described as *Nocardia matruchoti* from *O. edulis* in France. It was said to cause epithelial aplasia and weight loss in heavily infected oysters (Pettit, 1921).

Mackin (1962) described a 'mycelial disease' of *Crassostrea virginica.* The parasite grew as a filamentous mycelium varying between less than 0.5 μm and 5 to 6 μm in diameter, and branching irregularly in an arboreal fashion. Associated with most of the mycelial growths were deep-staining, amorphous masses which appeared to be part of the parasite. No structures interpreted as nuclei have been observed. In many oysters, 'stellate' bodies were found, which seemed to be formed by germination from spores in two or more

directions. The manner of spore formation suggested affinities of the organism to the genus *Micromonospora* of the Actinomycetes.

Growth of the agent in the host produced little cellular response. Mycelia occurring on external epithelia evidently lysed the underlying cells. First signs of infection were usually on the external epithelium of mantle, palps or gills. In advanced stages, colonies of mycelia were found on any external epithelial surface. They appeared to be loosely attached by a gummy material, and the filaments radiated outwardly, branching in a shrub-like manner. Frequently, penetration of the epithelia occurred. It was suspected that the organism may be toxic to the host. Heavily infected oysters stopped feeding. Mycelial disease was found

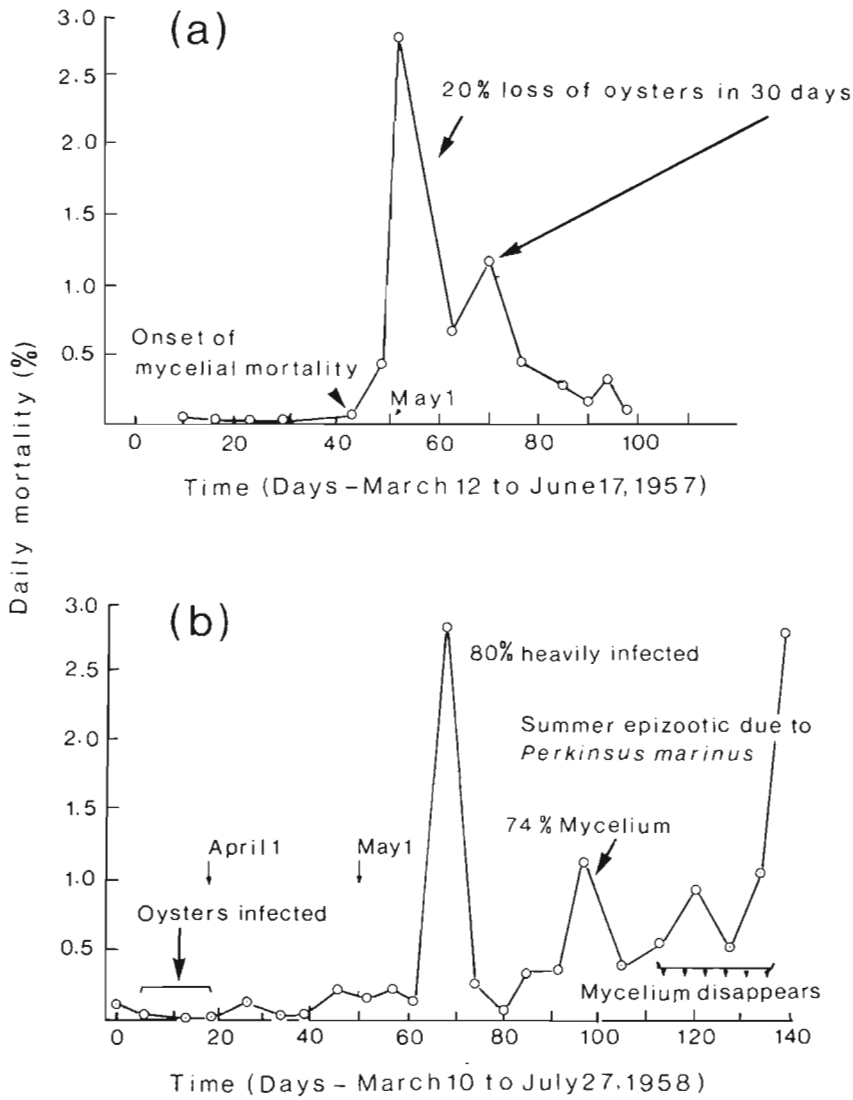

Fig. 13-9: *Crassostrea virginica*. Epizootics presumably caused by 'mycelial disease'. (a) Barataria Bay, Louisiana, 1957; (b) Bayou Rigaud, Louisiana, 1958. (After Mackin, 1962.)

in *Crassostrea virginica* from Texas, Louisiana, Virginia and Maryland, as well as in Olympia oysters *Ostrea lurida* from South Puget Sound, Washington.

In Louisiana, several summer-oyster epizootics, which were characterized by an abrupt onset of mortalities, have been attributed to 'mycelial disease'. Thus, in May 1957, about 20 % of 1,000 *Crassostrea virginica*, kept in trays in Barataria Bay for experimental purposes, died abruptly. Microscopic inspection disclosed the mycelial organism in about 75 % of the animals. A similar, explosive-type epizootic developed in Bayou Rigaud, Louisiana, in 1958 (Fig. 13-9). The mortalities caused by this presumed actinomycete were clearly discernible from those due to *Perkinsus marinus* infestations, which started when the former had already ceased.

Very little is known about the environmental factors controlling 'mycelial disease'. The agent appears to be definitely a spring form, but some heavily infected oysters have been found as late as August. Water temperatures in Louisiana bays from March to May, when the parasite blooms, range from 12 to 20 °C in March to 20 to 29 °C in May. Summer temperatures, in June to September, range from 27 to 32 °C. Only in the coldest months do oysters seem to be entirely free from the pathogen. Salinities between 15 and 30 ‰ S are likewise tolerated by the agent.

Another presumed actinomycete has been found in 16 of 243 *Crassostrea virginica* from Great South Bay, Long Island, New York. In most cases, septate filamentous forms, up to 10.6 μm in length and 0.7 μm in width, were diffusely distributed throughout the vesicular connective tissues with occasional focal concentrations. Many filaments had small terminal spores, about 0.8 μm in diameter, while larger spores, 1.5 to 2.0 μm in diameter, occurred free in the tissues. The organisms were Gram-positive and not acid-fast. The pathology of infected oysters included emaciation with generalized edema and thinning of the digestive tubular epithelium. Ulceration of the intestinal epithelium and moderate necrosis and haemocyte exudation of gill tissues were also seen (Meyers, 1981). The disease was somewhat similar to mycelial disease, but the causative agent differed from that described by Mackin (1962) by lack of associated sulphur granules or basic-staining amorphous masses, unbranched hyphal growth, uniform hyphal diameters and absence of stellate bodies. Moreover, its prevalence was highest in the summer (12.6 % of 87 oysters) and autumn (9.7 % of 41 oysters), but zero during the spring. Another presumed actinomycete was seen in *C. angulata* from France (Sindermann and Rosenfield, 1967).

There are other, apparently non-pathogenic associations between actinomycetes and adult bivalves. *Streptobacillus* infection of the outside surface of the gastric shield occurred in more than 90 % of mature *Crassostrea virginica* from Louisiana (USA). The bacteria, which sometimes formed long chains, caused lysis of the shield substance, but significant damage was not observed. The agent typically occurred in healthy, vigorously growing oysters (Mackin, 1962).

Spirochaetes *Cristispira balbianii*, first described as *Trypanosoma balbianii* from *Ostrea edulis* and *Crassostrea angulata* in France (Certes, 1882a, b), occur embedded in the substance of the crystalline style and in the intestinal tract. They appear to be non-pathogenic and occur in healthy and diseased oysters as well. Upon dissolution of the crystalline style, the liberated bacteria may spread to other parts of the body and, on superficial inspection, could give the impression of tissue invasion (Möbius, 1883; Orton, 1924). How these bacteria cope with the lysozymes present in the bivalves' crystalline style, has not been investigated.

Cristispira balbianii, as well as other spirochaetes, have also been reported from *Crassostrea virginica* and other bivalves (Dimitroff, 1926; Laird, 1961). Ryder (1883) named these organisms, obtained from the stomach of *C. virginica*, *Spirillum ostrearum*. Originally, the large (up to 120 μm long) spirochaetes were believed to be 'spirillum-like' flagellates related to the true protozoal haematozoans of fishes (Certes, 1882a, b, 1883). Although Laveran and Mesnil (1901) ascertained their bacterial nature, Pelseneer (1928), Cheng (1967) and van Banning (1979b) continue to list *'Trypanosoma balbianii'* and *Cristispira balbianii*, respectively, as flagellate protozoans (!).

Agents: Rickettsiae, Chlamydiae and Mycoplasmas

Rickettsiae are small, pleomorphic coccobacilli. Because most of them are obligate intracellular organisms that can survive only briefly outside animal cells, rickettsiae were long considered to occupy a special taxonomic niche between bacteria and viruses. Several properties, however, clearly indicate that they are bacteria. Chlamydiae are similarly obligate intracellular pathogens, which share a unique developmental cycle (Fig. 13-10, b). Although they have long been considered as viruses, their morphological and biochemical properties clearly identify them as bacteria. Mycoplasmas are the smallest known free-living organisms. They differ from bacteria in lacking cell walls. The members of all 3 groups are similar to bacteria (but different from viruses) with respect to their capability of independent protein synthesis, their mode of reproduction by binary fission, and their susceptibility to antibiotics.

In the past few years, beginning with 1977, evidence accumulates that rickettsiae, chlamydiae and mycoplasmas can live or survive in marine bivalves (Table 13-2). Some appear to be capable of causing disease and possibly death in these molluscs. All 3 groups contain forms that produce severe and sometimes fatal diseases in man (Rickettsiae: typhus, Q fever, rickettsialpox; Chlamydiae: trachoma, inclusion conjunctivitis, lymphogranuloma venerum, ornithosis (psittacosis); Mycoplasmas: primary atypical pneumonia). Mycoplasmas have been isolated from soil and sewage (Moulder, 1973; Nichols and Blyth, 1973; L. Thomas, 1973). Whether forms observed in bivalves originate from contaminated water, is at present unknown. Because they are potentially pathogenic to man, rickettsiae, chlamydiae and mycoplasmas occurring in commercially exploited marine bivalves deserve special attention.

The first rickettsia-like organisms detected in molluscs have been found in *Mya arenaria* from Chesapeake Bay. The ribosome-rich, undulating rods, measuring 300 × 2,000 nm (Fig. 13-10, d), occurred in roundish inclusions, up to 100 μm in diameter and usually located singly in the epithelial-cell cytoplasm or occasionally in the lumen of digestive diverticula (Harshbarger and co-authors, 1977; Otto and co-authors, 1979).

Similar vacuole-like inclusions, 10 to 30 μm in diameter and containing rickettsiae, have been found in the cytoplasm of epithelial cells of digestive diverticula of 1 of 10 *Crassostrea gigas* from Marennes-Oléron, French Atlantic coast (Fig. 13-11). The Feulgen-positive inclusions occurred in moderate numbers in the digestive gland but not in other tissues. The contained organisms ranged from 1.5 to 2.5 μm in length and from 0.5 to 0.6 μm in diameter (Comps and co-authors, 1977).

Another rickettsia, smaller than that in *Crassostrea gigas*, occurs in *Ostrea edulis* from

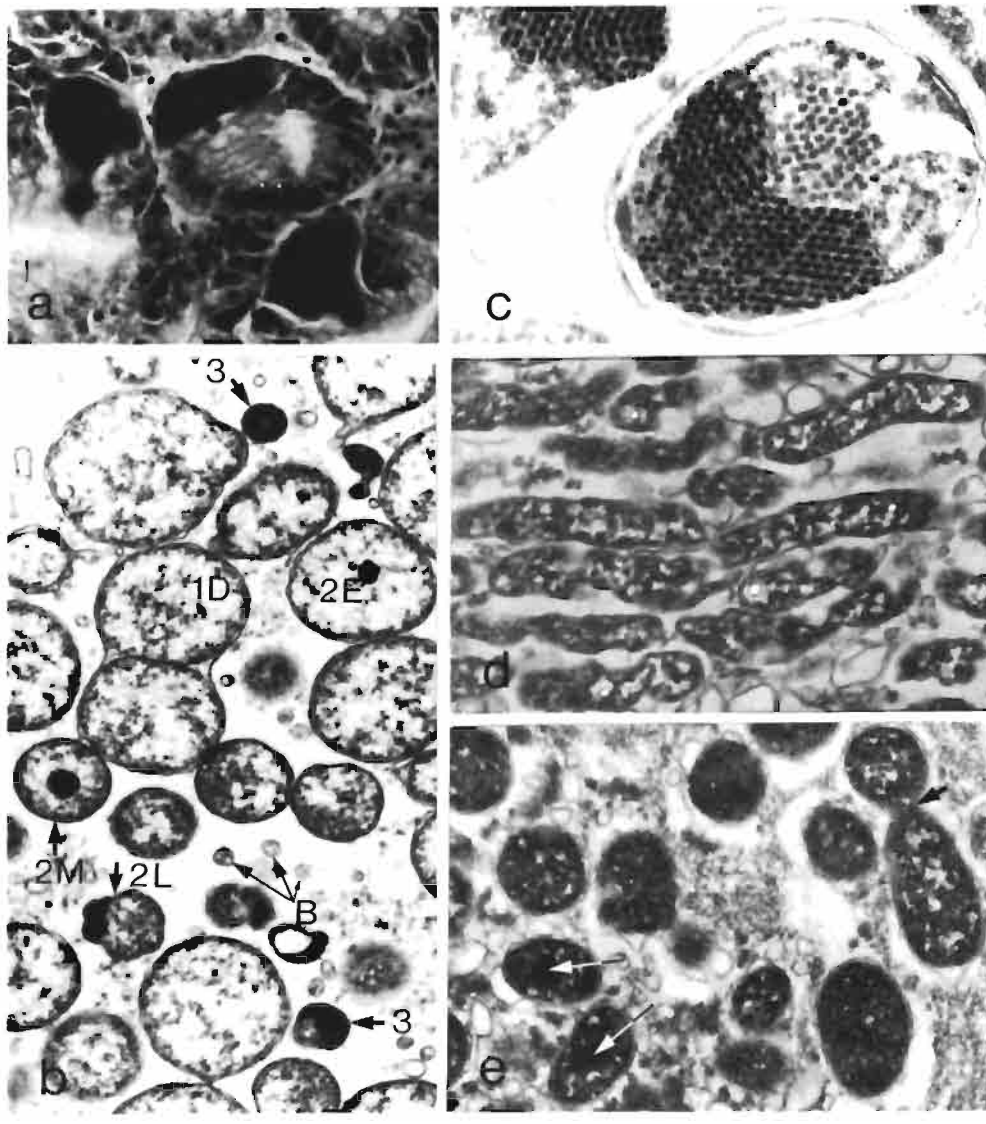

Fig. 13-10: Rickettsiae, Chlamydiae and Mycoplasmas from marine bivalves. (a) 3 intracytoplasmic chlamydial inclusions in epithelial cell of *Mercenaria mercenaria* digestive diverticulum. Inclusions are basophilic, finely granular, and irregular in size and shape. H & E stain, × 350. (b) Chlamydiae from *M. mercenaria* inclusion, showing all developmental stages: large reticulate initial bodies including a pair of daughter cells still united by a cell membrane following binary fission (1D); intermediate bodies in early stage (2E), middle stage (2M), and late stage (2L) of nucleoid condensation; and small, fully condensed elementary bodies (3). Small blebs (B), expelled from initial bodies, lie among the organisms. Two membranes are distinct on initial and intermediate bodies, × 20,335. (c) Greatly distended initial chlamydial body from *M. mercenaria* containing icosahedral viral particles in paracrystalline arrays, × 29,050. (d) Curved and recurved rod-shaped, ribosome-rich, double membrane-bound, Rickettsia-like organisms from inclusion in *Mya arenaria* epithelial digestive-diverticular cell. Lucent vacuoles are apparent in cytoplasm. 'Ghost' cells lie among organisms, × 20,335. (e) Ovoid ribosome-rich Mycoplasma-like organisms from inclusion in gut-goblet cells of *Crassostrea virginica*. Cell division (short arrow) and dense bodies (long arrows) are apparent, × 37,040. (After Harshbarger and co-authors, 1977.)

Table 13-2
Occurrence of Rickettsiae, Chlamydiae and Mycoplasmas in marine bivalves
(Compiled from the sources indicated)

Bivalve host	Rickettsiae	Chlamydiae	Mycoplasmas	Source
Crassostrea virginica	×		×	Otto and co-authors (1979)
			×	Harshbarger and co-authors (1977)
	×		×	Meyers (1981)
Crassostrea gigas	×			Comps and co-authors (1977, 1979)
	×			Comps (1980b, c)
Crassostrea angulata		×		Comps (1980b, c)
		×		Comps and Deltreil (1979)
Ostrea edulis	×			Comps and co-authors (1977, 1979)
	×			Comps (1980b, c)
Mytilus edulis	×	×		Yevich and Barszcz (1980)
Mytilus californianus	×	×		Yevich and Barszcz (1980)
Mercenaria mercenaria	×	× [1]		Otto and co-authors (1979)
		× [1]		Otto and co-authors (1975)
		× [1]		Harshbarger and co-authors (1977)
		×		Meyers (1979b)
	× [2]	×		Meyers (1981)
Mya arenaria	×			Otto and co-authors (1979)
	×			Harshbarger and co-authors (1977)
Tellina tenuis			× [1]	Hill (1976b)
	× [1]			Buchanan (1973, 1978, 1979b)
Donax trunculus	×			Comps (1980b, c)
Tapes decussatus		×		Comps (1980b, c)
Scrobicularia piperata (= *S. plana*)		×		Comps (1980b, c)

[1] with phage
[2] 2 distinct species present

the French Atlantic coast, sometimes concurrently with the oyster pathogen *Marteilia refringens* (see section 'Agents: Ascetospora'). Inclusions in *O. edulis* epithelial cells measure from 30 to 50 µm; the rickettsiae are 0.5 to 0.7 µm long and 0.45 µm in diameter. Host cells containing inclusions are grossly hypertrophied (up to 70 µm) and strongly vacuolated. Both cytoplasm and nucleus are profoundly altered. The agent was found in 4 of 80 *O. edulis* from Bretagne and Arcachon (France), as well as in 1 of 60 hatchery-reared European oysters obtained from California (Comps and co-authors, 1979; Comps, 1980b, c).

Rickettsiae, similar to the above-described, have been observed in digestive-epithelial cells of *Crassostrea virginica* from Chesapeake Bay and Long Island, New York, of *Mytilus edulis* and *M. californianus* from the U.S. Pacific, Atlantic and Gulf coasts, of *Tellina tenuis* from Scotland, and of *Donax trunculus* from France (Buchanan, 1978, 1979b; Otto and co-authors, 1979; Comps, 1980b, c; Yevich and Barszcz, 1980; Meyers, 1981). Other

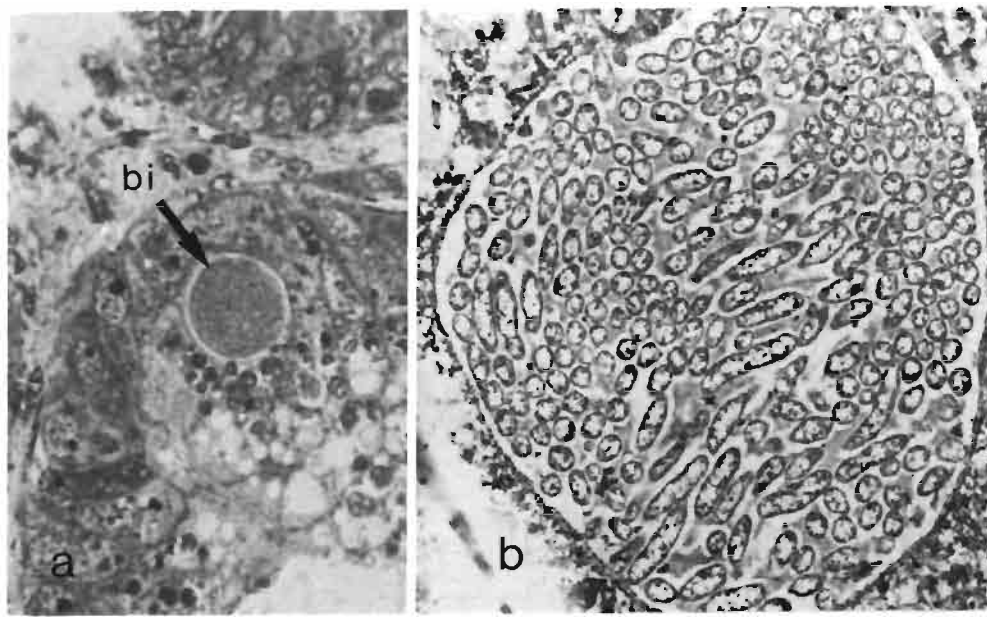

Fig. 13-11: *Crassostrea gigas*. (a) Basophilic inclusion (bi) containing Rickettsiae in epithelial cell of digestive diverticula, × 1,530. (b) Inclusion with contained Rickettsiae, × 8,500. (After Comps and co-authors, 1977.)

organisms of this type occur in gill-epithelial and -connective tissue cells of *Mercenaria mercenaria* from Chesapeake Bay and Long Island, New York (Otto and co-authors, 1979; Meyers, 1981).

Aside from the distinct pathological changes observed in individual infected host cells, the overall pathology caused by most of these agents appears to be light, since usually only a few cells are affected. Although Comps and co-authors (1977) found rickettsiae only in diseased *Crassostrea gigas*, they were unable to identify these organisms as primary cause of death of the oysters.

In contrast, the presence of rickettsiae in *Tellina tenuis* was definitely found to be associated with disease. Up to 75 % of the tellins from sheltered sandy beaches on the Scottish coast were found to harbour these organisms. The shells of affected individuals were markedly thinner and chalky in nature, and their digestive gland was pale yellow compared with the usual dark brown colour. A high proportion of digestive-gland cells were necrotic. Their cytoplasm contained membrane-bound inclusion bodies which, in turn, contained viral particles. No such particles were found in healthy tellins. At first, the disease was believed to be caused by the virus (Buchanan, 1973), but later was identified as a rickettsiosis (Buchanan, 1978, 1979b).

The organisms were found in secretory cells of the digestive gland but not in resorptive cells. First sign of infection was the appearance of a small eosinophilic inclusion body in the region normally occupied by the Golgi apparatus. Progressive enlargement of the inclusion, brought about by growth of the microcolony, resulted in the normally pyramidal cell becoming hypertrophied and spherical in outline. Infected cells were believed eventually to burst, thus releasing masses of rickettsiae into the digestive-tubule lumen. The persistent skeletal outlines of such ruptured 'ghost' cells were a useful

diagnostic feature of infected tellins. Electron microscopy of 'ghost' cells showed the thickened cell wall to consist of necrotic remnants of the host-cell nucleus and cytoplasm that had become compressed to the periphery by expansion of the vacuole containing the microcolony.

In the nucleus of affected cells, first pathological signs were a pale nucleoplasm and prominent nucleoli with pronounced margination, or rimming, of the chromatin. The nuclear envelope appeared swollen and the nucleus shrunken to give a characteristic lobed and irregular outline. Karyorhexis and, eventually, karyolysis characterized the terminal stage (Buchanan, 1978, 1979b).

The life cycle and mode of transmission of bivalve-infecting rickettsiae are unknown. Direct passage from host to host appears probable. Rickettsiae pathogenic to man require passage through an arthropod host (flea, mite, tick). As an exception, *Coxiella burneti*, the etiological agent of Q fever, is unusually stable outside host cells and is transmitted to man by infected dust or droplets. Comps and co-authors (1977), Buchanan (1978, 1979b) and Comps (1980c) believe that the rickettsiae present in *Crassostrea gigas* and *Tellina tenuis* may be related to *Coxiella*. Buchanan (1978) was able to cultivate the *Tellina* agent in chick embryo yolk sac at 37 °C, which indicates that its life cycle possibly involves a warm-blooded host. Buchanan concluded that the micro-organism may have an alternate host in shore birds.

In the light of these findings — and because it is recognized that several zoonoses including coxiellosis (Bell, 1971) can be transmitted from birds to humans —, it has become a matter of urgency to establish the true nature of these rickettsiae present in marine bivalves (Buchanan, 1979b). As shown by Anderson and co-authors (1965), propagation of these organisms in cell cultures under controlled, identical conditions would facilitate their identification.

Even if the molluscan rickettsiae would lack the capacity to infect humans, some detriment must be expected to result from ingestion of heavily infected bivalves. These micro-organisms are known to produce strong toxins capable of killing small mammals within a few hours (Moulder, 1973).

Chlamydiae have been detected in several commercially important marine bivalve species. These organisms display a characteristic infectious cycle. A small 'elementary body' invades the cytoplasm of a host cell and differentiates into a large (non-infectious) 'reticulate body'. The latter undergoes successive divisions by binary fission. The daughter cells ('intermediate bodies') differentiate back into elementary (infectious) bodies which, upon rupture of the host cell, are released and repeat the cycle.

All developmental stages of a chlamydial agent have been observed in *Mercenaria mercenaria* from Chesapeake Bay (Fig. 13-10, a, b). These were contained in amorphous, intracytoplasmic, basophilic, Feulgen-positive, PAS-negative inclusions evoking no obvious host-defense response in epithelial cells of the clams' digestive diverticula. The inclusions ranged in size from barely detectable to huge, irregular bodies up to 100 μm in greatest diameter, which had dilated the host cell nearly 5 times its normal width (22 μm) and had pressed the nucleus and normal cytoplasmic organelles against the plasma membrane. There was rarely more than 1 inclusion in a single tubule at the level of the microscopic section (Harshbarger and co-authors, 1977; Otto and co-authors, 1975, 1979).

Considerably heavier chlamydial infections were found in wild and hatchery popula-

tions of *Mercenaria mercenaria* from Great South Bay, Long Island, New York. In hatchery-held clams, the average number of microcolonies per section was 23 (range 1 to 114) in 29 males and 32 (range 1 to 270) in 25 females, the difference between sexes being statistically significant at the 1 % level. In heavily infected clams, as many as 15 to 20 % of the digestive tubules were damaged or obliterated. Such high infection intensities are likely to compromise the absorptive efficiency of the organ, and to lead to nutritional deficiency and emaciation. However, no histological evidence for such conditions were seen (Meyers, 1979b, 1981).

Chlamydial organisms, similar to the above-described, have been observed in *Crassostrea angulata, Tapes decussatus* and *Scrobicularia plana* from France and in *Mytilus edulis* and *M. californianus* from the U.S. Atlantic, Pacific and Gulf coasts (Comps, 1980b, c; Yevich and Barszcz, 1980).

As with the rickettsiae, the mode of transmission of bivalve-infecting chlamydiae, as well as their possible pathogenicity to humans, are unknown. Fluorescent antibody tests demonstrated that the *Mercenaria mercenaria* agent shares the group antigen specific for chlamydiae, but to a lesser degree than a mammalian chlamydial strain used as control. Therefrom, Meyers (1979b) concluded that the clam agent differs from known strains of chlamydiae. Again, the possibility remains that bivalves are accidental carriers of these potential pathogens, and that warm-blooded alternate or main hosts exist. These may be shore birds, which are ever-present in areas from which infected bivalves have been collected. Since avian chlamydioses can be transmitted to humans (Burkhart and Page, 1971; Storz, 1971), these micro-organisms should be studied more closely (Harshbarger and co-authors, 1977; Buchanan, 1978, 1979b; Yevich and Barszcz, 1980).

Mycoplasmas, round to kidney bean-shaped, 400 to 1,000 nm in length and 250 to 350 nm in cross section (Fig. 13-10, e), have been found in 50 µm-inclusions in gut-goblet cells of *Crassostrea virginica* from Chesapeake Bay and Long Island, New York (Harshbarger and co-authors, 1977; Otto and co-authors, 1979; Meyers, 1981). A similar form was seen in *Tellina tenuis* from Scotland (Hill, 1976b). No further information is available on the host-parasite relationship and specific identities of these micro-organisms. It appears possible that the molluscan mycoplasma(s) do not produce disease symptoms in their invertebrate (carrier?) hosts. Thus, *Mycoplasma pneumoniae*, the etiological agent of human primary atypical pneumonia, can be passed serially in embryonated chick eggs without producing demonstrable lesions, although it is distinctly cytopathic in human tissue cultures.

As stated, mycoplasmas are the smallest free-living organisms, but they can also occur intracellularly (Anderson and co-authors, 1965). Because they are filterable and share other properties with viruses, they have long been included in this agent group. However, unlike viruses they can be cultivated in cell-free medium. Most (but not all) known species are strictly host-specific and produce a wide variety of diseases in homoeotherms including man (Smith, 1971; L. Thomas, 1973). Since the specific identity of the mycoplasma(s) detected in oysters and tellins in unknown, and because various other mycoplasmal species are suspected as the cause of several unrelated diseases in humans, the study of the molluscan mycoplasmas deserves special attention, particularly under the aspect that oysters are usually consumed raw.

There are 3 records of associations with phages, 1 in each of the above groups of

in large numbers, in most of the rickettsiae infecting *Tellina tenuis* from Scotland. The particles were either scattered throughout the cytoplasm or arranged in paracrystalline arrays. Virus-infected rickettsiae were sometimes giant forms, up to 2 μm in diameter, or filamentous forms or rods, up to 4 μm in length. The cytoplasm of these pleomorphic individuals had a low opacity. There appeared to be a clear association between the presence of phages and the more bizarre and variable shape of the rickettsiae (Buchanan, 1978, 1979b).

Chlamydiae, present in *Mercenaria mercenaria* from Chesapeake Bay, sometimes harboured icosahedral viral particles, about 50 nm in diameter and arranged in paracrystalline arrays (Fig. 13-10, c). Infected chlamydiae were so distended that they became discernible under the light microscope (Harshbarger and co-authors, 1977; Otto and co-authors, 1979). Hill (1976b) briefly mentioned the occurrence of a phage in mycoplasmas from *Tellina tenuis* in Scotland. The virus was said to be similar to the *Reovirus* isolated by him from that bivalve species (see section 'Agents: Virales').

The occurrence of phages in these obligate prokaryote organisms represents instances of hyperparasitism. The high host specificity normally displayed by these viruses would — theoretically — fit them ideally for biological control of bivalve-infecting, potentially human-pathogenic rickettsiae, chlamydiae and mycoplasmas, as suggested by Harshbarger and co-authors (1977). Buchanan (1978, 1979b) assumes that these viruses probably limit the spread of phage-susceptible prokaryotes throughout host populations, but that the high mutational capacity of these bacteria make the effectiveness of phage therapy questionable.

The recent discovery of obligate endocytoplasmic prokaryotes in marine bivalves may lead one to suspect that their occurrence might be somehow related to water pollution. However, amorphous basophilic inclusions (ABI's) containing either rickettsiae, chlamydiae or mycoplasmas seem to be ubiquitous for these molluscs regardless of species, location, environmental factors and depth. The ABI's have been termed 'blue bodies', because they stain blue in fresh squash preparations with toluidine blue.

ABI's were found in *Crassostrea virginica* from all 23 major sampling areas in Chesapeake Bay, collected from 1963 to 1973. The pooled mean prevalence for all sites and all times was 5.6 % (1,089 of 19,480). Thirty-two samples of *Mercenaria mercenaria*, collected monthly between June 1970 and April 1973 from Chincoteague Bay, had an ABI prevalence of 14.3 % (103 of 721). Of 28 monthly hard-clam samples, collected during the same period in lower, mid Chesapeake Bay, 10 % (69 of 690) had inclusions. Of 2,401 *Mya arenaria*, collected from 5 areas of Chesapeake Bay over a 7-year period, 20 % (482) were positive for 'blue bodies'. There were no significant yearly, monthly, or geographical differences in prevalence. The presence of inclusions did not seem to be related to industrial or domestic pollution (Otto and co-authors, 1979).

Inclusions containing either of these prokaryotes have also been seen in *Ischadium recurvum, Macoma baltica, Rangia cuneata* and *Tagelus* sp. from Chesapeake Bay (Otto and co-authors, 1979), in *Crassostrea virginica* from the Gulf of Mexico, in *Argopecten irradians, Placopecten magellanicus, Spisula solidissima, Cyprina (Arctica) islandica* and *Crassostrea gigas* from the U.S. west coast, Korea and Japan (Couch, Kern, Yevich and Barszcz in Otto and co-authors, 1979). Farley (in Otto and co-authors, 1979) observed ABI's in digestive-diverticular epithelial cells of *Macoma* sp. from a depth of 457 m off the Californian coast.

Whether the ubiquity and apparent low pathogenicity of these obligate prokaryotes to marine bivalves would indicate a long standing of the association, remains to be established. The possibility exists that the molluscs merely serve as tolerant carriers for these organisms, which inflict overt pathogenicity on other (vertebrate?) hosts.

In addition to the large number of terrestrial homoeothermic animals which are known hosts for these groups of pathogenic micro-organisms, there is an endless array of potential sources from poikilothermic, non-molluscan hosts. Insects and ticks are well-known vectors of rickettsiae and mycoplasmas. 'Salmon poisoning disease' is a unique helminth–transmitted rickettsiosis of canines (dog, fox, coyote). The canines acquire the disease by feeding on salmon or other fish infested with metacercariae of the digenetic trematode *Nanophyetus salmincola* which, in turn, are infected with *Neorickettsia helminthoeca*. The micro-organism affects neither the flatworm nor its fish host but produces fatal infections in canines (Philip and co-authors, 1953, 1954a, b; Philip, 1955). Infections occur in freshwater but are carried to the sea by catadromous salmon. Fishes returning from the sea after 33 months may carry viable metacercariae with rickettsiae still capable of producing fatal infections in dogs (Farrell and co-authors, 1964; Millemann and co-authors, 1964).

Rickettsia-like organisms have also been observed in marine crustaceans (Vago and co-authors, 1970; Federici and co-authors, 1974). Prokaryote inclusions, known as 'epitheliocystis', occur in fish skin and gill epithelium. The condition may cause severe disease and host death (Hoffman and co-authors, 1969; Wolke and co-authors, 1970; Paperna and Zwerner, 1976; Paperna, 1977, 1980; Paperna and co-authors, 1977, 1978, 1980; Zachary and Paperna, 1977; Paperna and Baudin Laurencin, 1979; Paperna and González, 1980). Possible relationships of the bivalve prokaryotes with fish-pathogenic species should, therefore, also be taken into consideration.

Agents: Fungi

Mycotic infections have long been listed among the most serious diseases of marine bivalves, until it was recognized that the most destructive 'fungus', *Dermocystidium marinum (Labyrinthomyxa marina)* is not a fungus at all, but a protistan related to the 'sporozoans' (see section 'Agents: Apicomplexa'). Fungi have been found to cause 'shell disease' in marine bivalves, and to produce mycotic infections in the soft parts of adult and larval stages. Some of these presumed fungal organisms may actually be members of the fungus-like labyrinthomorph protistans (see section 'Agents: Labyrinthomorpha').

Fungi play a prominent part in the biodegradation of calcareous substrates including animal shells and skeletons. Several species are known to ramify throughout the shells of both dead and living bivalves, utilizing the energy present in the organic shell matrix, which is composed of proteins, scleroproteins and polypeptides (Kölliker, 1860a, b; Bornet and Flahault, 1889; Zebrowski, 1937; Peyer, 1945; Cole, 1956a; Johnson and Sparrow, 1961; Johnson and Anderson, 1962; Yonge, 1963; Höhnk, 1969; Kohlmeyer, 1969; Cavaliere and Alberte, 1970; Vol. I, Chapter 12).

The occurrence of fungi in decaying molluscan shells is quite common. A chlamydospore-producing organism, tentatively assigned to the genus *Endogone*, has been found in a cast-off valve of a smooth jingle shell *Anomia simplex* from Pivers Island, North

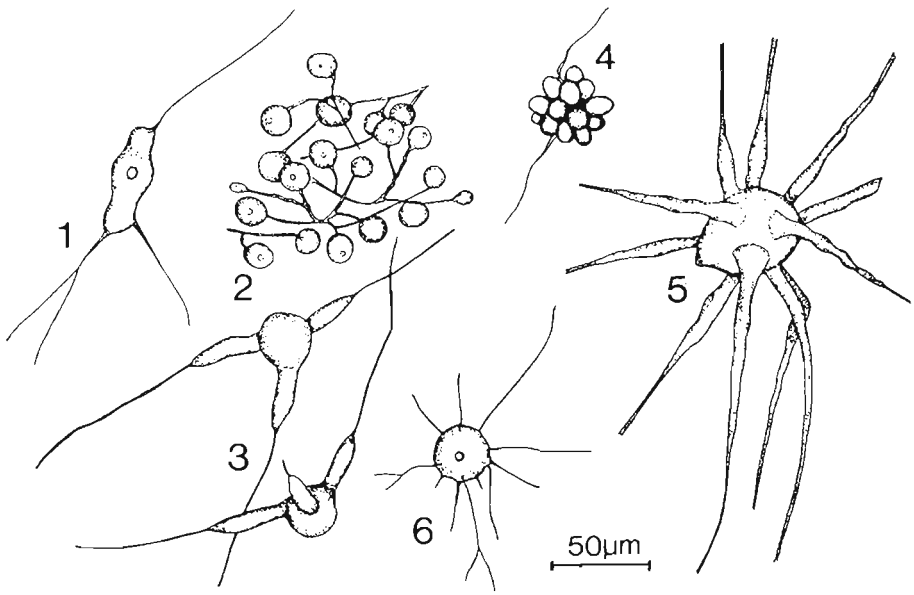

Fig. 13-12: Fungi from molluscan shell fragments. 1: Fruiting structure with central pore and hyphae; 2: *Endogone*-type fructifications with pores and subtending hyphae; 3: sporangia with oblong outgrowths; 4: sporangial clump; 5: large sporangium with tapering hyphae; 6: spherical fruiting body showing details of pore and hyphae. (After Cavaliere and Alberte, 1970.)

Carolina, USA (Johnson and Anderson, 1962). The fungus consisted of intra- and extramatrical hyphae and extramatrical chlamydospore-like bodies. Similar organisms have been observed in unspecified molluscan shell fragments collected along the North Atlantic coasts of the United States and Iceland (Cavaliere and Alberte, 1970; Fig. 13-12). It ist not known whether these organisms may also attack the shells of living molluscs. At least one species, *Ostracoblabe implexa* Bornet and Flahault, 1889, causes damage in living oysters (see below).

'Shell disease', in France known as 'maladie de la coquille', 'maladie du pied', or 'maladie de la charnière', is a mycosis affecting mainly *Ostrea edulis* in western European waters. A detailed description of the disease syndrome has been presented by Alderman and Jones (1971a). The causative agent lives in the shell. Initially, the affection appears as tiny chalky white specks below the surface of the sub-nacreous layer. The spots have a definite relief, giving the inner shell surface a rough texture. As the fungus proliferates, they develop into greenish, rubbery warts and knobs composed of conchiolin. The warts coalesce and may, eventually, cover one or both valves. In the advanced stage, infected shells may become grossly deformed and valve closure may become impossible. Such badly affected oysters usually die.

Squash preparations of shell material, decalcified in a 5 % solution of disodium diaminoethylenetetraacetate (Na_2 EDTA), disclose the presence of a dense mycelial network composed of hyphae which are straight and hyaline and 1.5 to 2.5 µm in diameter. At irregular intervals (40 to 100 µm) the aseptate mycelium bears small ovoidal dilations, 4 × 6 µm in dimension and termed 'pro-chlamydospores' by Alderman and Jones (1971a). Septa are formed only in senescent or dying mycelium, and chlamydospores form

from the pro-chlamydospores at low temperatures (5 °C). Growth of the phycomycetous fungus is optimal at higher temperatures (30 °C) and is inhibited by light. Despite quite extensive investigations, no fruiting structures have been found for this fungus, which Alderman and Jones (1971a) identified as *Ostracoblabe implexa,* a species originally (superficially) described by Bornet and Flahault (1889) from dead shells of *Ostrea edulis, Solen* sp. and other bivalves from the French coast of the English Channel.

Initially, *Ostracoblabe implexa* infections are confined to the shell. The burrowing fungus obtains its nourishment from breakdown of the proteinaceous shell matrix. Growth of the agent within the shell does no harm to the oyster's soft tissues. However, when the mycelium reaches the inner shell surface and continues to ramify between the mantle and shell, it sets up an irritation of the living tissues. As a result, adjacent mantle epithelium cells are converted from an almost squamous condition to a high columnar form, closely resembling those lying under, and secreting, the conchiolin material of the hinge ligament. Mantle cells irritated by the mycelium secrete similar material, which has a very much higher protein content than the normal shell matrix. The horny material of the developing warts sometimes consists of almost 100 % conchiolin — an ideal nutrient for the fungus which can then grow faster, whereby the disease becomes self-perpetuating. When the infection occurs under the adductor muscle attachment, a horny boss is produced. This boss-like type of wart is not found outside the muscle attachment and is the form of shell disease known in France as 'maladie du pied' (Giard, 1894). The latter term is an obvious misnomer because the foot is lacking in all adult oysters. In severe, advanced stages of the disease, the adductor muscle is weakened or even detaches from the shell. The oyster becomes emaciated, loses weight and eventually dies (Korringa, 1951d; Alderman and Jones, 1971a).

Severe pathological modifications may also develop in the hinge region of affected shells, with marked stimulation of shell growth resulting in excessive development of the hinge area in relation to the remainder of the shell, giving the oyster a beaked appearance. The centre hinge portion may become heavily warted and the edges of both valves may become thick and irregular in outline. Much of the wart material on the valve edges becomes eroded and the beaked area accumulates mud and bacteria. This is the form of shell disease known in France as 'maladie de la charnière'. The combination of the 2 types of damage — the 'pied' and the 'charnière' form —, occurring together in one oyster, is the most severe form of shell disease which is, however, encountered in only a small proportion of affected oysters (Alderman and Jones, 1971a).

Concomitant with the severeness of shell disease there is a decline in the oyster's condition index*. Alderman and Jones (1971a) found the C. I. of oysters from Althorne Creek, River Crouch (Essex, England) to drop from about 145 for disease-free specimens to 116 for individuals with heavy warting and possibly affected meats, and to even 79 for extremely heavily diseased oysters with visibly affected meats.

An atypical form of shell disease that exists in young *Ostrea edulis* is characterized by a thickening of the shells, accompanied by the formation of numerous white patches, but without deformation of the attachment area of the adductor muscle (Cole and Hancock, 1956).

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* Condition Index (C.I.) $= \frac{\text{Meat-dry weight}}{\text{Volume between valves}} \times 100$

Knowledge of the mode of spore dispersal and entry of *Ostracoblabe implexa* into molluscan shells is incomplete. There is, as yet, little evidence of any chemotactic attraction of the zoospores to host tissues. It has been observed, however, that many *O. implexa* infections are sited near the burrows produced by polychaete annelids, *Polydora* spp. (see section 'Agents: Annelida'). It appears, therefore, probable that infective stages of the fungus are swept into the oyster shell by the feeding currents of these worms (Alderman, 1976). Frequently also, the initial penetration of the oyster valves by shell disease is preceded or accompanied by the presence of burrowing algae, such as *Hyella* spp., which are common shell inhabitants (Alderman and Jones, 1971a; see section 'Agents: Algae').

Artificial infections, attempted by attaching pieces of diseased shell to the valves of healthy oysters or by adding fresh mycelium to the water of the culture containers, have, thus far, met with little success. Under natural conditions, a small percentage of disease-free 1-year-old Norwegian oysters, relaid in 1966 in trays at 3 sites in the Rivers Crouch and Roach (England), became infected. Of 1,050 oysters examined, 1.5 % developed shell disease that year, whilst in the following year 4 % of 450 of the then 2-year-old individuals were positive for *O. implexa*. Of another lot of 1-year-old Norwegian oysters, relaid in 1967, 25 % developed disease that year. Clearly, 1967 was the warmer year and infections were much higher (Alderman and Jones, 1971a). These findings indicate that progress of shell disease is usually slow, but that high water temperatures (above 19 °C) favour spreading and virulence of the pathogen. Mass mortalities due to shell disease have occurred only in warm summers. Water temperatures in excess of 20 °C for 2 or more weeks are necessary for high infection levels.

Korringa (1951d) suggested that young oysters are more susceptible to shell disease than older individuals — a view supported by the findings of Alderman and Jones (1971a). Apparently, older oysters may recover from an infection under good feeding conditions. Control of the disease could be achieved by bathing infected oysters in a weak solution of a mercury-base disinfectant. After the warm summer of 1947, more than 15 million one-year-old oysters had been saved from certain death by this treatment (Korringa, 1950a, 1951d; Lambert, 1951a; Cole, 1956a). Alderman and Jones (1971a), however, point out that mercuric chloride is a hazardous chemical to use, and that it is difficult to ensure adequate shell penetration. It would be unwise, therefore, to recommend its general use. Although careful application might eliminate shell disease for a while, there is far too great a risk of causing wide-spread pollution.

According to Alderman (1976), shell disease was first recognized in oysters from France in 1878. Bornet and Flahault (1889) described and named its causative agent, *Ostracoblabe implexa*, but did not describe the disease syndrome. Similarly, Dollfus (1921a) recorded *O. implexa* as growing in oyster shells but did not link it with shell disease, which he mentioned separately. Korringa (1951a) was the first to establish the mycotic nature of the disease but was unable to isolate the pathogen. Alderman and Jones (1967) achieved isolation and cultivation of the fungus in liquid yeast extract-peptone medium but were unaware of its specific identity. Eventually, Alderman and Jones (1971a) identified it as *O. implexa* and subsequently (1971b) described in detail its physiological requirements.

To date, shell disease has been reported from *Ostrea edulis* from the Netherlands, France, the United Kingdom, Ireland and Portugal but not from Norway, Denmark and

Germany. It has been recorded among imported French and Dutch oysters in the Limfjord, Denmark, but shows no signs of becoming established in Danish waters. Apparently, water temperature is the main factor limiting its distribution, summer temperatures of about 19 °C being the lower threshold for its activation and spreading (Dollfus, 1921a; Voisin, 1931; Korringa, 1950a, 1951d, 1952a; Cole, 1950, 1956a; Lambert, 1951a; Cole and Hancock, 1956; Cole and Waugh, 1956; Alderman and Jones, 1967, 1971a, b; Alderman, 1976). In addition to *O. edulis, Crassostrea angulata* is susceptible to shell disease but contracts only light infections.

During the thorough investigation on shell disease, conducted by Alderman and Jones (1971a), the authors were unable to trace symptoms referable to *Ostracoblabe implexa* infections in bivalves other than oysters. They concluded that oysters are the only susceptible organisms. Moreover, Alderman and Jones (1971a, p. 15) stated that "it has not been possible to trace any published report of damage to other shellfish". They probably overlooked Bornet and Flahault's (1889, p. CLXXII) statement that *O. implexa* has been found "sur diverses coquilles mortes", as well as the caption accompanying Plate XII of the latter authors' publication, which shows in Fig. 1 "une coquille de *Solen* toute pénétrée des canaux de *l'Ostracoblabe*". Korringa (1950a, 1951) reported the fungus from *Cardium edule* shells (see below). It appears that *O. implexa* is endemic in coastal waters throughout western Europe, but that it attacks only oysters — and only under certain well defined conditions — in a way severe enough to produce visible symptoms.

In Dutch waters, for example, the fungus was found to thrive abundantly in old rotting *Cardium edule* shells, which were used in tremendous quantities as oyster-spat collectors from 1920 to 1930. The epizootic nature of shell disease was attributable to the prolific production of fungal zoospores hatching under suitable environmental conditions. A better understanding of the character and epizootiology of the disease led to a thorough clearing of planting areas from cockles and other decaying molluscan shells which, in turn, resulted in substantial reduction of shell-disease incidences and oyster-stock improvement (Korringa, 1950a, 1951d). Removal of dead molluscan shells and diseased oysters, and their disposal on land as a method to prevent development of shell disease, has also been recommended by Alderman and Jones (1971a).

In Dutch oysters, shell disease has been known since 1902, but at that time occurred only in a limited percentage of *Ostrea edulis* and was never serious. It was in the early 1930's that the above-described introduction of methods intended to increase oyster production eventually led to such a dramatic spreading of the disease that the Dutch oyster industry was threatened with extinction (Korringa, 1950a). Among oysters imported into the region of Marennes, French Atlantic coast, from the Netherlands, losses amounted to 40 %, and most of the survivors were also infected. Although being fat and readily edible, affected oysters had so badly disfigured shells that they were hardly marketable (Voisin, 1931). Apparently introduced with infected individuals of *Ostrea edulis* relaid from Bretagne (France), shell disease was first recorded in Britain in 1949. Dispersal of imported oysters over various beds subsequently led to abundant infections of native *O. edulis*. Individuals of *Crassostrea angulata*, grown in England, also became infected. In 1953, up to 60.4 % of the oysters cultivated in shallow warm creeks in Essex had shell disease, which was regarded as a major cause of losses (Cole, 1950, 1956a; Cole and Hancock, 1956; Cole and Waugh, 1956). Alderman and Jones (1971a) diagnosed shell disease in 68 % of 987 oysters from Althorne Creek, River Crouch, Essex, and of these

28 % were considered unsuitable for sale due to their unpleasant appearance and weak and watery meats.

There are several inconclusive records of what appears to be shell disease or a similar affection from oysters in various parts of the world.

Galtsoff (1964) reported the rare occurrence of shell disease in *Crassostrea virginica* from the southern United States, but did not consider it a serious threat to the local oyster industry. 'Yellow pustule disease', described by Mackin (1962) from American oysters in Louisiana, was much like the European 'maladie du pied', and like the latter usually attacked the adductor muscle. In India, Durve and Bal (1960) recorded what they considered to be 'shell disease' in backwater oysters *C. gryphoides* cultivated in a farm near Bombay. Of nearly 3,000 individuals examined during a period of 2 years, only 7 were found affected. In the fall of 1956, a disease bearing much resemblance to shell disease occurred in 10 % of *C. gigas* of both Japanese and local origin from Pender Harbour, British Columbia (Canada). Mostly about 4–year-old oysters were affected. In these, the adductor muscle scars were partly covered with black protuberances up to 1.25 cm high and nearly 2.5 cm in diameter, the thin outer covering of which was of a conchiolinous nature. On some shells the base of the abnormality was expanded considerably by a raised portion of nacre covering an extension filled with puslike secretion containing a heavy concentration of bacteria in addition to circular bodies, 10 μm in diameter, which constituted the main portion of the material (Quayle, 1969). It appears that shell disease caused by *Ostracoblabe implexa* or similar fungi is worldwide distributed in oysters. However, Alderman and Jones (1971a) consider 'shell disease' reported from oysters outside Europe to be caused by agents other than *O. implexa*.

Unidentified, but apparently harmless fungi, which appear as a network of fine lines underlying the inner surface of the shell, have been seen in European oysters from England, and a condition similar to shell disease in oysters has been noted in mussels *Mytilus edulis*. The latter infection seems to be associated with malformation of the shells and loss of condition (Cole, 1956a).

The organism observed in oyster shells by Cole (1956a) may have been *Lithopythium gangliiforme*, another shell-burrowing fungus described by Bornet and Flahault (1889) in the same paper in which they described *Ostracoblabe implexa*. It consists of a horizontal network of interlaced filaments, 1.75 to 3.5 μm in thickness and sometimes profusely branched. The description of the fungus is incomplete in that only a vegetative mycelium has been reported. *L. gangliiforme* thrives in the shell at the level occupied by perforating algae (see section 'Agents: Algae'). There is no evidence that it produces disease. Bornet (1891) observed superficial resemblances between the mycelia of *L. gangliiforme* and the lichen *Verrucaria calciseda* but eventually concluded that they are different.

Althornia crouchii, a presumed biflagellate phycomycetous fungus, has been isolated, together with *Ostracoblabe implexa*, from *Ostrea edulis* exhibiting the 'wart stage' of shell disease (Jones and Alderman, 1971). The sporangial isolate had previously been mistaken for a stage in the development of *O. implexa* (Alderman and Jones, 1967). Although *A. crouchii* and *O. implexa* are similar with respect to their nutritive requirements, they were regarded as unrelated. Alderman and Jones (1971b) and Jones and Alderman (1971) maintain that *A. crouchii* has a number of characters in common with members of the Thraustochytriaceae (which are labyrinthomorphs, however; see section 'Agents: Labyrinthomorpha'). A relationship between the organism and shell disease of oysters has not

been established. Manier and Bouix (1979) regard *A. crouchii* as a 'fungus' of dubious affinities. In a review of fungal diseases of marine animals, Alderman (1976) does not even mention it in connection with shell disease. *A. crouchii* may not be a fungus at all.

Giard (1894) clearly described the symptoms of shell disease in the form of 'maladie du pied' from *Ostrea edulis* in the Gulf of Gascogne (France) but attributed the affection to a 'schizomycete' (i.e., a bacterium), named *Myotomus ostrearum.* Voisin (1931) isolated a fungus from shell-diseased Dutch oysters relaid on Marennes (French Atlantic coast) oyster beds, which he believed to be an ascomycete similar to *Monilia (= Candida)* or *Saccharomyces.* Neither of these authors, however, gave a clear description of the organism. It is possible that both of them saw free-floating sporangia and zoospores of *A. crouchii.*

Microscopic inspection of sectioned byssus material from *Mytilus galloprovincialis* from the Gulfs of Trieste and Genova (Italy) revealed fungal hyphae, scattered abundantly throughout the byssus stem (Vitellaro-Zuccarello, 1973). The hyphae appeared as typical refringent, branched filaments within the opaque homogenous byssal matrix (Fig. 13-13).

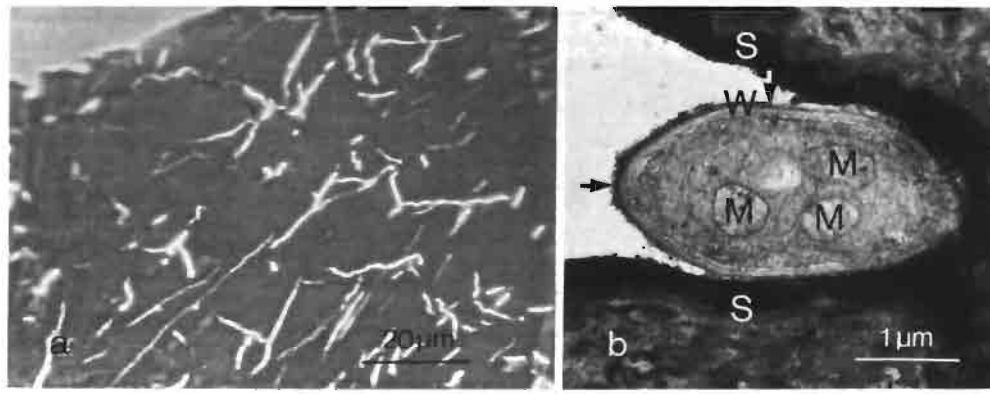


Fig. 13-13: *Mytilus galloprovincialis.* Fungal hyphae from byssus threads. (a) Hyphae appearing as refringent branching filaments within opaque matrix of byssus. Phase contrast, semithin section cut tangentially to byssus-stem surface, epoxy medium. (b) Hypha adhering to byssus surface (S). Note composite structure of cell wall (W), clearly visible where cell is not in contact with byssal matrix. M mitochondria. (After Vitellaro-Zuccarello, 1973.)

As shown by electron microscopy, the hyphae, which range in diameter from 2 to 4 µm, typically contain cytoplasmic vacuoles, and feature a cell wall with a septal apparatus characteristic of ascomycetous fungi (Fig. 13-14).

As mussels taken at different times from different stations all harboured the fungus in their byssus, it was assumed that the occurrence was neither accidental nor merely occasional. The byssus is composed of organic components, mostly tannate, that are fairly resistant to both chemical and physical agents. Drastic alterations of the distal portion of the byssus of *Mytilus galloprovincialis*, accompanied by extensive hyphal intrusion and greatly increased porosity, and subsequent invasion by algae and protozoans, appear to blame the fungus for being a major factor in the degradation of byssal material. It seems possible that such fungal breakdown of byssus could interfere with the mussel's ability to attach firmly to the substrate.

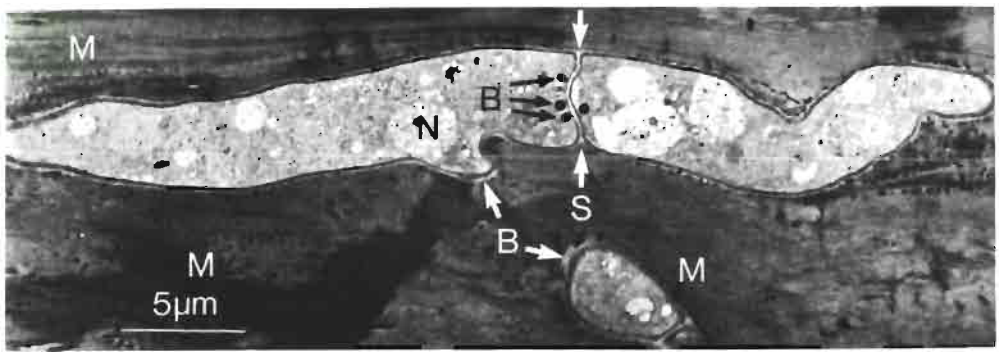


Fig. 13-14: *Mytilus galloprovincialis*. Fungal hyphae from byssus threads. Electron micrograph showing branching (B, white arrows), septation (S) of hypha and adhesion of byssal matrix (M) around hypha. N: nucleus; B (black arrows): Woromin bodies. (After Vitellaro-Zuccarello, 1973.)

Hirsch (1921) and Dollfus (1923c) reported on a disease of unknown etiology, affecting *Mytilus edulis* in Dutch waters and causing recurrent mass mortalities since its first appearance in 1901. The initial and characteristic sign of the disease consists of a degradation of the byssus threads, followed by weakening of the shell ligament and blackish discolouration of the mussel's soft body. Whether a fungus – similar to that reported from *M. galloprovincialis* by Vitellaro-Zuccarello (1973) – may have been responsible for the deterioration of the byssal and ligamental material, has not been determined.

Fungi may also infect the soft parts of pelecypods. *Sirolpidium zoophthorum*, a phycomycete of the family Sirolpidiaceae, was found responsible for some of the epizootic mortalities recorded in cultivated bivalve larvae. Not all species appear to be equally susceptible to the agent. In *Mercenaria mercenaria* and *Teredo navalis* larvae, infections were transmitted easily, often assuming epizootic proportions, while larvae of *Crassostrea virginica* were rarely affected. *Argopecten irradians* and *Tapes semidecussatus* frequently contracted infections, but rarely did the disease assume epizootic proportions.

Larvae of all ages, from the very early free-swimming veligers to those ready to undergo metamorphosis, as well as juveniles up to 400 µm in size, can be parasitized by the fungus. During the outbreak of an epizootic, many larvae may be observed in various stages of disintegration, with the fungus quite apparent in their interior as a contorted, looped and sparsely branched mycelium of torulose character with constrictions at intervals between the swollen and often lobed segments.

As the fungus develops rapidly, the segments mature into sporangia, each with an exit tube extending to the exterior and sometimes protruding considerable distances from the larval shells. Infection of the bivalves occurs by zoospores, which emerge through these tubes into the surrounding water. In heavily infected larval cultures, mortality is usually almost complete within 2 to 4 days, with a small number surviving as if they had acquired immunity (Davis and co-authors, 1954; Loosanoff and Davis, 1963).

When isolated on nutrient agar, *Sirolpidium zoophthorum* zoospores germinate and grow in the absence of bivalve larvae. The highly aerobic fungus makes good growth at temperatures ranging from 20 to 30 °C. Its thallus is branched and septate, 10 to 15 µm in diameter when young and up to 82 µm in diameter at maturity. In thalli having reached a

length of about 40 μm, large spherical vacuoles become visible. Sporangia, provided with a discharge tube 15 to 142 μm in length and 5 μm in thickness, are formed from swollen terminal cells. Planonts, measuring about 2 × 5 μm, are biflagellate, heterokont and monoplanetic. Occasionally produced resistant spores are ovoid, light golden-brown, thick-walled and about 45 to 90 × 40 to 80 μm in dimension. Gemmae may also be formed. Vishniac (1955) has given a detailed description of the morphology, physiology, nutrition and systematic position of the agent.

Sirolpidium zoophthorum also occurs in natural populations of oyster larvae, but the extent of infection and damage is unknown (Johnson and Sparrow, 1961). Davis and co-authors (1954) and Loosanoff (1954) speculated that this or related fungi may cause epizootics among bivalve larvae in nature.

Leptolegnia marina, a saprolegniaceous fungus affecting pea crabs *Pinnotheres pisum* (see section 'Agents: Decapoda'), has also been found to invade *Cardium echinatum* from Torquay (England). First signs of the infection were the appearance of white pustules on the foot and the mantle margin (Fig. 13-15, 1). No infection was seen in the gills. As the

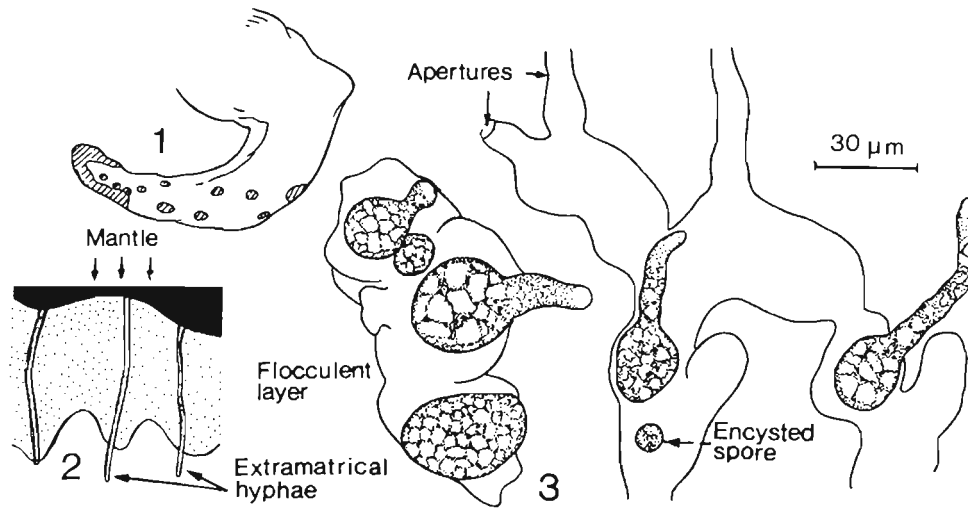


Fig. 13-15: *Leptolegnia marina* infecting *Cardium echinatum*. 1: White mycotic pustules on host foot; 2: hyphae extending from mantle and passing through flocculent layer composed of mucus, sloughed-off cells, protozoans and bacteria; 3: unusually coarse hyphae in mantle. Contents of most of these have been used up in zoospore formation, with a number of rounded bodies of vacuolated protoplasm remaining. Note formation of hyphae from several of these bodies, and apertures through which zoospores from old sporangium have been released. (After Atkins, 1954.)

mycosis progressed, the foot became almost entirely covered with a white flocculant layer, which, however, was largely composed of bacteria. Upon death of the cockle, the mantle tissue surrounding the pustules was found to be invaded by a network of hyphae, but there was little extramatrical growth (Fig. 13-15, 2). On the foot, the extramatrical hyphae reached a length of about 150 μm. In the mantle margin, broad vegetative, irregularly branching hyphae, 7.5 to 40 μm in diameter, were present (Fig. 13-15, 3). Filamentous sporangia, formed directly from unchanged hyphae, liberated biflagellate, pear-shaped zoospores, about 13 μm long. Encysted spores were 6 to 10 μm in diameter. It is not quite clear whether *L. marina* is a primary pathogen of *C. echinatum* or merely a secondary

invader, since the cockle was already moribund when the fungal infection was first noticed. A fungus, believed to be identical with the above-described one, was also found in a single individual of *Barnea candida*. In this lamellibranch, the subfilamental tissue of the gills was found to be infected (Atkins, 1929, 1954).

As pointed out by Johnson and Sparrow (1961) and Johnson and Pinschmidt (1963), Miss Atkins' description of *Leptolegnia marina* departs radically from the concept of the genus and rather fits that of *Leptolegniella*. Therefore, the organism should be restudied.

In certain years preceding 1920, New England oyster growers complained of pink discolourations appearing in oysters during shipment. Oysters which were in good condition when shipped were often found to be pink when reaching their destination. Microscopic examination revealed infection, of such oysters, with a 'pink yeast'. Cells were elliptical and 2.8 to 7.0 × 4.1 to 9.8 μm in dimension. Granules, lipid globules and vacuoles were present and budding forms were common. There was no mycelium and no spores were seen. In culture, the organism grew best in the absence of oxygen on dextrose agar at 20 °C, but not at 37 °C. The discolouration, which occurred in the mantle fluid ('shell liquor') or on the oyster itself, was found to be due to a water-insoluble pink pigment produced by the fungus. The agent was considered to belong 'to that group of yeast-like forms called Torulae', which are placed among the Deuteromycetes (Hunter, 1920).

The infection of oysters appeared to take place mainly during handling. Of samples taken from oyster houses and utensils, 73.9 % were positive. The yeast was found less frequently in oysters before they were brought to the oyster houses, only 26.3 % of oysterfield samples being positive. Occasionally, the agent could be isolated from sea water, particularly from surface samples (2.8 % positive), but not from the sediment. It appeared to be non-pathogenic to oysters (Hunter, 1920), but its non-pathogenicity to humans has not been examined. One might speculate about the relationship of the agent from the oysters with yeasts of the genus *Cryptococcus*, which contains marine or halophilic species. Fell (in Phaff and Fell, 1970) isolated 108 strains of *C. albidus* var. *albidus* from coastal waters, sediments, marine plants and invertebrates in Biscayne Bay (Florida), the Bahamas, the Caribbean and the Indian Ocean. This asporogenous yeast, which is worldwide in distribution and has been isolated commonly from both terrestrial and marine habitats, produces cream-coloured to slightly yellowish or tan-coloured colonies, but there are other species in the genus which produce a red or pink pigment, i.e., *C. laurentii* var. *laurentii*, which has also been isolated from marine sources. Last, not least, it should be mentioned that the genus *Cryptococcus* contains species pathogenic to humans, i.e., *C. neoformans*, the etiological agent of human cryptococcosis. In the United States, cryptococcosis (blastomycosis) was formerly termed torulosis because *C. neoformans* was originally named *Torula histolytica*. It produces cream-coloured to slightly pink growths.

Another deuteromycete, *Sterigmatomyces halophilus*, has been found in air samples taken at Biscayne Bay, Florida. Other strains were isolated from the Indian Ocean, at depths ranging to 1,997 m (Fell, 1966, 1967). Sawyer and Meyer (1977) reported the occurrence of what appears to be *S. halophilus* in the mantle fluid of *Crassostrea gigas* from Nanaimo, British Columbia, Canada (Fig. 13-16). When cultivated on corn meal-sea water agar medium, the fungus formed peculiar colonies of crystalline or flaky appearance, and substrate utilization was exactly as reported by Fell (1966) for *S. halophilus*. There was no evidence that the fungus is involved in disease of the Japanese oyster. Its non-

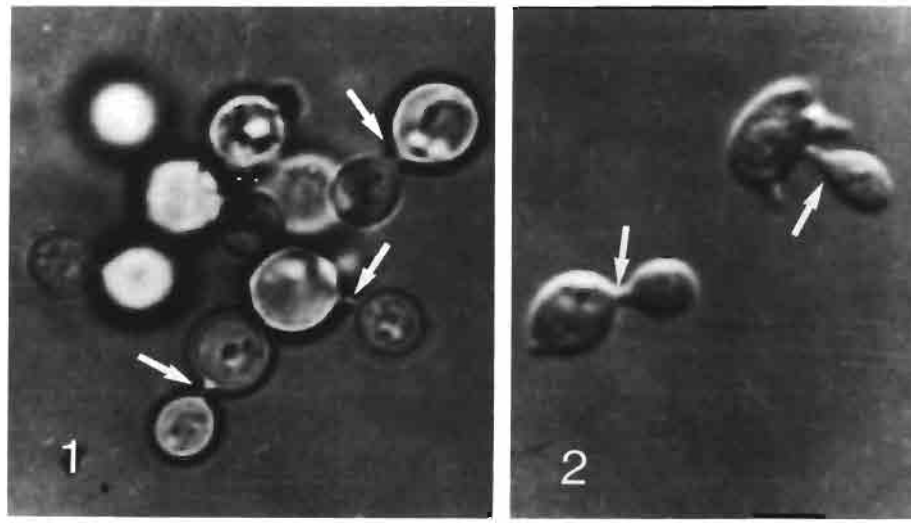


Fig. 13-16: *Sterigmatomyces halophilus* from *Crassostrea gigas*. 1: Spherical forms, bright field, × 1,600; 2: ovoid forms, Nomarsky interference contrast, × 1,600. Note formation of conidia on interconnecting sterigmata (arrows). (After Sawyer and Meyer, 1977.)

pathogenicity to humans has not been tested. It should be mentioned that a strain initially identified as *S. halophilus* has been isolated as a probable contaminant in a human case of keloid blastomycosis in Brazil. It may, however, represent a distinct taxon (Fell, 1970).

In addition to the presumed *Sterigmatomyces halophilus*, 2 other fungi were isolated by Sawyer and Meyer (1977) from the mantle fluid of *Crassostrea gigas*. On corn meal-sea water agar medium, one formed cream-coloured, and the other pink colonies. These yeasts were not identified further, but the latter may be identical with the one reported from *C. virginica* by Hunter (1920).

DISEASES CAUSED BY PROTISTANS

Agents: Sarcomastigophora (the 'Flagellates' and the 'Rhizopods')

In the modern classification scheme of the kingdom Protista, the classical 'zooflagellates' have been accomodated as class Zoomastigophorea, the 'rhizopods' or 'amoebae' as superclass Rhizopoda in phylum Sarcomastigophora (Table 13-3).

Zooflagellates have been reported as 'parasites' of marine bivalves, but probably are saprobiotes, invading moribund molluscs only. Several 'rhizopods', described as disease agents in bivalves, are probably referable to — or have been identified as — members of the Labyrinthomorpha (see below). It appears that the Sarcomastigophora do not contain true molluscan pathogens.

Zooflagellates, provided with 4 anterolateral and 2 posterior flagella and containing a contractile vacuole, have been reported as *Hexamita inflata* from *Ostrea edulis* from Marennes, France (Certes, 1882a, b). The flagellates were said to reproduce normally in the oyster's stomach. Van Banning (1979b) states that *'H. inflata'* has 6 instead of 4 anterior flagella. Schlicht and Mackin (1968) doubted the correctness of Certes' (1882a, b)

Table 13-3
Taxonomic position of protistan parasites of marine bivalves in the classification scheme based on Levine and co-authors (1980)

	Genera
PHYLUM I: SARCOMASTIGOPHORA (the 'Flagellates' and the 'Rhizopods')	
Subphylum I. Mastigophora	
Class 2. Zoomastigophorea	
Order 5. Diplomonadida	
Suborder 2. Diplomonadina	*Hexamita*
Subphylum III. Sarcodina	
Superclass 1. Rhizopoda	
Class 1. Lobosea	
Subclass 1. Gymnamoebia	
Order 1. Amoebida	
Suborder 3. Flabellina	*(Flabellula)*
Suborder 5. Acanthopodina	*Acanthamoeba, Hartmannella*
Order 2. Schizopyrenida	*(Vahlkampfia)* (= Labyrinthomorpha?)
PHYLUM II: LABYRINTHOMORPHA (the 'Fungus-like Protistans')	
Class 1. Labyrinthulea	
Order 1. Labyrinthulida	*Labyrinthula, Labyrinthuloides, Thraustochytrium, Thanatostrea, (Vahlkampfia?)*
PHYLUM III. APICOMPLEXA (*'Dermocystidium'*, the 'Gregarines' and the 'Coccidians')	
Class 1. Perkinsea	
Order 1. Perkinsida	*Perkinsus*
Class 2. Sporozoea	
Subclass 1. Gregarinia	
Order 2. Eugregarinida	
Suborder 3. Septatina	*Porospora, Nematopsis*
Subclass 2. Coccidia	
Order 3. Eucoccidia	
Suborder 1. Adeleina	*Klossia*
Suborder 2. Eimeriina	*Pseudoklossia, Merocystis*
PHYLUM IV. MICROSPORA (the 'Microsporidians')	
Class 2. Microsporea	
Order 1. Minisporida	*Steinhausia*
Order 2. Microsporida	*Microsporidium* (?)
Suborder 2. Apansporoblastina	*Nosema*
PHYLUM V. ASCETOSPORA (*Marteilia* and the 'Haplosporidians')	
Class 1. Stellatosporea	
Order 1. Occlusosporida	*Marteilia, Haplosporidium,*
Order 2. Balanosporida	*Urosporidium, Bonamia* (?)
PHYLUM VI. MYXOZOA All species parasitic in poikilothermic vertebrates. No known representatives in marine bivalves.	
PHYLUM VII. CILIOPHORA (the 'Ciliates') see Table 13-7 (p. 582)	

specific determination, since *H. inflata* has originally been reported by Dujardin (1841) as a brackish-water saprobiote. They described flagellates from *Crassostrea virginica, C. gigas* and *Ostrea lurida* from the U.S. Atlantic and Pacific coasts as *H. nelsoni* and believed them to be identical with those from *O. edulis* in Europe and *Saccostrea cucullata* in Australia. Living trophozoites (Fig. 13-17) are pyriform in shape, 14 to 17 µm long and 7 to 10 µm wide, and have 6 anterior and 2 posterior flagella.

Hexamita nelsoni was held responsible for mortalities of *Ostrea edulis* from 'pit disease' in Holland (Korringa, 1952a; Mackin and co-authors, 1952), and was also blamed

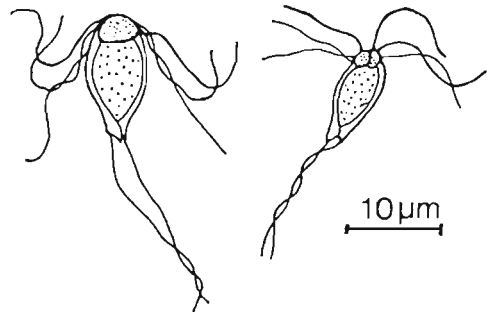


Fig. 13-17: *Hexamita nelsoni.* Trophozoites from intestine of *Crassostrea* spp. and *Ostrea* spp. (After Schlicht and Mackin, 1968.)

for mortalities of *Crassostrea virginica* and *O. lurida* in the United States (Laird, 1961; Stein and co-authors, 1961), although conclusive experimental evidence of the organism's pathogenicity was not presented. *H. nelsoni* is usually found as a free-living saprobic species in the proximity of oyster beds, affecting oysters as a facultative parasite apparently only under unfavourable environmental conditions, or as a secondary invader of individuals dying from other diseases.

Although 'intracellular stages' of the flagellate, resembling phagocytized oyster haemocytes in some respects, have been reported from the intestinal epithelium, Leydig tissue, gonad and other organs of oysters (Mackin and co-authors, 1952; Mackin, 1960), there is much doubt with respect to the identity of these structures. Thus, Sparks and Pauley (1964), in their studies on the normal postmortem changes in *Crassostrea gigas*, observed numerous peculiar multinucleate cells in the Leydig tissue of most of the dead oysters, first thought to be micro-organisms, but later identified as a multinucleate macrophage-type of cell. These 'giant cells', not previously recognized in oysters, developed usually at 72 h postmortem from the ingestion of numerous necrotic haemocytes and other cells, and probably represent a phenomenon characteristic of postmortem decomposition of the oyster. Perhaps, at least some of the structures believed by Mackin and co-authors (1952) to be intracellular stages of *Hexamita nelsoni* are identical with such macrophages.

Scheltema (1962) experimentally challenged individuals of *Crassostrea virginica* with *Hexamita nelsoni* trophozoites. After 33 days, there was a very dense population of these flagellates in the aquarium water. Of the 30 oysters employed in the experiment, only one died, and among the survivors, only 38 % proved to be infested. Furthermore, infestations were not heavy, in spite of the 4.5-week exposure to extremely high concentrations of *H. nelsoni* (ca. 10^4 organisms ml^{-1}). The author concluded that the relationship between *H.*

nelsoni and its pelecypod 'host' may lie somewhere between commensalism and parasitism, the respective condition depending largely on environmental factors, particularly temperature, and the physiological condition of the oyster. The flagellate occurred in high percentages of Delaware-Bay oysters only during the winter and early spring. Maximum infestations (54.5 %) occurred during March 1960 and coincided with minimum water temperatures. Salinity seemed unimportant in determining the occurrence of *H. nelsoni*. The reproductive rate of trophozoites cultivated in artificial media showed a clear temperature dependance, i.e., growth acceleration with increasing temperature up to approximately 20 °C. At 22 °C, peak concentrations of 56.4×10^4 organisms ml^{-1} were reached after 3 days; at 5 °C, the reproductive rate was much lower, reaching 24.1×10^4 organisms ml^{-1} after 20 days. Temperatures above 25 °C were lethal. Cultures maintained at room temperature, however, rapidly declined, and within 1 week, *H. nelsoni* could no longer be found (Scheltema, 1962). Axenic cultures of the organism, incubated at 15 to 18 °C, yielded concentrations of 10^6 trophozoites ml^{-1} after 5 days. Stock cultures have been maintained for 2 years (Khouw and co-authors, 1968).

Observations made in the course of experimental 'hexamitiasis' in *Crassostrea virginica* led Feng and Stauber (1968) to the conclusion that the invasive power of the flagellate has not yet been demonstrated conclusively, and that the role of bacteria usually associated with hexamitiasis has not been elucidated. In all, the role of *Hexamita nelsoni* as a pathogenic organism appears highly questionable (Shuster and Hillman, 1963).

Several 'amoeboid organisms' have been reported from marine bivalves. Most of these appear to be members of the Labyrinthomorpha or Apicomplexa rather than of the Rhizopoda (Table 13-3). As an example, *'Dermocystidium marinum' ('Labyrinthomyxa marina')*, for considerable time grouped with the Labyrinthulales ('Fungi'), is now recognized as an apicomplexan.

Two 'amoebae', *Vahlkampfia calkensi* and *V. patuxent*, have been isolated from the digestive tract of *Crassostrea virginica*. They could not be found in tissues of the oysters and were regarded as purely commensal, feeding on the bacterial flora in their hosts' digestive tract. The amoebae closely resembled oyster blood cells but could be distinguished, in stained material, by the morphology of the nucleus. Hogue (1915, 1921) was able to cultivate both species on agar plates. Their life cycles were not studied in detail, but cyst formation has been observed in some instances. Infestation was presumed to occur by ingestion of cysts or excysted trophozoites.

Vahlkampfia calkensi, measuring 10 to 30 μm in greatest diameter, occurred in most of the oysters from the New York City area, while oysters from Cape Cod, Massachusetts (USA), were found to be particularly free from this amoeba. The intensity of infestation was quite variable. *V. patuxent*, which may be more than 200 μm in length when extended, has been found in the stomach and intestine of *Crassostrea virginica* from Patuxent River, Maryland. It resembles *V. calkensi* in almost every respect, but was said to have strinkingly different cysts. While those of *V. patuxent* are spherical and vary greatly in size, *V. calkensi* cysts are rather uniform in size, dorsoventrally flattened and irregular in outline (Hogue, 1915, 1921).

Bovee (1965) transferred the 2 amoebae in *Crassostrea virginica* to the emended genus *Flabellula* (Table 13-3). Page (1971) isolated a free-living amoeba from a marine habitat, made a critical study, and identified it as *Flabellula calkensi*. He stated that the cysts Hogue (1915) figured for *F. calkensi* are quite similar to those of an *Acanthamoeba*.

Such cysts may frequently be encountered in biological material and result from air- or water-borne contamination (Sawyer and Buchanan, 1971). As stated by Page (1971), the promitotic configurations reported by Hogue (1921) for *F. patuxent* may have been derived from a vahlkampfiid contaminant present in her cultures, which could also have formed the cysts.

Vahlkampfiid amoebae are commonly grouped with the Schizopyrenida in subclass Gymnamoebia, class Lobosea, superclass Rhizopoda (Table 13-3). This placement is incorrect. Mackin and Schlicht (1976) discovered a proteomyxan amoeba stage in the development of *Vahlkampfia patuxent*. Hence, this 'rhizopod' is related to the Labyrinthomorpha rather than to the Rhizopoda (see section 'Agents: Labyrinthomorpha').

Studies on *Crassostrea virginica* from Chesapeake Bay, Maryland, revealed 4 different 'amoeboid organisms', of which 2 show affinities to the Amoebidae and 2 to the Labyrinthulidae and Vampyrellidae. Microscopic studies of the trophozoites, cysts, flagellate swarm cells and zoospores suggested that all amoeboid organisms reported previously from *C. virginica* are of uncertain taxonomic status (Sawyer, 1966). An unidentified amoeboid organism was also seen in oysters from Connecticut. The parasite occurred in a round form, up to 10.5 µm in diameter, and in an elongate, apparently motile form. The former was seen in the gut epithelium, while the latter often occurred extracellularly. The gut epithelium of infested oysters had a vacuolated and disorganized appearance. In one heavily infested host, the parasites were also found in the digestive diverticula (Newman, 1971).

'Rhizopods' *Thanatostrea polymorpha,* reported by Arvy and Franc (1968) and Franc and Arvy (1969) from the gill epithelium of *Crassostrea angulata* in France, as well as a similar (or identical) amoeboid organism observed by Gutiérrez (1977a) in the same host species in Spain, are probably referable to either the Labyrinthomorpha or to the Perkinsea (see section 'Agents: Labyrinthomorpha').

In addition to 'gill amoebas', individuals of *Crassostrea angulata* from the Cádiz region (Spain) had unidentified 'amoeboid cysts' in their digestive tract and diverticula. Up to 66 % of the oysters were found to harbour these cysts, which ranged from 6 to 15 µm in diameter. Heavily infested hosts exhibited progressive emaciation, and associated mortalities ranged from 40 to 90 % (Gutiérrez and Pascual, 1976; Gutiérrez, 1977a). The amoeboid organism was believed to resemble the agent described by Newman (1971) from *C. virginica* (p. 548).

Unless restudied by means of modern methods, including *in vitro* cultivation and electron microscopy, none of the above 'rhizopods' or 'amoeboid organisms' can be assigned with certainty to any known group of organisms. Probably, most of these are referable to the Labyrinthomorpha.

Hartmannella tahitiensis, usually known as a soil amoeba, has been found associated with mass mortalities of *Saccostrea cucullata* in Tahiti (French Polynesia). Although trophozoites have been observed intermingled with the epithelial lining of the alimentary tract, Leydig tissue, gonadal trabeculae, and ctenidia, the amoebae were not believed to be the cause of the mass mortalities. Rather, *H. tahitiensis* appeared to be a secondary invader of oysters which had been rendered moribund by water pollution (Cheng, 1970).

Cysts of *Acanthamoeba* sp. have been found on tissue sections of *Crassostrea virginica*. Their presence, however, resulted from external contamination (Sawyer and Buchanan, 1971). Amoebae of the *Hartmannella-Acanthamoeba* group are potentially

pathogenic to man (Culbertson, 1971). The possible role of marine or estuarine bivalves in the transmission of such pathogens to humans would merit further study (Cheng, 1973b).

Agents: Labyrinthomorpha (the 'Fungus-like Protistans')

The fungus-like Labyrinthomorpha encompass species which are highly destructive to marine bivalves. Many of these pathogens have not yet been identified or described adequately. The taxonomic position of the labyrinthulids has been the subject of considerable uncertainty. For long time they have been grouped with the Fungi as 'Labyrinthulales', although affinities of the labyrinthulids with protozoans and even algae have been noted (Johnson and Sparrow, 1961; Perkins, 1974a; Moss, 1977). Levine and Corliss (1963) granted the 'Labyrinthulia' subclass rank in the Rhizopodea (Honigberg and co-authors, 1964). In the most recently revised classification of the Protozoa, the Labyrinthomorpha have been raised to phylum rank (Table 13-3).

Members of the genera *Labyrinthula, Labyrinthuloides* and *Labyrinthomyxa* are mostly saprobic or parasitic on marine algae (Duboscq, 1921; Perkins, 1974a, b; Quick, 1974a, b). *Labyrinthula thaisi* has been isolated from the gills of oyster drills *Thais haemastoma floridana* (Cox and Mackin, 1974). Several true or presumptive labyrinthulids have been reported from marine bivalves. The exact systematic position of most of these, particularly of the various *'Labyrinthomyxa'* spp., is at present problematic.

The genus *Labyrinthomyxa* has been erected by Duboscq (1921) to include *L. sauvageaui*, a parasite of *Laminaria lejolisi*. Mackin and Ray (1966) erroneously assigned to the same genus a protistan parasite of *Crassostrea virginica*, previously (mis-)named *Dermocystidium marinum* (Mackin and co-authors, 1950). Electron microscope studies (Perkins, 1974a, 1976a, b) have shown that *'Labyrinthomyxa marina'* from oysters lacks any labyrinthulid characteristics and is, in fact, an apicomplexan. It has, therefore, been removed from genus *Labyrinthomyxa* and included by Levine (1978) in the newly established apicomplexan genus *Perkinsus* (see section 'Agents: Apicomplexa'). In addition to the oyster pathogen *'L. marina'*, several *Labyrinthomyxa* spp. or *'Labyrinthomyxa*-like organisms' have been reported from marine bivalves. Whether these are true labyrinthulids of the genus *Labyrinthomyxa sensu* Duboscq, 1921, or apicomplexans of the genus *Labyrinthomyxa sensu* Mackin and Ray, 1966 (= *Perkinsus* Levine, 1978), remains to be demonstrated by future (electron microscope) studies. In this review, all these organisms are provisionally retained in the Labyrinthomorpha.

A major distinguishing feature appears to be that the oyster pathogen *'Labyrinthomyxa marina'* (= *Perkinsus marinus*) is easily cultured in Ray's (1952) fluid thioglycollate medium and stains deeply blue-black with Lugol's iodine solution, whereas members of the Labyrinthomorpha, as well as numerous other organisms, especially fungi and protozoans, do not or only incompletely respond to this culture medium and staining technique. While Labyrinthomorpha can be cultivated on nutrient media, *P. marinus* can not (Quick, 1972a; Cox and Mackin, 1974). Whether Ray's thioglycollate assay method will consistently permit to distinguish Perkinsida from Labyrinthulida, remains to be studied. At least most of the *'Labyrinthomyxa*-like organisms' isolated from marine bivalves can not be assayed on thioglycollate medium.

Members of the Labyrinthomorpha are among the most destructive pathogens of marine bivalves. One oyster disease with a history of long and frustrating scientific study

was first observed in the Gulf of Saint Lawrence (Canada). In 1915, large-scale mortalities of *Crassostrea virginica* in Prince Edward Island waters followed an importation of apparently healthy seed oysters from New England in 1914. The losses were attributed to a contagious disease of unknown etiology, which soon became known as 'Malpeque Bay disease'. Oyster mortalities in epizootic areas were high and persistent. Less than 2 % of the stock survived after the 6-year duration of the initial epizootic. From 1915 to 1933, the disease spread around Prince Edward Island, destroying most of the oyster stocks, some of which required 20 years to return to previous levels of abundance. Survivors apparently developed resistance to the unknown causative agent (Needler, 1931).

In 1936 there was another outbreak of Malpeque Bay disease in the Prince Edward Island area, again accompanied by extreme losses (Fig. 13-18). Mortalities spread to the

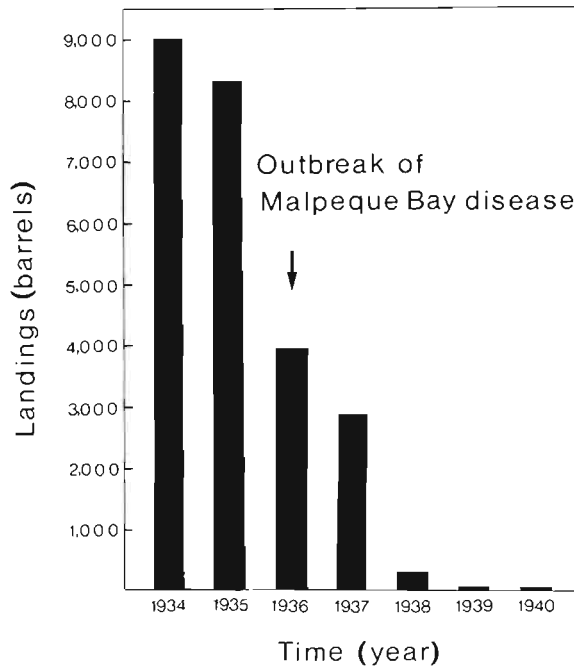


Fig. 13-18: *Crassostrea virginica*. Effect of 'Malpeque Bay disease' on annual oyster landings in Queens County, Prince Edward Island, Canada. (After Needler and Logie, 1947.)

adjacent mainland of New Brunswick and to Nova Scotia across Northumberland Strait in 1954. Oyster populations along the entire coast were severely decimated. Eventually, mass transfer of disease-resistant seed from Prince Edward Island waters, beginning in 1957, promoted gradual recovery of the fishery (Needler, 1941; Needler and Logie, 1947; Logie, 1956; Logie and co-authors, 1960; Drinnan and England, 1965; Quayle, 1969). However, recovery of the oyster stocks in affected areas was never complete. At present, Bras d'Or Lake, Cape Breton Island, is the sole remaining disease-free area of any commercial importance (Li and co-authors, 1975).

Oysters suffering from Malpeque Bay disease exhibit symptoms of weakness, extreme weight loss, stunted growth, and failure to spawn. A secondary symptom is the occasional

appearance of yellow-green pustules in the mantle and digestive diverticula in the early stages of an epizootic. Histopathological studies reveal haemocytic infiltration, an increase in 'brown cells' (p. 535), heavy deposits of collagen and the development of deep crypts in the lesions. Just before death, affected individuals may be emaciated, or may be quite fat. All ages are susceptible, and death may occur in late winter, early spring or late summer. Disease-free Cape Breton oysters, transferred to the epizootic area of Malpeque Bay, showed a consistently low condition index and often suffered a mortality of over 90 % in 2 years. Losses among younger transferred oysters were significantly lower (Needler and Logie, 1947; Li and co-authors, 1975; Sindermann, 1977).

Malpeque Bay disease is highly contagious, but its causative agent remained unknown for many years. Needler (1941) found an organism which "may be a mycetozoan" in sick oysters. He probably saw the true disease agent, but studies on its nature were apparently discontinued. Li and co-authors (1966) obtained evidence for the presence of 1 to 4 'spherical bodies' in haemocytes of affected oysters, but their precise nature has not been determined. In addition, unidentified bacteria have been isolated from diseased individuals. Pathogenicity of these organisms has not been demonstrated. Repeatedly, viral etiology has been suspected but never been ascertained (Sindermann, 1977). Eventually, Li (1976) identified a species of *Labyrinthula* as the possible causative agent of Malpeque Bay disease. The organism grew well in liquid Vishniac medium containing 2 to 3 % NaCl at 20 °C. This medium had been adopted by Booth and Miller (1968) for the cultivation of 'aquatic phycomycetes' of the genus *Thraustochytrium* (which, however, actually are Labyrinthomorpha, not Fungi!). Growth of the Malpeque Bay disease agent dropped dramatically in medium containing only 1.5 % NaCl. Oyster haemolymph (20 %) as a supplement to the medium (2.5 % NaCl) appeared to enhance growth. Electron microscopy of the organisms cultivated *in vitro* revealed cells differentiating into presporangia and multiple-cell sporangia, which produce infestive bodies (zoospores?). These, in turn, can differentiate into uninucleate cells or into a plasmodial stage. Identical stages were frequently observed in tissues of diseased oysters (Li, 1976; Li and Clyburne, 1979).

The Malpeque Bay disease agent has been believed to be host–specific for *Crassostrea virginica* in the Gulf of Saint Lawrence, but subsequently a very similar organism has been isolated from *Mytilus edulis* at Prince Edward Island.

During January 1977, many mussels on the Ascumpeque Bay side of River Bridge, Prince Edward Island, were found to be dead or weakened (gaping). Mortalities occurred in an area which had been a major mussel production centre for decades. Spheres, similar to those of *Labyrinthomyxa* spp., were detected in all the specimens incubated in thioglycollate–dextrose medium for 2 weeks at 25 °C, but they stained only poorly (very light brown) with dilated Lugol's iodine solution, suggesting that they are not *Perkinsus* sp. Histological examination showed most of the diseased mussels to be heavily infested with cysts or sporangia of the same microparasite. Rupture of the sporangia liberated 'infestive bodies' (zoospores?), approximately 1 μm in size and consistently showing filamentous structures. Infestive bodies were found to differentiate into presporangia (Li and Clyburne, 1979). The mussel pathogen was tentatively identified as *Labyrinthomyxa* sp.

Two specifically unidentified presumptive *Labyrinthula* spp., parasitizing *Crassostrea virginica* in Gulf of Mexico waters, were held responsible for large-scale oyster mortalities in Texas, Louisiana and Mississippi and possibly also in Alabama and Florida, during most if not all summers. The disease syndrome observed was similar to that caused by *Perkinsus*

marinus, but could not be diagnosed by Ray's (1952) thioglycollate-culture technique. Instead, the organisms grew on beef serum. The pathogens stripped off the oysters' gill and mantle epithelium. When entering the tissues or haemolymph, the agents caused general lytic attack and, eventually, death of the host. In contrast to *P. marinus*, the pathogens are acclimated to low salinities, but apparently are active only above 18 °C (Mackin, 1970).

A thraustochytridaceous agent (i.e., a member of the Labyrinthulida), almost indistinguishable from *Perkinsus marinus* in oyster tissues, has been isolated in agar culture from individuals of *Crassostrea virginica* in Apalachicola Bay, Florida. The organism did not respond to the thioglycollate assay, and cultivation in sea water demonstrated that it is not congeneric with *P. marinus*. Quick (1972b), who named it *Thraustochytrium inglei*, believed it to be responsible for early summer mortalities of oysters. Another organism, isolated from *Brachidontes recurvus* (= *Ischadium recurvum*), responded poorly to Lugol's iodine staining used in the fluid thioglycollate technique (Quick, 1972a). It may also be a labyrinthulid.

Labyrinthuloides saliens, normally found on sea grass *Halophila engelmanni* (Quick, 1974b), has also been isolated from *Crassostrea virginica* (Quick, 1974a). Medium to heavy *Labyrinthomyxa*-like infestations were observed in histological preparations of *C. virginica* tissues when thioglycollate assays of the same oysters had shown negative or light infestations. In almost every case, close examination revealed certain morphological differences from *Perkinsus marinus* (Quick, 1971; Quick and Mackin, 1971). The behaviour of these organisms in culture suggests that they may be labyrinthulids.

'Amber disease' represents a condition diagnosed in *Crassostrea virginica* from Bayou Rigaud, Louisiana. Gaping oysters, collected during a small mortality, were found to have a light amber colour. Microscopic examination revealed the presence, in the tissues, of various stages of a hitherto unknown parasite. The most numerous stages were more or less spindle-shaped amoeboid cells, which commonly had 1 to several filopodia or thread-like extensions of the cytoplasm (Fig. 13-19, 1, 2). The amoebae occurred mostly in the

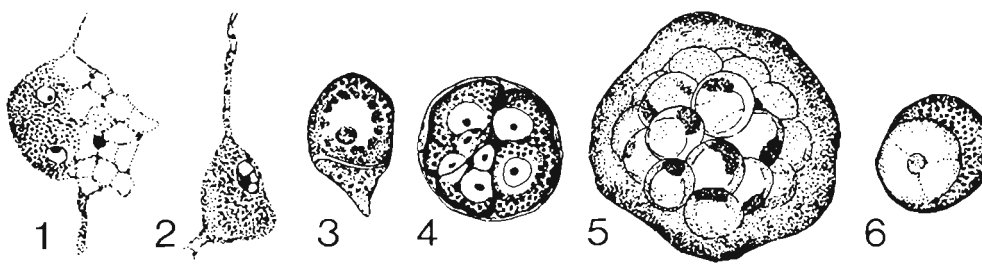


Fig. 13-19: 'Amber disease' organism from *Crassostrea virginica*. 1, 2: Uninucleate amoeboid cells; 3: small amoeba with endogenous bud; 4: advanced proteomyxan amoeba; 5: large myxamoeba; 6: cyst (?). Note similarities with *Vahlkampfia patuxent* (Fig. 13-20). (After Mackin, 1962.)

Leydig tissue, but may penetrate any tissue, and tended to congregate around the basement membranes of the gut epithelium and between the digestive-gland tubules. Often they were found to line the interior of blood vessels. Stages resembling presporangia and sporangia were less common.

Histopathology associated with Amber disease is intense. The Leydig-cell system is largely destroyed, and digestive-gland tubules lose their normal pigmentation, shrink and

become 'knobby-looking' in cross section. Infiltration of the various epithelia with parasites and haemocytes is common. Gonad atrophy is apparent but may be due, at least in part, to post-spawning conditions (Mackin, 1962). The author did not assign the agent to any group of organisms, but noted similarities with 'MSX' *(Haplosporidium nelsoni)*, as well as with sporangium formation in the Synchytriaceae. It appears, however, that the organism may be a labyrinthulid.

Vahlkampfia (Flabellula) patuxent has until recently been grouped with the Rhizopoda (see section 'Agents: Sarcomastigophora'). The discovery of a flagellate stage in the life cycle of a terrestrial vahlkampfiid (Darbyshire and co-authors, 1977; Page, 1977) casted doubt upon the correctness of this allocation. Mackin and Schlicht (1976) observed a proteomyxan amoeba stage in the development of *V. patuxent* which, under certain environmental conditions, may cause lethal epizootic disease in *Crassostrea virginica* populations. The authors believe (p. 16) "that all serious students of molluscan pathology have observed these protistans, but have failed to recognize their significance". Earliest uninucleate amoebae, as removed from the haemolymph of *C. virginica* in advanced disease, are rounded to oval, about 6 to 12 μm in length, and may have 1 or 2 polar pseudopods (Fig. 13-20, a). Growth and further development of these cells results in (i) production of endogenous buds (Fig. 13-20, b) ranging from 1 to many, depending on the size of the amoebae; (ii) growth to giant amoebae, which may be more than 200 μm in length when extended; and (iii) production of multibranched and anastomosing pseudopodia originating from flattended ectoplasmic lamellae. Endogenous buds may produce secondary gemmae, and rarely even more. Early buds are rounded uninucleate cells, which resemble (and may be mistaken for) oyster haemocytes in stained sections. The buds can reproduce by binary fission to produce massive concentrations in host tissues and haemolymph. Advanced endogenous buds may form small plasmodia, presporangia and sporangia, often in the same amoeba (Fig. 13-20, c).

Mackin and Schlicht (1976) state that the development of the organism studied by them closely resembles that of *'Labyrinthomyxa marina'* (= *Perkinsus marinus*, an apicomplexan, however), and consequently (although incorrectly) changed its name to *L. patuxent*. The various characteristics of the stages of *Vahlkampfia patuxent* in *Crassostrea virginica*, as described by these authors, are congruent only with those of a single group of protistans — the Labyrinthulida. *V. patuxent* also differs from *P. marinus* in its incomplete response to Ray's (1952, 1954a) thioglycollate diagnostic culture method.

There are striking similarities between the 'Amber disease' organism, as described by Mackin (1962), and the proteomyxan amoeba stages of *Vahlkampfia patuxent*, as reported by Mackin and Schlicht (1976) (Figs 13-19 and 13-20). Although, in their 1976 paper, the latter authors make no reference to Mackin's (1962) description of the 'Amber disease' organism, the reviewer feels inclined to regard both agents as identical and probably referable to the Labyrinthomorpha.

'Labyrinthomyxa-like' organisms have also been reported from oysters in Europe. *Thanatostrea polymorpha*, a 'rhizopod' resembling *L. sauvageaui*, was held responsible for causing 'Maladie des branchies' in *Crassostrea angulata* in France (Arvy and Franc, 1968; Franc and Arvy, 1969, 1970; see section 'Agents: Virales'). Comps (1969), who studied the parasite in detail, noted its striking similarity with Mackin's (1962) 'Amber disease' organism. No attempts have been made to cultivate the protistan, but Arvy and Franc (1968) state that it does not stain with iodine as does *Perkinsus marinus*. There can be little

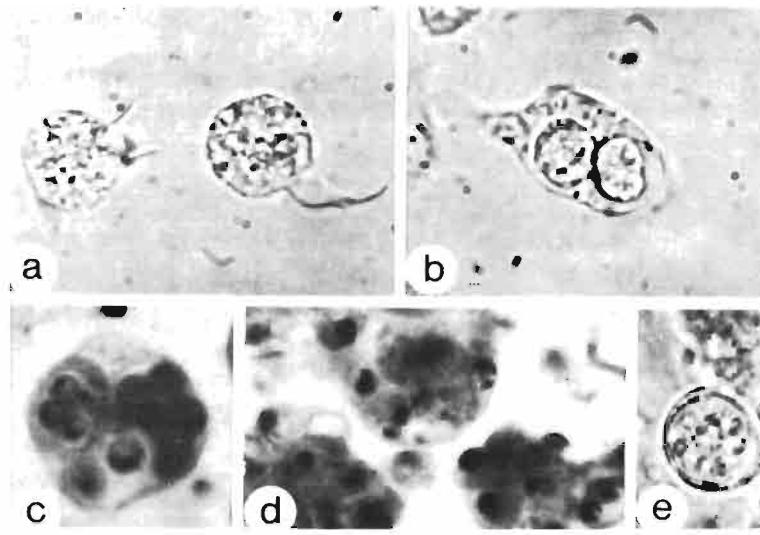


Fig. 13-20: *Vahlkampfia patuxent ('Labyrinthomyxa patuxent')*. Stages in *Crassostrea virginica*. (a) Uninucleate amoeboid cells with polar pseudopods; (b) small proteomyxan amoeba with early development of 2 endogenous buds; (c) proteomyxan amoeba in late disease stage. Rounded vegetative cells now replaced as endogenous buds by presporangia (left), small sporangia with tightly appressed nuclei (right), and only 1 vegetative cell; (d) 3 large myxamoebae with vegetative cells, most of which are ready to leave mother cell; (e) living cyst attached to gill tissue. All × 1,100. (After Mackin and Schlicht, 1976.)

doubt that this agent is a labyrinthulid closely related to or identical with both *Vahlkampfia patuxent* and the 'Amber disease' organism from *C. virginica*. Alderman and Gras (1969) isolated a *Labyrinthomyxa*-like protistan from gill-diseased *C. angulata*, imported into Britain from the River Tagus (Portugal). It could be cultivated in liquid yeast-peptone medium. The same authors reported on finding of a *Labyrinthomyxa*-like agent in a single individual of *Ostrea edulis* at Cancale (France). Gras (1969) was able to grow a similar organism from oysters in France in Ray's fluid thioglycollate medium but not an Sabouraud peptone-agar media. This entity may be a true *Perkinsus* species, not a labyrinthulid. Unfortunately, it has not been stated in Gras' report whether the organism has been isolated from *C. angulata* or *O. edulis*.

The role of these organism(s) in the etiology of 'Maladie des branchies' is not clear. Gill disease in *Crassostrea angulata* is generally believed to be a virosis, caused by an iridovirus (Comps and Duthoit, 1976). However, Gutiérrez (1977a), who refound in *C. angulata* from Cádiz (Spain) what appears to be Arvy and Franc's (1968) *Thanatostrea polymorpha*, observed the presence of the large (25 to 80 μm) uninucleate amoebae in gill and mantle epithelia to be associated with massive histopathological alterations and haemocytic infiltration.

Intracellular parasitic stages, believed to be similar to *'Labyrinthomyxa marina'*, have been observed in cultures of cardiac tissue of *Crassostrea gigas* from the French Atlantic and Mediterranean coasts. Other forms, occurring free in the culture medium, resembled sporangia. The parasite appeared frequently in tissue cultures obtained from apparently healthy oysters. Affected cells showed progressive vacuolization and lysed within 8 to 15 days (Cousserans and co-authors, 1974).

Moribund individuals of *Donax gouldi*, examined during recurrent bean-clam mass mortalities occurring on the coast of southern California, contained vast numbers of unicellular organisms, 2 to 6 μm in diameter. The organisms were found in enlarged blood vessels, connective tissues, digestive diverticula, gut epithelium and within host-haemocytes. The affected clams, both young and old, had unusually soft tissues and a slightly greenish tinge. Although the brief description given by Coe (1955) is remotely reminiscent of unicellular algae (*Coccomyxa*? See section 'Agents: Algae'), the author felt that the cells, which were irregularly spherical or ovoidal in shape, were similar to *'Dermocystidium marinum'*.

One can speculate about the incrimination of labyrinthulids in other molluscan diseases and associated (mass) mortalities. In most cases, however, histologic evidence for the presence of such agents is weak, although the reported pathology is highly suggestive of labyrinthulid infestation. One such example is 'Winter disease' of *Saccostrea cucullata (Crassostrea commercialis*))* in New South Wales (Australia), as described in detail by Roughley (1926). Gross signs of the disease include yellowish spots on the body surface, formation of abscesses in the stomach wall, ulcerations on the epithelium of the gills, palps, mantle and gonads and massive haemocytic infiltration. The causative agent has not been determined. At one time, Mackin (1961) thought that *Hexamita* was involved in some important way, but later (Mackin, 1969) believed that 'Winter disease' of *S. cucullata* may be caused by *Labyrinthomyxa*.

'Winter disease' is continuing to be the main problem in the New South Wales oyster industry, killing up to 70 % of the oyster stocks. Geographically, it is confined to the middle and southern part of the New South Wales coast, and takes epizootic proportions in autumns and winters with low rainfall. After a wet autumn and early winter, disease prevalence is either very low or absent. Mortalities involve chiefly mature oysters during their third winter, just before marketing. Wolf (1979) noted similarities between Australian oyster-winter disease and Canadian 'Malpeque Bay disease', but was unable to identify the causative agent. Histological sections of diseased oysters did not reveal any bacteria or fungi or other microparasites. Wolf believed that a virus may be involved. The fact that wild, uncultivated stocks do not suffer from epizootic winter mortality suggests that physiological stress, resulting from cultivation techniques, may also play a certain part. Roughley (1926) speculated on the involvement, in the disease, of bacteria, but eventually concluded that the mortalities are directly related to low environmental temperatures.

Devastating mortalities of *Ostrea edulis* occurred early in this century in various parts of Europe. Deaths began in 1919 in Mar Piccolo near Taranto, Italy (Cerruti, 1941a), and soon after were reported in England, The Netherlands, Germany and other European countries, where they persisted until 1923. In spite of intensive studies, which have been summarized by Orton (1923, 1924) and Korringa (1952a), the causative agent(s) have never been discovered although, at times, flagellates *Hexamita* and copepods *Mytilicola* have (unjustifiedly) been incriminated. Physiological stress, mainly poor food supply and low water temperatures, were also claimed to be the cause of these catastrophic mortalities, which depleted large areas of cultivated oyster beds, as well as natural populations

*) Stenzel (1971) pointed out that the Sydney rock oyster is not a member of the genus *Crassostrea* and suggested to change its name into *Saccostrea cucullata*. Although justified, this change has, for the most part, not yet found its way into the scientific literature.

(Gaarder and Alvsaker, 1941; Spärck, 1950). Since oysters in the various countries were affected almost simultaneously, i.e., under a large variety of environmental conditions, such an explanation is untenable.

Orton (1924) observed "tiny spindle-shaped bodies somewhat resembling the spores of sporozoan parasites" in the tissues of diseased oysters. These bodies, which were met with from the beginning of the disease studies, were interpreted as "the products of disintegration of muscular tissue of some kind in some part of the body of the oyster not yet identified". Orton also saw massive diapedesis of haemocytes in affected hosts. In the reviewer's opinion, there can be little doubt that Orton saw stages in the life cycle of a lethal labyrinthulid parasite.

Agents: Apicomplexa (*'Dermocystidium'*, the Gregarines and the Coccidians)

The phylum Apicomplexa, established by Levine (1970), contains serious molluscan pathogens — such as the causative agent of '*Dermocystidium* disease', previously believed to be a mycotic infection of oysters —, as well as relatively harmless parasites, such as gregarines, and moderately pathogenic forms, such as coccidians. As a result of repeated revisions (Sprague, 1966, 1977, 1979; Levine, 1970, 1978; Levine and co-authors, 1980), members of the old 'Sporozoa' have now been assigned to 4 phyla — the Apicomplexa, Microspora, Myxozoa and Ascetospora (Table 13-3). The most recent addition to the Apicomplexa is the class Perkinsea, established by Levine (1978) to accomodate *Perkinsus marinus*.

This protistan parasite has a long and fascinating history, during which it has been pushed from one taxonomic group into another. Mackin and co-authors (1950) described *Dermocystidium marinum* as a protistan parasite causing recurrent heavy summer mortalities in *Crassostrea virginica* from the Gulf of Mexico. Mackin (1951) and Ray (1952) felt that the cells of *Dermocystidium* are morphologically somewhat like those of the human blastomycosis-producing fungi, such as *Blastomyces* and *Cryptococcus*. In contrast, Sprague (1954a) listed the organism as a haplosporidian. Mackin and Boswell (1956) returned it to the Fungi and related it to the Synchytriaceae. Scheer (1957) appears to be the first author noticing that *D. marinum* is strongly different from fish-invading *Dermocystidium* spp. and should be removed from that genus. On the basis of *in vitro* studies, Mackin and Ray (1966) believed it to be a 'fungus' of the Labyrinthulales, and consequently changed its name to *Labyrinthomyxa marina*. Perkins and Menzel (1967) made a detailed electron microscope study and suggested that *L. marina* is allied to the Phycomycetes, order Saprolegniales. Following the classification scheme of Honigberg and co-authors (1964), Cheng (1973a) listed the organism in the protozoan subphylum Sarcomastigophora, superclass Sarcodina, class Rhizopodea, subclass Labyrinthulia, order Labyrinthulida. As a result of continued studies, Perkins (1974a) considered the oyster pathogen a doubtful member of the genus *Dermocystidium* and an equally doubtful member of the genus *Labyrinthomyxa*. He concluded (p. 58) that "at present *Dermocystidium marinum* appears to have no close affinities". Perkins (1974a, 1976a, b) also provisionally retained the generic name *Dermocystidium* and stated that the designation *L. marina* should be rejected in the absence of any labyrinthulid characteristics, the assumption being made that *Labyrinthomyxa* spp. are labyrinthulids. Nevertheless, *D. marinum* continued to be

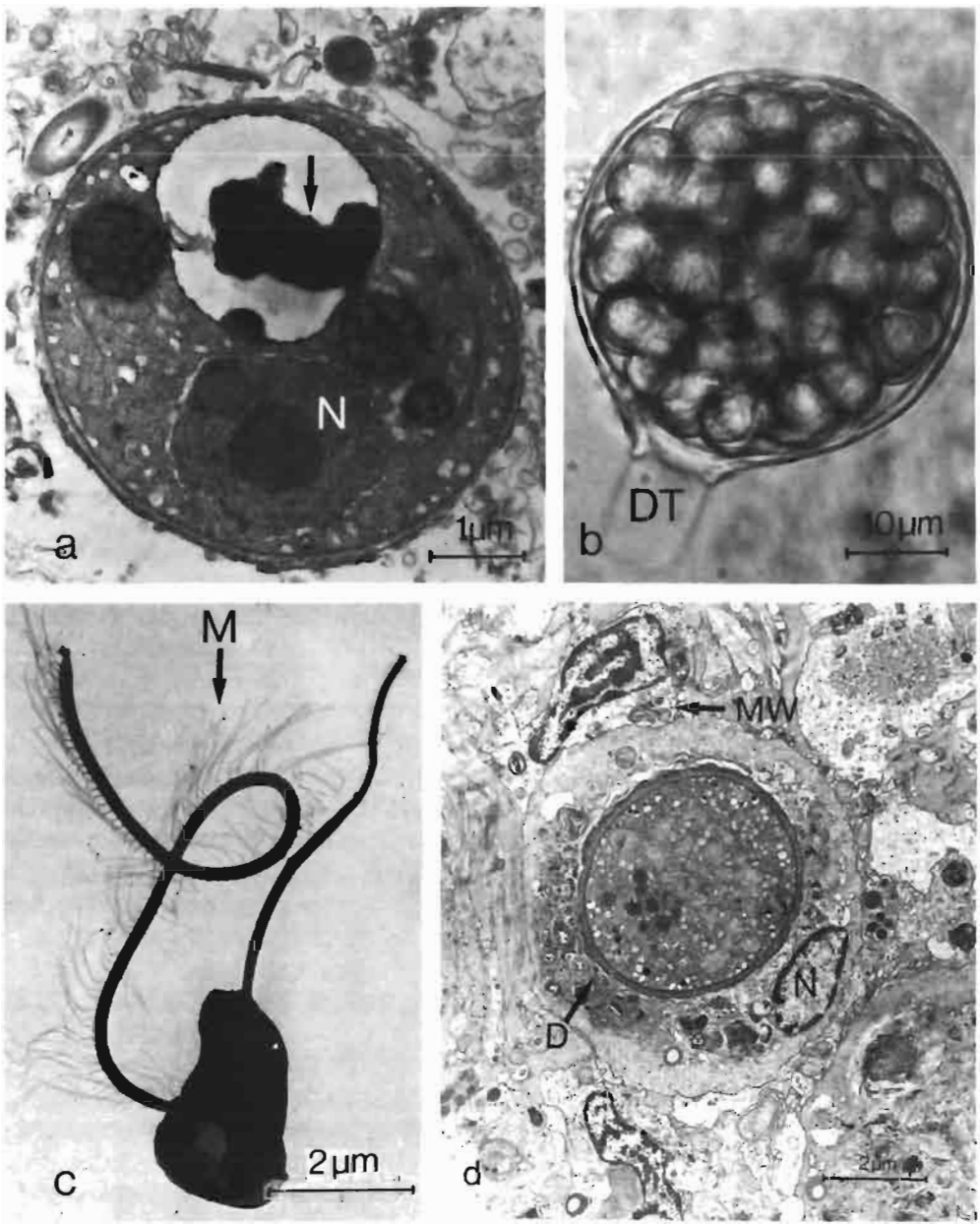


Fig. 13-21: *Perkinsus marinus*. (a) Developing aplanospore with eccentric vacuole with vacuoplast (arrow) and nucleus (N); (b) zoosporangium with successively bipartitioning protoplast leading toward zoospore formation, and discharge tube (DT); (c) zoospore with mastigonemes (M) along one side of anterior flagellum; (d) aplanospore engulfed by *Crassostrea virginica* haemocyte in mantle-connective tissue, causing degenerative changes in host cell. Haemocyte nucleus (N) pyknotic and cell debris (D) accumulating around parasite. Adjacent connective-tissue and muscle cells less severely affected, but myelin whorls (MW) present in cells nearest to haemocyte. (a, c, d after Perkins, 1976a; b after Perkins and Menzel, 1966.)

listed as a fungus, *L. marina*, in the literature (Little and Quick, 1976; Marteil, 1976; Quick, 1977).

In the course of another thorough electron microscope study, Perkins (1976b) found that the zoospores of *Dermocystidium marinum* have an 'apical complex', composed of organelles typically (and only) present in protozoans of the (at the time sub-)phylum Apicomplexa. He concluded that this discovery placed the organism in the latter taxon, rather than in the Fungi, as generally believed. As pointed out by Levine (1978), the sporangium of *D. marinum* is reminiscent of a gregarine gametocyst, its sporangium-discharge tube is reminiscent of a gregarine sporoduct, and its 'zoospores' resemble apicomplexan sporozoites. Other morphological, as well as life-cycle features (i.e., apparent absence of sexual stages), however, are sufficiently different to justify the establishment of a new class for this species and to separate it from other named apicomplexans. Therefore, Levine (1978) established the following new taxa in the protozoan phylum Apicomplexa in order to accomodate this species: Class Perkinsea, order Perkinsida, family Perkinsidae, genus *Perkinsus*. At present, *P. marinus* is the sole named species in the class Perkinsea (Table 13-3).

For long time, recurrent oyster mortalities, occurring in the Gulf of Mexico since the 1940's, had been blamed on oil pollution, until the oil industry contracted the Texas A. & M. Research Foundation to investigate the problem in the 1950's (Overstreet, 1978). This initiative led to the discovery of *Perkinsus marinus* by Mackin and co-authors (1950). The information, which has subsequently accumulated on the morphology, host-parasite relationships and epizootiology of *Perkinsus marinus* is considerable. The most pertinent papers will be discussed below. For details on the ultrastructure of the vegetative and sporulation stages of *P. marinus* see Perkins and Menzel (1966, 1967) and Perkins (1969b, 1976b). The ecology and epizootiology of the oyster pathogen have been studied most thoroughly by Ray (1954a), Andrews and Hewatt (1957), Mackin (1962) and Quick and Mackin (1971). The most recent summarizing accounts of *P. marinus* infestations in *Crassostrea virginica* are those of Perkins (1976a) and Andrews (1979).

In brief, the life cycle of *Perkinsus marinus* consists of vegetative reproduction in which uninucleate aplanospores enlarge and undergo successive bipartition (alternating karyokinesis and cytokinesis) to form 4- to 64-cell sporangia, from which are liberated uninucleate, coccoid or cuneiform aplanospores, which measure 2 to 4 μm in longest axis. As the aplanospores enlarge and mature, a large eccentric vacuole develops, which may occupy more than 50 % of the total cell volume. At this stage, the cells are about 5 to 10 μm in diameter. The vacuole membrane synthesizes a free-floating, refringent vacuoplast (Fig. 13-21). Alternating nuclear and cytoplasmic divisions then result in the formation of aplanospores which, upon rupture of the mother-cell wall, re-initiate the cycle.

In moribund oysters, as well as under culture conditions (see below), uninucleate prezoosporangia may rarely be formed by enlargement of aplanospores to about 15 to 20 μm (up to 100 μm in fluid thioglycollate medium). These large cells have a very large eccentric vacuole, which may comprise 90 % of the total cell volume. Upon liberation from oyster tissue or culture medium into sea water under aerobic conditions, the prezoosporangia develop into zoosporangia. Subsequent sporulation produces biflagellate 'zoospores' (sporozoites?), which are released through 1 or 2 discharge tubes in the sporangial wall (Fig. 13-21, b, c). The zoospores are capable of invading new hosts and

initiate infestations by establishing themselves or being established by host haemocytes in epithelial sites. It has been estimated that an 85-μm sporangium contains some 1,000 to 2,000 zoospores (Perkins and Menzel, 1966, 1967; Perkins, 1969b, 1976a, b). Other stages, which rarely occur in oysters, such as amoeboid cells, spindle cells, multinucleate plasmodia with long, rhizoid-like mucoid processes, have been described from observations of cultured cells. Their significance in the life cycle of *Perkinsus marinus* remains obscure (Mackin and Ray, 1966). Hoese (1964) detected *P. marinus* in the digestive tract and faeces of fishes, oyster drills and crabs that had fed on dying or dead infested oysters. He speculated that transmission of the pathogen might be furthered by scavangers.

Perkinsus marinus produces systemic infestations in *Crassostrea virginica*. Gross signs of 'Dermo disease' are severe emaciation, gaping and a pale appearance of the digestive gland. Considerable shrinkage of the soft tissues is common in heavy infestations. Maceration of the adductor muscle is sometimes apparent and pus-like pockets of haemocytes are also seen. In advanced stages, the shell-secreting function of the mantle is damaged and the mantle shrinks away from the outer edge of the shell, leading to cessation of shell growth (Mackin, 1951; Menzel and Hopkins, 1955). Any cell stage of *P. marinus* appears capable of initiating infestations, except possibly prezoosporangia. It appears that parasite cells contained intracellularly in diapedesed haemocytes remain infestive. Any tissue of the oyster is susceptible to invasion, although external epithelia and peripheral nerves are not usually penetrated. Initial host response to infestation is marked haemocytosis and migration of phagocytes to the sites of parasitic lodgement, resulting in extensive inflammation. Haemocytes invading the columnar epithelium may be numerous. Separation of the basement membrane from the underlying connective tissue often takes place, and the resulting cavity fills with haemocytes and *P. marinus* cells if the basement membrane has been penetrated by the latter.

Heavy accumulation of *Perkinsus marinus* cells ultimately results in liberation of the pathogens into the lumen of the digestive system. Large areas of the epithelium are sloughed off the basement membrane, and necrotic host tissue and parasite cells fill the lumen. The agent gains access to the connective tissue through lysis of the basement membrane. *P. marinus* cells may lie between or within host cells (Fig. 13-21, d). Multiplication results in clusters of aplanospores, which are engulfed and dispersed through the host tissues by haemocytes. Once in the connective-tissue mesenchyme, the organisms may be distributed by the haemolymph to all parts of the body. In advanced stages of the disease, numerous *P. marinus* cells and haemocytes may completely obliterate the haemolymph sinuses and obstruct all but the largest blood vessels. The connective tissue, particularly in the digestive-gland region, mantle and gills, is highly susceptible to invasion. Host response in the early stages often consists of an attempt at 'encapsulation' by several layers of haemocytes, but, as the organisms increase in number, the 'capsule' disappears. In advanced stages, infestation foci may attain several hundred micrometers in diameter and contain thousands of *P. marinus* cells mixed with host-cell debris.

Heavy connective-tissue infiltration may result in the conversion of the entire visceral mass into a large abscess. Histolysis ensues at the final stage of the disease. There is no indication that *Perkinsus marinus* produces exotoxins in any significant amount. Damage to the host is almost entirely due to lysis of tissues (Mackin, 1951, 1962; Perkins, 1976a).

Tissues of oysters infested with *Perkinsus marinus* exhibit a significant increase in number and size of 'brown cells' (Mackin, 1951, 1962; Stein and Mackin, 1955). The exact

function of these structures — which, in the literature, are variously referred to as 'pigment cells', 'ceroid bodies' or 'Keber's organ' — remains unknown, but appears to be related to the internal defense system or to the processing of biological fluids (Ruddell and Wellings, 1971). Cheng and Rifkin (1970) have exhaustively reviewed the literature on 'brown cells'. It is now well established that their number increases in response to various disease agents and parasites (Cheng and Burton, 1965a, 1966; Gutiérrez, 1977a). Mackin (1951, 1962) and Stein and Mackin (1955) attribute the increase in brown cells in *Crassostrea virginica* infested with *P. marinus* to a change in host-fat metabolism caused by the parasite.

The average meat weight of 198 heavily *Perkinsus marinus*-infested *Crassostrea virginica* was found to be about 33 % less than that of 227 healthy or lightly infested individuals (Ray and co-authors, 1953; Ray, 1954a). However, these figures may have been somewhat exaggerated by a concomitant loss in condition due to post-spawning conditions (Quick and Mackin, 1971).

Perkinsus marinus prevalences vary with the age of the oyster. Adult oysters are most susceptible to infestation immediately after spawning. Spat are refractory to 'Dermo disease' until they are 3 to 4 months old, and death rates are low during the first year of life. Both prevalence and mortality rates increase with host age and size. Thus it is important to plant the largest seed oysters available, and to harvest beds as soon as the oysters have reached market size (Ray, 1954a; Ray and Chandler, 1955; Andrews and Hewatt, 1957; Mackin, 1962; Sindermann, 1970c).

Laboratory experiments, as well as field studies, have shown that *Perkinsus marinus* is

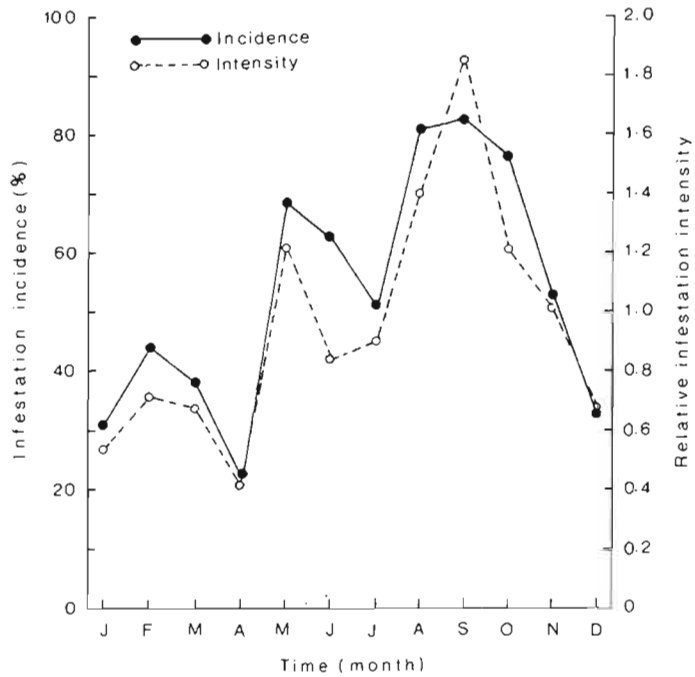


Fig. 13-22: *Crassostrea virginica*. Incidence and intensity of *Perkinsus marinus* infestation in relation to season. (After Quick and Mackin, 1971.)

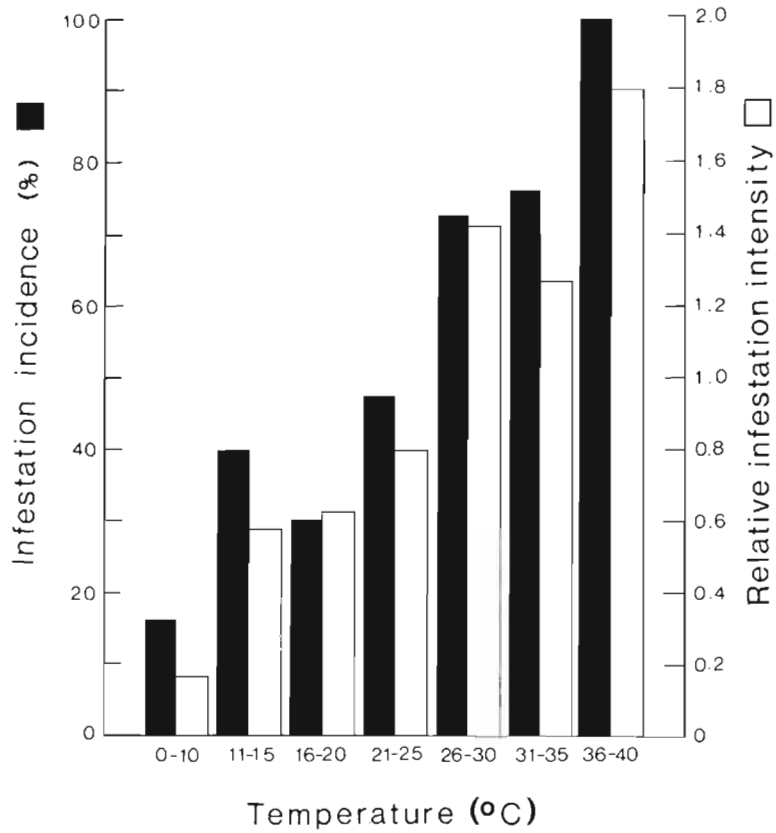


Fig. 13-23: *Crassostrea virginica*. *Perkinsus marinus* infestation in relation to temperature. (After Quick and Mackin, 1971.)

transmitted directly from oyster to oyster. Prevalence is markedly increased by crowding of hosts on densely seeded beds (Mackin, 1951, 1962; Andrews, 1965). Experimentally, *P. marinus* has been transmitted to healthy oysters by two methods. 'Proximity' experiments, in which healthy and infested oysters were placed in the same container, resulted in an infestation of 38 (95 %) and the death of 20 (50 %) of 40 test individuals, as opposed by a mortality of 3 (20 %) of 15 control oysters. In feeding experiments, in which finely minced infested oyster tissue was suspended in the enclosure water, 26 (87 %) of 30 previously healthy oysters became infested and 25 (83 %) of them died, while in the control aquarium 6 (40 %) of 15 *P. marinus*-free individuals died. The 'proximity' method required 16 weeks to produce the first acutely infested dead oysters, while 9 weeks were sufficient with the 'feeding' method (Ray, 1954b). 'Proximity' experiments conducted in the field indicate that most new infestations occur from dying infested oysters (Andrews, 1965, 1979).

Perkinsus marinus is easily cultured in fluid thioglycollate medium, fortified with penicillin and streptomycin or, preferably, mycostatin and chloramphenicol, to inhibit bacterial contamination. Subsequent iodine staining discloses the presence of aplanospores (Ray, 1952, 1966b, c; Quick, 1972a). The method has many advantages over histological examination, particularly in large amounts of material to be processed, and proved to be likewise useful for studies of the behaviour of the pathogen *in vitro* (Mackin, 1962; Mackin

and Ray, 1966). Since, at that time, *P. marinus* was considered a fungus, 12 antifungal antibiotics were screened to determine their effect on the agent. Two of these, cycloheximide and streptimidone, arrested its proliferation at concentrations of 0.25 to 1.0 μg ml^{-1} *in vitro*. Administration of 1 μg ml^{-1} cycloheximide per week was considered useful for inhibition of *P. marinus* in laboratory stocks of oysters (Ray, 1966c, d).

Ecological factors governing the distribution and virulence of *Perkinsus marinus* have been studied in great detail. Temperature appears to be the major factor checking epizootiology. The agent proliferates readily only at temperatures above 20 °C (Figs 13-22 and 13-23). Associated mortalities rise during the warm months with peaks in late summer and early fall and decline during colder periods, probably due to reduced parasite metabolism rather than to elimination of the pathogen, although occasional loss of infestation may occur, particularly at low temperatures. Both infestation incidence and intensity increase almost linearly with temperature throughout the thermal range sampled (Fig. 13-23). Due to decreased winter-water temperatures, the agent is dormant for almost half the year in Chesapeake Bay, while it is active for most of the year in the Gulf of Mexico (Ray, 1954a; Hewatt and Andrews, 1954, 1956; Ray and Chandler, 1955; Andrews and Hewatt, 1957; Mackin, 1962; Andrews, 1965, 1979; Quick and Mackin,

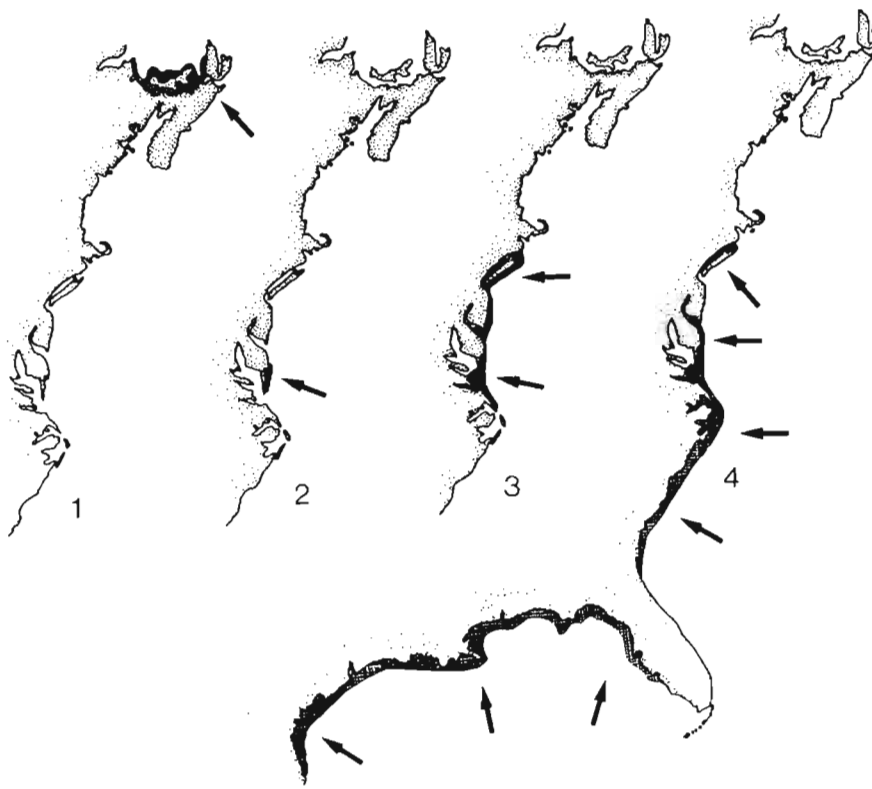


Fig. 13-24: Distribution of serious diseases of *Crassostrea virginica* on the Atlantic coast of North America. 1: 'Malpeque Bay disease', caused by *Labyrinthula* sp.; 2: 'Seaside disease', caused by *Haplosporidium costale;* 3: 'Delaware Bay disease', caused by *H. nelsoni;* 4: 'Dermocystidium disease', caused by *Perkinsus marinus*. (After Sindermann, 1968a.)

1971). The geographic range of *P. marinus* extends from the Gulf of Mexico to Connecticut (Fig. 13-24, 4). Failure to detect the pathogen consistently north of Chesapeake Bay suggests that prolonged low temperatures may be a significant limiting factor.

Perkinsus marinus requires salinities above 12 to 15 ‰ S for its proliferative development. Lower salinities alone inhibit activity but do not eradicate the parasite from oysters. Excessively high salinities may also be inhibitory. The absence of infestations from low-salinity waters may indicate a lack or scarcity of spores. It appears likely that, in estuaries, spores are dispersed and carried downstream by the addition of freshwater. The disease persists and intensifies in infested oysters transplanted to low–salinity areas (Ray and co-authors, 1953; Ray, 1954a, c; Ray and Chandler, 1955; Mackin, 1956, 1962; Andrews and Hewatt, 1957; Hoese, 1963; Quick and Mackin, 1971; Andrews, 1979).

Perkinsus marinus has been found responsible for recurrent devastating oyster mortalities, particularly in waters of the southern United States. Ray (1966a) reported the agent from 35 of 39 samples from various localities in the Gulf of Mexico, with prevalences as high as 100 %. In 1967, some 3,000 acres of oyster beds in Alabama waters suffered 73 to 98 % losses. Examination of oysters taken during the heaviest mortality revealed a 60 % incidence of *P. marinus* infestations. Subsequent sampling in producing areas disclosed light to heavy infestations in 0 to 85 % of the oysters (May, 1968). In affected areas, annual losses of *Crassostrea virginica* due to *P. marinus* are frequently in excess of 50 % (Ray, 1954a; Ray and Chandler, 1955; Mackin, 1962; Quick, 1971; Little and Quick, 1976; Ogle and Flurry, 1980).

In the 1950's, 'Dermo disease' was the primary cause of oyster deaths in Virginia waters during the warm season. Increased mortality rates experienced since about 1940 may reflect its introduction into Chesapeake Bay. With initial seed-oyster counts of 1,000 to 2,000 per bushel, total losses in 2 or 3 years of culture were 65 to 85 % of the number planted. *Perkinsus marinus* caused a major part of these losses in most high-salinity areas. Recruitment on public beds and repeated plantings on private beds insured that a few old infested oysters functioned as reservoir for infestation of newly planted, healthy oysters. Spread of the disease into new areas without residual infested oysters is very slow. Scarcity of oysters caused a decline in *P. marinus* abundance in lower Chesapeake Bay since 1960, when plantings ceased due to *Haplosporidium nelsoni* epizootics experienced in that area (Andrews, 1979; see section 'Agents: Ascetospora').

In Chesapeake Bay, *Perkinsus marinus* does not multiply at temperatures below 20 °C and salinities lower than 12 to 15 ‰ S. Hence, the parasite is inactive from early November to late May each year. Expulsion of active stages occurs in late autumn and winter. Subclinical overwintering infestations persist at temperatures below 5 °C and salinities of less than 5 ‰ S. They are usually too rare to be detected by the thioglycollate test, but become active when oysters with cryptic stages are placed in heated aquaria in April or May. Deaths occur in about a month (Andrews and Hewatt, 1957; Andrews, 1966).

Interaction of *Perkinsus marinus* with *Haplosporidium nelsoni* may limit spread of the former within areas where the latter is enzootic. Thus, disease-free oysters, introduced into lower York River, Virginia, required 1 to 3 years to contract *P. marinus* infestations. During this period, *H. nelsoni* decimated oyster populations, thereby preventing *P. marinus* from becoming epizootic (Andrews, 1967).

When, after the severe outbreaks of *Haplosporidium nelsoni* in Chesapeake Bay

waters of southern Maryland during the mid-sixties, oysters were drastically decimated, *Perkinsus marinus* vanished from that area. After gradual replenishment of the collapsed oyster beds, *P. marinus* became epizootic again in 1974, with prevalences up to 100 %. However, the outbreak was not accompanied by the high mortalities experienced previously. It is not known whether this decrease in severity was due to a less virulent strain, a greater resistance of the oysters, or external factors. Data from the epizootic area for 1977 show an even lesser prevalence (0 to 18 %), but this sharp drop may be attributable to the extremely cold winter of 1976–77 (Otto and co-authors, 1979).

Oysters of different geographic origin may exhibit pronounced differences in their susceptibility to *Perkinsus marinus* infestation. Such differences, which have important implications for oyster planters, probably reflect, to a large extent, the duration of host-parasite contact. Chesapeake Bay-native *Crassostrea virginica* were found to be more susceptible to *P. marinus* than introduced South Carolina oysters, and seed from seaside bays of Virginia was so susceptible that its planting in enzootic areas was precluded if more than a single summer's exposure to *P. marinus* was required (Ray and Chandler, 1955; Andrews and McHugh, 1956; Andrews and Hewatt, 1957).

Although prevalences of *Perkinsus marinus* may at times attain epizootic proportions in particular areas, its most significant effect is probably that of continuing attrition, year after year, during periods of high sea-water temperatures. Adverse effects of the disease on commercial oyster beds are now controlled, at least to some extent, by planting and harvesting at prescribed times of the year and by planting oysters thinly on beds. Losses could be minimized by limiting the number of summers that oysters are held in enzootic areas and by the maximum use of low-salinity areas (Sindermann, 1968a).

Perkinsus marinus does not appear to be host–specific to *Crassostrea virginica*. Although it prevails in American oysters, specifically identical or very closely related organisms have been reported from leafy oysters *Ostrea frons* in Florida and from horse oysters *Ostrea equestris* in Texas. The agent was not found, however, in mangrove oysters *Crassostrea rhizophorae* from Puerto Rico, in European oysters *Ostrea edulis* from Holland, or in rock oysters *Saccostrea cucullata (Crassostrea commercialis)* from Australia. Olympia oysters *Ostrea lurida* were experimentally infested by exposure to diseased *C. virginica* (Ray, 1954a).

Outside the North American continent, *Perkinsus marinus* or a closely related form has been reported from Hawaii. Kern and co-authors (1973) found a 1972 mass mortality of *Crassostrea virginica* from West Loch, Pearl Harbor, to be associated with the presence of a "fungus parasite similar to *Labyrinthomyxa marina*". Losses in that area ranged from 90 to 99 %, or approximately 30 to 34 million oysters.

Whether the occurrence of *Perkinsus marinus* is restricted to oysters, is not clear. Ray (1954a) and Ray and Chandler (1955) reported '*Dermocystidium*-like' parasites from *Anadara transversa* (fam. Arcidae), *Argopecten irradians* (Pectinidae), *Anomia simplex* (Anomiidae), *Laevicardium mortoni* (Cardiidae), *Mercenaria mercenaria* (Veneridae), *Macoma baltica, M. tenta, M. phenax* (Tellinidae), *Tagelus plebeius* (Sanguinolariidae), *Ensis minor* (Solenidae), *Mulinia lateralis* (Mactridae), *Mya arenaria* (Myacidae), *Martesia* sp. (Pholadidae) and *Lyonsia hyalina* (Lyonsiidae). In some host species, these parasites were found in Chesapeake Bay, but were absent in the Gulf of Mexico. The possibility that some of these bivalves act as reservoir hosts for *P. marinus* cannot be excluded.

Andrews (1955) found '*Dermocystidium*-like' organisms in 12 of 16 bivalve species from the Gloucester Point, Chesapeake Bay, area. Infestations were usually systemic, but the parasite cells were more clustered and appeared to be enclosed in cysts. In several of the host species, all individuals examined were infested, but intensities were rarely rated above light. *'Dermocystidium'* stages taken from *Macoma baltica* enlarged in Ray's (1952) thioglycollate medium developed for the cultivation of *Perkinsus marinus*. Identical results were obtained by Mackin (1962) with a (presumably identical) *M. baltica* parasite.

Ray (1954a) and Andrews and Hewatt (1957) conducted host–specificity studies in which they injected minced, *Perkinsus*–infested *Macoma baltica* tissue into the mantle cavity of *Mya arenaria, Mercenaria mercenaria* and *Crassostrea virginica*. Transfer of the pathogen was not achieved, which led Ray to the conclusion that the *M. baltica* parasite is different from that found in *C. virginica*. In view of the ease with which *C. virginica* is normally infested experimentally with *P. marinus*, Ray's assumption appears to be justified. Electron–microscopic examination of zoospores of the *M. baltica* parasite revealed but minor morphological differences (i.e., longer mastigonemes on the anterior flagellum) (Perkins, 1968a), but the sporangium differed markedly in morphology from that of *P. marinus* (Valiulis and Mackin, 1969). Farley (1977) reported the parasite from 19 of 39 *M. baltica* from Tred Avon River, Maryland, used in pesticide toxicity tests. Twelve of the 19 infested clams died in the course of the experiments, which reflects a considerable degree of pathogenicity of *Perkinsus* sp. for *M. baltica*.

Although there can be little doubt that the oyster and the clam parasite are distinct species, it is not quite clear whether *Perkinsus marinus* in *Crassostrea virginica* represents a single species. Two major differences were noted between pathogens from oysters in Virginia and in Florida. Thus, Perkins (1969b), in his study on the ultrastructure of the vegetative stages of *P. marinus*, observed virus-like particles, 46 to 53 nm in diameter, in the nucleus and cytoplasm of *P. marinus* from Virginia oysters. Morphologically, the particles consisted of a limiting dense line as well as an electron-light cortex and a core separated by a dense line. They are probably icosahedrons. There was usually an electron-light halo, 670 to 740 nm in diameter, around these particles and a very large halo, 1,500 to 2,000 nm in diameter, around the whole system. The significance of these halos is not known. The virus–like particles were not seen in *P. marinus* from Florida oysters.

Secondly, Perkins and Menzel (1967) observed a paranuclear structure of unknown significance in all *Perkinsus marinus* zoospores from Gulf oysters, but in none of the Virginia pathogens. This may suggest that 2 congeneric species, commonly regarded as *P. marinus*, are involved (Perkins, 1969b).

The occurrence of *Perkinsus* spp. or *'Labyrinthomyxa*-like' organisms is not restricted to bivalves. Recently, a new species of *Perkinsus* has been found in abalones *Haliotis ruber* (Gastropoda) from Australia. The organism was not assayed on thioglycollate medium, but its belonging to *Perkinsus* has conclusively been evidenced by light and electron microscopy. Host pathology was similar to that described from *P. marinus*-infested oysters, and pathogenicity increased with temperature (Lester, 1980).

With respect to the large number of *'Labyrinthomyxa*-like' organisms detected in oysters and other bivalves, Quick and Mackin (1971) and Quick (1972a) expressed the opinion that we are actually dealing with a group of related species. It appears more likely, however, that a considerable proportion of these organisms are not apicomplexans but labyrinthulids (see section 'Agents: Labyrinthomorpha').

Several species of 'cephaline' (= septate) gregarines — members of the Eugregarinida (Table 13-3) — have life-cycle stages in marine bivalves. While the majority of these 'sporozoans' parasitize arthropods and usually complete their entire life cycle in one host individual, members of the family Porosporidae exhibit host alternation, with intermediate stages in marine pelecypods.

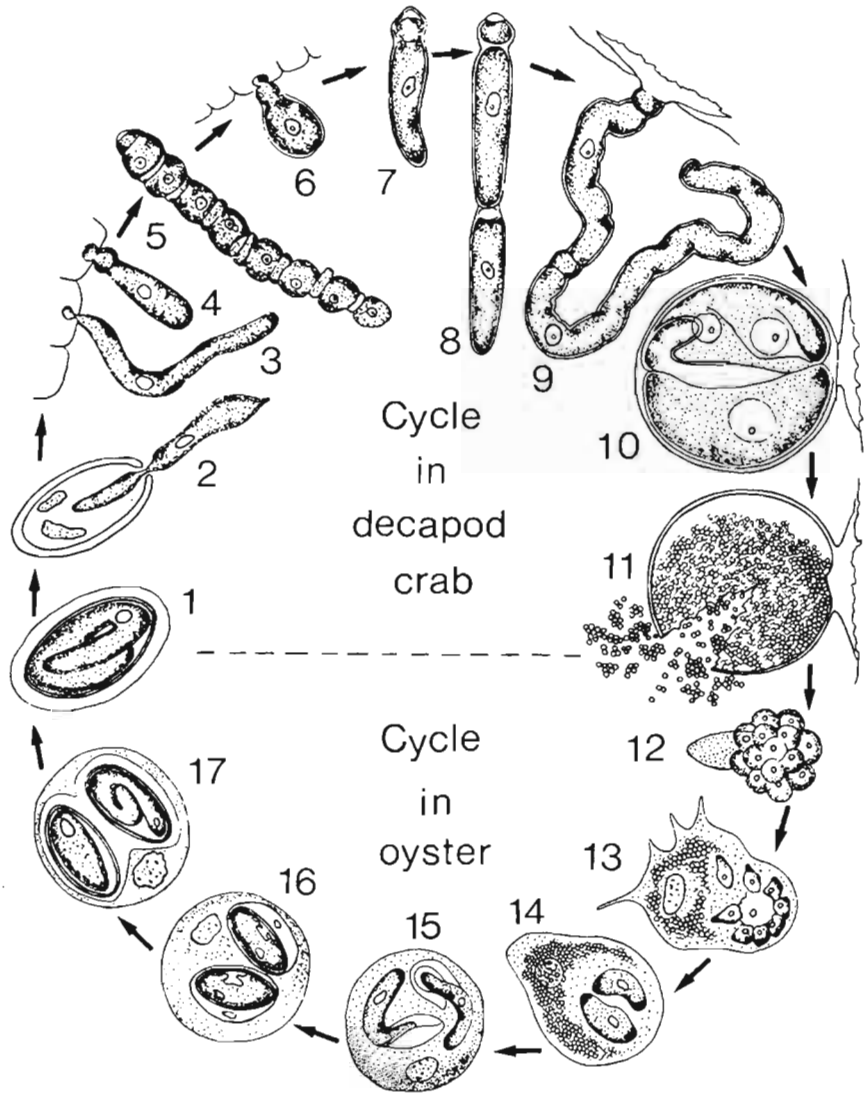


Fig. 13-25: *Nematopsis ostrearum*. Life cycle. 1: Oocyst from *Crassostrea virginica* containing sporozoite; 2: sporozoite escaping from spore in crab intestine; 3: attachment to host's intestinal epithelium; 4: development into trophozoite; 5: association of trophozoites; 6: temporary reattachment to intestinal epithelium; 7: development into mature gamont; 8–9: syzygy followed by attachment to crab's rectum; 10: gametocyst formation; 11: liberation of gymnospores from ruptured gametocyst; 12: single gymnospore; 13: engulfment of gymnospore by oyster phagocyte and disruption of gymnospore; 14–16: sporozoite growth within phagocyte; 17: formation of resistant oocysts within phagocyte. (After Prytherch, 1940.)

With respect to the designation of the various gregarine life-cycle stages, the terminology proposed by Levine (1971) is adopted here. It deviates markedly from that used by previous authors. For the sake of clarification, it may be stated that the gregarine 'spores' encountered in molluscan hosts actually are oocysts containing one or more uninucleate sporozoites, which represent the stage capable of developing into trophozoites (gamonts) in the intestine of the arthropod final host.

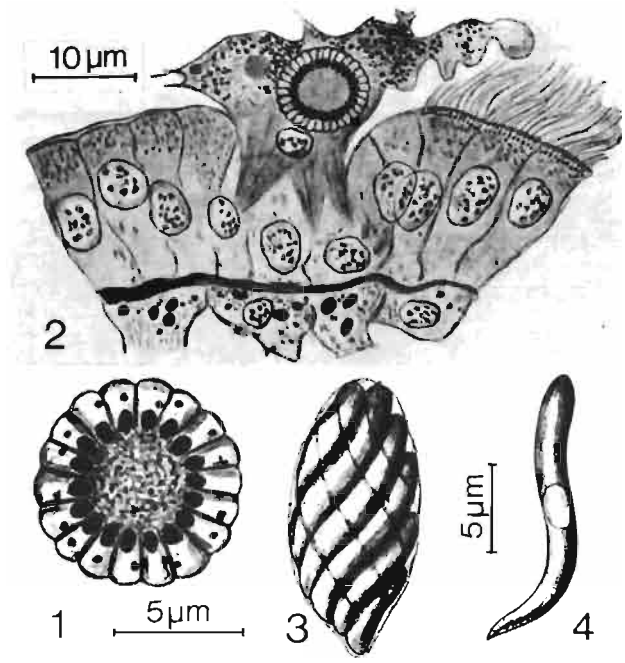


Fig. 13-26: Porosporidae. Life-cycle stages in molluscs. 1: Gymnospore of *Nematopsis legeri;* 2: *Porospora gigantea* gymnospore engulfed by mussel phagocyte re-entering host gill; 3: bundle of naked *P. gigantea* sporozoites; 4: single sporozoite. (After Hatt, 1931.)

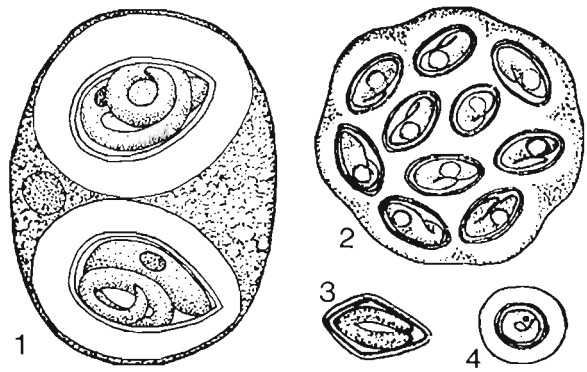


Fig. 13-27: Gregarine oocysts from marine bivalves. 1: *Nematopsis* sp. Two oocysts enclosing vermiform sporozoites in gill-connective tissue cell of *Solen vagina;* 2: *N. legeri* oocysts from *Mytilus galloprovincialis;* 3: *N. veneris* from *Venus fasciata* gill; 4: *N. pectinis* from *Chlamys varia.* (1: After Schneider, 1892; 2–4: After Léger and Duboscq, 1925.)

The basic life-cycle pattern in the Porosporidae is as follows (Fig. 13-25): Trophozoites in the arthropodan intestine undergo syzygy, thereby giving rise to gametocyst and gamete formation. Subsequent sporulation produces 'gymnospores' (Fig. 13-26, 1), which are released from ruptured gametocysts. The gymnospores, which are typical of the Porosporidae, represent the stage infesting molluscs. When gymnospores are drawn into the mantle cavity of a compatible bivalve-intermediate host and come into contact with the

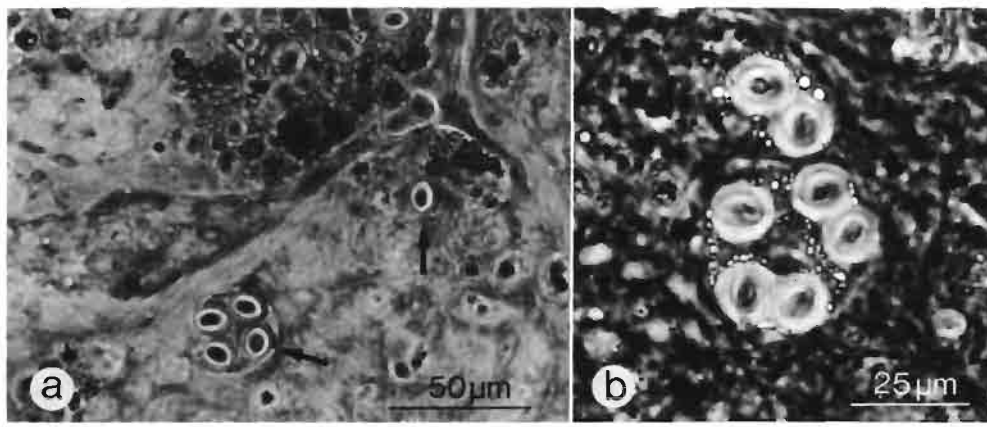


Fig. 13-28: *Nematopsis (schneideri?)* oocysts from gill connective tissue of *Cardium edule*. (a) Bright field, (b) phase contrast. (Original.)

gill or mantle epithelium, the host responds by sending out phagocytes to engulf them. The gymnospore-laden phagocytes then pass back through the epithelium (Fig. 13-26, 2). In this process, the gymnospore provides the stimulus, while the phagocyte takes the active role. Within the cytoplasm of the host cell, disruption of the gymnospore ensues, followed by growth of the individualized sporozoites. Eventually, a fusiform bundle of naked sporozoites, enclosed by a host cell, results in members of the genus *Porospora* (Fig. 13-26, 3, 4), while in *Nematopsis* monozoic resistant oocysts ('spores') are formed. These have thick hyaline walls and contain a vermiform sporozoite resembling a little nematode (Figs 13-26 to 13-28). Koulman (1970) mistook *Nematopsis* spores from *Cardium edule* for encysted larval trematodes.

Porospora gigantea gymnospores normally develop into sporozoites in various gastropods (Vol. I, Chapter 12). Experimental infestations were achieved in individuals of *Brachidontes (Mytilus) minimus*, although with limited success. The gills of mussels challenged with gymnospores were seen to undergo considerable atrophy. Similar changes, however, occurred after prolonged exposure to India ink suspensions. The final host of *P. gigantea* is the European lobster, *Homarus gammarus* (Hatt, 1927a, 1928, 1931).

Gregarines of the genus *Nematopsis* utilize marine bivalves as normal intermediate hosts. The first described species — only known in its oocyst stage (Fig. 13-27, 1), and consequently not given a specific name — has been found in the mantle of razor clams *Solen vagina* in France. Schneider (1892), with some reservation, assigned it to the Coccidia. *N. schneideri* oocysts, ovoid in shape, about 12 × 8 µm in dimension, and slightly pointed at one end (Fig. 13-28), have been recovered from the gills of *Mytilus edulis, Spisula solida, Donax vittatus, Tapes pullastra, Macoma baltica, Cardium edule* and

other pelecypods. The vegetative stages of this gregarine (which possibly represents an assemblage of several distinct species) occur in decapods *Carcinus maenas* and *Macropipus depurator* (Léger, 1903; Léger and Duboscq, 1925; van Banning, 1979b). *N. portunidarum* has oocysts, 12 to 15 μm long, in the gill vessels of *C. edule* and vegetative and sexual stages in *M. depurator* (Léger and Duboscq, 1913a, b).

Nematopsis legeri, originally named *N. mediterranen [nomen oblitum]* by Léger (1905) and redescribed as *Porospora galloprovincialis* by Léger and Duboscq (1925), is a common parasite of various bivalves, chitons and gastropods from the French Atlantic and Mediterranean coasts. Its oocysts (Fig. 13-27, 2), which measure approximately 14.5 × 8.5 μm, have been recorded from *Mytilus galloprovincialis, Brachidontes minimus, Lasaea rubra* and *Cardita calyculata*. Crabs *Eriphia spinifrons* serve as the decapod definite host (Hatt, 1927b, 1931). Other European species of *Nematopsis* include *N. veneris* from Venus clams *Venus fasciata* and *N. pectinis* from scallops *Chlamys (Pecten) varia* (Léger and Duboscq, 1925; Fig. 13-27, 3, 4). Their decapod hosts are unknown. Originally, *N. veneris* and *N. pectinis* were placed in the genus *Porospora*, but later have been transferred to *Nematopsis* by Sprague (1970b).

Crassostrea virginica from the Atlantic and Gulf coasts of the United States frequently harbours the gymnospore and oocyst stages of *Nematopsis ostrearum* (Fig. 13-25). The oocysts, which measure approximately 14 × 10 μm, occur in all organs of the oyster, but are most conspicuous in the mantle. *N. ostrearum* has a weak host specificity, at least with respect to its intramolluscan stages. Other common bivalves, such as *Ostrea equestris, Argopecten irradians, Anomia simplex, Venus ziczac, Geukensia demissa* and *Martesia cuneiformis* have also been found susceptible to gymnospore infestation. The trophozoite, gamont and gametocyst stages of *N. ostrearum* parasitize in the gut of at least 4 species of decapod crustaceans (Prytherch, 1938, 1940; Sprague, 1949, 1954b, 1970b; Landau and Galtsoff, 1951; Sprague and Orr, 1955).

Nematopsis prytherchi is another parasite of *Crassostrea virginica*, which sometimes occurs in concurrent infestations with *N. ostrearum*. Its oocysts, although larger (ca. 19 × 16 μm) and mostly concentrated in the gills, have been mistaken by Prytherch (1940) for those of *N. ostrearum*. Vegetative and sexual stages of *N. prytherchi* occur in stone crabs *Menippe mercenaria* (Sprague, 1949; Sprague and Orr, 1955). In addition to *Nematopsis* oocysts located mainly in the gills and mantle — which probably represent *N. ostrearum* and *N. prytherchi* —, Quick (1971) observed numerous *Nematopsis* oocysts internal to the gonad near the stomach, intestine or larger digestive diverticula of tank-held individuals of *C. virginica* from the Gulf of Mexico. It appeared that the oocysts had entered the oysters via the intestinal wall, "leaving a thin trail of lysed cells in their wake". Whether these oocysts represent a third species of *Nematopsis* infesting *C. virginica* has not been studied. Oysters from northern Florida consistently lacked these near-digestive tract oocysts.

Oocysts of *Nematopsis duorari*, measuring 19 × 10 μm, were found in *Argopecten irradians, Cardita floridana, Chione cancellata* and *Macrocallista nimbosa* from Florida. The decapod host of this gregarine is the pink shrimp *Penaeus duorarum* (Kruse, 1966; Sprague, 1970b).

Specifically unidentified or unnamed *Nematopsis* spp. have been observed in *Mercenaria mercenaria* from Long Island, New York (oocyst: 11.8 × 8.2 μm), in *Crassostrea rhizophorae* from French Guayana (oocyst: 14 to 20 × 10 to 14 μm) and in *Perna*

canaliculus from New Zealand (oocyst: 12 × 7 μm) (Comps and co-authors, 1972; Jones, 1975; Meyers, 1981). Unidentified *Nematopsis* spp. from North and South American bivalves have been listed by Sprague (1954b), and 'gregarine–like organisms' have been observed in the kidney and pericard of *Macoma baltica* from Tred Avon River, Maryland, USA (Farley, 1977).

Reports concerning the pathogenicity of *Nematopsis* spp. infestations in bivalves are inconclusive. Although entrance of gymnospores into the intermediate host's tissues is believed to be a passive process accomplished by host phagocytosis (Fig. 13-26, 2; Hatt, 1931), thin trails of lysed cells were found to mark the path of the sporozoan from the epithelial surface to the definite site (Quick, 1971). The resulting immune reaction was quite localized.

Prytherch (1938, 1940) believed that *Nematopsis ostrearum* was an important factor in oyster mortalities observed in Mobjack Bay, Virginia, and Lake Barre, Louisiana (USA), its pathogenicity depending on the intensity of infestation, age and general condition of the host, and other factors. Heavy oocyst infestations were accompanied by paralysis, gaping and cessation of shell growth. In experiments, in which oysters were infested with *N. ostrearum* by exposing them to infested crabs, mortalities varied between 46 and 84 %, as opposed by 3 and 7 % in controls. Mackin (1962) found no support for Prytherch's contention and suggested that the latter author's results were obscured by *Perkinsus marinus* infestations.

Andrews (1979), on the other hand, points out that the 1930 winter mortality of *Crassostrea virginica* in Mobjack Bay, discovered by Prytherch in March, could not have been caused by *Perkinsus marinus*, for it does not kill oysters in late winter. Neither was it caused by *Nematopsis* as proposed by Prytherch, for these gregarines have not been demonstrated to kill oysters in Virginia (Feng, 1958). Anyhow, Prytherch's proposal to limit *Nematopsis* in oysters by reducing mud-crab abundance was largely effected in the mid-sixties by accidental importation of the parasitic sacculinid cirriped *Loxothylacus panopaei* into Chesapeake Bay from the Gulf of Mexico in oyster shipment (van Engel and co-authors, 1966; Andrews, 1979). This appears to be an excellent example of biological control.

Likewise, Landau and Galtsoff (1951), Owen and co-authors (1951) and Quick (1971) obtained no evidence to indicate that *Nematopsis ostrearum* killed oysters or reduced the quality of the meats. Sprague and Orr (1955) introduced extremely large numbers of *N. ostrearum* and *N. prytherchi* oocysts into *Crassostrea virginica* in an attempt to kill the host. The results were superimposed by unknown mortality factors and were neither consistent nor decisive, although a slight trend toward greater mortality in experimentally infested hosts was noticed. Heavy oocyst invasion could result in clogging of the oyster's gill-blood vessels. It was felt, however, that the numbers of oocysts observed to occur naturally in *C. virginica* were no significant factors in mortality of oysters in the field. Oocyst counts in oysters showed to be positively correlated with the abundance of xanthid crabs, which serve as definite hosts for *N. ostrearum*. The infestation intensity was usually cumulative and increased with the oysters' age. Since the oocysts do not undergo schizogonic multiplication in the oyster, their concentration depends solely on acquisition from the environment. Feng (1958) demonstrated the existence of a dynamic equilibrium between uptake and elimination of oocysts, with numbers declining rapidly in the absence of crabs.

Microscopic examination of large numbers of oysters revealed no significant histopathological changes associated with the presence of *Nematopsis ostrearum* or *N. prytherchi* oocysts, nor was there evidence that the oocysts produced toxins of any kind, as believed by Prytherch (1938, 1940). The latter author assumed that oocysts in a heavily infested individual of *Crassostrea virginica* reached concentrations between 1 and 5 millions. Simple calculations, however, indicate that in a 5-cm-long oyster the number of oocysts per 10-μm cross section would be 500 if there were 2.5 million oocysts in one oyster. In contrast, only a maximum of a couple of dozen oocysts has been observed in any one section, though thousands of sections have been examined (Mackin, 1962). Sindermann (1970a), on the other hand, observed massive *N. ostrearum* infestations in *C. virginica*, in which extensive areas of the oyster were occupied by oocysts. He suggested that such mechanical interference with host physiology must have some harmful effect.

It might be tempting to associate the presence of *Nematopsis* oocysts in the mantle veins and tissues of *Mytilus edulis* with the production of pearls or calcareous excrescences on the inner surface of the valves. Léger (1903) found the valves of mussels from Luc-sur-Mer (France), which were heavily infested with *Nematopsis schneideri* oocysts, to be "irregular in thickness". Although Léger did not specifically report the presence of pearls or distinct calcareous ridges on the shell, the condition described by him is much like the early stage of pearl formation usually attributed to larval trematode infestation (see section 'Abnormalities'). Götting (1979a) reported unidentified 'ova' from the centre of *M. edulis* pearls. The electron-light core of a pearl shown in Fig. 13-29 measures 13.8 × 10.0 μm, and thus fits exactly into the size range of *Nematopsis* oocysts frequently encountered in mussels.

Immature stages of a specifically unidentified gregarine-like 'amoeboid' organism, which apparently uses oysters as definite host, have been reported from *Crassostrea*

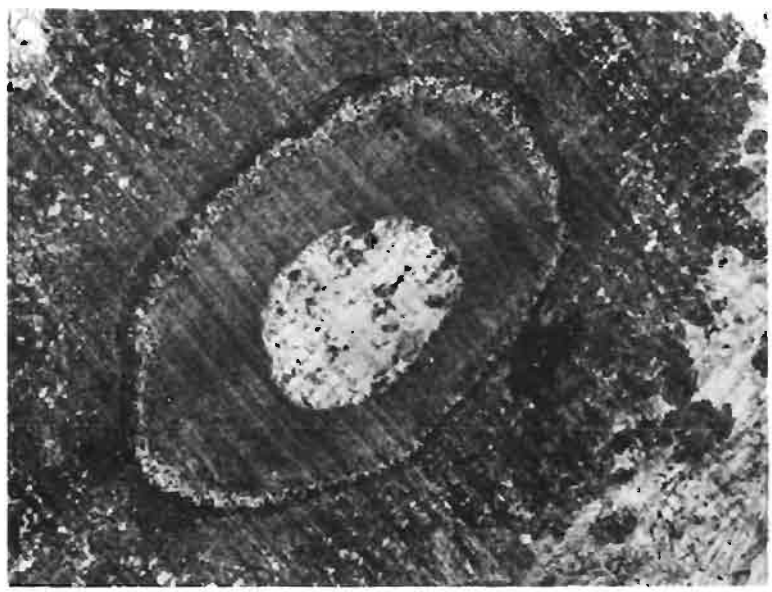


Fig. 13-29: *Mytilus edulis*. Electron micrograph of ultrathin cut of pearl nucleus from mantle tissue. Note 'ovum' (*Nematopsis* oocyst?) in centre. (Photograph courtesy K.-J. Götting.)

virginica in New Haven Harbor, Connecticut, and Chesapeake Bay, Maryland (USA). Spherical forms resembling encysting trophonts were present in the stomach, and young sporonts and trophozoites occurred in the cells and lumen of the digestive gland. The parasites, which have not been observed to penetrate the tissues beyond the basement membranes, caused short-term intestinal inflammation and focal sloughing of cells of the columnar epithelium. Infestations had an apparent springtime seasonality, followed by recovery without residual tissue pathology. There are indications that the parasite overwinters in hibernating oysters, undergoes vegetative growth in the spring, and is cleared from the digestive tract with concurrent transient host-tissue response. The 'amoeboid organism' was found in 14 of 1,337 oysters from Connecticut and in 19 of 150 individuals from Chesapeake Bay (Newman, 1971; Sawyer and co-authors, 1973). Upon restudy, Sawyer and co-authors (1975) tentatively identified the parasite as a gregarine, possibly of the *Nematopsis-Porospora* type. Its occurrence in oysters was regarded as atypical, since the observed stages would more appropriately be expected to reside in the digestive tract of a crustacean host.

Although Gutiérrez and Pascual (1976) and Gutiérrez (1977a) noted similarities between Newman's (1971) 'amoeboid organism' from *Crassostrea virginica* and the 'amoeboid cysts' found by them in *C. angulata* (p. 524), a direct relationship appears to be unlikely.

Several eimeriine and adeleine coccidians (Table 13-3) are known to infest marine bivalves. Anisogamy and sporogony occur in the molluscan host, mostly in the kidney. Further life-cycle stages are unknown. Alternate hosts harbouring schizogonic (merogonic) stages very probably exist but have not yet been identified. A single known eimeriine completes its entire life cycle in an oyster.

Eimeriines *Pseudoklossia glomerata* parasitize in the kidney and occasionally in the visceral ganglia of *Tapes floridus* and *T. virgineus* from the Mediterranean Sea. Micro- and macrogamonts measure respectively 18 to 36 and 40 μm in diameter. The sexual stages occur in the epithelial cells of the renal tubules. By the end of sporogony, 2 sporozoites develop in each of the numerous sporocysts contained in the oocysts, which are about 33 μm in diameter. Sporocysts measure 4 to 5 μm across and have very delicate walls. Host cells penetrated by gametocytes become enormously hypertrophied, the space normally occupied by the cytoplasm being entirely replaced by the parasite (Fig. 13-30, 1-3). Affected cells may detach and heavy infestation may cause serious kidney damage (Léger and Duboscq, 1915).

Pseudoklossia pectinis, with oocysts 32 to 35 μm in diameter, occur in the kidney of *Pecten maximus* from Roscoff, France (Léger and Duboscq, 1917), and *Pseudoklossia (Hyaloklossia) pelseneeri*, with oocysts 75 to 80 μm in diameter, has been reported from the nephridia of *Tellina* sp. and *Donax* sp. (Léger, 1897b). A coccidian, similar to *P. pectinis*, parasitizes in the renal epithelium of *Ostrea edulis* from Auray, France (Tigé and co-authors, 1977). Affected cells undergo considerable hypertrophy, similar to that produced by *P. glomerata*, but overall damage to the kidney appears to be light.

Klossia tellinae, an adeleine coccidian, invades the epithelial cells of the kidney of *Tellina tenuis* from Scottish waters. Its spherical to subspherical oocysts measure about 41 μm in diameter and contain 32 or more sporocysts, 6 μm in diameter, each with 3 flexed sporozoites, 8 μm in length and 1 μm in width. Merogony was not observed and presumably occurs outside the bivalve host. Long-term studies suggest that *K. tellinae* has little if

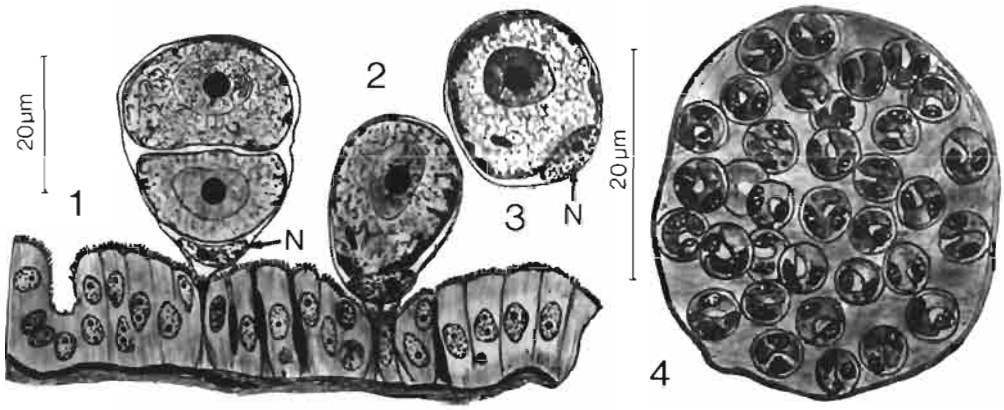


Fig. 13-30: *Pseudoklossia glomerata* in epithelial cells of *Tapes floridus* kidney. 1, 2: Greatly hypertrophied host cells containing *P. glomerata* gamonts; infested host cells still attached to renal epithelium by delicate stalk; 3: dislodged host-epithelial cell enclosing parasite, floating free in lumen of renal tubule; 4: mature oocyst enclosing numerous sporocysts, each containing 2 sporozoites. N hypertrophied host-cell nucleus. (After Léger and Duboscq, 1915.)

any effect on the density and distribution of *T. tenuis* populations known to be heavily parasitized by this and another coccidian (see below) (Buchanan, 1979a). Unidentified coccidians have been observed in the kidney of *Anomia aculeata* from Plymouth, England (Atkins, 1933a).

While all above-described coccidians presumably have — yet unknown — alternate hosts in which merogony occurs, eimeriines *Merocystis tellinovum* complete their entire life cycle in a bivalve, *Tellina tenuis*. Sporozoites occur in the primary germ cells of the host's ovarian tubules. Merogony gives rise to merozoites, which infest other germ cells. After repeated schizogony, young merozoites develop into gamonts. These produce macrogametes and swarms of spherical, biflagellate microgametes. Subsequent karyogamy results in the formation of oocysts, 28.4 µm in diameter, with 64 thin-walled sporocysts, 4.5 µm across, each containing 2 flexed sporozoites, 3 µm long, lying at right angles to each other, and having a conspicuous refractile globule at the posterior end. *M. tellinovum* has only been observed in the primary germ cells of the ovarian tubules of *T. tenuis*; male hosts were not infested. The coccidians brought about partial to complete loss of the reproductive potential of female clams owing to mechanical disruption of primary germ cells. In Kames Bay, Millport, Isle of Cumbrae (Scotland), approximately 90 % of the *T. tenuis* females were found to be infested (Buchanan, 1979a).

Almost 57 % of the females of blacklipped oysters *Crassostrea echinata* from Darwin Harbour, Northern Territory (Australia), were found to be seasonally infested by an unidentified protistan parasite. One or 2 cysts, each with 2 unicellular organisms, occurred in a single host ovum in the yolk portion adjacent to the egg nucleus. Individual cysts were spherical, with a clear wall, and divided into 2 chambers by a transverse membrane (Fig. 13-31). With some reservation the parasite was assigned to the Coccidia. It was seen only in ripe and developing ova of *C. echinata*; males were not affected. It was assumed that the protistan, which was considered non-pathogenic, uses the oyster's ova simply as a vehicle for distribution, although with an obviously castrating effect on the host. Nevertheless, most infested individuals examined were in good condition, with their gonads ripe and

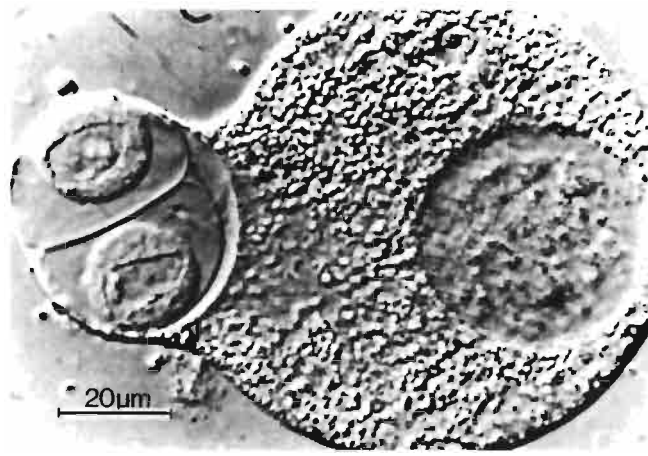


Fig. 13-31: *Crassostrea echinata*. Unidentified (coccidian?) parasite in host ovum. Squash preparation; interference contrast. (Photograph courtesy P. H. Wolf.)

running, inspite of the infested ova. It was suspected that the parasite's life cycle perhaps involves a final host other than the oyster. Stages resembling schizogony were not seen (Wolf, 1977, 1979). Further study, however, might reveal a life cycle similar to that reported for *Merocystis tellinovum*.

'Coccidia-like organisms', measuring 14.3 × 6.8 µm (probably meronts or gamonts), were observed within epithelial cells lining the intestine of an individual of *Crassostrea virginica*. Each organism was ellipsoidal and protruded from the apical portion of a host cell. Approximately 100 adjacent epithelial cells were each infested with a single parasite, some of which contained rod-shaped bodies resembling developing merozoites within meronts. A single oyster from Long Island, New York, was found to be infested with this organism (Meyers, 1981). This appears to be another coccidian either completing its entire life cycle in a single host individual or — as gametogonic and sporogonic stages were not observed — using the oyster as intermediate host.

Agents: Microspora (the Microsporidians)

A few members of the Microspora (Table 13-3) are known to parasitize marine bivalves. These intracellular protozoans are comparatively small (3 to 6 µm) organisms completing their entire life cycle in a single host cell. Intermediate hosts are not known. Transmission of the agents is accomplished by spores liberated from ruptured infested host cells. Microsporan spores have chitinous walls and enclose an amoeboid sporoplasm ('amoebula') and a long protrusible, hollow polar filament. When ingested by a compatible host and coming into contact with the intestinal epithelium, the spore releases the polar filament, which anchors the spore to the host, and through which the amoebula escapes into the host cell. Sporoplasms usually invade and remain in the epithelial cells of the intestine but may spread to other body parts, using the host's phagocytes as vehicle. Inside the host cell, the parasite undergoes schizogony (merogony) and sporogony.

Steinhausia ovicola has been found to parasitize the ova of individuals of *Ostrea edulis* from Marennes (France). The stage most frequently seen consisted of intracytoplasmic

spherical or ovoidal cysts, 18 × 20 µm in dimension and containing 40 to 60 spherical spores, 2.3 µm in diameter. The cysts, which were closely associated with the host-cell nucleus, had characteristic tough, elastic membranes, and caused conspicuous invaginations of the host-nuclear membrane. Less frequently, multinucleate sporogonial plasmodia, about 8 µm in diameter, have been encountered. Host ova harbouring a single cyst appeared to be affected but slightly. Rare cases of multiple infestation, however, were accompanied by a diminution of yolk granules in the cytoplasm and a tendency toward lysis of the nucleus (Léger and Hollande, 1917).

Steinhausia mytilovum parasitizes in the ova of *Mytilus edulis* from the Atlantic coast of the United States (Field, 1923; Sprague, 1965, 1970a) and has also been found in about 10 % of *Mytilus galloprovincialis* from the Gulf of Naples, Italy (Vincentiis and Renzoni, 1963). The cysts, which are spherical and up to 20 µm in diameter, are typically found in the cytoplasm but sometimes occur also in the nucleus of the host ova and contain spherical spores, about 4 µm in diameter (Fig. 13-32). Field (1923) recognized amoeboid organ-

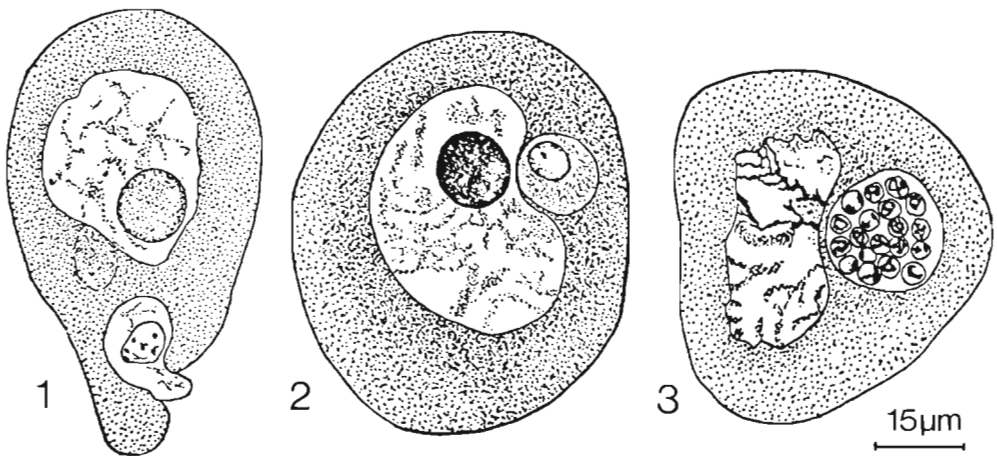


Fig. 13-32: *Steinhausia mytilovum*. 1: 'Amoeboid stage' invading *Mytilus edulis* ovum; 2: sporogonial plasmodium in cytoplasm; note characteristic invagination of host-cell nucleus; 3: cyst with mature spores. (After Field, 1923.)

isms, moving about freely among the follicles and in the genital canals of female mussels, which actively penetrated host ova. Sprague (1965a), who restudied *S. mytilovum* in detail, recognized small, uninucleate cysts in the cytoplasm of infested ova as the earliest developmental stage of the microsporan.

According to Field (1923), many thousands of eggs in a mussel may be parasitized and destroyed by *Steinhausia mytilovum*. Sprague (1965), on the other hand, found that the proportion of infested eggs was quite small in *Mytilus edulis* collected near Ocean City, Maryland (USA), and considerable searching was usually required to find the parasite. Infested ova usually contained only a single cyst. Multiple infestations with as many as 4 or 5 cysts per egg were infrequent. There is no reason to believe that this microsporan is economically important.

The two species of *Steinhausia* were originally believed to belong to the Balanosporida ('Haplosporidia') and have been described as *Chytridiopsis ovicola* (Léger and

Hollande, 1917) and *Haplosporidium mytilovum* (Field, 1923), respectively. Dollfus (1921a) regarded *C. ovicola* as a coccidian but gave no indication that its systematic position might be in doubt. Sprague (1963) felt that *H. mytilovum* is improperly classified. Manier and Ormières (1968), who studied the ultrastructure of the type species of the genus *Chytridium, C. socius*, demonstrated that it is actually a microsporan. Subsequently, Sprague and co-authors (1972) created the new microsporan family Chytridiopsidae and erected the genus *Steinhausia*, in which they included the two species from *Ostrea edulis* and *Mytilus edulis*.

Steinhausia ovicola and *S. mytilovum* affect only female hosts, and both appear to be strictly host-specific. In addition to the usual route of transmission via the alimentary tract, transovarial transmission has been suggested for both species. Since the effect of single cysts on the viability of the ova is but faible, infested eggs presumably develop normally and carry the microsporan over to the offspring (Léger and Hollande, 1917; Maurand and Loubès, 1979).

Approximately 10 % of 109 *Ostrea lutaria* from Foveaux Strait, New Zealand, were found to be infested with a sporozoan, described as a new species, *Microsporidium rapuae*. Spores from frozen tissue in aqueous mount measured $5.20 \pm 0.35 \times 2.57 \pm 0.28$ µm ($n = 50$); those embedded in Araldite were $4.70 \pm 0.39 \times 2.39 \pm 0.28$ µm ($n = 25$) in dimension. They showed a polar cap at the spore apex, with an attached long polar filament of 19 to 22 coils, and a well-developed endospore. The spores occurred in oval or spherical cysts, 20 to 70 µm in long axis, in the connective tissue surrounding the gut, with each cyst containing more than 100 spores. No gross histopathological signs were observed in the infested oysters (J. B. Jones, 1981). As sporogenesis was not seen and the number of spores formed within the pansporoblast membrane is unknown, the parasite could not be assigned to a family and was, therefore, provisionally retained within the 'collective group' *Microsporidium* of Sprague, 1978 (J. B. Jones, 1981).

Sporogonial plasmodia of an unidentified microsporan, with spores measuring about 2.5×1.3 µm, have been observed in epithelial cells of the digestive gland of *Cardium edule* from Auray (France). The parasite appears to be of rare occurrence; its pathogenicity has not been studied (Comps and co-authors, 1975). Another specifically unidentified microsporan infests the ova of *Macoma baltica*. About 20 % of the clams from Tred Avon River (Maryland) were found to harbour this parasite (Farley, 1977). Microsporans of the (heterogenous) genus *Nosema* are known as hyperparasites of larval trematodes infesting marine molluscs (see section 'Agents: Trematoda'). Species of that genus infesting arthropods have been transferred to the genus *Ameson* by Sprague (1977).

A' protistan *incertae sedis*' has been observed in the cytoplasm of maturing ova of individuals of *Crassostrea gigas* from Humboldt Bay (California). Earliest observable stages were solitary, unencysted, uninucleate amoebulae. Division occurs within a cyst, primarily by successive division into about 8 to 16 advanced stages. No plasmodia were seen. Later stages comprised ovoidal to spherical cysts with nearly indistinct membranes, 4.0×5.0 to 18.0×20.0 µm in size. The parasite superficially resembles *Steinhausia ovicola* and *S. mytilovum* but does not possess a multinucleate stage, or form mature spores (Becker and Pauley, 1968). The authors discussed its affinities to the Microspora and the 'Haplosporida' but were unable to assign it to either of these groups.

Closer examination of bivalves for the presence of Microspora will most likely reveal, in the future, the existence of further species. A certain handicap is the small size and

inconspicuousness of these intracellular parasites. Detection of live spores requires observation with a phase-contrast microscope at a magnification of 600 to 1,000 ×. Once spores are exposed to tissue fixatives, identification becomes more difficult. Palmieri and Sullivan (1977) have described a technique for the location of spores in host tissues, by means of which Microspora can be differentiated from molluscan and trematode tissues at magnifications as low as 100 ×.

Agents: Ascetospora (*Marteilia* and the 'Haplosporidians')

As a result of recent revisions of protozoan classification, particularly of the Haplosporida (Sprague, 1979; Levine and co-authors, 1980), the new phylum Ascetospora has been created. Representatives of this group occurring in marine bivalves are all members of the new class Stellatosporea. These are *Marteilia* (Marteiliidae, in order Occlusosporida) and *Haplosporidium* and *Urosporidium* (Haplosporidiidae and Urosporidiidae, respectively, in order Balanosporida). The exact systematic position of *Bonamia*, the most recently discovered ascetosporan, has not yet been determined. It is probably related to the Balanosporida (Table 13-3). All ascetosporans are serious pathogens.

From 1968 on, recurrent serious mortalities have been recorded in *Ostrea edulis* populations in France, particularly in the region of Marennes on the Atlantic coast and in Bretagne. The disease syndrome became soon known as 'Maladie des Abers', named after a locality in Bretagne where unusual oyster mortalities were first experienced, and was later named 'Maladie de la glande digestive' (Digestive-gland disease), after the main infestation site in the oyster. The causative agent was first seen and superficially described by Comps (1970b), who discussed its affinities with the haplosporidians. Herrbach (1971a) hesitatingly assumed that it might be a fungal member of the Chytridiales. Grizel and Tigé (1973) believed it to be a member of the genus *Labyrinthomyxa*. Grizel and co-authors (1974a), who studied the organism in detail and named it *Marteilia refringens*, tended to relate it to the Microspora. Perkins (1976c), on the other hand, grouped it with the Haplosporea (now Balanosporida). Considering the arguments of Desportes and Ginsburger-Vogel (1977), Sprague (1979) eventually removed *M. refringens* from the latter group and assigned it to the newly created order Occlusosporida.

Marteilia refringens has an unusual life cycle previously not observed in protozoans. Its sporulation involves a series of endogenous buddings that produce sporoplasms within sporoplasms. Mature spores have an entire wall and do not possess an operculum covering an orifice as do the Balanosporida. The various stages of *M. refringens* have been studied intensively by means of electron microscopy (Grizel and co-authors, 1974a, b; Perkins, 1976c). Only a brief summary of the complex picture can be given here. For detailed information the reader is referred to the above papers, as well as to those of Bonami and co-authors (1971) and Herrbach (1971a).

Presporulation, vegetative stages of the parasite consist of ovoidal plasmodia found in the lumina of digestive–gland tubules and intestine, between the epithelial cells of the tubules and intestine, and in the connective tissue surrounding both structures. These small (5 to 8 μm) plasmodia, which are delimited by a plasmalemma with no cell wall or fibrillar layer, have been termed 'cellules primaires' ('primary cells') by Grizel and co-authors (1974a).

Sporulation commences with the delimitation ('endogenous budding') of uninucleate

segments within the plasmodial cytoplasm, which become presporangia or sporangial primordia ('secondary cells'), about 8 μm in longest axis. At this stage, the plasmodium corresponds to a sporangiosorus. As sporulation progresses, the sporangiosorus enlarges from about 15 to 30 μm. About 8 sporangial primordia are formed within each sporangiosorus. A single nucleus (sometimes 2) remains in the plasmodial cytoplasm ('primary-cell nucleus') and is not included in the sporangial primordia. A wall is then formed around each developing sporangium, followed by nuclear multiplication and cytoplasmic cleavage, which eventually results in the formation of 3 or, more often, 4 spore primordia ('tertiary cells'). Each spore primordium consists of 3 uninucleate sporoplasms of graded sizes. The smallest lies in an eccentrically located vacuole of the medium-sized one, the medium–sized one lies in an eccentrically situated vacuole of the largest sporoplasm. As spore maturity is attained, the sporangial cytoplasm not included within the spore walls degenerates, as does the 'primary-cell nucleus'. Polymorphic inclusion bodies, 2.3 to 4.0 μm in longest axis, appear in the extraspore cytoplasm during spore maturation, eventually becoming a dominant feature of the sporangium. When viewed with light optics, these inclusion bodies are highly refringent, thus the species name. Mature spores of *Marteilia refringens* are spherical to subspherical in shape, with a diameter ($\bar{x} \pm$ S.E., n = 22; living cells, interference contrast optics) of 3.9 ± 0.08 μm. Nuclear diameters, as seen in electron micrographs, range from 1.02 to 1.52 ($\bar{x}$ = 1.24, n = 19) μm (Perkins, 1976c; Fig. 13-33).

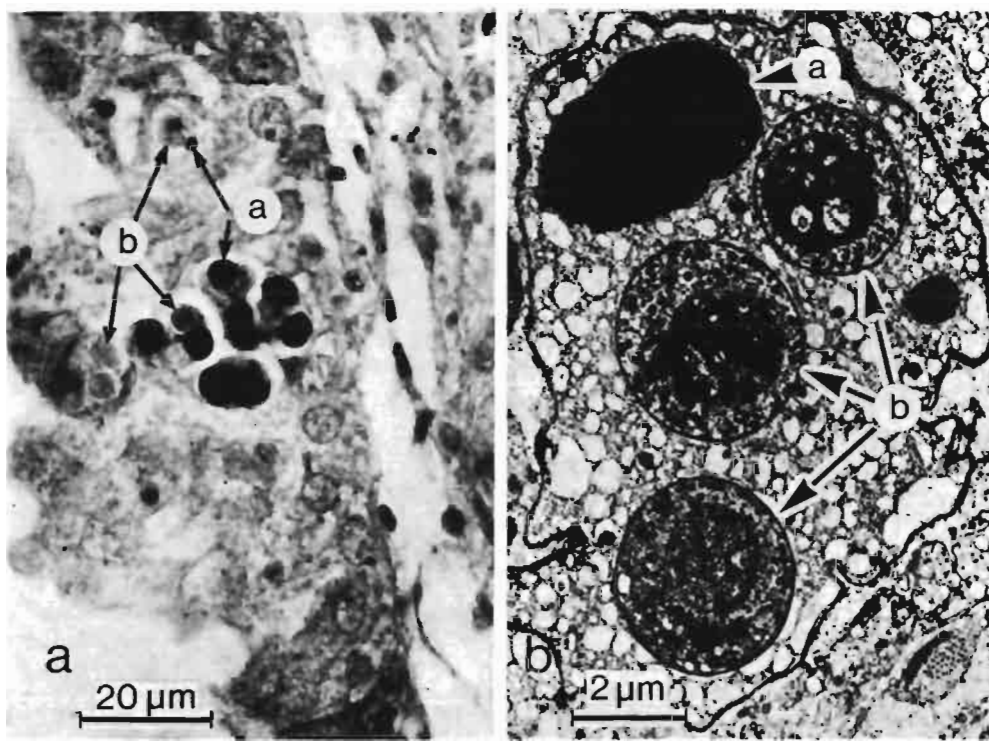


Fig. 13-33: *Marteilia refringens*. (a) Mature sporangium in digestive-gland tubule of *Ostrea edulis;* (b) electron micrograph of sporangium with 3 fully formed spores (b) and 1 refringent inclusion body (a). (After Bonami and co-authors, 1971.)

From the occurrence of the various developmental stages in different tissues of *Ostrea edulis*, Grizel and co-authors (1974a) inferred a tentative life cycle for *Marteilia refringens* (Fig. 13-34). Primary infestations presumably occur in epithelia of the gut and/or the gills. Sporangia mature in the digestive diverticula and are discharged via the gut. How the oyster becomes infested, and by which stage, has not yet been determined. Experimental

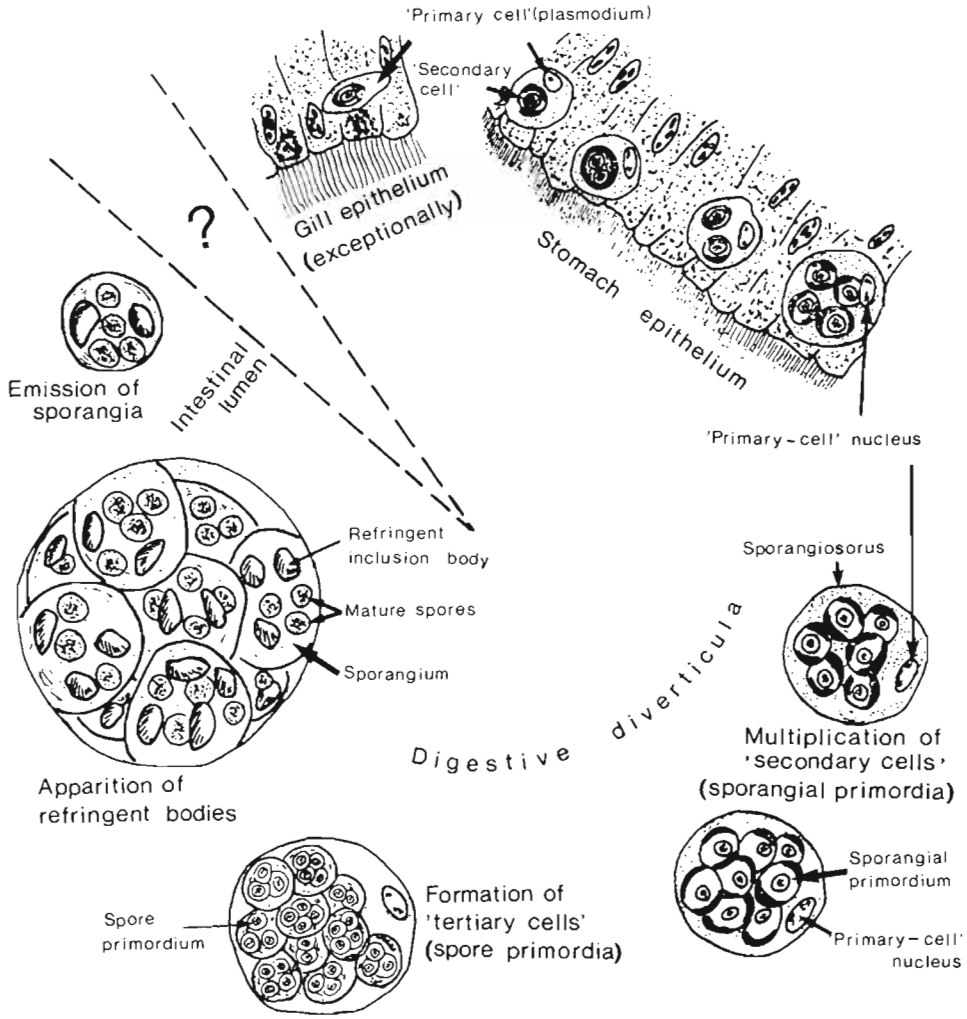


Fig. 13-34: *Marteilia refringens*. Life-cycle stages observed in *Ostrea edulis*. Presumed life cycle. (After Grizel and co-authors, 1974a.)

attempts to transmit the disease to healthy oysters in the laboratory have met with failure, although field experiments were successful.

Recent evidence suggests that the life cycle of *Marteilia refringens* may be even more complex than outlined by Grizel and co-authors (1974a). Franc (1980) maintains that the 'primary', 'secondary' and 'tertiary' cells of Grizel and co-authors (1974a) represent 3 independent categories of cells, which may be part of several subcycles in the development of the organism. The author observed stages resembling schizogony, as well as small

amoebulae, 4 to 5.5 μm in size, which hatched from sporangia of a certain type. On the surface of the epithelium of mouth and oesophagus, uninucleate cells, 9 to 13 μm in diameter, were seen in different stages of fusion. Paired cells were different in size. These and comparable stages, which are suggestive of a sexual phase, were also seen in the digestive gland. Furthermore, a new type of sporangium — believed to be derived from one of the 3 distinct cell categories — was seen in the visceral connective tissue, "à bonne distance" of the digestive diverticula and the intestinal tract, i.e., the sites harbouring the hitherto known stages of the parasite. The presence of at least some of these stages appears to depend on the season. These highly interesting observations have been made with light optics and should be confirmed by electron microscopy in the future. It appears likely that *M. refringens* will hold good for further surprising discoveries. More detailed insight into its life history might even place *M. refringens* in another taxon.

'Aber disease' is accompanied by severe pathology. Infested oysters become progressively emaciated and the digestive gland assumes a brownish to pale yellow colour. Upon deprivation of its glycogen reserves, the mantle becomes transparent and shell growth ceases. The visceral mass loses its pigmentation and, in heavily infested individuals, appears shrunken and 'slimy'. First clinical signs become apparent in oysters in the autumn of the year of planting and aggravate until death ensues in the autumn of the following year. These signs, however, are not specifically diagnostic, since they may also be exhibited by oysters not suffering from Aber disease. On the other hand, *Marteilia refringens* may also be found in apparently healthy, well–growing individuals with normal gonads. The factors governing or influencing the virulence of *M. refringens* are not yet understood (Grizel and co-authors, 1974a; Grizel, 1977; Cahour, 1978).

Recordings of the valvular activity of oysters by means of an 'ostreograph' revealed abnormal periods of shell closure in *Marteilia refringens*-infested individuals. First signs of abnormal activity coincided with the appearance of parasite stages in the digestive gland. In the terminal disease stage, the oysters are no longer capable of closing their valves (His and co-authors, 1976; Grizel, 1977).

The severe effect of *Marteilia refringens* and its bearing on the oyster industry are best illustrated by comparison of the total wet weights of affected and healthy individuals of *Ostrea edulis*. Lots of 1,000 oysters each have been weighed on March 26 and November 15, 1974. The respective figures were 16 and 31 kg for 18-month-old, healthy oysters, and 11 and 23 kg for diseased individuals of the same age. Two-year-old healthy oysters weighed 36 and 53 kg, while infested ones weighed only 29 and 34 kg (Morel and Tigé, 1974).

Initially, *Marteilia refringens* was held responsible for causing 'affection branchiale' in *Ostrea edulis*, a syndrome similar to 'gill disease' in *Crassostrea angulata*, caused by a virus (see section 'Agents: Virales'; Comps, 1970b). With a single exception, *M. refringens* was never found again in the gills of numerous individuals of *Ostrea edulis* examined over a period of several years. 'Affection branchiale' must, therefore, have another etiology (Grizel and co-authors, 1974a; see section 'Agents: Protista incertae sedis').

Kills from Aber disease usually commence in May, attain their maximum in June to August, and then gradually diminish, with decreasing losses persisting until December or January. From March to April, the parasite is quiescent. In southern Bretagne oyster beds, *Marteilia refringens* could not be found during this period, while in the colder waters of northern Bretagne, subclinical infestations persisted throughout the winter. From trans-

plantation experiments it became evident that new infestations are acquired between May and August (Grizel and Tigé, 1973, 1979). Long-term observations suggest that old mature sporangia containing refringent inclusions are present from May or June on, but are eliminated completely by the end of January, whereas young plasmodia persist during the winter and re-initiate new clinical infestations in the following May (Grizel, 1976; Cahour, 1978; Balouet and co-authors, 1979).

Incidences of *Marteilia refringens* infestation ran as high as 100 % in oysters from Aber Wrach and Aber Benoît, Bretagne; similarly high prevalences have been reported from other localities in France, while some areas have, thus far, remained unaffected (Bonami and co-authors, 1971; Balouet and co-authors, 1979). Initially, only *Ostrea edulis* was found to be attacked; *Crassostrea angulata* and *C. gigas* were examined with negative results. Therefore, the parasite was considered to be specific to *O. edulis* (Grizel and Tigé, 1973; Marteil, 1976). In May 1977, however, Balouet and co-authors (1979) detected young stages of *M. refringens* in individuals of *C. gigas* from Carantec near Roscoff (France). These findings were interpreted as a change in susceptibility of this oyster species.

During a search for possible alternate hosts expected to harbour unknown life-cycle stages of *Marteilia refringens*, Comps and co-authors (1975) found stages in 2 % of *Mytilus edulis* from Auray and Penzé, as well as in 10 % of *Cardium edule* from Auray. Gutiérrez (1977b) observed a 3.3 % infestation in 92 *M. edulis* from Arosa on the northwest coast of Spain. Stages of the parasite were easily detected histologically by a modified staining technique (Gutiérrez, 1977c). While the forms found in the mussels were indistinguishable from those in oysters, the cockles harboured, in addition to sporangia ('tertiary cells'), amorphous, proteinaceous, spherical masses, 0.3 μm in diameter. Corresponding forms found in *Ostrea edulis* measured 3 to 4 μm. While the latter may be identical with the amoebula described by Franc (1980; see above), the significance of the former remains obscure. It appears possible that the parasite found in *C. edule* is a distinct species of *Marteilia*. Its effects on the cockle have not been studied, but it was noticed that there were unusually high numbers of empty shells on the beds where the parasite occurred (Grizel, 1977).

'Virus-like particles', observed in the parasite in the cockle, appear to be identical with those previously reported by Bonami and co-authors (1971) and Grizel and co-authors (1974a) from *Marteilia refringens* in *Ostrea edulis*. These particles probably represent 'haplosporosomes', unique cytoplasmic organelles of unknown significance found exclusively in members of the Stellatosporea (Perkins, 1976c). Although Comps and co-authors (1975) and Gutiérrez (1977b) have shown that *M. refringens* can exist in *M. edulis*, the observations of Tigé and Rabouin (1976) suggest that the mussel is merely an accidental host for the oyster pathogen. It appears that the presence of *M. refringens* does not affect the health of mussels (Grizel, 1977).

The obvious lack of consistent correlation between the degree of *Marteilia refringens* infestation and *Ostrea edulis* mortality is one of the most puzzling facts. Oysters kept in high-prevalence areas for extended periods of time may show the characteristic disease signs without yielding a notable number of parasites. Inversely, oysters heavily infested with young plasmodia, as well as with mature sporangia, may exhibit virtually no histological alterations. Therefore, the mode of action of *M. refringens* remains obscure. As one possible explanation, the production of toxins, by the parasite, has been taken into

consideration. It is also possible, that the occlusosporidan acts in concert (synergistically) with other, yet unidentified pathogens. Environmental conditions (stress) may also play a prominent part (Cahour, 1978; Ormières and Grizel, 1979).

High mortalities have also been reported from *Ostrea edulis* imported into Spain from France. In 1974-75, losses in some rias (estuaries) on the northwest coast amounted to 50 to 70 % and were most serious in areas where *O. edulis* and *Mytilus edulis* were grown in close proximity. Microscopic examination, however, revealed the presence of *Marteilia refringens* in only a small fraction of the affected oysters. It was concluded that poor adaptation of the seed oysters to the physical environment of the planting areas was the main reason for the high mortalities (Massó Bolivár, 1978). In the Netherlands, *M. refringens,* introduced with seed oysters imported from Bretagne, failed to demonstrate any significant virulence. Although the parasite stayed alive and 'in good condition' in imported and replanted oysters, it did neither infest the disease-free stock of native Zeeland oysters nor did it cause abnormal mortalities among the imported lots (van Banning, 1979a, b). The differences in apparent virulence displayed by *M. refringens* in French and Dutch waters are unexplained. It appears that much remains to be learned about the biology and epizootiology of this presumed oyster pathogen.

Three other species of *Marteilia* have, thus far, been reported from oysters. The first, originally reported by Wolf (1972) as an unnamed haplosporidian from Sydney rock oysters *Crassostrea commercialis (= Saccostrea cucullata)* in Moreton Bay, Queensland (Australia), was later identified as a member of the genus *Marteilia*, named *M. sydneyi* (Perkins and Wolf, 1976). Its developmental sequence, infestation site and size are essentially the same as in *M. refringens,* with the exception that the mature spores of *M. sydneyi* are surrounded by a heavy layer of concentric membranes, which are lacking in *M. refringens.* The pathogen has been found only in subtropical and tropical regions of the eastern Australian coast, not in the colder waters south of Richmond River. An organism, probably identical with *M. sydneyi,* occurs in *C. echinata* from the same area.

Marteilia sydneyi has been recovered from 1 moribund oyster and from 4 oysters which were in poor condition. While the moribund specimen contained numerous spores, fewer were found in the others. High oyster mortalities occurring in Moreton Bay were believed to be caused by this occlusosporidan. Its incubation period is less than 60 days from early infestation to death of the host. *M. sydneyi* appears to prefer lower salinities, at least for a part of its development. Higher growing levels in the intertidal zone will avoid invasion, or at least reduce pathogen numbers. Up to 80 % of the oysters in southern Queensland and northern New Wales are lost during severe epizootic outbreaks of this pathogen (Wolf, 1972, 1979).

Marteilia lengehi parasitizes *Saccostrea cucullata* in the Persian Gulf. Plasmodia, 8 to 15 μm in diameter, and presporulation stages were found in the stomach epithelium and the digestive gland. Mature spores were not seen (Comps, 1976a). The third named species of *Marteilia* is *M. maurini,* found in *Mytilus galloprovincialis* imported into France from the lagoon of Venice, Italy. Its morphology and developmental sequence closely resemble those of *M. refringens* (Comps and co-authors, 1982).

Several members of the classical order Haplosporidia are known to parasitize marine bivalves. There has been much interest in this group during the past decade, mainly because it contains species which are highly destructive pathogens of oysters. Electron microscopic studies by Perkins (1968b, 1969a, 1971, 1975, 1979), Rosenfield and co-

authors (1969) and Perkins and co-authors (1977) have contributed much to our understanding of the haplosporidians. As a result of several revisions of the group (Sprague, 1963, 1966, 1978), a classification scheme has been proposed (Sprague, 1979), which has been adopted by the 'Committee on Systematics and Evolution of the Society of Protozoologists' in their 'Newly revised Classification of the Protozoa' (Levine and co-authors, 1980). As shown in Table 13-3, the ordinal name 'Haplosporida' has been replaced by the newly created 'Balanosporida', which includes the families Haplosporidiidae (with *Haplosporidium*) and Urosporidiidae (with *Urosporidium*). The latter genus contains species which are hyperparasites of larval trematodes and a nematode parasitizing marine bivalves.

Haplosporidians are cytozoic and histozoic parasites. Details of their life cycles are largely unknown. Infestation of the invertebrate host is believed to be accomplished by characteristic operculate spores, which lack internally coiled polar filaments. Amoebulae, released by the spores, undergo a modified type of schizogony, which gives rise to multinucleate plasmodia. These develop into sporonts, which cleave into uninucleate portions and eventually differentiate into spores. The sporangium enclosing the spores is known as the sporocyst. However, other routes of transmission must exist, at least in species of *Haplosporidium* infesting oysters, since massive infestations are frequent although sporulation is extremely scarce.

The first named balanosporidan parasitizing marine bivalves is *Haplosporidium costale*; it was described from *Crassostrea virginica* by Wood and Andrews (1962). Unfortunately, Sprague (1963) transferred it, together with several members of the genus described earlier, to the restored genus *Minchinia*. For reasons outlined in Chapter 14, p. 964, this transfer is unjustified, as is the assignment of subsequently described haplosporidians to that genus. For quite other reasons, Sprague (1978) retransferred all of these to *Haplosporidium*.

Haplosporidium nelsoni and *H. costale* are known to cause serious diseases and extreme commercial losses among *Crassostrea virginica* populations on the North American Atlantic coast (Fig. 13-24). Both were first recognized in the late fifties and have since been studied in great detail. An immense body of literature on these two haplosporidians has accumulated. Recent accounts of the epizootiology and reports of long-term observations of *H. nelsoni* and *H. costale* have been presented by Andrews and Frierman (1974), Farley (1975), Andrews (1976a, 1979), Haskin (1976), Kern (1976a), Andrews and Castagna (1978), and others. Ford and Haskin (1982) and Haskin and Ford (1982) discuss the results of long-term studies (1957 to 1980) of *H. nelsoni* in the New Jersey portion of Delaware Bay. In spite of these gigantic efforts, the life cycles, mode of infestation and general biology of both pathogens remain largely obscure — after more than 24 years of intensive research.

In the spring of 1957, about 50 % of the *Crassostrea virginica* planted on New Jersey oyster grounds in Delaware Bay died within 6 weeks. In the fall, the overall mortality at the centre of the outbreak approached 85 %. Histological examination revealed a protistan previously unreported from *C. virginica*. Initially, it was referred to as 'MSX' (multinucleate sphere X) from the spherical shape of the plasmodium, and the disease syndrome became known as Delaware Bay disease. Later, the causative agent was identified as a haplosporidian, originally named *Minchinia nelsoni* (Haskin and co-authors, 1965, 1966).

The first appearance of Delaware Bay disease in Chesapeake Bay in 1959-60 dramatically changed the whole industry of oyster culture in that area. MSX rapidly replaced and displaced *Perkinsus marinus* as the major cause of oyster mortalities in the bay. *Haplosporidium nelsoni* infestations spread throughout lower Chesapeake Bay in 1 year (1960), affecting oysters over long distances from a Mobjack Bay focus in 1959. As a

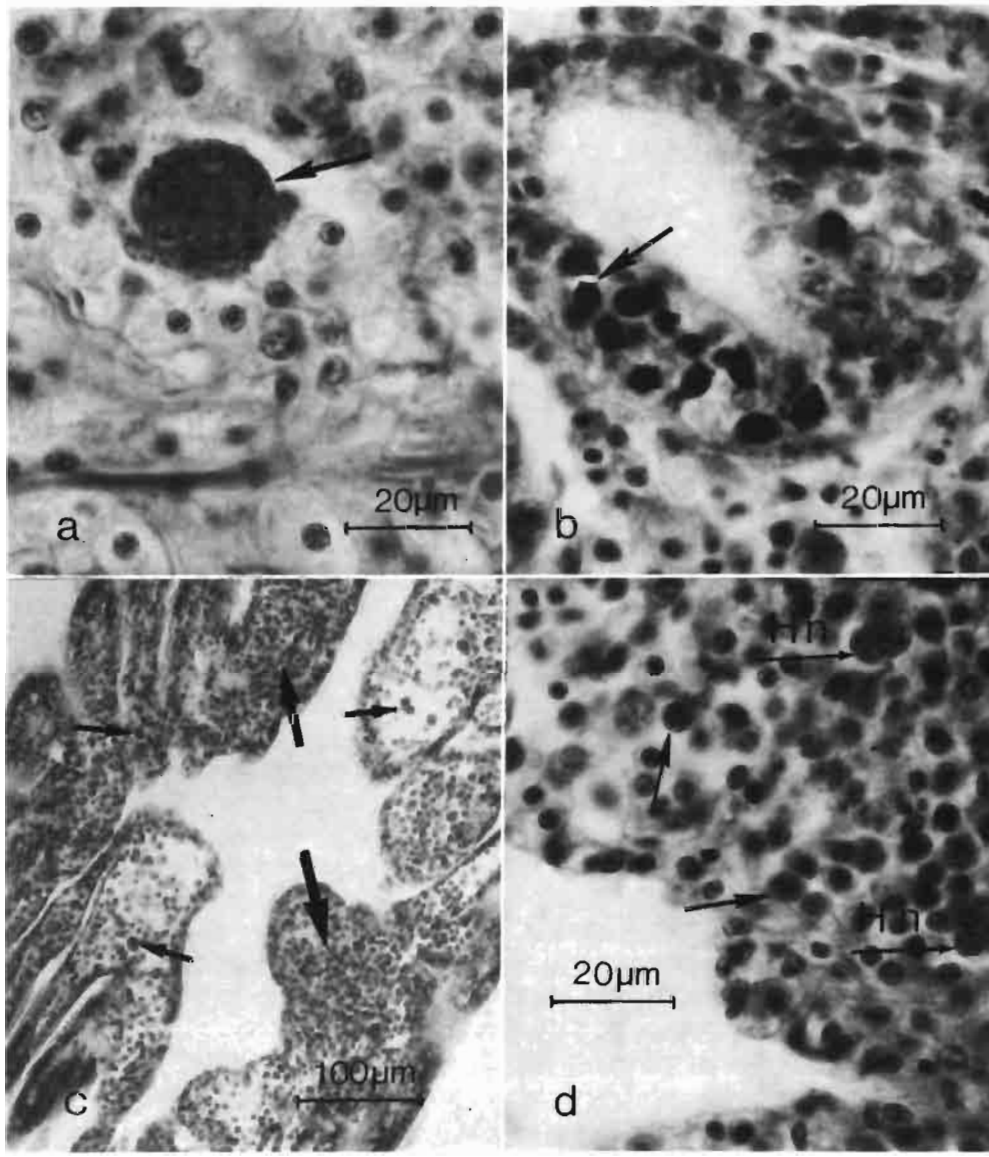


Fig. 13-35: *Haplosporidium nelsoni* in *Crassostrea virginica*. (a) Multinucleate spherical plasmodium; (b) acid-fast operculate spores in tubule of digestive diverticula; (c) oyster-gill filament infested with *H. nelsoni* plasmodia (small arrows) showing massive haemocytic infiltration (large arrows); (d) gill lesion at higher magnification showing accumulation of hyaline haemocytes (arrows) in response to *H. nelsoni* (Hn). (After Kern, 1976a.)

consequence, previously flourishing oyster beds collapsed, and after 1960, only trial plantings were made in high-salinity waters (> 15 ‰ S) (Andrews, 1966, 1968, 1979).

Perkins (1968b) and Rosenfield and co-authors (1969) provided detailed descriptions of the ultrastructure of the plasmodial and spore stages of the parasite. The multinucleate plasmodia, roughly spherical in shape and usually from 4 to 30 μm in diameter, occur throughout the gills, palps, suprabranchial chambers and Leydig tissues of *Crassostrea virginica* (Fig. 13-35, a). The number of nuclei, which measure from 1.5 to 7.5 μm in diameter, may vary from 1 to more than 60 per plasmodium (Fig. 13-36, K). Sporulation stages are restricted to the epithelial layer of digestive tubules. Plasmodial and prespore stages predominate; sporocysts containing mature spores are extremely rare. It was concluded that *Haplosporidium nelsoni* is highly detrimental, since host death usually ensues before sporulation occurs (Andrews, 1968). Couch and co-authors (1966) first saw sporulation and spore stages in the digestive gland of oysters naturally infested with the plasmodial stage of the haplosporidian. Spores, measuring 7.5 × 5.4 μm unfixed, as well as prespore stages, were found in 12 of 266 oysters from Chincoteague Bay and adjacent Chesapeake Bay waters. This is an unusually high record, since Andrews (1979), who monitored the occurrence of *H. nelsoni* sporulation in oysters from Virginia waters of Chesapeake Bay, found only 44 cases of sporulation in 16 years, although about 10,000 oysters were examined microscopically each year. Fifteen oysters containing spores were resistants and 29 were susceptibles. Haskin and co-authors (1966) observed sporulation and mature spores in only 2 of over 10,000 oysters from Delaware Bay harbouring the plasmodial stage, which led them to suspect that these spores belong to another species of *Haplosporidium*. However, Barrow and Taylor (1966) demonstrated that a fluorescent antibody is produced against the plasmodia of *H. nelsoni*, which also reacts with the sporulating and spore stages present in *C. virginica*, indicating conspecificity of the stages. No such reaction occurred with any stage of *H. costale*. The rare occurrence of sporulation in *H. nelsoni* renders a transmission of Delaware Bay disease by spores unlikely. Moreover, the persistance of 'hot spots' for infestation in areas where oysters are sparse, the lack of spores in hosts infested with plasmodia, and failure to transmit the disease experimentally, led to the hypothesis that an alternate or reservoir host produces the infestive stages of MSX (Ford and Haskin, 1982).

Treatment of *Haplosporidium costale*-infested oyster tissues with a modified Ziehl-Neelsen carbol fuchsin technique results in a very specific staining reaction. All mature spores contain bright red sporoplasm, while spore walls and host tissue fail to retain any of the stain (Farley, 1965). No such acid-fast spore stages were seen in tissues of oysters infested with *H. nelsoni*. The author concluded that this could place the latter organism in another taxon. However, the apparent rareness of sporulation stages in *H. nelsoni*-infested oysters, as reported by Couch and co-authors (1966), Haskin and co-authors (1966) and Andrews (1979), may have accounted for the absence of such acid-fast stages from Farley's (1965) material. *H. nelsoni* spores were later found to stain similar to those of *H. costale* (Kern, 1976a; Fig. 13-35, b).

Gross signs of *Haplosporidium nelsoni* infestation (which are, however, unspecific) include mantle recession, gaping, emaciation, pale colour of the digestive gland and (as rare specific signs) the occasional formation of pustules on the oyster's inner shell surface. Microscopic signs include diapedesis, relative decrease in numbers of phagocytes, relative increase in numbers of hyaline haemocytes (Fig. 13-35, c, d), phagocytosis, fibrosis,

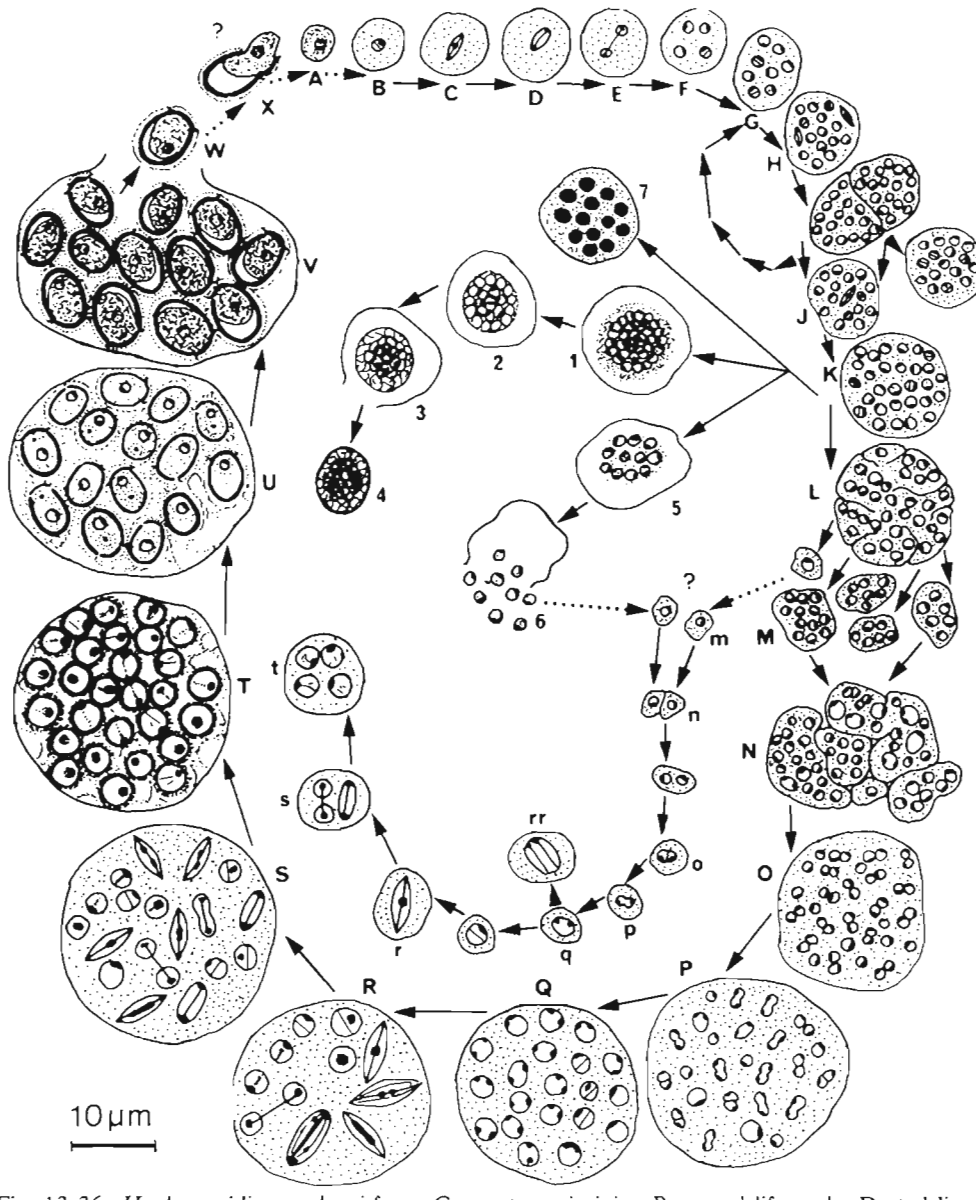


Fig. 13-36: *Haplosporidium nelsoni* from *Crassostrea virginica*. Proposed life cycle. Dotted lines: nuclear or unobserved (inferred) sequences. A–H: Early karyokinetic development of multinucleate plasmodium from uninucleate amoebula; I: plasmotomy producing daughter plasmodia (J); K: enlarged plasmodium; L: unequal plasmotomy forming gametic (?) plasmodia (M); N: aggregation and fusion of 'gametic' plasmodia; O: protozygote stage with paired nuclei; P: prozygote stage with fusing nuclei; Q: synkaryon stage with enlarged diploid (?) nuclei; R, S: first and second meiotic (?) divisions; T: early sporont; U: immature sporocysts containing 'haploid' spores; V: mature sporocyst and spores; W: free spore; X: escape of sporoplasm. 1–4: Encystment sequence. 1: Cytoplasmic contraction; 2: formation of dense outer coat; 3: rupture of plasmodial membrane; 4: mature cyst; 5, 6: release of nuclei. 7: Moribund plasmodium. m–t: Atypical sexual sequence and meiosis (?). m: 'Gametes'; n: fusion of 'gametes'; o: prozygote with paired nuclei; p: nuclear fusion; q: synkaryon with 2 endosomes; rr: anaphase figure exhibiting double endosomes and spindles; r: first division, metaphase; s: second division, possibly reduction; t: quadrinucleate, post-division plasmodium; this form, when found, usually appears to be degenerating. (After Farley, 1967.)

cellular infiltration, abscess formation, ulceration, excessive pigment cell formation, mechanical disruption, pyknosis and necrosis. Initial infestations occur in the gills and palps. The pathogen is thought to enter the host through the epithelia of the filtering organs. The intermediate disease stage is characterized by local infestation and infiltration of connective tissue in and adjacent to the epithelia of gills, palps, oesophagus, stomach, gut, digestive diverticula and gonadal alveoli. In advanced infestations, there is a general invasion and infiltration of the connective tissue and the circulatory system by hyaline haemocytes. Terminal infestations show histologically massive pyknosis of nuclei and necrosis of tissues before outward signs of death become apparent (Farley, 1965, 1968).

In contrast to 'Seaside disease', caused by *Haplosporidium costale* and characterized by a sharp seasonality and unique timing (Fig. 13-37), Delaware Bay disease kills oysters

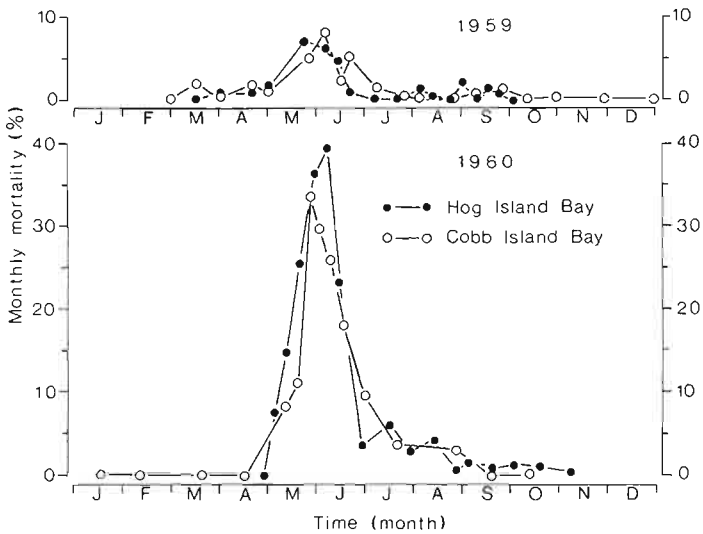


Fig. 13-37: *Crassostrea virginica*. Typical pattern of monthly death rates of oysters, resulting from *Haplosporidium costale* infestation. (After Andrews and co-authors, 1962; modified.)

throughout the year. June to July infestations become patent in about 5 weeks, and mortalities usually begin by August 1, reaching their peak in September. Infestation intensity may be very variable in gapers; therefore, gaping is not diagnostic. Most deaths due to *H. nelsoni* occur in the warm season, but another peak may result from death of weak oysters near the end of the winter, i.e., in March. However, prevalence changes do not appear to occur in the winter during dormancy of host and pathogen, except by deaths of weak oysters. Localized or subpatent infestations may remain 'hidden' for periods of up to 9 months (Andrews, 1977b, 1979).

All attempts to transfer *Haplosporidium nelsoni* to healthy oysters in the laboratory by placing infested and healthy oysters in the same aquarium or by transplantation of invaded tissues have failed so far. Likewise, routes of invasion and methods of transmission of the pathogen in the field, as well as its entire life cycle, remain unknown (Andrews and Frierman, 1974; Andrews, 1976a; Haskin, 1976; Ford and Haskin, 1982). It appears that long incubation periods may account for the failure of laboratory infestation experiments (Andrews, 1979).

On the basis of histological observations made during a 5-year study, Farley (1967) proposed a tentative life cycle for *Haplosporidium nelsoni*, involving hypothetical or poorly understood stages and sequences (Fig. 13-36). Other life-cycle patterns implicating additional hosts have been discussed. The prime questions about the life cycles of both *H. nelsoni* and *H. costale* are: (i) What is the source and the cellular form of the infestive particles? (ii) Does an alternate host exist? (iii) What is the true nature of the 'hidden infestations'? (iv) How may infestations be initiated artificially? (Andrews, 1979). None of these questions have been answered even remotely.

Some considerations which may indicate that no alternate or co-hosts are required are: General distribution of the haplosporidian in all moderately salty waters of lower Chesapeake Bay, implying a rather simple method of transmission; the long period of infestivity from May to November, suggesting no close timing with another host; and the apparent lack of any new immigrant host in Chesapeake Bay. Evidence suggesting involvement of another host includes: Failure to infest oysters experimentally under laboratory conditions; apparent absence of any relation between population size of oysters and the intensity of *Haplosporidium nelsoni* infestations; a steady level of the parasite's activity over wide areas, suggesting a rather stable source of infestive particles; and no apparent effect of proximity of infested oysters (Andrews, 1966, 1979; Ford and Haskin, 1982).

Although Andrews (1966), on the basis of a 10-year period of intensive oyster mortality studies, concluded that *Haplosporidium nelsoni* was new to Chesapeake Bay in terms of patterns of mortality, circumstantial evidence favours the view that the pathogen was present years before the epizootics (Andrews and Wood, 1967; Andrews, 1968).

Haplosporidium nelsoni is highly detrimental to *Crassostrea virginica*. Besides the histopathological changes observed by Farley (1965, 1968) and Haskin and co-authors (1965, 1966), massive biochemical alterations have been demonstrated. Thus, enzyme levels decrease significantly in heavily infested oysters. Particularly alkaline and acid phosphatase, as well as non-specific esterase, are reduced. Activities drop to near hibernation levels in the digestive gland. As a consequence, normal digestive physiology appears deranged (Eble, 1966 a, b). Malic dehydrogenase, a respiratory enzyme, was found to be reduced in mantle homogenates of *H. nelsoni*-infested *C. virginica* individuals, and haemolymph levels of phosphohexose isomerase activity were also significantly depressed (Table 13-4). Consequently, the normal glycolysis and glycogen synthesis could be blocked since the latter enzyme catalyzes the interconversion of glucose-6-phosphate and fructose-6-phosphate (Mengebier and Wood, 1967, 1969).

There appears to be a distinct correlation between changes in enzyme activity and the stage of the disease. Douglass and Haskin (1974, 1976) assayed the haemolymph levels of phosphohexose isomerase (PHI), alanine aminotransferase (AlAT) and aspartate aminotransferase (AsAT) in 4 categories of oysters, namely (1) normal, (2) individuals with prepatent lesions, (3) oysters with gill lesions and (4) individuals with generalized infestations. Contrary to the findings of Mengebier and Wood (1969), PHI levels showed a significant increase (+ 300 %) in Group 3 and a 270 % increase in Group 4. AlAT levels were elevated (+ 475 %) in Group 3 but normal in Group 4-individuals. AsAT activity increased (+ 890 %) in Group 3 but showed only a slight elevation in Group 4. In the remaining categories, respective enzyme levels in diseased oysters did not deviate significantly from those in Group 1 or in previously unexposed, introduced individuals kept as

Table 13-4

Crassostrea virginica. Seasonal phosphohexose isomerase (PHI) activities in haemolymph of healthy and *Haplosporidium nelsoni*-infested individuals. In brackets: number of individuals in test. PHI activity expressed as µg fructose formed in 30 min (After Mengebier and Wood, 1969; modified)

Time of sampling	Normal mean PHI values	Diseased mean PHI values	Significance level p (%)
Oct 1965	(10) 43.65 ± 11.20	(13) 12.53 ± 4.58	< 0.01
Dec 1965	(11) 49.36 ± 9.40	(25) 16.21 ± 2.80	< 0.01
Feb 1966	(30) 22.79 ± 3.45	(21) 12.93 ± 3.75	< 0.05
Apr 1966	(33) 38.29 ± 6.03	(37) 14.50 ± 3.36	< 0.01
Jul 1966	(46) 95.47 ± 3.24	(3) 69.91 ± 30.00	< 0.075
Oct 1966	(13) 35.00 ± 5.75	(5) 9.37 ± 6.40	< 0.05
Dec 1966	(18) 33.27 ± 3.55	(2) 15.00 ± 0	< 0.10

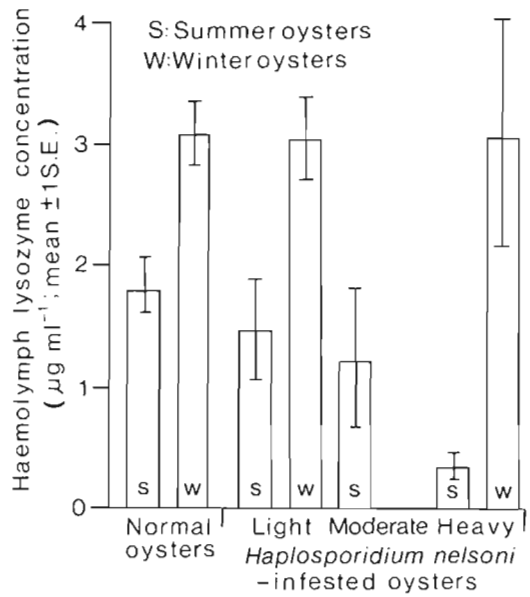


Fig. 13-38: *Crassostrea virginica*. Variations in oyster-haemolymph lysozyme activities associated with season and *Haplosporidium nelsoni* infestation. See also Fig. 13-79. (After Feng and Canzonier, 1970; modified.)

controls. The alterations of haemolymph enzyme activities were discussed in terms of host metabolism and possible humoural defense mechanisms.

Haemolypmh lysozyme activities in *Crassostrea virginica* exhibit a distinct seasonal pattern, being higher in winter than in summer. In *Haplosporidium nelsoni*-infested individuals, enzyme concentrations are significantly reduced during the summer but show little or no deviation from that of uninfested hosts in the winter when the parasite is presumably quiescent (Fig. 13-38). Polyacrylamide gel electropherograms of haemolypmh proteins revealed 1 major fast migrating anodal fraction (I) and 3 slow moving cathodal fractions (II, III, IV). Total haemolymph protein was not greatly altered in infested oysters but displayed significant increases in Fraction II with concurrent diminutions in Fraction

IV (Figs 13-39 and 13-40). The quantitative changes in fractions II and IV could be correlated with the severity of the infestations and were interpreted as evidence of host–humoural responses to the parasite (Feng and Canzonier, 1970).

Individuals of *Crassostrea virginica* from Navesink River, New Jersey, when exposed to *Haplosporidium nelsoni* infestation by transplanting them to Delaware Bay, showed consistently lower haemolymph-total free amino acid (FAA) concentrations than normal oysters. The differences between healthy and infested *C. virginica* reached a minimum during the winter, probably reflecting the relative quiescence of the pathogen (Fig. 13-41;

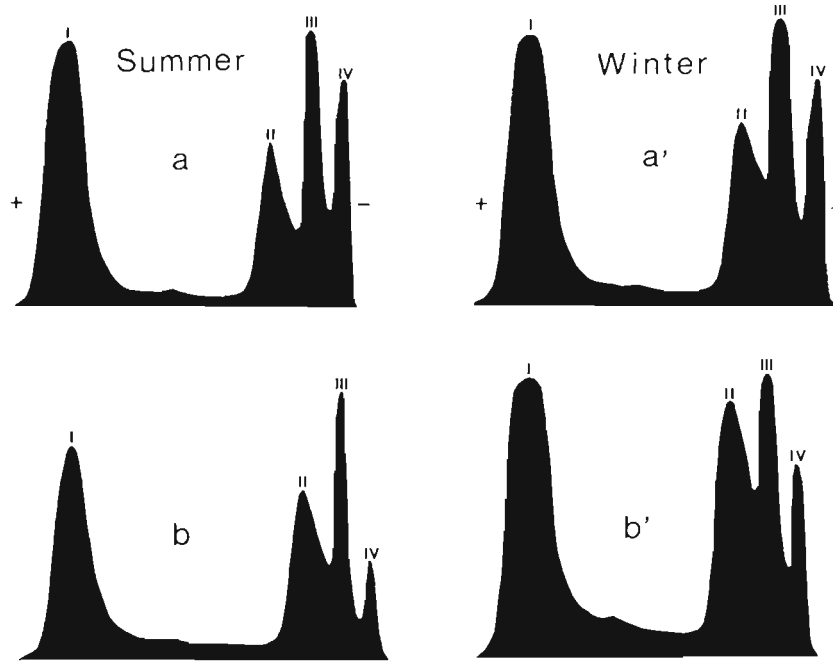


Fig. 13-39: *Crassostrea virginica*. Polyacrylamide gel electropherograms showing effect of *Haplosporidium nelsoni* infestation on haemolymph proteins. (a) Pool of 8 summer-uninfested oysters; (a') pool of 8 winter-uninfested individuals; (b) pool of 3 summer-*H. nelsoni*-infested oysters; (b') pool of 4 winter-*H. nelsoni*-infested individuals (Anode on the left, cathode on the right; see also Fig. 13-80). (After Feng and Canzonier, 1970.)

Table 13-5). Lowering of ambient temperatures may conceivably affect the rate of transport and utilization of host-FAA by the parasites. With respect to individual amino acids, summer depletion of free taurine, alanine, glycine, serine, phosphoethanolamine, β-alanine, ornithine, arginine, leucine and isoleucine was accompanied by a concomitant increase in free aspartic acid, glutamic acid, threonine and γ-aminobutyric acid (Feng and co-authors, 1970).

Salinity is an important factor controlling the distribution of *Haplosporidium nelsoni*. The agent occurs only in waters with salinities consistently in the range > 15 ‰ S < 25 ‰ S. Oysters living in estuaries with low salinities are essentially free from the pathogen. However, as a consequence of repeated droughts, which affected the mid–Atlantic coast during 1961-66, there has been a marked increase in salinity in several inshore regions with a resultant spread of *H. nelsoni* to these areas. In the James River oyster seed

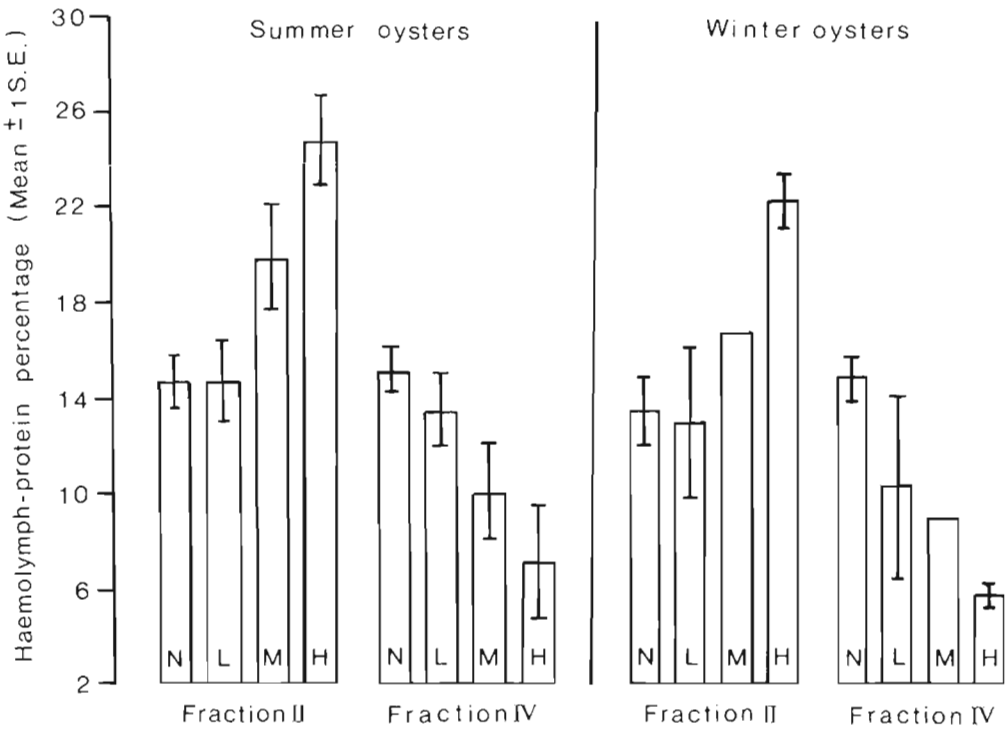


Fig. 13-40: *Crassostrea virginica*. Effect of *Haplosporidium nelsoni* infestation on haemolymph protein Fractions II and IV of normal oysters (N) and individuals with light (L), moderate (M) and heavy (H) infestations in relation to season. See also Fig. 13-81. (After Feng and Canzonier, 1970.)

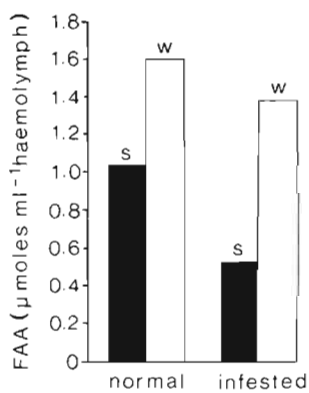


Fig. 13-41: *Crassostrea virginica*. Effect of *Haplosporidium nelsoni* infestation on haemolymph-total free amino acids (FAA). S: Summer concentrations, W: winter concentrations. See also Fig. 13-82. (After Feng and co-authors, 1970; modified.)

beds of Virginia, simultaneous monitoring of salinity, *H. nelsoni* prevalences and associated mortalities disclosed that infestations disappeared from areas of marginal disease occurrence during low seasonal salinities, as well as during several years of exceptionally high rainfall. *Crassostrea virginica* is capable of readily eliminating *H. nelsoni* at salinities

Table 13-5
Crassostrea virginica. Effect of *Haplosporidium nelsoni* infestation on concentration (nanomoles ml^{-1}) of free amino acids and related compounds in oyster haemolymph in relation to season. See also Table 13-12 (After Feng and co-authors, 1970; modified)

Amino acid or compound	Summer oysters		Winter oysters	
	normal	infested	normal	infested
Urea	725	460	–	438
Ammonia	520	219	299	589
Taurine	250	154	112	189*
Alanine	229	168	330	219
Glycine	124	56	109	108
Serine	120	26	87	93*
PE (footnote)	72	15	434	588*
β-alanine	71	18	104	74
Ornithine	69	30	111	53
PS (footnote)	62	–	142	10
Arginine	27	–	24	12
Lysine	20	–	17	43*
Glutamic acid	12	33*	114	14
Leucine	8	–	5	+
Isoleucine	3	–	6	+
GABA (footnote)	3	8*	11	10
Aspartic acid	+	32*	7	2
Threonine	+	4*	+	–
Methionine	–	–	+	+
Total amino acids	1072	544	1615	1418
No. of oysters	3	13	3	7

GABA γ-aminobutyric acid; PE phosphoethanolamine; PS phosphoserine; + trace quantities or less than 1 nanomole ml^{-1} haemolymph. * increases in amino acid and amine concentrations in infested oysters as contrasted with their appropriate controls. Histidine, proline, cystine, valine, tyrosine and phenylalanine not detected in samples.

below 10 ‰ S during the active growth period. In the high-salinity waters of the eastern shore of Virginia (> 30 ‰ S), infestations may occur, but oysters overcome these often and relatively few deaths result (Andrews, 1964, 1976a; Couch and Rosenfield, 1968; Andrews and Frierman, 1974; Andrews and Castagna, 1978). Salinity reduction has been found to entail a decrease in *H. nelsoni* infestations in oysters in the laboratory (Sprague and co-authors, 1969). On the basis of their intensive studies, Haskin and Ford (1982) concluded that salinity has little effect on the dispersal of the infestive stages of MSX or on their ability to infest oysters, but that it severely limits the parasite's capacity to develop once it has entered the host.

Temperature may also have some effect on the epizootiology of *Haplosporidium nelsoni*. Infestations persist through the winter, but death rates drop with decreasing temperatures in autumn and rise again in February to March. Peak mortalities and infestation of new hosts occur primarily during the warm season, but there is no abrupt onset of extreme mortalities, as is typical of *H. costale* infestations. Death of oysters from

H. nelsoni is probably attributable to the combined action of physical factors and the parasite (Andrews, 1966; Farley, 1975).

Delaware Bay disease, which sometimes caused mortalities in excess of 95 %, is by far the most serious protozoan malady of *Crassostrea virginica* along the mid–Atlantic coast of the United States. In the late 1940's and early 1950's, oyster landings in New Jersey waters of Delaware Bay had fluctuated around 6 million pounds of shucked meats. With the onset of the *Haplosporidium nelsoni* epizootic, landings fell precipitously to a low of 167,000 pounds in 1960, and have not recovered significantly (Fig. 13-42). Comparable effects

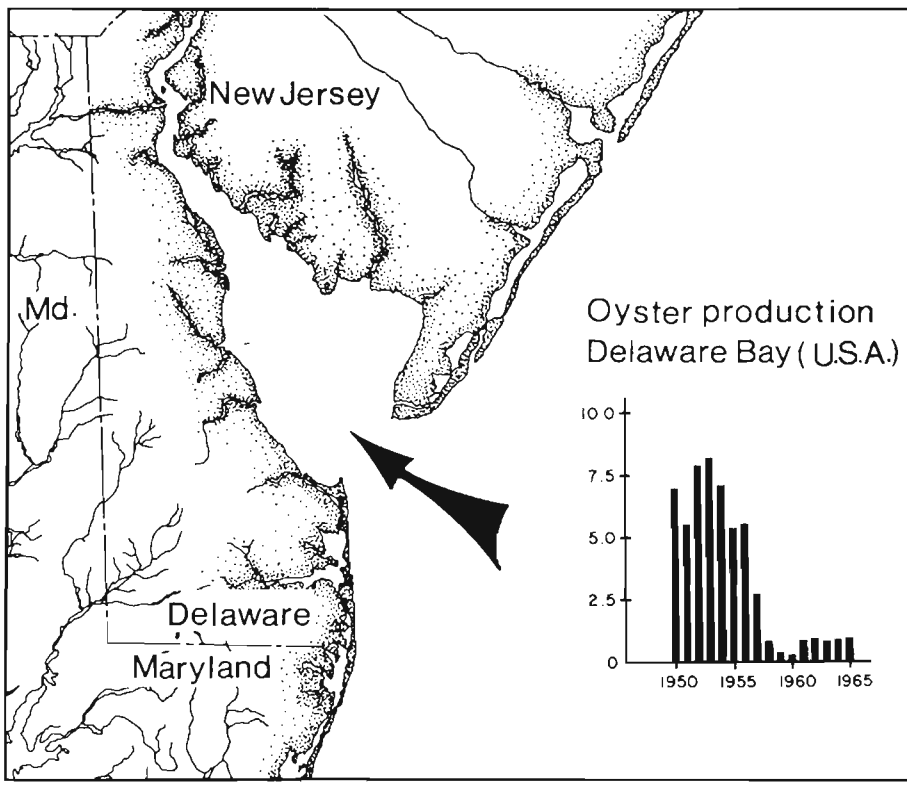


Fig. 13-42: *Crassostrea virginica*. Effect of *Haplosporidium nelsoni* infestation on oyster production in New Jersey waters of Delaware Bay, 1950–65. (After Sindermann, 1968a.)

have been experienced in the high-salinity areas of lower Chesapeake Bay, another major oyster-producing area. Millions of dollars worth of oysters were lost in 1960 alone (Andrews and Wood, 1967; Sindermann, 1968a, 1970a).

Prevalence of Delaware Bay disease and resultant losses have fluctuated widely since records commenced in 1958 to 1959. In a second high period, during the drought of the mid-sixties, prevalence peaks often reached 70 to 80 %, while during quiescence periods — in the early sixties and in 1971 — they were generally below 40 %. In 1972, MSX levels rose dramatically to new heights, with an average prevalence of over 70 % and a 50 % total mortality. The 1972-73 resurgence of *Haplosporidium nelsoni* persisted for several further years. With activity peaks in 1957–59, 1964–67 and 1972–75, interspersed with

periods of low prevalence in 1960–62 and 1971, there appears to be emerging an activity periodicity with peaks spaced to 7 to 8 years apart (Haskin, 1976).

During the period from 1963 to 1967, losses due to *Haplosporidium nelsoni* were high in all of Chesapeake Bay. In 1967, mortality and prevalence levels began to decline rapidly, and by 1970 the parasite had nearly disappeared from the Maryland portion of Chesapeake Bay. Currently, *H. nelsoni* is only found in a small area of Tangier Sound, where the microscopic appearance of the haplosporidian (moribund plasmodia, reduced infestation intensities and pronounced host response) suggests the development of a resistance by the oyster (Otto and co-authors, 1979).

In lower Chesapeake Bay, however, many ravaged oyster planting areas did not recover and remain barren. Nearly half of Virginia's pre-epizootic rented oyster grounds are no longer planted. Scarcity of oysters in epizootic areas has not reduced the activity level of *Haplosporidium nelsoni*, and oyster-population density or proximity have no effect on the pattern and development of Delaware Bay disease (Andrews and Frierman, 1974). There are marked epizootiologic differences between oysters native to disease areas and introduced juvenile, previously unexposed (to *H. nelsoni*) populations. Infestations in native individuals tend to be less serious, and frequently are delayed or attenuated, while those in introduced oysters progress to advanced and terminal stages (Farley, 1975).

Attempts to produce MSX-resistant oysters have been encouraging. Experiments with laboratory-reared progeny from susceptible (unexposed) and selected (by MSX) parents indicate that early exposure to *Haplosporidium nelsoni* appears to be important for subsequent survival of large oysters. Observed resistance appears to be both hereditary and acquired (Powell and Andrews, 1967; Andrews, 1968; Myhre and Haskin, 1970; Andrews and Frierman, 1974; Haskin, 1976). By selection of survivors from successively exposed generations of laboratory-reared oysters it was possible to obtain a 4-fold survival increase in first-generation resistants, a 5.5-fold increase in second-generation resistants and, as far as the limited data permitted to extrapolate, possibly a 6-fold increase in the third generation. The latter group had an overall survival of 50 %, as opposed by only 8 % in a group of susceptible, unexposed progeny believed to be closely comparable to the native susceptible Delaware Bay stocks present at the onset of the MSX epizootic in 1957 (Haskin, 1974). Most recent tests have shown that the 'resistance factor' of selected third-generation stocks may rise to as much as 7.5 times that of susceptible controls (Haskin, 1976).

The geographic range of *Haplosporidium nelsoni* now extends from southern Massachusetts to North Carolina (Sindermann and Rosenfield, 1967; Krantz and co-authors, 1972). *H. costale*, the second haplosporidian pathogen of *Crassostrea virginica*, has a more restricted distribution. It was initially referred to as 'SSO' (Seaside organism), because it occurred only on the seaside of Virginia and in Chincoteague Bay, Maryland, where salinities are higher than approximately 25 ‰ S (Fig. 13-24).

Earliest stages of *Haplosporidium costale*, observed in oyster-connective tissues, are small multinucleate (usually 4 to 12) plasmodia, more or less irregular in outline, and 6.1 × 7.8 μm in size. Mature sporocysts, 7 to 14 μm in diameter, each contain 20 to 50 spores, which measure 3.09 × 2.58 μm in fixed and 4.3 × 3.3 μm in fresh material (Wood and Andrews, 1962; Couch, 1967). In contrast to *H. nelsoni*, *H. costale* sporulates regularly during the May – June epizootics. All plasmodia sporulate synchroneously throughout the connective tissues of all organs. Infestations are intense at this time, and

tissues are riddled with sporonts and sporocysts. A typical disease syndrome of clustered cells and large sporulation stages gives a curdled appearance to tissues, which is readily recognizable at low magnification (x 100). Epithelia are usually not involved. Heavily *H. costale*-infested oysters have the tissues disrupted and essentially become sacks of whitish sporocysts. Sporulation results in immediate death of the host. If sporulation is not achieved, regression of plasmodia often ensues, but not after sporulation has begun (Andrews and Castagna, 1978; Andrews, 1979). Sporulation of *H. costale* has been studied in detail by means of electron microscopy (Perkins, 1969a). As in other haplosporidian parasites of bivalves, the fate of fully developed spores, as well as the mode of entry of the agent into the oyster, remain unknown.

Gross signs of 'Seaside disease' caused by *Haplosporidium costale* are gaping, emaciation, discolouration of the digestive gland, mantle recession and failure to add new 'bill' or shell in May. Epizootics, restricted to waters of high salinity (over 25 ‰ S), are characterized by sharp seasonality. The disease is first evident in live oysters in February. Prevalence of infestations in mid-May, when oysters begin to die abruptly, may run as high as 39 %. Mortalities peak in early June and stop early in July. During 2 weeks in June 1960, the death rate caused by *H. costale* exceeded anything ever experienced on the seaside of Virginia with other oyster pathogens. SSO mortalities are sharp but of short duration. By the end of June, the parasite lapses into obscurity for another year (Fig. 13-37). Losses are most serious in older oysters, which had been held beyond the usual period of culture. Yearlings tend to escape the disease, but 2- and 3-year-olds are usually severely affected. New infestations acquired in June remain subpatent without evidence of sickness or localized tissue affection until March of the following year. Oysters imported into epizootic areas in early August do not exhibit an epizootic until 22 months later (Andrews and co-authors, 1962; Wood and Andrews, 1962; Couch and Rosenfield, 1968; Andrews, 1977b).

Andrews and Castagna (1978) and Andrews (1979) have summarized their 19-year long-term studies (1959 to 1977) on the epizootiology of *Haplosporidium costale* infestations in *Crassostrea virginica* in seaside bays of the eastern shore of Virginia. SSO-caused death peaks could readily be identified by their unique timing. In this respect, Seaside disease differs markedly from Delaware Bay disease, which is irregular in level and timing of epizootiological events and causes deaths throughout the year. Interference of *H. costale*-caused losses with other diseases could practically be ruled out if mortalities did not extend beyond July 1. Years of high SSO mortality (50 % or over) alternated with long periods of the parasite's quiescence. Mortalities were higher in seed oysters imported from James River, Virginia, than in native oysters. It appears that the latter have developed, via selection, some degree of natural resistance against *H. costale* infestation, similar to oysters exposed to *H. nelsoni*.

The two *Haplosporidium* diseases are sympatric in seaside bays of Virginia and Maryland, although *H. nelsoni* is of rare occurrence there and plays a minor role, because salinities above 30 ‰ S inhibit this pathogen, while they seem to favour *H. costale*. In concurrent infestations, the plasmodial stages of both haplosporidians are difficult to distinguish with routine staining. However, eventually all *H. costale* plasmodia sporulate, as indicated by their absence in advanced infestations, whereas those of *H. nelsoni* remain as plasmodia when sporulation occurs. Moreover, the spores of MSX are about twice the size of those of SSO (Table 13-6), and all stages of MSX are larger than those of SSO.

Table 13-6
Haplosporidium spp. from marine bivalves (Original)

Parasite	Host	Spores	Sporulation site	Incidence	Host pathology	Locality	Source
Haplosporidium nelsoni	*Crassostrea virginica*	7.5 × 5.4 μm	Epithelium of digestive diverticula	Variable, up to 95 %	General emaciation, mostly unspecific signs; shell blisters (specific sign) rare	Chesapeake Bay	Couch and co-authors (1966), Haskin and co-authors (1966), Andrews (1977a, 1979)
Haplosporidium costale	*Crassostrea virginica*	4.3 × 3.3 μm	All connective tissues	80 % and over, mortalities 60 % and over	General emaciation, discolouration of digestive gland, cessation of shell growth	Chesapeake Bay to Delaware Bay	Wood and Andrews (1962), Andrews and Castagna (1978)
Haplosporidium sp.	*Crassostrea gigas*	Operculate; size interm. between *H. nelsoni* and *H. costale*	Leydig tissue	?	Recovered from gapers and 1 moribund oyster	Humboldt Bay, California	Katkansky and Warner (1970a)
Haplosporidium sp.	*Crassostrea gigas*	6.3 × 4.6 μm	Digestive diverticulum tubules	4 of 1,438	No gross pathology, no oyster mortality	Chung Mu, Korea	Kern (1976b)
Unnamed (*H. armoricanum?*)	*Ostrea edulis*	No spores seen, only plasmodia	Multinucleate plasmodia, 4 to 8 μm long	In 1 individual of sample of unknown size	Massive gill necrosis	La Tremblade, France	Comps (1976b)
Haplosporidium armoricanum	*Ostrea edulis*	5.0 to 5.5 × 4.0 to 4.5 μm with 2 projections 70 to 100 μm long	Connective tissues	3 of 3,700 4 of 5,400	General emaciation, shrunken meats	Oysters imported into The Netherlands from France	van Banning (1977, 1979a)
Unnamed haplosporidian	*Ostrea lurida*	No spores seen; only plasmodia	Plasmodia in Leydig tissue of visceral mass, mantle and palps	up to 5 %	Complete destruction of affected connective tissue	Yaquina and Alsea Bays, Oregon	Mix and Sprague (1974)
Haplosporidium tumefaciens	*Mytilus californianus*	8 to 11 × 5 to 8 μm	Digestive gland and kidney	23 of 1,114	Tumefactions in digestive gland and kidney	Little Corona Beach, Corona del Mar, California	Taylor (1966)
Haplosporidium tapetis	*Tapes decussatus*	12 × 5–7 μm	Gills	4 of 96	Reduced gonad development (?)	Portugal coast	Vilela (1951)
Unnamed haplosporidian	*Tresus capax*	No spores seen; only plasmodia	Plasmodia in cysts in mantle, siphon, gills, digestive gland, Leydig and muscular tissues	97 of 226	General emaciation, reduced gonad development	Yaquina Bay, Oregon	Armstrong and Armstrong (1974)
Haplosporidium sp.	*Teredo navalis, T. bartschi, T. furcifera*	6 to 8 μm long	All connective tissues, mainly gills	127 of 256 *T. navalis*	Considerable tissue damage	Barnegat Bay, New Jersey	Hillman (1978), Hillman and co-authors (1982)
Haplosporidium (*ascidiarum?*)	*Cardium edule*	10 × 7 μm	?	?	?	?	Harant (1931)

Couch (1967) reported concurrent infestations with *H. costale* and *H. nelsoni* in 6 of 101 individuals of *Crassostrea virginica* from Chincoteague Bay, Virginia.

Probably the most puzzling feature in the biology of *Haplosporidium costale* is the fact that the site of subclinical, 'hidden' infestations in the oyster has not been discovered. The earliest stage thus far observed are uninucleate cells in digestive-tubule epithelia. These appear very sparingly in June but do not appear to develop into systemic infestations until the following March (Andrews and Castagna, 1978). Although *H. costale* has not been recognized as a disease agent until the late fifties (Andrews and co-authors, 1962), recurrent oyster mortalities in Chincoteague Bay, dating back to the mid-forties, and perhaps earlier (Mackin, 1960), suggest a long-standing existence of the agent in Maryland and Virginia coastal waters. The known geographic distribution of SSO now extends from Long Island Sound to Cape Charles. Occasionally, oysters near the mouths of Delaware and Chesapeake Bays are affected. Probably, the agent has an even wider distribution (Andrews, 1977b). Katkansky and Warner (1970b) reported plasmodia, morphologically identical with those of *H. costale*, from *Crassostrea virginica* imported into California from the vicinity of New Haven, Connecticut, and held in Tomales Bay for periods of up to 18 months prior to sale. Sporulation was not observed in these individuals. Although the pathogen evidently could survive for extended periods of time on the west coast, infestations were not observed to spread to adjacent stocks of native *C. gigas*.

Several other species of *Haplosporidium*, as well as unidentified haplosporidians, have been described from marine bivalves. *H. tumefacientis* parasitizes *Mytilus californianus*. Of 1,114 mussels from Corona del Mar, California, 23 were infested (Taylor, 1966). Host specificity is indicated, since the organism was found in none of 1,000 *M. edulis* from the same area. Multinucleate plasmodia in the digestive gland and kidney of the host caused considerable swelling of the affected tissues. The life cycle of *H. tumefacientis* remains unknown. Attempts to infest *M. californianus* and *M. edulis* by injection of tumefaction homogenates met with failure. The fate of infested mussels in the field is not known but it is unlikely that the parasite may cause serious mortalities among *M. californianus*.

Haplosporidium tapetis has been found in 4 of 96 *Tapes decussatus* from Portuguese waters. Uni- and binucleate plasmodia, measuring 12 to 15 μm in diameter, occurred in the lumen (!) of the digestive tract. Multinucleate stages of approximately the same size were seen in connective tissues, and a larger (34 μm) sporogonic stage, as well as fully developed spores, 12 × 5 to 7 μm in dimension, occurred in the gill-connective tissue. The parasite appeared to have caused a delay in gonad development in one host (Vilela, 1951).

Unidentified haplosporidians were discovered in 13 of 314 Olympia oysters *Ostrea lurida* taken from Yaquina and Alsea Bays, Oregon (USA). This is the first report of a haplosporidian from *O. lurida* and perhaps any *Ostrea* species. Only plasmodial stages were seen. These were roughly spherical when small (5 μm in diameter) and slightly elongate when large (50 μm in length). Some plasmodia contained as few as 2 nuclei, 2 to 3 μm in diameter, but most had large numbers. The plasmodia were present throughout the Leydig tissue of the visceral mass, mantle and palps. Infested tissue, being lysed by the pathogen, was seen in various stages of disintegration and was entirely destroyed in the most heavily affected individuals (Mix and Sprague, 1974).

Haplosporidium armoricanum (originally referred to as *Minchinia armoricana*) affects *Ostrea edulis* imported into The Netherlands from Bretagne (France). Plasmodia (17 to 25 μm in diameter), sporonts (30 to 45 μm) and sporocysts (35 to 50 μm) containing 100

to 150 spores (5.0 to 5.5 × 4.0 to 4.5 μm), were present throughout the connective tissues. The outer spore wall has 2 characteristic long (70 to 100 μm) projections situated at opposite ends of the spore (Perkins and van Banning, 1981). Affected oysters showed a peculiar brown discolouration of the tissues and were thin and glassy looking and tough in consistency. Thus far, *H. armoricanum* appears to be of rare occurrence. During the study period (1974–77), only 4 of about 5,400 oysters were found to harbour the pathogen (van Banning, 1977, 1979a, b). Cahour and co-authors (1980) found *H. armoricanum* in 2 *O. edulis* individuals from Aber Benoît and Élorn, Bretagne.

In the course of recent mass mortalities of *Ostrea edulis* experienced at Saint-Philibert, French Atlantic coast, Vivarès and co-authors (1982) observed another new haplosporidian within the connective tissues of the gills and digestive gland of a single diseased oyster. Characteristic stages of the parasite are large plasmodia, approximately 20 μm in diameter, and smaller ovoidal forms, 4 to 5 μm in size (Fig. 13-43). Small

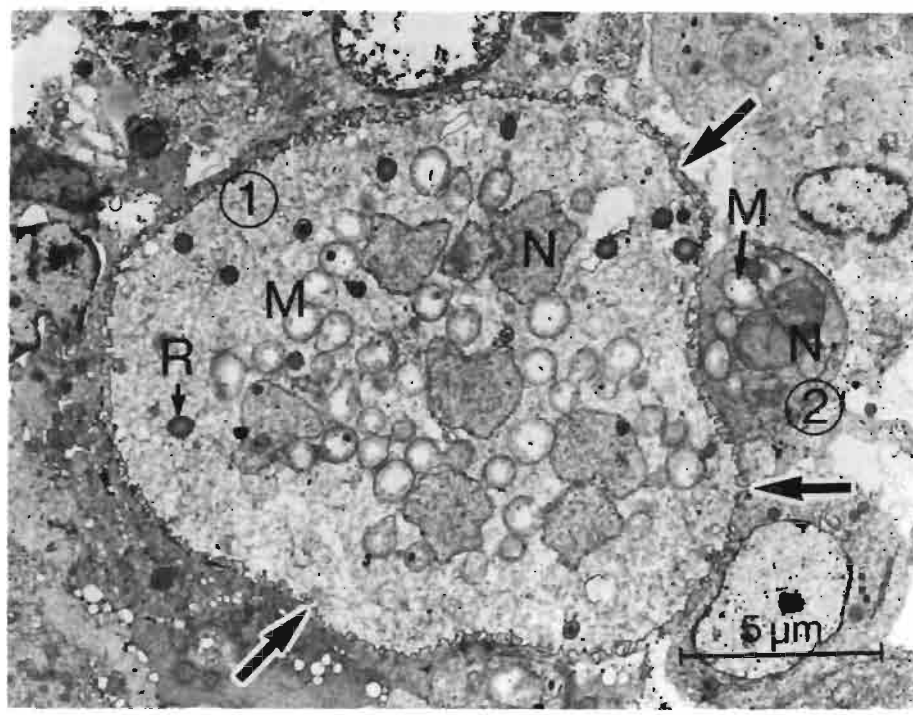


Fig. 13-43: *Haplosporidium* (?) sp. from *Ostrea edulis*. 1: 'Clear' plasmodial form; 2: 'dense' ribosome-rich, binucleate form. N diplokaryotic nucleus, M mitochondrion, R reserve body. Note indented plasmodium wall with external electron-dense layer (arrows). (After Vivarès and co-authors, 1982.)

plasmodia contain a single nucleus in diplokaryon stage, about 1.5 μm in diameter and 2.7 μm in length, while large ones have several diplokaryons (up to 6 per section), which measure approximately 5.2 × 2.3 μm. At the cell periphery the plasmodia contain haplosporosomes, about 320 nm in length and 130 nm in diameter. In addition, numerous plasmodia harbour, in their cytoplasm, sometimes large numbers of virus-like particles,

about 60 nm in diameter (Fig. 13-44). No typical spore stage of the parasite has yet been seen.

On gross examination, the affected oyster exhibited slightly indented gills while the visceral mass appeared normal. Histologically, however, there was an intense haemocytic infiltration of the connective tissue. According to Vivarès and co-authors (1982), this haplosporidian is distinct from *Haplosporidium armoricanum*.

Acid-fast spores, believed to belong to a haplosporidian of the genus *Minchinia* (= *Haplosporidium*), have been seen in a single, apparently moribund individual of *Crassostrea gigas* from Humboldt Bay, California (USA). The organism sporulated throughout the connective tissues of the oyster, as does *H. costale*, but was intermediate in

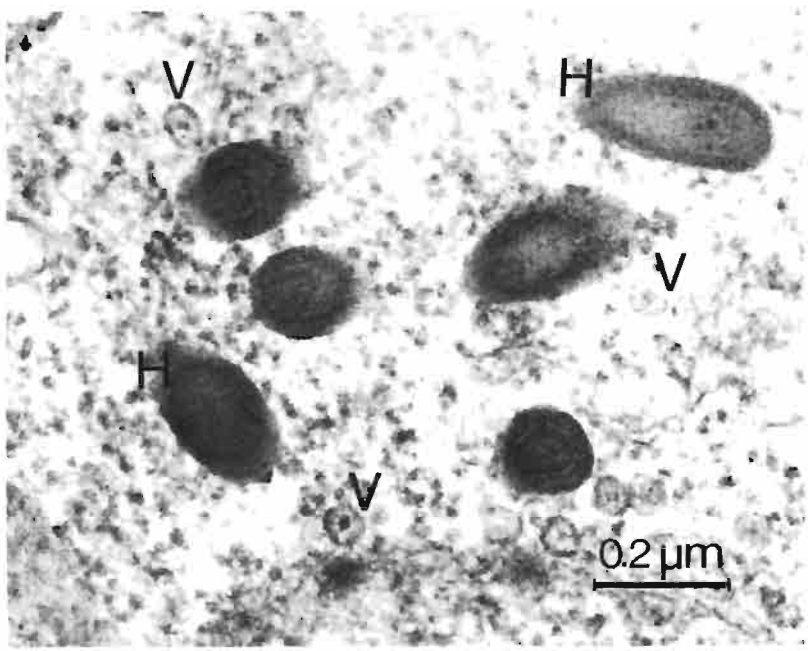


Fig. 13-44: *Haplosporidium* (?) sp. from *Ostrea edulis*. Haplosporosomes and virus-like particles within cytoplasm of plasmodium. (After Vivarès and co-authors, 1982.)

size between *H. costale* and *H. nelsoni* (Katkansky and Warner, 1970a). Haplosporidians, very similar to *H. nelsoni* in both size and structure of the plasmodial and spore stages, have been detected in 4 of 1,438 *C. gigas* from Korea (Kern, 1976b). Plasmodia, very similar to those in the American oyster, occur in *C. gigas* from Taiwan (Sindermann and Rosenfield, 1967). Katkansky and Warner (1970a), as well as Kern (1976b), indicated that there was no evidence that their parasites were responsible for any unusual oyster mortalities.

An unidentified haplosporidian, only seen in its plasmodial stage, parasitizes gaper clams *Tresus capax* in Yaquina Bay, Oregon (USA). Gross signs of infestation were white, spherical cysts ranging from 0.25 to 2.0 mm in diameter, which were primarily located in the mantle overlying the viscera and in the siphon under the epithelium bordering the inhalant and exhalant channels. The cysts were formed by host-cellular response and

consisted of massive aggregations of haemocytes and fibroblasts. Cyst formation was accompanied by displacement and destruction of normal tissues. In more heavily infested clams, cysts were also present in the gills, digestive gland, Leydig tissue and throughout the musculature of the body wall and foot. The cysts contained spherical to oval plasmodia, 10 to 35 μm in length, with 2 to over 60 nuclei. These were of 2 distinct size groups, with modes at about 1.5 and 3.0 μm, and probably represent 2 stages in nuclear development. Necrotic plasmodia were frequently observed and some were in the process of being phagocytized. Of 226 gaper clams, 97 (43 %) were found to be infested with this haplosporidian. Of these, 80 % were rated as light and caused no apparent damage to the clams. Heavily infested individuals were emaciated, sluggish in response to prodding, had dark-coloured meats and little gonad development even during the spawning season (Armstrong and Armstrong, 1974).

A further species of *Haplosporidium* was found to infest shipworms *Teredo navalis, T. bartschi* and *T. furcifera* from Barnegat Bay, New Jersey (USA). Stages from all 3 hosts appeared to be morphologically identical and are probably the same species. Infestations were not observed in *Bankia gouldi,* the most common molluscan woodborer in the bay. Overall prevalences in hosts collected from wooden experimental panels exposed at 20 stations from August 1975 throughout December 1980 were 451/1,117 (*T. navalis*), 24/345 (*T. bartschi*) and 9/23 (*T. furcifera*).

The plasmodia of this haplosporidian are highly variable in size, and some are as long as 60 μm and 10 μm wide. Sporulation occurs in the connective tissues of all host organs throughout most of the year. Spores are about 6 to 8 μm long in fixed material and almost 10 μm in smears of fresh material. When infestations were observed, they were usually intensive, suggesting rapid proliferation. There was considerable tissue damage or response to the pathogen. Thus, in infested hosts, the gill epithelium was found to be reduced from normal cuboidal to a squamous-like layer which sloughed off in many places, often forming large lesions on the gill surface and releasing the parasite into the mantle cavity. Systemic infestations could be accompanied by haemocytic infiltration into the connective tissues, metaplasia and hyperplasia of the digestive-tubule epithelium and occlusion of blood vessels by masses of haemocytes and life-cycle stages of the parasite.

Statistical analyses of the data obtained during the 1975–1980 long-term observations provided circumstantial evidence for the assumption that the haplosporidian may be responsible, at least in part, for controlling shipworm abundance in Barnegat Bay. High prevalences of the parasite in one year were followed by a significant decrease in *Teredo* abundance during the following year (Hillman, 1978, 1979, 1980; Hillman and co-authors, 1982).

The fact that all 3 *Teredo* species in Barnegat Bay are susceptible to the parasite, and that *Bankia gouldi,* by far the most common teredinid in that area, is not, may lead to speculation as to the origin and host specificity of this haplosporidian. It was not observed in Barnegat Bay until 1975, i.e., until after both *T. bartschi* and *T. furcifera,* 2 subtropical species, became established in the bay, possibly in consequence of water temperature elevation caused by the effluent of a nuclear power plant. It is possible, therefore, that the parasite has been brought in with the subtropical shipworms. It has also been speculated that it may be the oyster pathogen *Haplosporidium nelsoni,* from which it is thus far morphologically indistinguishable (Hillman, 1978).

For purposes of comparison, the *Haplosporidium* spp. reported from marine bivalves

have been listed in Table 13-6. Species of *Urosporidium* occurring as trematode or nematode hyperparasites in bivales have been discussed in Vol. I, Chapter 10, as well as in sections 'Agents: Trematoda' and 'Agents: Nematoda' of the present chapter.

Bonamia ostreae is the most recently discovered ascetosporan pathogenic to bivalves. It has been identified as the (possible) causative agent of unusual *Ostrea edulis* mortalities, first noticed at Ile Tudy, Bretagne (France), during June 1979 (Cahour and co-authors, 1980; Comps and co-authors, 1980; Pichot and co-authors, 1980; Tigé and co-authors, 1980). The protistan locates in the cytoplasm of haemocytes and causes gill ulcerations and breakdown of connective tissues, accompanied by massive haemocytic infiltration. Occasionally, internal lesions involving the stomach epithelium may be observed. Oysters heavily infested with *B. ostreae* display a characteristic yellowish colour (Comps and co-authors, 1980; Tigé and co-authors, 1980; Comps, 1982).

Two cell types of the parasite are apparent. The most frequently seen 'dense forms', generally round and 2 to 3 μm in diameter, have a dense cytoplasm rich in ribosome-like structures. The nucleus, limited by 2 unit membranes, contains granular, electron-opaque material. In addition, the cells contain the following elements: (i) 1 or 2 mitochondria, 0.5 to 1.8 μm in diameter; (ii) densely structured particles, 130 to 170 nm in diameter and in peripheral location; (iii) a very dense body, about 0.5 μm in diameter and without apparent structure, later identified as reserve material (Fig. 13-45).

'Clear forms', subspherical to elongate and amoeboid, 2.5 to 4 (exceptionally up to 7) μm in length, possess a nucleus with a voluminous nucleolus and differ from the 'dense form' with respect to the structure of their mitochondria and the densely structured particles (Fig. 13-46). A third type of organelle is present in the form of membraneous 'saccules', which sometimes are arranged in groups of 2 to 4. Morphologically comparable to a Golgi apparatus, they are associated with vesicles, 50 to 90 nm in size, which originate from lateral budding of the saccules. Some saccules have a circular profile, thus forming 100 to 150 nm vesicles, within which morphogenesis of the 'densely structured particles' — later identified as haplosporosomes — takes place.

Electron microscope examination of the various stages of *Bonamia ostreae* present in *Ostrea edulis* seems to indicate that the 'clear forms' of the parasite represent the vegetative, schizogonic stage multiplying by binary fission, while the 'dense forms' are resistant stages frequently found free (extracellularly) in tissues altered by the disease. These may represent the stage infesting new hosts. Occasionally, binucleate plasmodial forms of the approximate size of 'clear cells', are seen within infested host cells. These are somewhat reminiscent of the binucleate plasmodia of *Haplosporidium* spp. (Pichot and co-authors, 1980). Very recently, Brehélin and co-authors (1982) found true plasmodial forms of *B. ostreae,* which measured up to 6 μm in diameter and had 3, 4 or 5 nuclei. Such multinucleate plasmodia occurred only in dying or dead oysters (Fig. 13-47).

Initially, *Bonamia ostreae* was considered to be of doubtful taxonomic position. Cahour and co-authors (1980) believed it to represent 'young stages' of a haplosporidian. Comps and co-authors (1980), Pichot and co-authors (1980), Tigé and co-authors (1980) and Comps (1982) stated that this protistan shares several characteristics with both *Marteilia* and *Haplosporidium* but were unable to assign it with confidence to either the Occlusosporida or the Balanosporida. Although spores characteristic of the genus *Haplosporidium* have not yet been seen in *B. ostreae,* the discovery, by Brehélin and co-authors (1982), of true plasmodial forms of the parasite clearly relates it to the latter group.

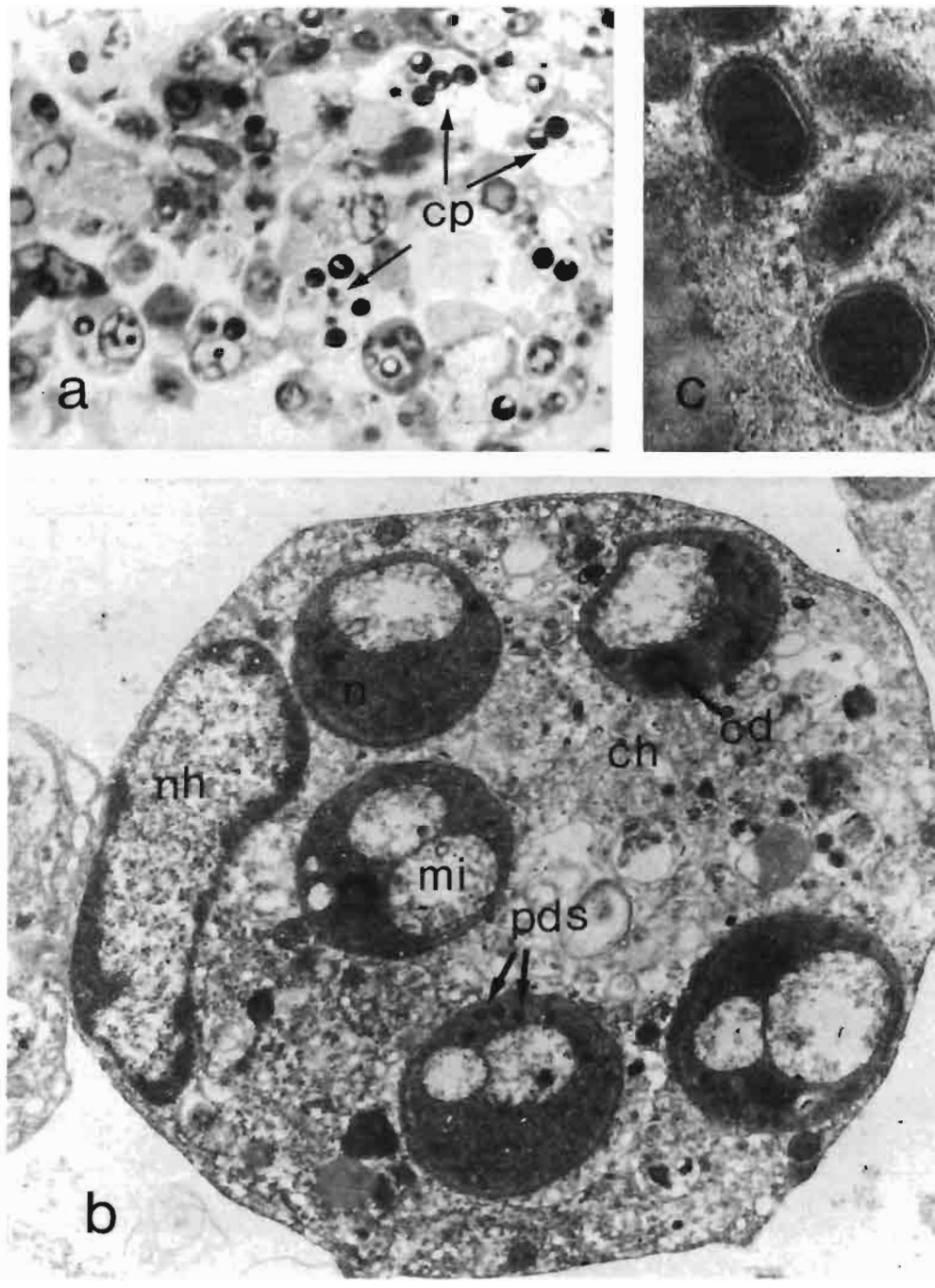


Fig. 13-45: *Bonamia ostreae* from *Ostrea edulis*. (a) Connective tissue in advanced stage of infestation showing numerous parasitized cells (cp), × 910. (b) Infested host haemocyte with 5 'dense forms' of parasite. cd dense (reserve) body, ch host cell, mi mitochondrion, n parasite nucleus, nh host-cell nucleus, pds 'densely structured particles' (haplosporosomes), × 12,400. (c) haplosporosomes at high magnification, × 118,000. (After Pichot and co-authors, 1980.)

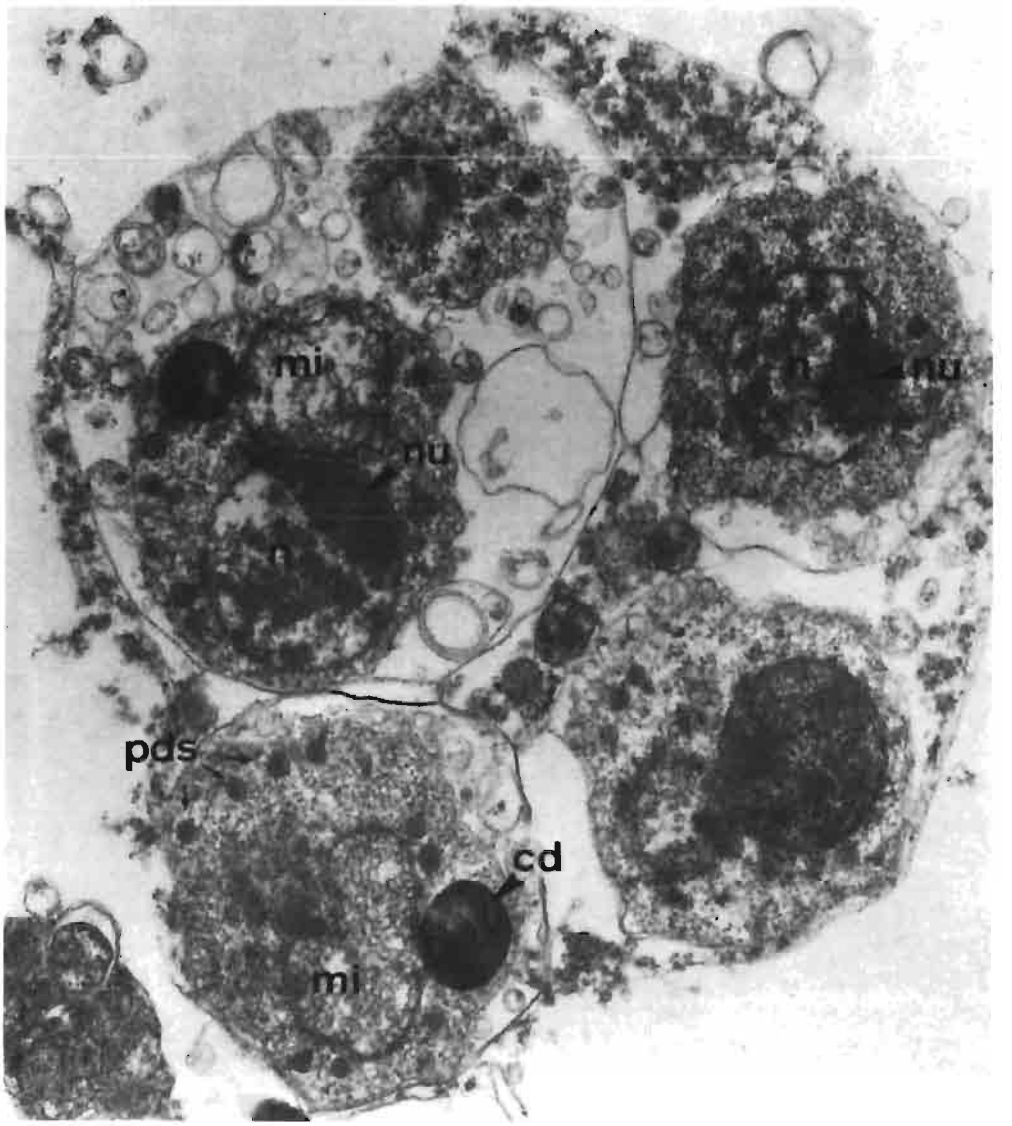


Fig. 13-46: *Bonamia ostreae* from *Ostrea edulis*. Host cell infested with 'clear form' of parasite. cd dense (reserve) body, mi mitochondrion, n nucleus, nu nucleolus, pds 'densely structured particles' (haplosporosomes), × 16,000. (After Pichot and co-authors, 1980.)

Prevalences of *Bonamia ostreae* are normally moderate to low (5 to 10 %) in healthy-appearing *Ostrea edulis* but were always higher than 70 % in dying or dead oysters from Saint-Philibert, Bretagne (France). Moribund individuals constantly contain higher parasite numbers than 'normal' ones. There appears to be an explosive multiplication of *B. ostreae* in the terminal stages of the disease. These observations, as well as the occurrence of multinucleate plasmodia in dying or dead oysters only, led Brehélin and co-authors (1982) to suspect that there are 2 different kinds of multiplication in *B. ostreae.* The first — simple binary division, as described by Pichot and co-authors (1980) — may

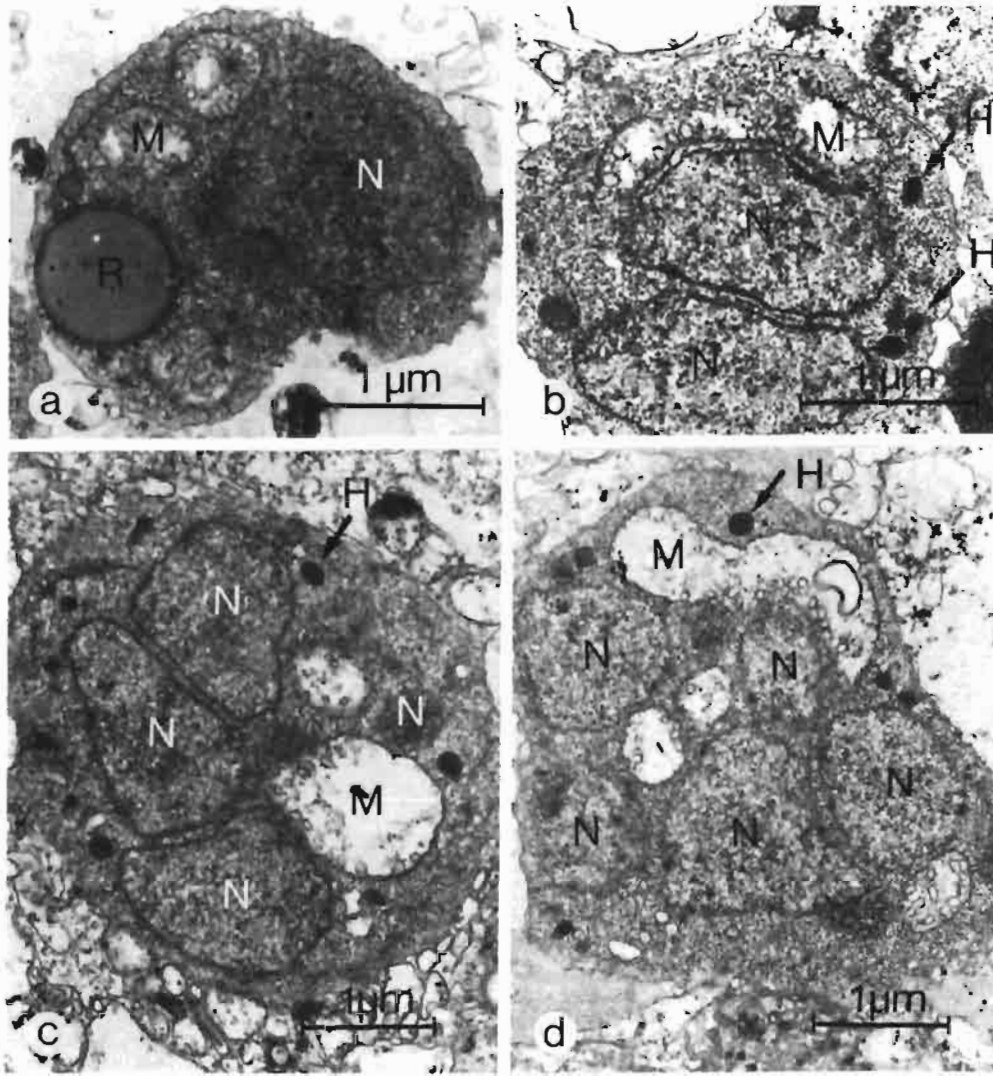


Fig. 13-47: *Bonamia ostreae* from *Ostrea edulis*. 'Classical' forms (a, b) of the parasite: (a) electron-dense, mononucleate stage, (b) clear, binucleate stage, (c) plasmodium with 4 nuclei, (d) plasmodium with 5 nuclei. H haplosporosome, M mitochondrion, N nucleus, R reserve body. Note tubular crests of mitochondria, which are characteristic of the species. (After Brehélin and co-authors, 1982.)

represent the initial stage of the disease. The second — multiplication *via* multinucleate plasmodia — may either represent a rare, fugacious event, or may be typical of the terminal stage of the disease.

Agents: Ciliophora (the Ciliates)

A considerable number of ciliates — members of the classical orders Thigmotrichida, Peritrichida, Heterotrichida and Hypotrichida — are known to associate with marine bivalves. The majority of these appear to be harmless commensals but evidence is

accumulating that others are true parasites. Many of the ciliate–bivalve associations are certainly transitional, established somewhere on the continuous scale leading from commensalism to parasitism, the equilibrium of the association depending on a complex of factors, such as physiological state of the host, number of agents present, environmental stress, etc. Defining the term 'parasitism' and being aware of the problem of doing so rightly, Noble and Noble (1976, p. 5) state:

> "One difficulty with this definition, however, lies in its emphasis on harm or lack of benefit. How can we be certain that a symbiont does *not* affect its host in a way more subtle than causing obvious physical damage or change in behavior? Numerous parasites apparently act as commensals most of the time, but are pathogenic when their numbers become unusually high."

This statement precisely circumscribes the situation governing most ciliate–bivalve associations.

Ciliate systematics are an ever-changing world, particularly in the era of modern electron microscopy. During the past two decades, the scheme of classification for the ciliated protozoans has been revised several times. While the Ciliophora has previously been conceded subphylum rank (Honigberg and co-authors, 1964), it was later raised to phylum status (Raabe, 1964; Corliss, 1974; de Puytorac and co-authors, 1974) and refined further (Corliss, 1975; Levine and co-authors, 1980).

The taxonomic position of the ciliates discussed below within the classification scheme proposed by Corliss (1975, 1979) is summarized in Table 13-7. Comparison with the scheme adopted by Fenchel (1965) shows several major differences. The genera *Ancistrum* and *Ancistrocoma*, for example, previously accomodated in the order Thigmotrichida, have now become separated remotely as members of two different classes, the Kinetofragminophorea (*Ancistrocoma*) and the Oligohymenophorea (*Ancistrum*).

More than 150 species of ciliates belonging in the classes Kinetofragminophorea, Oligohymenophorea and Polyhymenophorea, are known to live in the mantle cavity, on the gill surfaces or (rarely) in the digestive tract of marine bivalves. Most are regarded as commensals but the subtle physiological relationship with their hosts is yet poorly understood. Only a few species will be discussed below, with emphasis on those occurring with more common or commercially important marine bivalves.

Host specificity is typical of many ciliates associating with bivalves. Of the 42 Scandinavian species studied by Fenchel (1965, 1966), for example, 21 are strictly host-specific. *Abra alba* and *A. nitida*, living side by side in some localities in Gullmar Fjord, Sweden, both harbour a characteristic ciliate fauna with no species in common. Ciliates which are not strictly host-specific, occur only in a few — usually systematically or ecologically related — host species.

Bivalve-associating ciliates can be classified as belonging to one of three types adapted to live in the molluscan mantle cavity. They are either (i) filter feeders utilizing the suspended food items made available by the host's ciliary currents, (ii) particle feeders collecting food and/or mucus from the gills or mantle epithelium, or (iii) parasites feeding on the contents of epithelial cells of the host's gills.

The arhynchodine Scuticociliatida possess a cytostome and are thus predisposed to ingest particulate matter (bacteria, unicellular algae, etc.). Their members belong to the first (i.e., filter-feeding) and second type of bivalve-associating ciliates. Thigmotrichs *Ancistrum mytili* occur, often in great numbers, on the gills of *Mytilus edulis* (DeMorgan,

Table 13-7
Taxonomic position of ciliates from marine bivalves in the classification scheme based on Levine and co-authors (1980)

PHYLUM: CILIOPHORA	
	Families (after Corliss, 1979) and Genera
Class 1. Kinetofragminophorea	
Subclass 3. Hypostomatia	
Superorder 3. Rhynchodea	
Order 1. Rhynchodida	Ancistrocomidae: *Ancistrocoma, Crebricoma, Goniocoma, Holocoma, Hypocomagalma, Hypocomatidium, Hypocomella, Hypocomides, Hypocomidium, Insignicoma, Isocomides, Raabella.* Sphenophryidae: *Gargarius, Pelecyophrya, Sphenophrya*
Subclass 4. Suctoria	
Order 1. Suctorida	
Suborder 2. Endogenina	Endosphaeridae: *Endosphaera*
Class 2. Oligohymenophorea	
Subclass 1. Hymenostomatia	
Order 2. Scuticociliatida	
Suborder 1. Philasterina	Uronematidae: *Uronema* Thigmophryidae: *Conchophyllum, Thigmophrya*
Suborder 2. Pleuronematina	Peniculistomatidae: *Peniculistoma*
Suborder 3. Thigmotrichina	Ancistridae: *Ancistrum, Ancistrumina, Boveria, Proboveria*
Order 3. Astomatida	Anoplophryidae: *Anoplophrya*
Subclass 2. Peritrichia	
Order 1. Peritrichida	
Suborder 1. Sessilina	Ellobiophryidae: *Ellobiophrya*
Suborder 2. Mobilina	Urceolariidae: *Urceolaria.* Leiotrochidae: *Leiotrocha.* Trichodinidae: *Trichodina*
Class 3. Polyhymenophorea	
Subclass 1. Spirotrichia	
Order 1. Heterotrichida	
Suborder 6. Licnophorina	Licnophoridae: *Licnophora*
Order 4. Hypotrichida	
Suborder 2. Sporadotrichina	Euplotidae: *Uronychia*

1925; Kahl, 1931; Kidder, 1933a; Raabe, 1934, 1936, 1949; Fenchel, 1965). Although generally considered as a commensal, Pauley and co-authors (1966) regard *A. mytili* as a potential parasite, capable of producing pathological changes in hosts weakened by adverse environmental conditions. *A. isseli*, previously confounded with the former species by Issel (1903), occurs in the mantle cavity of Mediterranean *Modiolus barbatus* and North American Atlantic *M. modiolus* (Issel, 1903; Kidder, 1933a, b). Its food vacuoles normally contain the yellow pigment found in the host's cells, but it is not known whether the ciliate actively ingests host tissue or whether the pigment is derived from the plankton food. The latter appears more likely.

The morphology and morphogenesis of *Ancistrum mytili* have been studied in great detail by Hatzidimitriou and Berger (1977). As revealed by sophisticated multivariate

morphometric analyses, 3 morphologically distinguishable sympatric 'forms' of *A. mytili* occur in *Mytilus edulis* and *Modiolus modiolus* from the North American Atlantic coast (Berger and Hatzidimitriou, 1978). Raabe (1936) recognized 5 distinct forms of *A. mytili* in European mussels. Other species of *Ancistrum* have been described from *Mytilus galloprovincialis, Musculus niger, Mya arenaria, Macoma baltica, Tapes* spp., *Donax trunculus, Venus gallina, Scrobicularia plana, Dosinia exoleta, Tellina* spp., *Abra nitida, Nucula turgida* and other pelecypods (Maupas, 1883; Issel, 1903; Chatton and Lwoff, 1926, 1949; Kahl, 1931, 1934; Raabe, 1934, 1936; Fenchel, 1964b, 1965; and others). *Ancistrina* (= *Ancistrumina*) *kofoidi* associates with *Petricola pholadiformis* (Bush, 1937). Species of *Boveria* and *Proboveria* occur on the gills or in the mantle cavity of *Tapes decussatus, Cardita sulcata, Loripes lacteus, Pinna nobilis, Capsa fragilis, Tellina* spp., *Tellimya* (*Kellia*) *suborbicularis, Teredo navalis, T. furcifera* and *Nausitora hedleyi* (Issel, 1903; Nelson, 1923; Chatton and Lwoff, 1926, 1936, 1949; Delphy, 1938; Santhakumari and Balakrishnan Nair, 1973).

Strictly speaking, these thigmotrichs are filter–feeding commensals, not parasites. When occurring in low abundance, they presumably do not harm their carriers. High ciliate population densities on the gills, however, may interfere with laminary water flow, essential for adequate particle transport across the epithelial surfaces (Jørgensen, 1966; J. R. Blake, 1971). Tactile stimuli produced by the swarming ciliates may cause temporary cessation of the strokes of the gill cilia, whereby the filtering activity and particle retention may become reduced (Winter, 1969, 1970). Irritation due to the presence of large ciliate numbers may also stimulate excessive mucus production (Kellogg, 1915). Finally, the effect of food competition between host and commensals should not be underestimated. Most experimenters who are familiar with mass cultivation of unicellular algae, have experienced occasionally how rapidly and readily accidentally introduced thigmotrichous ciliates can cause the breakdown of algal cultures. This gives an impression of the enormous quantities of food consumed by dense ciliate populations per unit time. Harry (1979) has delineated a method for highlighting filter-feeding ciliates, which is also suited to estimate the amount of food consumed by these protozoans. In his 1980 paper, he could show that a single individual of *Licnophora auerbachi*, a filter-feeding heterotrichous ciliate, ingests between 400 and 600 yeast cells within a time interval of 10 to 15 min (p. 601). Food availability is a factor limiting the population size of ciliates living on the gills of bivalves (Fenchel, 1966).

In cases in which the mass occurrence of gill–inhabiting thigmotrichous ciliates coincides with host debility and death, the role of these protozoans is difficult to evaluate. The thigmotrichs are definitely no parasites *sensu stricto,* but large population densities are likely to result, by their mere presence, in a considerable degree of irritative interference with the proper function of the gills that hastens death of otherwise moribund or weakened hosts. But also healthy bivalves may be severely affected by excessive numbers of thigmotrichs. Thus, juvenile laboratory-reared *Cardium edule*, exposed to masses of (specifically unidentified) thigmotrichs obtained from decaying, heavily infested adult cockles, soon developed signs of disease and, eventually, died. No mortalities occurred in non-infested controls (Lauckner, unpubl.). The 'sequence of events' determining the shift, of these organisms, from non-pathogenic scavengers to pathogenic agents may be the same as that outlined for *Trichodina* spp. (p. 597).

Pleuronematine and philasterine ciliates represent the particle-feeder type of bivalve-

associated Protozoa. Pleuronematines of the family Peniculistomatidae (frequently confounded with the Conchophthiridae) are large forms resembling *Paramecium*, which crawl about on the epithelial surface of the foot and mantle, feeding on particles removed from the host's body surface.

Peniculistoma (*Conchophthirus, Kidderia, Morgania*) *mytili* (Fig. 13-48) occurs on the foot epithelium and adjacent surfaces of *Mytilus edulis*, to which it adheres loosely by the thigmotactic cilia of its left (physiologically ventral) side (DeMorgan, 1925; Kidder, 1933c, d). Almost 100 % of mussels from various localities in the North and Baltic Seas,

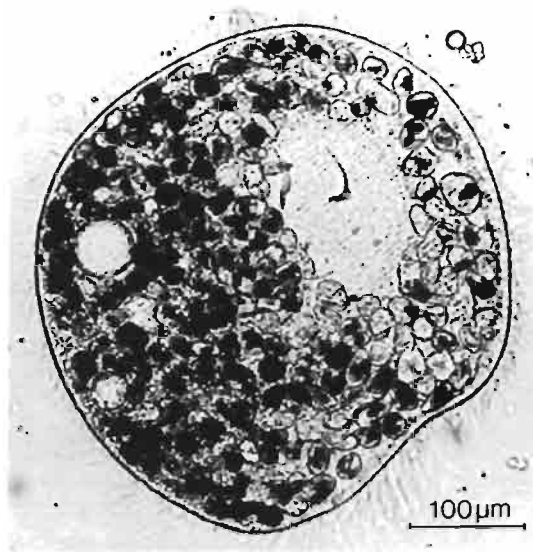


Fig. 13-48: *Peniculistoma mytili* from foot of *Mytilus edulis*. Note characteristic dark contents of food vacuoles. (Original.)

and more than about 20 mm in length, were found to be infested, some harbouring up to 300 of these ciliates (Beers, 1959; Fenchel, 1965). Although about 5 % of these were regularly in division, there was no appreciable increase in ciliate population density. It was concluded, therefore, that considerable numbers of *P. mytili* are constantly lost to the environment, probably via the host's exhalant current. In pure sea water, *P. mytili* can survive for 48 to 72 h at 14 °C, a period of time that seems adequate to ensure its maintenance and dissemination in a *Mytilus* population. *P. mytili* can feed, grow and divide once or twice in sea water to which a small amount of mussel haemolymph has been added, but the contribution supplied by the blood is unidentified. Rod-shaped bacteria and colourless flagellates were found in the food vacuoles of *P. mytili* maintained *in vitro*. In individuals collected freshly from the foot of *M. edulis*, food vacuoles may contain irregularly–shaped masses that have the same brownish colour as the pigment of the ciliated epithelium of the foot (Beers, 1959; Fenchel, 1965). Kidder (1933c) observed food vacuoles in *P. mytili* which were crowded with mussel spermatozoa.

In *Mytilus edulis* simultaneously infested with *Ancistrum mytili* and *Peniculistoma mytili*, both species occupy separate ecological niches in the host. *A. mytili*, which feeds on smaller particles than the latter, is exclusively found on the gills, whereas *P. mytili* occurs

predominantly on the foot of the mussel (Fenchel, 1965, 1966). *P. mytili* is strictly host-specific to *M. edulis*. Attempts to experimentally infest *Modiolus modiolus* with this ciliate have invariably met with failure (Fenchel, 1965).

Philasterines *Thigmophrya* spp. resemble *Peniculistoma* with respect to their *Paramecium*-like shape. *T. bivalviorum* occurs on the gills of *Mactra* (= *Spisula*) *solida* and *Tapes pullastra* from the French coast of the English Channel (Chatton and Lwoff, 1923b). *Macoma baltica* from the Baltic Sea and French coasts are host for *T. macomae* (Chatton and Lwoff, 1926; Raabe, 1936; Fenchel, 1965). Other species of *Thigmophrya* have been reported from *Tapes pullastra, Saxicava* (= *Hiatella*) *arctica* and *H. striata, Cultellus pellucidus, Spisula elliptica* and *Cardium ovale* (Chatton and Lwoff, 1926; Fenchel, 1964a, 1965). *T. annella* is 'hyperparasitic' in the intestine of nemerteans *Malacobdella grossa* living commensally in the mantle cavity of *Cyprina* (*Arctica*) *islandica* (Vol. I, p. 307).

Thigmophrya macomae was rare in *Macoma baltica* from Hel, Polish Baltic Sea coast, and in clams from shallow water at Askö, Sweden, but in individuals from 20 m depth infestations were nearly 100 % (Raabe, 1936; Fenchel, 1965). The ciliate was said to behave like a parasite, as its food vavuoles were found to be filled with cells of the host's gill epithelium (Chatton and Lwoff, 1926). Raabe (1936), however, believed that these cells are merely 'waste products' of the gill epithelium. Algae, but no host-epithelial cells, were observed in the food vacuoles of *Conchophyllum* ('*Conchophthirius*') *caryoclada,* a thigmophryid occurring in the mantle cavity of *Siliqua patula* from Seaside, Oregon (Kidder, 1933e).

Philasterines *Uronema marinum* were found to invade hatchery-reared cultchless oysters *Crassostrea virginica* and *Ostrea edulis*. Heavy mortalities among oyster larvae coincided with high ciliate abundance. Closer examination revealed that *U. marinum* is a bacteriophage, not a histophage. Ciliate numbers increased in correlation with the expanding bacterial population and entered an exponential growth phase. Addition of oyster tissue to the bacterial suspension did not enhance ciliate reproduction (Plunket and Hidu, 1978). Similarly, Davis and co-authors (1954) and Tubiash and co-authors (1965) assumed that ciliates, observed by them in bivalve-larval cultures, function as scavengers only on individuals weakened or killed by bacterial or fungal infections. However, as has been pointed out above, the irritative effect that excessive numbers of ciliates may have on healthy bivalve larvae, can considerably accelerate time to death in the cultures.

The Rhynchodida clearly represent the third (i.e., parasite-)type of bivalve-associating ciliates. In the members of this order the cytostome is functionally replaced by an anterior 'suctorial tentacle' by which the ciliates attach to epithelial cells of the host, mainly the gills or palps, and suck out nutrients (Figs 13-49 to 13-52). The structure and function of the suctorial tube have been studied by means of light microscopy (Chatton and Lwoff, 1939, 1949, 1950) and electron microscopy (Lom and Kozloff, 1968). The extensible and retractable tube is composed of compact sheets of microtubules which form both the outer wall of the tube and its internal radial septa. The cytoplasm on the inside of the tube may be regarded as phagoplasm. During the process of fixation of the ciliate to the host, a knob-like organelle, which is continuous with the suctorial tentacle, is embedded in the cytoplasm of the attacked host-epithelial cell. The knob serves for food uptake as well as for attachment. The mechanism of feeding has still not been elucidated entirely.

Most — if not all — of the rhynchodines reported from marine bivalves are members of the Ancistrocomidae and, less frequently, of the Sphenophryidae (Table 13-7; Figs 13-

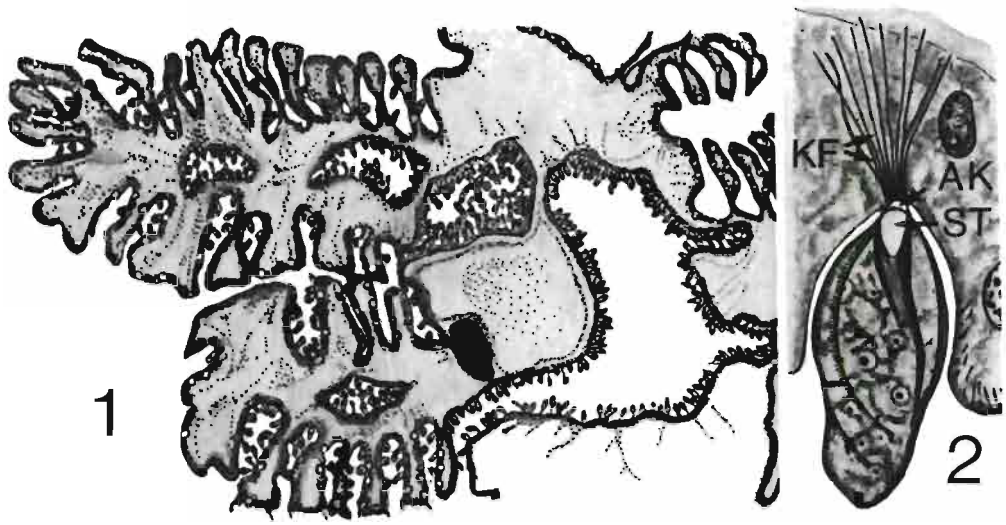


Fig. 13-49: *Cardium edule*. 1: Section of gill filament showing numerous attached *Hypocomella cardii;* 2: *H. cardii in situ* on epithelial cell; iron haematoxylin staining. Note keratin fibres (KF) converging upon attachment knob (AK) on suctorial tube (ST). (After Chatton and Lwoff, 1950.)

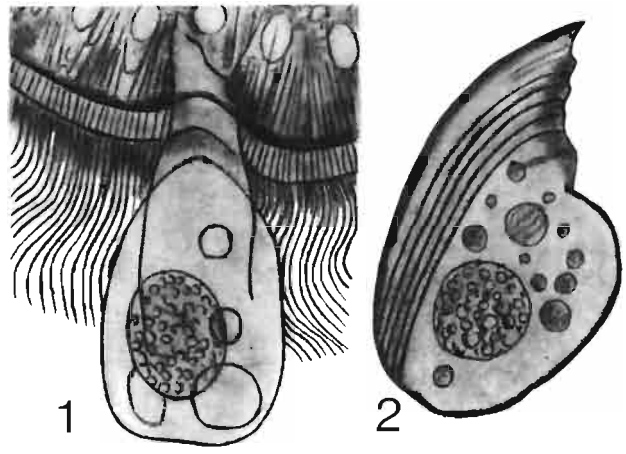


Fig. 13-50: *Pelecyophrya tapetis*. 1: Individual *in situ* on gill epithelium of *Tapes aureus*; 2. right lateral view (*in vivo*) of detached individual. (After Chatton and Lwoff, 1950.)

49 to 13-52). A considerable number of species has been described, several of which are of doubtful validity. Some ancistrocomid and sphenophryid rhynchodines infesting marine bivalves and their prevalence in their respective hosts are listed in Table 13-8. Several rhynchodines, probably *Ancistrocoma* sp. (or spp.) have been reported as internal parasites of oysters (Mackin, 1962; Burton, 1963; Engle and Rosenfield, 1963; Cheng, 1967; Gutiérrez, 1977a; Otto and co-authors, 1979; Meyers, 1981).

Among the ciliates associating with marine pelecypods, host specificity is most pronounced in the Rhynchodida. Thus, of 14 representatives of this order studied by Fenchel (1965) in the Baltic Sea, only 4 have been found in more than 1 host species.

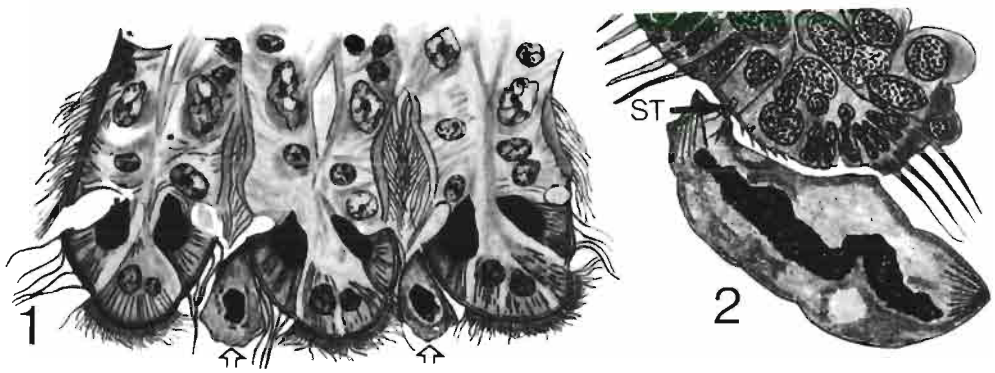


Fig. 13-51: *Sphenophrya dosiniae*. 1: Two individuals (arrows) *in situ* on gill filament of *Venus ovata*, transverse section; 2: longitudinal section of *S. dosiniae in situ*. Note suctorial tube (ST) embedded in host cell. (After Chatton and Lwoff, 1950.)

Sphenophrya dosiniae occurs in 5 bivalve species; *S. cardii* and *Hypocomella raabei* invade 2 closely related, sympatric lamellibranchs, *Cardium edule* and *C. lamarcki*; *Goniocoma macomae* is found in *Macoma baltica* and *Abra alba*. On the other hand, of the 3 closely related but morphologically distinct *Hypocomides* spp., each lives in one of 3 different *Musculus* species (Fenchel, 1965). *Gargarius gargarius* is strictly host-specific to *Mytilus edulis* and is only found in mussels carrying insignificant numbers of other ciliates (Raabe, 1938; Chatton and Lwoff, 1950). *Pelecyophrya tapetis* selectively invades *Tapes aureus* at Roscoff, France, but never occurs in *T. decussatus* and *T. pullastra* from the same localities (Chatton and Lwoff, 1922b, 1950). Similarly, *Raabella* (*Hypocomides*) *botulae* was found in 12 of 34 *Botula californiensis* from Moss Beach, California, but not in *B. falcata* from the same localities (Kozloff, 1946b).

In the laboratory it is easy to infest bivalves with their typical ciliates by adding these to the water containing the hosts. On the other hand, it is difficult to transmit ciliates to untypical hosts. Even when such infestations are initially successful, the protozoans survive for only a short time (Fenchel, 1965).

Once established, host specificity displayed by rhynchodines can be very strict. Thus, *Abra alba* and *A. nitida*, living side by side in Gullmar Fjord, Sweden, both have a characteristic ciliate fauna with no species in common. In *A. alba*, *Goniocoma macomae* was found in large numbers but no other ciliates were encountered in this bivalve. *A. nitida*, in turn, harboured 4 ciliate species but no *G. macomae* (Fenchel, 1965).

Where 2 ryhnchodine species occur concomitantly in the same host individual, a delicate equilibrium exists. Thus, *Hypocomidium fabius* can establish itself in *Cardium lamarcki* simultaneously infested with *Hypocomella raabei* only if the other species is represented by low numbers of individuals, and *vice versa* (Raabe, 1938). *Ancistrocoma pelseneeri* occurs in mixed infestations with *Goniocoma* (*Hypocomella*) *macomae* in *Macoma baltica* from the Bay of Gdansk, Poland, but the prevalence of either species is particularly high only in hosts in which the abundance of the other ciliate species is low (Raabe, 1934). Similar other cases of (competitive?) exclusion have been documented in the literature.

Mixed infestations involving arhynchodines or peritrichs are more common. Thus, *Holocoma primigenius* occurs in *Macoma baltica* from Askö, Sweden, in association with

Table 13-8
Some ancistrocomid and sphenophryid ciliates from marine bivalves (Original)

Host species (in alphabetical order)	Parasite	Prevalence	Locality	Source*)
Abra alba	*Goniocoma macomae*	100 %, high intensity	Gullmar Fjord (Sweden)	*12*
Abra nitida	*Ancistrocoma thorsoni*	about 20 %		
Astarte montagui	*Hypocomides astartae*	15 to 50 %, up to several 100 per host		
Barnea (Pholas) candida	*Ancistrocoma pholadis*	?	Wimereux (France)	*7*
Botula californiensis	*Raabella* (as *Hypocomides*) *botulae*	small numbers in 12 of 34 hosts	Moss Beach (California)	*15*
	Raabella (as *Hypocomides*) *parva*	small numbers in 19 of 34 hosts		
	Insignicoma venusta	small numbers in 9 of 34 hosts		
Brachidontes (Mytilus) minimus	*Isocomides* (?) (as *Hypocomides*) *mytili*	20 % of 150	Split (Yugoslavia)	*25*
Cardium edule	*Hypocomella* (originally as *Hypocoma*) *cardii*	?	Roscoff, Banyuls (France)	*3, 6, 9*
	Hypocomella raabei	low prevalence	Øresund (Denmark), Askö (Sweden)	*12*
	Sphenophrya cardii	about 20 %	Roscoff/Øresund	*9/12*
Cardium exiguum	*Hypocomella raabei*	?	Roscoff	*9*
Cardium lamarcki	*Hypocomella raabei*	low prevalence	Øresund, Askö	*12*
Cardium lamarcki (as *C. edule*)	*Hypocomella raabei* (as *H. cardii*)	?	Hel (Poland)	*24*
	Hypocomidium fabius	ca. 20 %, intensity sometimes extremely high	Hel	*24*
Cardium lamarcki	*Sphenophrya cardii*	about 20 %	Roscoff/Øresund	*9/12*
Cardium lamarcki (as *C. edule*)	*Sphenophrya cardii* (as *S. dosiniae*)	?	Hel	*24*
Corbula gibba	*Sphenophrya dosiniae*	ca. 40 %, high intensity	Roscoff/Gullmar Fjord	*5, 9/12*
Crassostrea gigas	*Ancistrocoma* (?) sp.	?	Purdy (Washington)	*22*
Crassostrea virginica	*Ancistrocoma* (?) sp.	up to 37.5 %	North American Atlantic coast	*2, 10, 17, 18, 20, 21*
	Sphenophrya (?) sp.	up to 29 %	North American Atlantic coast	*1, 18, 20, 21*
Cryptomya californica	*Ancistrocoma myae* (as *A. pelseneeri*)	low prevalence	Tomales Bay (California)	*16*
Dosinia exoleta	*Sphenophrya dosiniae*	?	Roscoff	*5,9*
Kellia (Tellimya) laperousii	*Raabella* (as *Hypocomides*) *kelliae*	in 9 of 28 hosts	Moss Beach	*15*
Macoma baltica	*Ancistrocoma pelseneeri*	fairly high prevalence	Wimereux/Hel	*7/23, 24*
	Goniocoma (originally as *Hypocomella*) *macomae*	fairly high prevalence	Wimereux, Boulogne (France)/Hel	*7, 9/23, 24*
	Holocoma primigenius	about 33 %, low intensity	? (France)/Askö	*9/12*
Macoma inconspicua	*Ancistrocoma myae* (as *A. pelseneeri*)	low prevalence	S. Francisco Bay (California)	*16*
Macoma irus	*Ancistrocoma myae* (as *A. pelseneeri*)	low prevalence		

Table 13-8 (continued)

Macoma nasuta	*Ancistrocoma myae* (as *A. pelseneeri*)	low prevalence	S. Francisco Bay	*16*
Macoma secta	*Ancistrocoma myae* (as *A. pelseneeri*)	low prevalence	Tomales Bay	*16*
Musculus discors	*Hypocomides musculus*	1 to 18 ciliates in 7 of 19 hosts	Gullmar Fjord	*12*
Musculus (Modiolaria) marmoratus	*Hypocomides modiolariae*	?	Roscoff	*3*
Musculus niger	*Hypocomides elsinora* (initially as *H. modiolariae*)	almost 100 %	Øresund	*11,12*
Mya arenaria	*Ancistrocoma myae* (as *A. pelseneeri*)	very common	San Francisco Bay	*16*
	Ancistrocoma myae (as *Parachaenia myae*)	?	San Francisco Bay	*13*
	Ancistrocoma myae	100 %, up to 1,100 per host	Helsingør (Denmark), Kristineberg (Sweden)	*12*
	Hypocomidium granum	very low prevalence	Hel	*24*
	Sphenophrya dosiniae	?	Hel/Woods Hole (Mass.)	*24/9*
Mya truncata	*Sphenophrya dosiniae* (as *S. myae*)	?	Kandalakschka Bay (White Sea)	*19*
Mytilus edulis	*Isocomides* (originally as *Hypocomides*) *mytili*	low to very low prevalence	Roscoff, Sète (France)/Helsingør, Kristineberg, Askö	*3,9/12*
	Raabella helensis (as *Hypocomides mytili*)	very low prevalence	Hel/Helsingør, Kristineberg, Askö	*24/12*
	Crebricoma (originally as *Hypocomina*) *carinata*	very low prevalence	Hel/Helsingør, Kristineberg, Askö	*23,24/12*
	Crebricoma kozloffi (as *C. carinata*)	?	San Francisco Bay	*14*
	Raabella sp. (as *Hypocomides mytili*)	about 80 %	San Francisco Bay	*15*
	Gargarius gargarius	low prevalence	Roscoff, Sète/Hel	*8/24*
Mytilus galloprovincialis	*Isocomides* (as *Hypocomides*) *mytili*	about 1 % of over 200	Split, Monaco	*25*
Nucula nucleus	*Hypocomatidium jarockii*	?	Banyuls	*9*
Pholadidea penita	*Ancistrocoma dissimilis*	in 21 of 36 hosts	Moss Beach	*16*
	Hypocomagalma pholadidis	in 28 of 36 hosts		
Saxicava (Hiatella) arctica	*Hypocomides hiatellae*	0 to 50 %, low intensity	Gullmar Fjord	*12*
Saxicava striata	*Hypocomides hiatellae*	0 to 50 %, low intensity		
Tapes aureus	*Pelecyophrya tapetis*	?	Roscoff	*4, 9*
Venus ovata	*Sphenophrya dosiniae*	?	Roscoff	*5, 9*
Zirfaea (Pholas) crispata	*Anisocomides* (as *Hypocomides*) *zyrphaeae*	?	Wimereux	*7, 9*

*) Sources: *1* Anonymous (C. A. Farley) (1965), *2* Burton (1963), *3* Chatton and Lwoff (1922a), *4* Chatton and Lwoff (1922b), *5* Chatton and Lwoff (1922c), *6* Chatton and Lwoff (1924), *7* Chatton and Lwoff (1926), *8* Chatton and Lwoff (1934), *9* Chatton and Lwoff (1950), *10* Engle and Rosenfield (1963), *11* Fenchel (1964b), *12* Fenchel (1965), *13* Kofoid and Bush (1936), *14* Kozloff (1946a), *15* Kozloff (1946b), *16* Kozloff (1946c), *17* Mackin (1962), *18* Meyers (1981), *19* Mjassnikowa (1930), *20* Newman (1971), *21* Otto and co-authors (1979), *22* Pauley and co-authors (1966), *23* Raabe (1934), *24* Raabe (1938), *25* Raabe (1949).

Ancistrum macomae, Thigmophrya macomae and *Trichodina macomarum. Hypocomides elsinora* is found together with *Ancistrum caudatum* in nearly all *Musculus niger* from Øresund (Fenchel, 1965).

Incidence and intensity of infestation of bivalves with ancistrocomids vary with parasite and host species involved (Table 13-8), and with ecological factors. About one third of the *Macoma baltica* from Askö, Sweden, were found to be infested with *Holocoma primigenius*, usually with only a few individuals. *M. baltica* from Øresund lacked this ciliate. In Gullmar Fjord, between 15 % and 50 % of the *Astarte montagui* carried *Hypocomides astartae*. Infestation intensities were normally low but in one case several hundred ciliates were recovered from a single host. Although most of the ciliate species follow their hosts into waters of lowered salinity, this is not invariably the case. At Helsingør and Kristineberg, for example, *Mya arenaria* is almost 100 % infested with *Ancistrocoma myae*, and the numbers of ciliates per host are very high. In one clam, 34 mm in length, 1,100 indviduals were counted (Fenchel, 1965). *A. myae* does not follow its host in the Baltic proper. Neither Raabe (1938), who studied the ciliate fauna of *Mya* at Hel, Poland, nor Fenchel (1965), who examined hosts from Askö, Sweden, recorded *A. myae* although large numbers of clams from these localities were screened.

Host population density is another factor determining composition and density of the ciliate fauna. In the Baltic Sea, where *Mytilus edulis* and *Macoma baltica* are abundant, these bivalves harbour the greatest number of ciliate species, i.e., 7 and 6, respectively. *Ancistrocoma thorsoni* was found on the gills of about 20 % of the *Abra nitida* from Gullmar Fjord where this lamellibranch forms dense populations. At other sites where *A. nitida* occurs more sporadically, the frequency of *A. thorsoni* infestations is lower or even zero (Fenchel, 1965, 1966).

Infestation intensities may also largely be determined by peculiarities in the life cycle of a given ciliate species. If a bivalve is infested by, say, a species of *Sphenophrya*, the parasite is always present in large numbers. Chatton and Lwoff (1921, 1922b, 1950) have studied the mode of multiplication in *Sphenophrya* spp. which leads to high ciliate numbers in individual hosts. Fully developed sphenophryids are devoid of cilia. During budding, a ciliated 'embryo' resembling an ancistrocomid is formed (Fig. 13-52). Upon detachment, the ciliated larva swims to another place on the host's gills where it attaches. It is probably also this stage which invades new hosts.

In *Crassostrea virginica* from upper Chesapeake Bay, all developmental stages of a *Sphenophrya* species, ranging from the ciliated larva to the non-ciliated adult, were found attached to the gill-epithelial cells of 4 to 5 % of the oysters. Occasionally, these ciliates induced the formation of a host structure known as a 'xenoma' (Weissenberg, 1922), in which one of the organisms enters a host cell and proliferates. Concurrently, the affected cell undergoes repeated karyokinesis and hypertrophies to an enormous size to accomodate the hundreds of ciliates contained therein. The xenomas, which occurred in less than 1 % of the *Sphenophrya*-infested oysters, ranged from a normal sized cell harbouring a single ciliate to a macroscopic, palpable cell about 3 mm long. Neither the sphenophryids nor the xenomas appeared to distress the oysters. Xenomas have not previously been reported from molluscs (Otto and co-authors, 1979).

Despite the great number of records of rhynchodine ciliates occurring in bivalves, very little has been reported on their pathology. At the cellular level, damage to the host has been revealed by electron microscope studies. Pathological manifestations include the

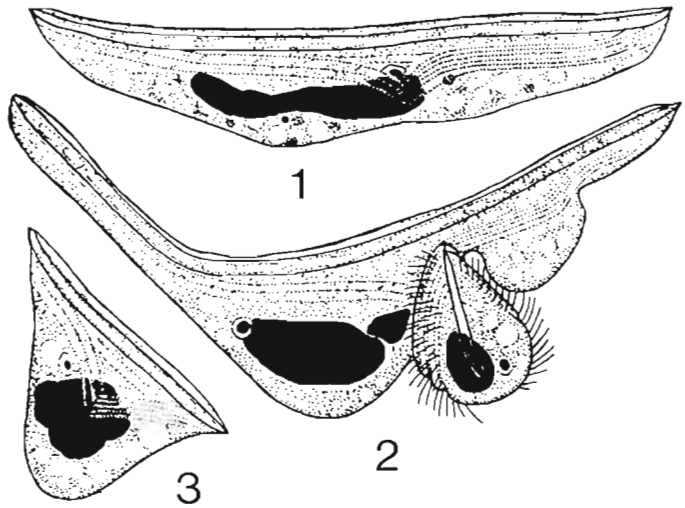


Fig. 13-52: *Sphenophrya dosiniae*. 1: Fully developed 'adult' individual; 2: parental individual with developing ciliated 'embryo'; 3: young stage recently settled on host-gill epithelium. (After Chatton and Lwoff, 1950.)

formation of strands of keratin fibres inside the affected host cell, which converge upon the surface of the attachment knob of the ciliate's suctorial tentacle, and an accumulation of lipid inclusions close to the knob. This may indicate a seriously disturbed host-cell metabolism. Damage to the cell, particularly when the ciliate leaves an open wound after its detachment, may perhaps also be important (Lom and Kozloff, 1968). Such wounds no doubt provide entrance ports for secondary microbial invaders, such as bacteria and fungal spores.

In heavy infestations, rhynchodine ciliates attached to the epithelial cells of the host's gills appear like pearls on a string in sectioned material (Fig. 13-49, 1). In *Crassostrea gigas* infested with a species tentatively identified as *Ancistrocoma* sp., the tissue around such parasitic foci generally appeared necrotic and in many cases only liquefied tissue debris remained. The normally tall, ciliated, columnar epithelium of the palps, to which ciliates became attached, exhibited metaplastic changes into a low cuboidal epithelium. Ciliates entering the gill ostia and attaching to the epithelium of the water tubes, appeared to occlude these chambers. Such occlusion undoubtedly interferes with normal respiration in the oyster and may cause anoxia of large areas of host tissue (Pauley and co-authors, 1967). Distinct pathological gill-tissue alterations, leading to functional deficiency, have also been observed in individuals of *C. angulata* infested with ciliates of the genera *Ancistrocoma* and *Sphenophrya* (Gutiérrez, 1977a).

Oysters injected with turpentine for experimental purposes, and having weakened adductor muscles, harboured by far more *Ancistrocoma* sp. than healthy controls or individuals submitted to other procedures, which identifies the ciliates as stress parasites. It was assumed, however, that small, latent natural infestations were prevalent in the *Crassostrea gigas* populations under study, as 2 to 3 % of the lightly stressed or control oysters also yielded these rhynchodines. In heavily infested hosts the organisms were also seen in necrotic kidney tubules and in Leydig cells below the rectum. In one case ciliates

were observed in gonadal tubules in which no sperm was present, while the unparasitized portions of this oyster's gonad contained sperm of normal appearance. The invading agents caused a general inflammatory reaction with a thick band of haemocytes surrounding the ciliates, possibly as an attempt to prevent further spreading of the infestation. In addition, the adductor muscles of the turpentine-injected oysters harbouring ciliates were swollen, edematous, heavily congested, and had hypertrophied muscle fibres (Pauley and co-authors, 1967). In spite of the clearly parasite-associated pathology, the authors (in all probability incorrectly) concluded that *Ancistrocoma* sp. in *C. gigas* is a secondary invader rather than a primary pathogen.

An ancistrocomid — similar to, or identical with, the one from *Crassostrea gigas* — occurs as an internal parasite in *C. virginica* from the North American Atlantic and Gulf coasts (Mackin, 1962; Burton, 1963; Cheng, 1967; Newman, 1971; Otto and co-authors, 1979; Meyers, 1981). Some of these workers believed it (probably erroneously) to be identical with *Ancistrocoma pelseneeri*. Cheng (1967) pointed out that it might not be a species of *Ancistrocoma* at all. The agent is usually rare in healthy oysters but may be found in large numbers in the stomach, intestine, intertubular spaces of the digestive gland and, occasionally, in the gonad. Burton (1963) observed the presence of the ciliates to be associated with an abnormal histological appearance of the host's tissues and unusual concentrations of haemocytes. Mackin (1962) concluded that the agent may become a complicating factor in *Dermocystidium marinum* (= *Perkinsus marinus*) infestations but that there is no reason to assume that it has much independent effect on the host. In contrast, Gutiérrez (1977a) concluded that *Ancistrocoma* sp. infestations may be a factor responsible for, or contributing to, mortality in *Crassostrea angulata*.

Crassostrea virginica from Long Island, New York, were found to carry unidentified ancistrocomids on the gills. Prevalences ranged from 5.7 to 37.5 % in a total of 243 oysters examined, and were higher in the winter and spring than in the summer and autumn. Concomitant infestations, ranging from 2.7 to 5.8 %, were observed in the epithelium of the intestine and the tubules of the digestive gland. The agent was provisionally, and probably incorrectly, identified as *Ancistrocoma pelseneeri* (Meyers, 1981). It appears possible that both forms — the one on the gills and the internal parasite — are specifically identical. One may conclude that all the above ancistrocomids reported as internal agents in *C. gigas* and *C. virginica* are normally gill parasites which invade the host's tissues only under abnormal conditions of physiological stress.

Sphenophrya-like ciliates have repeatedly been observed in *Crassostrea virginica* from North American Atlantic waters (Anonymous [C. A. Farley], 1965; Newman, 1971; Otto and co-authors, 1979; Meyers, 1981). Prevalences were usually low but reached a maximum of 29.2 % in Long Island oysters in the winter. Infestations seldom involved more than 1 or 2 gill filaments but when present on a filament, the parasites occurred in large numbers, occasionally causing a moderate exudate of haemocytes at the host's gill surfaces. Multiple xenomas containing sphenophryids were also observed. In most cases, however, parasite numbers were too small to cause observable pathology.

No further information concerning the pathology of bivalve-associated, gill-inhabiting rhynchodine ciliates is available, most of the papers cited above and in Table 13-8 being merely descriptive. Wherever host relationships have been taken into consideration, the role of these agents in the production of disease has clearly been underestimated and needs to be re-evaluated. Damage to the host at the cellular level has conclusively been

demonstrated. In heavy infestations, massive damage at the tissue level is to be expected. Furthermore, it must be assumed that the mere presence of large numbers of these protozoans on the gill surfaces produces irritative effects similar to those caused by commensal thigmotrichs and peritrichs. Finally, the role of rhynchodines in clearing the way for secondary microbial infections of the host remains to be studied.

Peritrichous ciliates of the genera *Trichodina, Urceolaria* and *Leiotrocha* — members of the suborder Mobilina — play a considerable role as associates of marine Mollusca. The body of *Trichodina* is slightly dome-shaped and equipped with adoral and aboral bands of cilia. The concave adhesive or basal disc bears a circle of conspicuous hooklets (denticles), the number of which is important as a diagnostic feature (Fig. 13-53). Contrary to previous

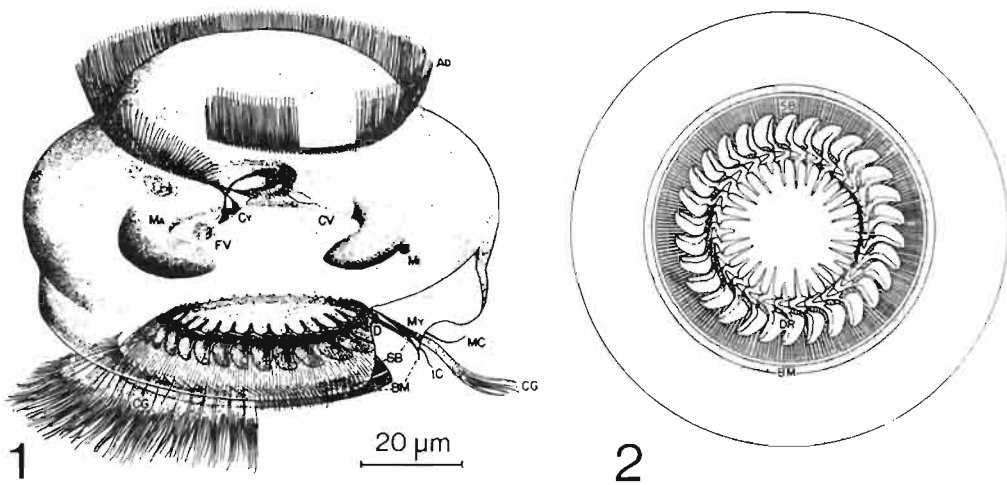


Fig. 13-53: *Trichodina myicola* from palps of *Mya arenaria*. 1: Ventro-lateral aspect. Semidiagrammatic reconstruction of entire organism from living and stained material, silver preparations and sections. Cutaway shows detail of basal disc and associated ciliature in section. 2: Posterior aspect (semidiagrammatic) of basal disc showing details of denticle ring, striated band and border membrane. AD adoral cilia, BM border membrane, CG ciliary girdle, CV contractile vacuole, CY cytopharynx, D denticle, DR denticle ring, FV food vacuole, IC inner cilia, MA macronucleus, MI micronucleus, MC marginal cilia, MY myoneme, SB striated band. (After Uzmann and Stickney, 1954.)

assumptions, the denticles are *not* used for attachment but merely represent skeletal elements for the reinforcement of the basal disc. Trichodines are mainly ecto- and endosymbiotes of amphibians and fishes (for parasite-host list see Uzmann and Stickney, 1954). Most of them appear to be bacterivorous commensals although some have been labelled parasitic.

Cyclochaeta cardii, very superficially described from *Cardium edule* at Arcachon, France, is the first trichodine reported from a marine bivalve. Uzmann and Stickney (1954) transferred it to the genus *Trichodina* but Raabe and Raabe (1959) considered *T.* (*Cyclochaeta*) *cardii* Delphy, 1938, a *nomen nudum* and described *T. cardiorum* from *'C. edule'* on the Baltic Sea coast of Poland. However, the host species given by these authors is, in fact, *C. lamarcki*, not *C. edule*. A species of *Trichodina*, occurring on

individuals of *C. edule* in the nothern Øresund, could not be identified to species level but was considered different from *T. cardiorum* (Fenchel, 1965).

Trichodines occurring with marine pelecypods are listed in Table 13-9. Whether all of these represent valid species remains to be studied. At least some appear to be host-specific. Thus, *Cardium edule* and *C. lamarcki* from the Baltic Sea harbour different species of *Trichodina*. Experimental cross–infestations were either negative or the ciliates died after a very short time (Fenchel, 1966). *T. polandiae* and *T. macomarum*, on the other hand, have each been reported from 2 different, unrelated host species (Raabe and Raabe, 1959; Fenchel, 1965; Santhakumari and Balakrishnan Nair, 1973; Stein, 1974). *T. pectenis*, reported from the mantle cavity of *Pecten (Patinopecten) yessoensis*, is also found in association with sea urchins *Echinorachnis parma* (Stein, 1974).

Incidence and intensity of *Trichodina* infestation varies with locality and season. Delphy (1938) found about 50 % of the *Cardium edule* from Arcachon to be infested with *T.* (*Cyclochaeta*) *cardii*. Hancock and Urquhart (1965) reported what might be the same ciliate from only 12 % of 438 cockles on Llanrhidian Sands, Wales. It was observed at all seasons and in all age groups of cockles, including 4 out of 5 individuals examined at 6 months of age. A 13-mm cockle nearly a year old contained at least 4 trichodines. *T. myicola* occurred in 0 to 62 % of the *Mya arenaria* from Sagadahoc Bay, Maine, and Plum Island Sound, Massachusetts, with highest percentages and intensities of infestation in late spring. Greatest concentrations of these peritrichs were found on the outer faces of the palps where numbers may run as high as 100 or more to each of the four palps. Occasional specimens were found on the wall of the visceral body and on the internal face of the pallial muscle (Uzmann and Stickney, 1954). Apparently, *T. myicola* does not occur in European *M. arenaria* (Fenchel, 1965).

Trichodina macomarum was present in almost 100 % of the *Macoma baltica* from Askö, Sweden, but in only 20 % of the same host species from Øresund, Denmark (Fenchel, 1965), and was rare in *M. baltica* from Gdynia, Poland (Raabe and Raabe, 1959). Infestation of *Cardium lamarcki* from Askö with *T. polandiae* and that of *C. edule* from Øresund with *Trichodina* sp. was close to 100 %, while *C. lamarcki* from Øresund and Gullmar Fjord were never infested (Fenchel, 1965).

There is some controversy with respect to the pathogenicity of *Trichodina* spp. Epizoic representatives of the genus are usually regarded as harmless commensals while endozoic species are mostly labelled parasitic (Fulton, 1923; Lom and Prasad Haldar, 1976). Uzmann and Stickney (1954, p. 149), commenting on the trichodines known from invertebrates, state that "there is little evidence that any of these are significant parasites, if parasitic at all". From their morphology, peritrichs are filter-feeders, capable of collecting small suspended particles, such as bacteria. In fact, Hirshfield (1949) found the food vacuoles of *Trichodina tegula* from marine turban shells *Tegula funebralis* to contain only bacteria. On the other hand, food vacuoles of trichodines living on the gill epithelium of fishes contain host-red blood cells, which is indicative of tissue destruction. Irritation from heavy infestation may lead to complete abrasion of the gill epithelium, hyperplasia, extravasation, secondary infection and, eventually, death of the fish (Padnos and Nigrelli, 1942; Nigrelli, 1948; Reichenbach-Klinke, 1956a). In the New York Aquarium, between 4 and 12 % of the losses occurring among marine fishes in the period 1939 to 1941 have been attributed to *Trichodina* sp. infestation (Nigrelli, 1940, 1943).

Comparable studies on the pathology caused by *Trichodina* spp. invading marine

Table 13-9
Trichodina spp. from marine bivalves (Original)

Host	Parasite	Diameter (µm): Average (range)	Height (µm)	Ring of denticles (Ø in µm)	Denticle number	No. of radial rods per denticle	Geographic area	Source
Cardium edule	*Trichodina (Cyclochaeta) cardii*	60–80	40	–	22	–	Arcachon (France)	Delphy (1938)
Cardium edule	*Trichodina* sp.	52 (44–56)	–	21 (20–24)	24 (22–25)	about 5	Øresund (Baltic Sea)	Fenchel (1965)
Cardium edule	*Trichodina* sp.	–	–	–	–	–	Norderney (Germany)	Hausmann (1978)
Cardium edule	*Trichodina* sp.	60–70	–	–	24	–	Dutch North Sea coast	van Banning (1979b)
C. lamarcki	*T. polandiae* (syn. *T. domerguei* f. *cardii*)	52 (47–60)	–	25 (21–29)	21 (19–23)	7–10	Askö (Kattegat)	Fenchel (1965)
C. lamarcki	*T. polandiae*	38–50	20	25 (21–28)	20 (18–22)	10	Baltic Sea (Poland)	Raabe and Raabe (1959)
C. lamarcki (as *C. edule*)	*T. cardiorum*	75 (54–90)	20	36 (30–41)	25 (24–29)	10–11	Baltic Sea (Poland)	Raabe and Raabe (1959)
Mya arenaria	*T. myicola*	81 (62–103)	31–86	36 (29–46)	29 (26–36)	–	Maine and Massachusetts (USA)	Uzmann and Stickney (1954)
Macoma baltica	*T. macomarum*	60 (50–65)	20	25.5 (21–33)	23 (20–27)	10	Baltic Sea (Poland)	Raabe and Raabe (1959)
Macoma baltica	*T. macomarum*	68 (60–74)	–	29 (26–34)	27 (24–30)	6–8	Askö (Kattegat), Øresund (Baltic Sea)	Fenchel (1965)
Crassostrea angulata	*Trichodina* sp.	56	–	–	26–28	–	Seudre (French Atlantic coast)	Besse (1968)
Ostrea edulis	*Trichodina* sp.	75–80	–	–	33–35	–	Dutch North Sea coast	van Banning (1979)
Chlamys sp.	*T. polandiae*	34.0–50.3	–	22.4–34.0	24 (20–26)	7–10	Gulf of Peter the Great (Sea of Japan)	Stein (1974)
Pecten (Patinopecten) yessoensis	*T. pectenis*	–	–	26.2–33.5	22–31	7–9	Gulf of Peter the Great (Sea of Japan)	Stein (1974)
Nausitora hedleyi	*T. balakrishnia**)	44 (33–55)	–	28 (20–35)	22–30	8–10	Southwest coast of India	Santhakumari and Balakrishnan Nair (1973)
Teredo furcifera	*T. balakrishnia**)	44 (33–55)	–	28 (20–35)	22–30	8–10	Southwest coast of India	Santhakumari and Balakrishnan Nair (1973)
Martesia striata	*T. macomarum**)	50 (40–60)	18 (15–20)	31 (25–37)	22–30	6–9	Southwest coast of India	Santhakumari and Balakrishnan Nair (1973)

*) not stated whether the 2 *Trichodina* species occur in 1 or more of the 3 host species.

invertebrates have not been conducted, but from the morphology and function of the basal disc of peritrichs (Fauré-Fremiet, 1943, 1950; Fauré-Fremiet and Thaureaux, 1944; Favard and co-authors, 1963; Lom and Corliss, 1968; Lom, 1973) one may conclude that the effects produced by bivalve-inhabiting *Trichodina* must be similar to those documented for *Licnophora auerbachi* (p. 601). In fact, Portuguese oysters *Crassostrea angulata*, heavily infested with *Trichodina* sp., exhibit distinct hyperplasia or destruction of the gill filaments (Besse, 1968). Bivalve-invading *Trichodina* spp. presumably ingest host-blood and epithelial cells but this has not been proven positively. Pigmented host cells are easily detected in *L. auerbachi* and fish-invading trichodines. But demonstration of colourless, inconspicuous haemocytes and epithelial cells among the contents of the digestive vacuoles of species invading bivalve gills would require more scrutinized methods of detection. Furthermore, the possible role of peritrichs in clearing the way for secondary microbial infections merits investigation.

Trichodina sp. (Fig. 13-54) is held responsible for recurrent large-scale mortalities among recently settled *Cardium edule* on tidal flats at Sylt, German North Sea coast,

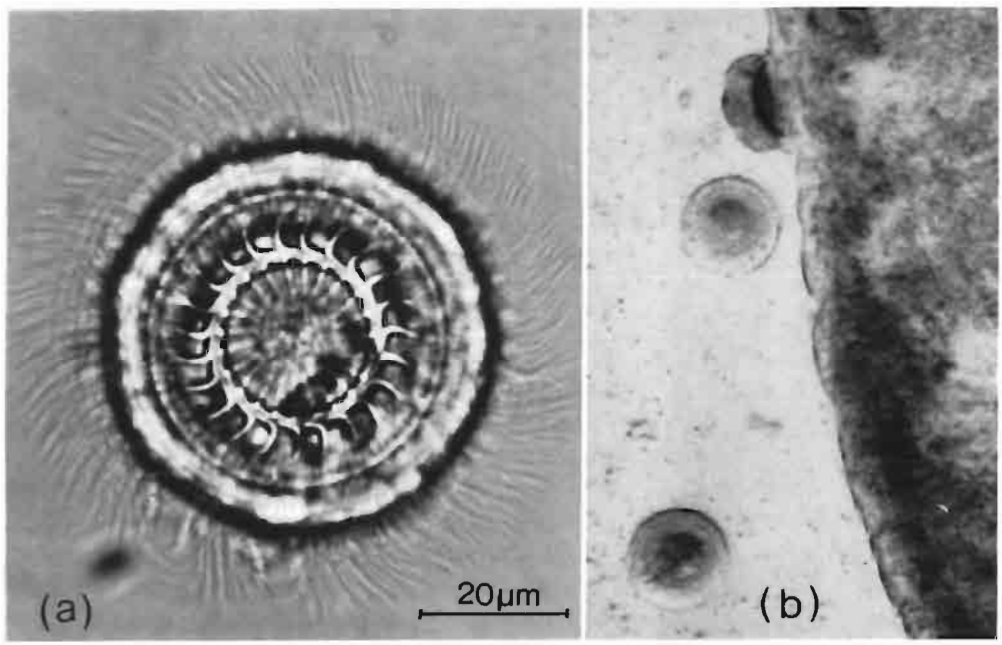


Fig. 13-54: *Trichodina* sp. from *Cardium edule*. (a) Posterior aspect showing denticle ring and ciliary girdle; (b) *Trichodina* sp. invading mantle epithelium of *C. edule*. (Original.)

particularly in unusually hot summers. The peritrichs are normally found in low abundance and over the whole year in the mantle cavity of adult cockles but occur in vast densities in 0-group individuals of 5 mm or less shell length in August. The rapid spreading of *Trichodina* infestation on beds carrying spat is distinctly correlated with the population density of juvenile cockles and with the presence, on these beds, of adult individuals. *Trichodina* sp. does not survive long outside the host. High cockle abundance significantly increases the ciliates' chance of finding a new host. Adult cockles serve as a 'reservoir' for

the ciliates, supplying the stock for initial infestation of the offspring. Therefore, *Trichodina* infestations are less severe in spat from flats where adult *C. edule* are lacking (Lauckner, unpubl.). Similar observations have been made on Dutch cockle beds:

> "The fact that the presence of even a small number of older cockles causes the mortality among one-year-old cockles to be higher than in places where there are no older cockles, points to the possibility that infection by parasites raises the mortality. Such parasites, however, could not be identified" (Kristensen, 1957, p. 99).

Michaelis (1977) and van Banning (1979b) found *Trichodina*-infested *Cardium edule* to be "most frequent in some mortality areas" in the German and Dutch Wadden Sea.

Infested juvenile cockles soon become emaciated and moribund. At Sylt, resultant epizootic mortalities last from about mid-July until the end of August or mid-September. Adult cockles sharing the same beds with the spat are usually only lightly infested although *Trichodina*-associated deaths may occur occasionally (Lauckner, unpubl.). Similar to *Cardium edule* from North Sea tidal flats, *Crassostrea angulata* from Seudre, French Atlantic coast, appears to suffer appreciable losses from infestation by trichodines. Diseased oysters were found to be emaciated to such an extent that clapping of their valves gave a 'hollow noise'. Their soft parts had an acrid taste, and the digestive gland was unusually slender and of a greyish tinge. The gills of such oysters were heavily eroded and deformed, and carried numerous *Trichodina* individuals. Centrifuged mantle-cavity water from healthy oysters did not yield these organisms (Besse, 1968). It cannot be excluded, however, that Besse's oysters actually suffered from "Maladie des branchies", a virus-caused disease (p. 481).

One might assume that, in these cases, trichodines are secondary invaders rather than primary pathogens, feeding on bacteria present on the gills of bivalves suffering from another disease. However, experimental evidence indicates that this is not the case. Laboratory-raised, healthy juvenile *Cardium edule* soon became moribund, and eventually died, when exposed to masses of *Trichodina* sp. obtained from the gills of naturally infested cockles. Adult cockles, kept as controls in the same containers with the juveniles, became only lightly infested and survived the experiment (Lauckner, unpubl.).

The rapid sweeping of *Trichodina* sp. infestations through previously flourishing cockle populations gives the impression that the virulence of the agent is massively enhanced by its passage through a succession of hosts. But, supposedly, a multitude of factors acting in concert triggers the switching of *Trichodina* from an apparently harmless commensal to a disease-causing agent. The sequence of events might be as follows: The ciliates normally occur in low abundance in the mantle cavity of healthy adult hosts. There is an equilibrium between the rates of multiplication and loss due to 'accidental' expulsion of individuals from the mantle cavity. Minor gill lesions caused by the attachment of the epibiotes are overcome by the regenerative capacity of the host tissues. Under environmental stress, the ciliary activity of the host's gills decreases and, in consequence, less ciliates are expelled from the mantle cavity; their abundance increases. Constant irritation of the gill epithelium by masses of *Trichodina* now produces persistent degenerative alterations of the epithelium. The activity of the ctenidial cilia decreases due to the constant tactile irritation by the swarming protozoans. Excessive mucus, produced in defense against the epithelial stimulation, begins to cover the gill lamellae. Eventually, host death ensues. Whether this is *directly* due to respiratory impairment caused by the

sheet of mucus and protozoans covering the gill surface and preventing proper gas exchange, or *indirectly* due to secondary infection by bacterial pathogens entering the host tissues through wounds in the gill epithelium, presumably produced by the attached ciliates, remains to be studies.

Perturbation of the gills' respiratory function and resultant reduced metabolism were held responsible for the massive emaciation of *Trichodina*-infested *Crassostrea angulata* (Besse, 1968). A similar impairment was believed to be caused by dense 'clouds' of trichodines covering the gills of fishes (Reichenbach-Klinke, 1956a).

Fenchel (1965) observed that, in some instances, the number of ciliates found in the mantle cavity of individual bivalves remains fairly constant despite the rapid multiplication of these organisms. He concluded that excessive parasites are constantly lost to the environment. This also holds true for the population density of *Trichodina* sp. in adult, healthy *Cardium edule*. In the laboratory, ciliates were repeatedly seen being discharged through the exhalant sipho. Under identical conditions, the number of trichodines in juvenile hosts increased steadily (Lauckner, unpubl.). The absence or low abundance of ciliates in the mantle cavity of pectinid bivalves may be due, in part, to the strong respiratory currents produced by these molluscs (Fenchel, 1966). In contrast, the ciliary activity of small hosts may be too weak for an effective ejection of excessive numbers of ciliates. Field observations tend to support this hypothesis. Thus, adult *C. lamarcki* from Askö constantly carry less *T. polandiae* than juvenile cockles (Fenchel, 1965). On the Polish coast of the Baltic Sea, *T. cardiorum* occurs exclusively in very young cockles, measuring up to 5 mm in shell length, and is most common in spat 2 to 3 mm in length. Adult cockles from the same localities are never infested (Raabe and Raabe, 1959).

According to Hausmann (1978), *Trichodina* sp. infestations in *Cardium edule* from the East Frisian (Federal Republic of Germany) coast have increased in recent years. Large-scale mortalities among cockles from those areas, due to unknown causes (but suspected, by some workers, to be correlated with man-made pollution) clearly characterize the East Frisian tidal flats as stress habitats (Michaelis, 1977, 1978a, 1981; Tougaard and Helweg Ovesen, 1981). It should be borne in mind that this coastal strip receives the highly polluted discharges of the rivers Ems and Weser, and of the Jade estuary. At Seudre, French Atlantic coast, increases in *Trichodina* sp. infestations in *Crassostrea angulata* were found to be correlated with concomitant changes in water quality (Besse, 1968). Interestingly, in this context, Padnos and Nigrelli (1942) diagnosed trichodiniasis in puffers *Spheroides maculatus* from the (presumably more heavily polluted) New York and New Jersey coasts but could not find *Trichodina* in fishes from Massachusetts waters. If future investigations would prove the occurrence of *Trichodina* disease in (invertebrate as well as vertebrate) hosts from polluted areas to be a constant phenomenon, then *Trichodina* spp. would qualify as sensitive stress or pollution indicators.

Ellobiophrya donacis, a peritrich of the suborder Sessilina, grasps gill filaments of *Donax vittatus* by means of a pair of remarkable aboral 'arms'. These contractile structures, which are cylindrical projections on either side of the scopula, encircle the gill filament and join tightly at their tips to form a closed ring (Fig. 13-55). *E. donacis* was found on about 50 % of the *D. vittatus* but on none of 25 other lamellibranch species occurring abundantly in the region of Roscoff, France (Chatton and Lwoff, 1923a, 1929). Nothing has been reported on its pathology.

A single case of hyperparasitism has been observed in peritrichs associating with

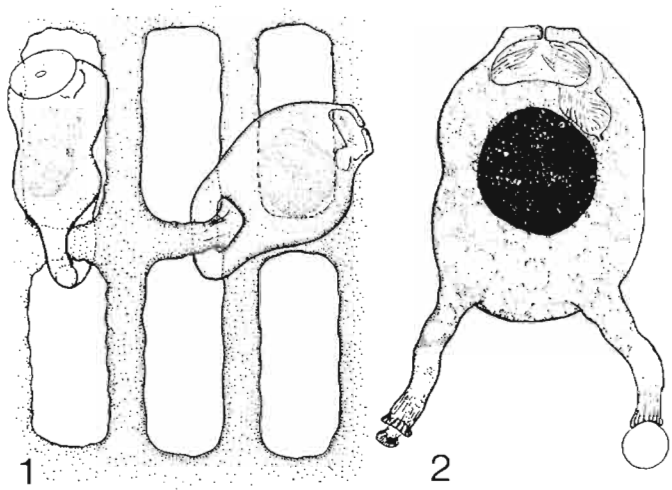


Fig. 13-55: *Ellobiophrya donacis* from *Donax vittatus*. 1: Two living individuals *in situ* on host gill (gill cilia omitted), × 1,000; 2: forcibly detached, fixed and stained specimen showing contractile aboral extensions. (After Chatton and Lwoff, 1923a.)

marine bivalves. Uzmann and Stickney (1954) found several individuals of *Trichodina myicola*, occurring in *Mya arenaria* from Sagadahoc Bay, Maine, and Plum Island Sound, Massachusetts, to harbour endoparasitic suctorians *Endosphaera* sp. (Vol. I, p. 111), possibly *E. engelmanni*, as reported by Padnos and Nigrelli (1942, 1947) from *T. spheroidesi* and *T. halli* infesting *Spheroides maculatus*.

Heterotrichs *Licnophora auerbachi* are ectoparasitic on the eyes of *Chlamys* (*Aequipecten*) *opercularis*. The body of these rare and exotic ciliates is divisible into 3 distinct regions: a basal disc for attachment, a flexible 'neck' region, and an oral disc supporting a prominent and quite distinctive adoral zone of membranelles (Figs 13-56, 1 and 13-57, b).

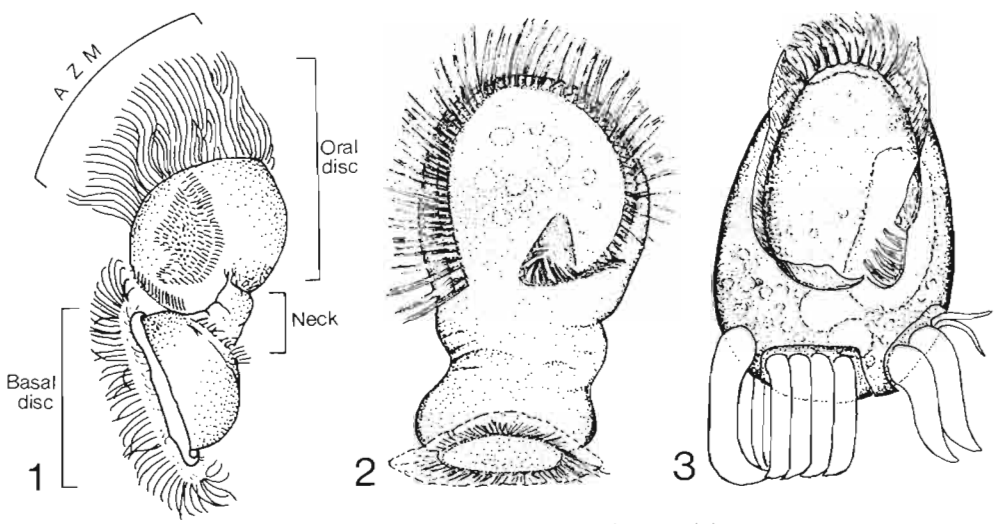


Fig. 13-56: Ciliates of class Polyhymenophorea from marine bivalves. (1) *Licnophora auerbachi* from *Chlamys opercularis*; (2) *Licnophora* sp. and (3) *Uronychia bivalvorum* from *Thyasira sarsi*. AZM adoral zone of membranelles. (1 after Harry, 1980; 2 and 3 after Fenchel, 1965.)

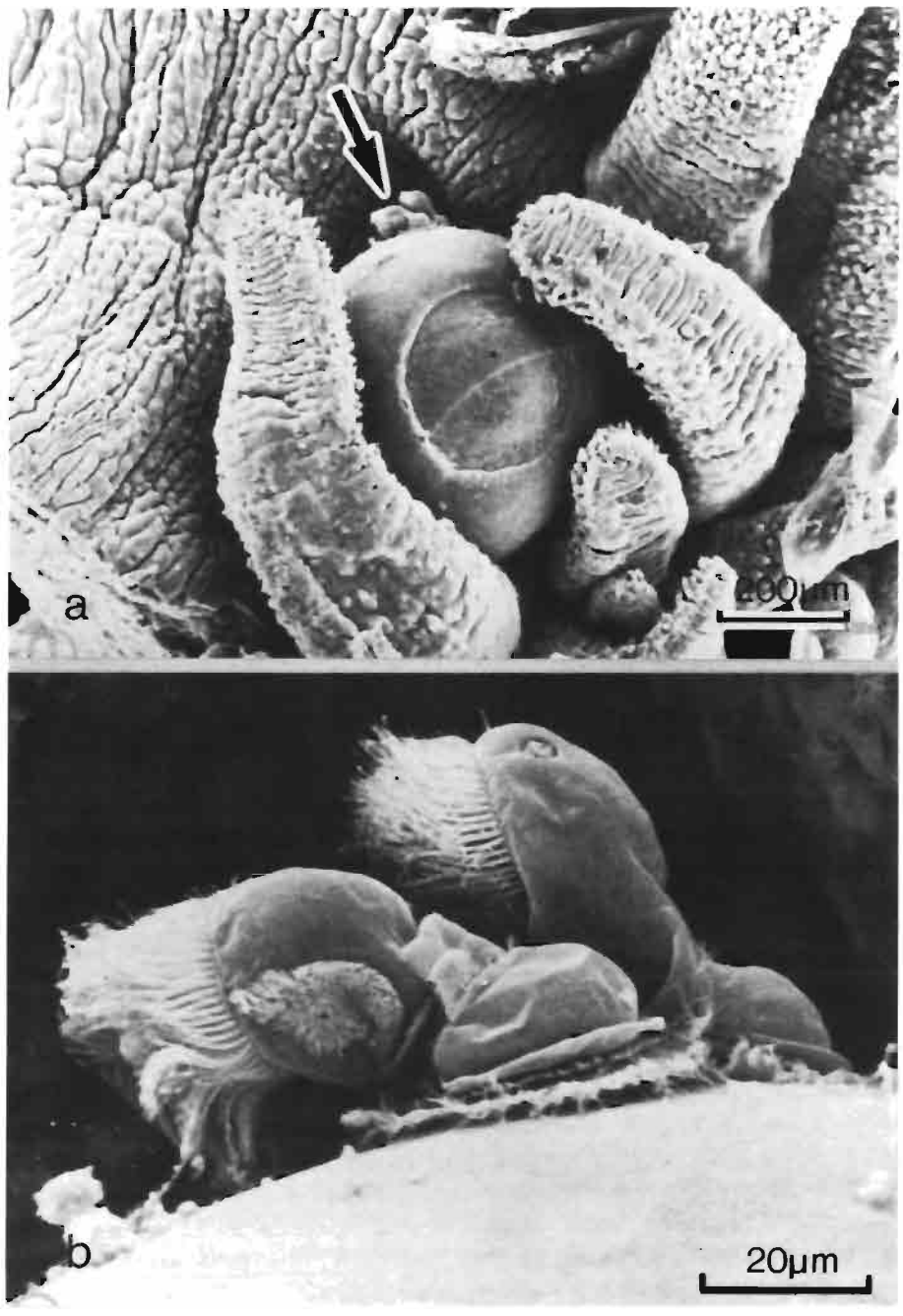


Fig. 13-57: *Chlamys opercularis*. (a) Scanning electron micrograph of eye and tentacles. Note 2 *Licnophora auerbachi* attached to eye (arrow). (b) Close-up (lateral view) of *L. auerbachi* individuals shown in (a). (After Harry, 1980.)

In Strangford Lough, Northern Ireland, 50 of 58 and 85 of 88 Queen scallops, respectively, were found to harbour varying numbers of *L. auerbachi*. As many as 120 occurred on individual eyes, all of the about 100 eyes around the mantle margin of some hosts being infested. One randomly selected and moderately infested scallop carried a total of 1,748 *L. auerbachi* on 92 of its 103 eyes (Fig. 13-58). Nine of the eyes of another host supported between 70 and 100 ciliates each (Harry, 1980).

Like *Trichodina*, *Licnophora* is normally a filter-feeder probably thriving on bacteria. As shown experimentally, it would also feed on killed stained yeast cells (Harry, 1979).

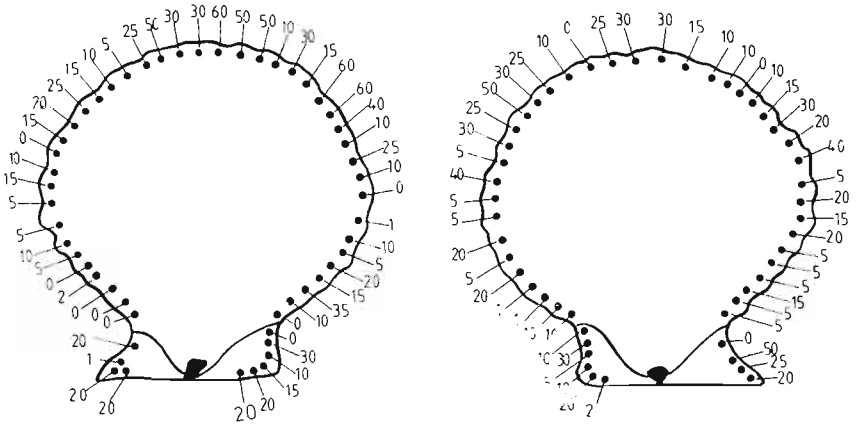


Fig. 13-58: *Chlamys opercularis*. Number and distribution of *Licnophora auerbachi* (n = 1,748) on all the individual eyes (n = 103) on the left (upper) and right (lower) mantle margin of a moderately heavily infested scallop. (After Harry, 1980.)

However, in a large number of *Licnophora auerbachi* removed from *Chlamys opercularis*, black pigment granules of host origin were visible in the food vacuoles in the region of the feeding membranelles. Ciliate-populated eyes showed loss of pigment in the region of the iris, and some heavily invaded eyes exhibited signs of disintegration (Fig. 13-59, b). It seems that the dual effect of the ciliary bands of the basal disc and the clamping action of the rough abrasive surface of its central portion (Fig. 13-59, d) causes mechanical damage to the epidermis of the eye. In the attached state, the basal disc appears to exert considerable suction, as it leaves a distinct 'footprint' at the site of attachment (Fig. 13-59, c). Although *L. auerbachi* is highly motile, undergoing exploratory excursions from time to time, it tends to return to the same position before reattaching to the eye epidermis. Forcible removal from the host invariably damages the ciliate.

Mechanical injury to *Chlamys opercularis* may be quite extensive if the number of *Licnophora auerbachi* is high. Attachment of the ciliates' basal disc causes opacity and hypertrophy of the columnar epithelial cells covering the cornea, and the clear glassy lens may assume a fuzzy and irregular appearance. Heavy infestation may result in entire disintegration of affected eyes.

Individual *Chlamys opercularis* eyes, surgically removed from scallops and kept in cool, filtered sea water, retain their structure for up to 9 days, and *Licnophora auerbachi* continues to feed and reproduce on these eyes. When the eyes eventually distintegrate and a freshly removed eye is brought into contact with a degenerating one, the ciliates jump

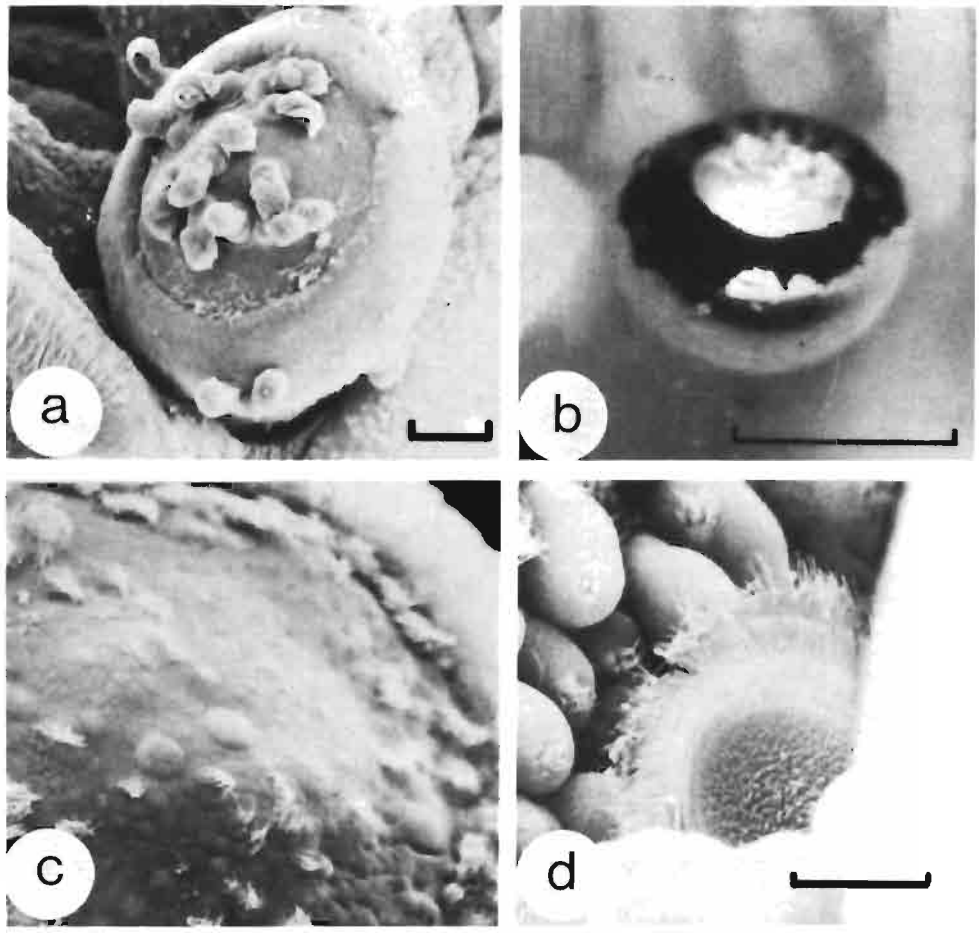


Fig. 13-59: *Chlamys opercularis* infested with *Licnophora auerbachi*. (a) Individual eye supporting 18 ciliates. Note their occurrence on both lens and iris. Bar 100 μm. (b) Eye with heavy infestation with 30 *L. auerbachi*. Note damage to edge of iris. Bar 500 μm. (c) Basal disc impressions ('footprints') left on eye after detachment of ciliates. (d) Underside of basal disc of *L. auerbachi* showing bands of cilia and rough abrasive surface of central disc. Bar 20 μm. (After Harry, 1980.)

from the donor to the recipient eye in one rapid movement (Harry, 1977). The possibility to cultivate *L. auerbachi* continuously on eyes removed from hosts at weekly intervals clearly qualifies *L. auerbachi* as a parasite, rather than as a commensal. Since the eyes of *C. opercularis* are not capable of image formation, it was considered unlikely that the presence of ciliates would impair their visual response to any great extent. Destruction of individual eyes may also be a minor problem because these organs are capable of regeneration. However, *L. auerbachi* might have a significant effect on young developing scallops (Harry, 1977, 1980).

Although G. Williams (1954) has recorded *Licnophora auerbachi* from the tentacles of individuals of *Pecten maximus* from Northern Ireland, it appears to show a distinct preference for *Chlamys opercularis*. Harry (1977) found only a few individuals of the 2 closely related scallop species *P. maximus* and *C. varia* from Strangford Lough to be

infested. Attempts to induce *L. auerbachi* to move *in vitro* from degenerating eyes of *C. opercularis* to freshly removed eyes of the above species revealed a remarkable reluctance of the protozoans to move to the 'wrong' recipient eyes. In all trials, *L. auerbachi* would move *en masse* to surgically isolated eyes of *C. opercularis* immediately after they had rejected those of the other scallop species.

Heterotrichous ciliates, believed to be *Licnophora auerbachi,* have also been reported from bivalves *Tellina exigua,* as well as from other marine invertebrates including nudibranchs and echinoderms (Villeneuve-Brachon, 1940). *Thyasira sarsi* is host for another, specifically unidentified ciliate of the genus *Licnophora* (Fig. 13-56, 2), about 10 % of the clams from Gullmar Fjord on the west coast of Sweden being heavily infested. A *Licnophora* species was also present in 2 of 115 *Abra nitida* from the same region. Whether both forms are identical, is unknown (Fenchel, 1965). Another specifically unidentified *Licnophora* is a rare inhabitant of the mantle cavity of wood-boring bivalves *Martesia striata, Teredo furcifera* and *Nausitora hedleyi* from the southwest coast of India (Santhakumari and Balakrishnan Nair, 1973).

Thyasira sarsi from Gullmar Fjord also harbours hypotrichous ciliates *Uronychia bivalvorum* (Fig. 13-56, 3). Infestation incidences varied between 25 and 50 %, and intensities ranged from 1 to 16 ciliates per clam. Both incidence and intensity of infestation were somewhat higher in *T. flexuosa* from Øresund (Fenchel, 1965). The nature of the association has not been studied in detail. Other species of *Uronychia* are free-living (Kahl, 1933).

Large astomes *Anoplophrya mytili,* about 120 μm long and of doubtful taxonomic status, have been reported on a single occasion from the mantle cavity and intestine of *Mytilus edulis* from Arcachon, France (Delphy, 1938). Anoplophryids are normally known as endosymbiotes of terrestrial oligochaetes.

Agents: Protista *incertae sedis*

Mass mortalities or serious diseases of marine bivalves, suspected to be caused by unidentified micro-organisms and protistans, have been witnessed for decades in various parts of the world. Some continue to hamper today's commercial shellfisheries. However, most of the previously obscure disease agents have been identified in the past few years, mainly by the aid of electron microscopy. These organisms have been discussed in the preceding sections of this review.

A disease of still unknown etiology, affecting Pacific oysters *Crassostrea gigas,* is 'Denman Island disease'. It is characterized by deep pustules on the surface of the body and mantle and/or pus-filled sinuses (Fig. 13-60) which, however, may not be the primary manifestation of the disease, since pustule formation seems to be an unspecific reaction of oysters to many forms of stress (Quayle, 1961, 1969; Sprague, 1971).

Thus far, neither viruses nor inclusion bodies of any kind have been detected in oysters affected by Denman Island disease. Instead, a peculiar cell type (so-called microcells), which may be a life-history stage of an unknown pathogen, has been found in intracellular, as well as extracellular location in diseased individuals. Similar microcells occurred in European oysters *Ostrea edulis* planted experimentally in California (Lindsay, 1969; Katkansky and co-authors, 1969a; see below).

The disease was first observed in the area of Henry Bay on Denman Island in Baynes

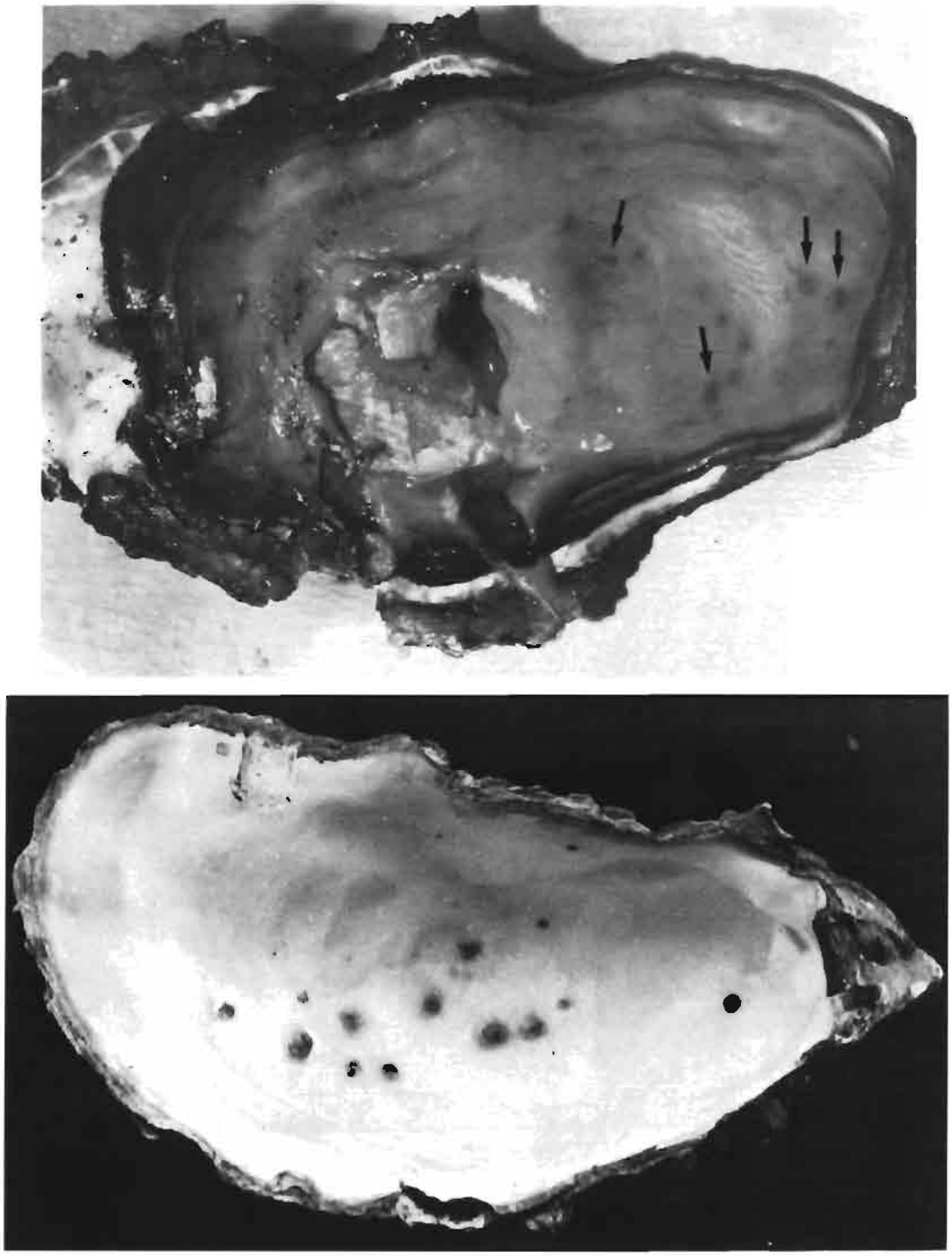


Fig. 13-60: *Crassostrea gigas*. Pustules caused by 'Denman Island disease', appearing as dark focal areas on mantle (arrows, top) and corresponding lesions on inner shell surface (bottom). (After Quayle, 1969.)

Sound, British Columbia (Canada) in 1960. In this area, oysters were nearly always in excellent condition, and affected individuals also had a very high condition factor. The mortality from the initial outbreak in 1960 was estimated to be more than 30 %. In 1961, much the same pattern recurred in Henry Bay, but the disease was also reported from adjacent areas, namely from Ladysmith Harbor and from Crofton, but not from more remote places. There were no reports of the disease from other areas in subsequent years. It therefore appears to be endemic to locations along the east coast of Vancouver Island.

Denman Island disease usually develops in early spring at water temperatures above approximately 8 °C, and disappears by about mid-July when water temperatures have reached about 18 °C. Highest mortalities occur in oysters at lowest tide levels. Older age groups, from two years onward, seem to be affected most heavily. Laboratory tests have demonstrated the highly contagious nature of the disease (Quayle, 1961, 1969). Denman Island disease of Pacific oysters resembles Malpeque Bay disease affecting Virginia oysters on the Canadian Atlantic coast with respect to pustule formation, as well as in that 'microcells' have also been found in tissues of Malpeque Bay oysters (Sindermann, 1977). However, the etiological agent of the latter disease has meanwhile been identified as a species of *Labyrinthula* (see section 'Agents: Labyrinthomorpha').

Comps and co-authors (1980) noted superficial similarities between *Bonamia ostreae* and the 'microcell' agent reported from *Ostrea edulis* imported into California (Lindsay, 1969; Katkansky and co-authors, 1969a), and identified as a serious pathogen (Glude, 1974). Because the available descriptions of the 'microcells' are merely based on examination with light optics, a detailed comparison with *B. ostreae* is impossible. However, the description given by C. J. Sindermann (in Katkansky and co-authors, 1969a) matches at least some of the peculiarities of the various life-cycle stages of the European flat oyster pathogen (see section 'Agents: Ascetospora'). The 'microcells' in *O. edulis* were 2 to 3 μm in diameter and had a single nucleus of about 0.8 μm. Binucleate cells were also seen. The organism was cytozoic within oyster haemocytes (as many as 15 cells were observed in a single haemocyte) or occurred free in the haemolymph.

Oysters affected by 'microcells' were thin, watery and transparent. Histologically, they exhibited a generalized haemocytic infiltration which was particularly evident in the area of the digestive tubules. The latter generally lacked the normal crypt structure, with the epithelial cells being reduced to a low cuboidal epithelium. Prevalence of 'microcells' was 14/48 in oysters from Morro Bay and 11/19 in oysters from Elkhorn Slough. Gapers becoming available during the studies were all infested. Of 18 oysters from Tomales Bay, none had 'microcells'. Survival of *Ostrea edulis* at the latter site was considerably higher than in the other study areas. 'Microcells' were also found in individuals of *Crassostrea angulata* within 2 months after exposure to infested *O. edulis* but did not occur in stocks of *C. gigas* adjacent to infested European oysters (Katkansky and co-authors, 1969a).

Another microcell parasite, identical with the above-described with respect to size and location in host haemocytes, has been observed in a single out of 5 *Argopecten irradians* from Millstone Point, Connecticut. The heaviest concentration of parasite-containing blood cells was in the gonad where large macrophages appeared to have replaced the testicular and ovarian follicles (Hillman and Marconi, 1980).

Another microparasite has been recovered from moribund individuals of *Pinctada maxima* from Northern Australia. It consists of spherical bodies, 1.8 to 3.3 μm (mean 2.62 ± 0.43 μm, n = 60) in diameter, found in large quantities within epithelial cells of

digestive diverticula. The bodies, believed to be 'spores', have a thick, brownish wall, stain very faintly yellow with H & E stain and light pinkish-brown with PAS, the latter being regarded as a negative reaction. When sections stained with acridine orange were viewed under incident fluorescent light, many 'spores' had a circular spot on the wall resembling an aperture. Inside was an amorphous spherical body that did not fill the lumen, or there were 3 to 4 smaller spherical bodies. These stained red with safranin and Gomori and orange with acridine orange (Wolf and Sprague, 1978).

First signs of infestation are mantle shrinkage and cessation of feeding. Tissue damage associated with the microparasite was intense, and it was believed to be the causative agent of epizootic pearl-oyster mortalities observed in northern Australia. Up to 50 % of the *Pinctada maxima*, kept on rafts after collection from natural beds, died during an outbreak of the disease. *P. maxima* is of commercial importance because it produces large culture pearls. First considered a fungus, the organism was subsequently grouped with the *Protista incertae sedis* (Wolf and Sprague, 1978; Wolf, 1979).

According to P. H. Wolf (pers. comm.), F. O. Perkins, who restudied the microparasite, was not able to obtain clear electron micrographs of the spherical bodies which had no nucleus and contained only diffuse, electron-dense material. Perkins does not believe that these bodies have anything to do with the observed *Pinctada maxima* mortalities. Wolf, on the other hand, saw the enigmatic structures only in diseased pearl oysters. Japanese workers tend to associate the mortalities with the presence of a *Vibrio* species.

Similar or identical spherical bodies, 2 to 3 μm in diameter, were seen in large numbers in the digestive-gland epithelium of diseased individuals of *Pinctada margaritifera* from Dongonab Bay, Sudanese Red Sea coast. Each of these consisted of an indistinct wall and 1 to 4 rounded bodies inside. Only mature pearl oysters were affected and suffered heavy mortality. Epizootics, presumably caused by this unidentified microparasite, occurred in Dongonab Bay in 1969 and 1973 (Nasr, 1982). According to P. H. Wolf (in Nasr, 1982), the spherical bodies in the Sudanese *P. margaritifera* are identical with those observed in Australian *P. maxima*.

Another 'protistan *incertae sedis*' has been observed in the cytoplasm of maturing ova of individuals of *Crassostrea gigas* from Humboldt Bay, California. Earliest observable stages are solitary, unencysted, uninucleate amoebulae, irregularly oval and about 2.5 μm in diameter (Fig. 13-61, 1). These are found primarily in the cytoplasm of host ova, but a few may occur in the surrounding connective tissue and rarely in the epithelium lining the digestive tract. They probably represent the initial invasive stage. The single amoebula nucleus is located eccentrically, spherical in shape, basophilic and strongly Feulgen-positive.

All other vegetative forms are encompassed by a cyst. Their nuclei are basophilic, Feulgen-positive and PAS-negative. Subsequent development proceeds primarily by successive division into about 8 to 16 advanced stages. No plasmodia were seen. Later stages comprise ovoidal to spherical cysts with nearly indistinct membranes, measuring up to 18.0 × 20.0 μm (Fig. 13-61, 10). Two to 4 cysts per ovum are common, but up to 10 have been observed in eggs of heavily infested oysters. Large cysts may invaginate, but do not penetrate, the nuclear membrane of the host egg. The parasite superficially resembles *Steinhausia ovicola* and *S. mytilovum* but does not possess a multinucleate plasmodium or form mature spores (Becker and Pauley, 1968). The authors discussed its affinities with the Microspora and the 'Haplosporida' but were unable to assign it to any of these groups.

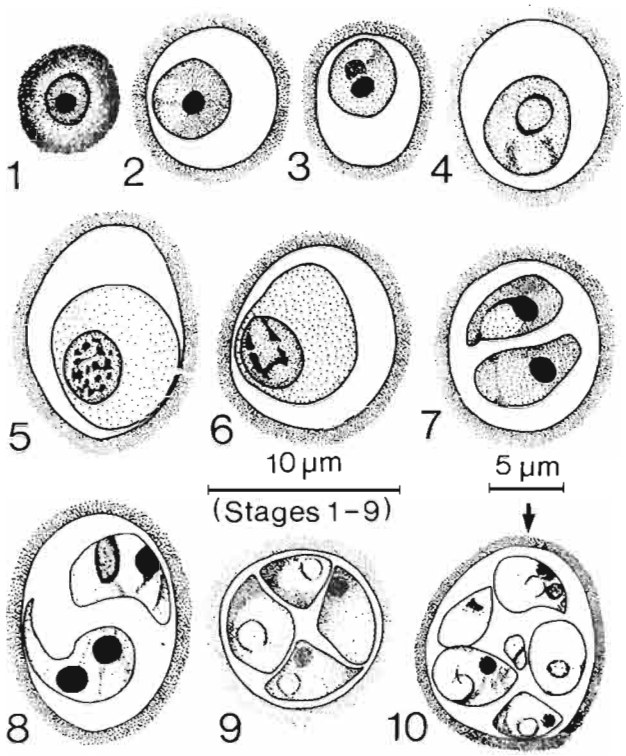


Fig. 13-61: Unidentified ovarian parasite from *Crassostrea gigas*. Developmental stages. 1: Uninucleate amoebula in host-egg cytoplasm; 2: slightly larger amoebula after cyst formation; 3: amoebula with trace of second nucleus; 4: amoebula with nucleus in ring formation; 5, 6: large amoebulae with vesicular nucleus; 7: primary (infestive?) cells arising from first division of amoebula; 8: binucleate cells resulting from first division; 9: early developmental stages arising from first division of quadrinucleate amoebula; 10: advanced developmental stage illustrating variation in size, shape and staining reaction of cells. (After Becker and Pauley, 1968.)

In Humboldt Bay oysters, infestation incidences varied from 44 to 71 %, but intensities were usually light. In most infested females, only between 1 and 10 % of the ova carried the parasite. Infested eggs sometimes were necrotic and elicited an inflammatory host response. There were no indications that the organism killed oysters. Seasonality of infestation, related to the reproductive cycle of *Crassostrea gigas*, appears likely but requires confirmation. Male oysters were never found to harbour the presumed prostistan (Becker and Pauley, 1968).

A serious disease was found to affect hatchery-reared larval *Crassostrea gigas* in Washington (USA). The earliest diagnostic sign in seemingly healthy larvae was the appearance of numerous zoospore-like bodies on the velar cilia. As they progressively increased in number, the velum and mantle became eroded. Concomitantly, thick-walled spheres, 4 to 24 µm in diameter and with an eccentric vacuole, appeared in the stomachs of 7-day-old umbo larvae. Morphologically, these spheres resembled the hypnospores (aplanospores) of *'Dermocystidium marinum' (= Perkinsus marinus)*. Moribund larvae were seen to regurgitate fascicular collections of the zoospore-like and thick-walled spherical bodies from the stomach. The terminal stage of the infestation was characterized by

general necrosis of the mantle and velum, complete loss of motility, numerous enlarged spherical bodies in the stomach, dilated darkened and sometimes ruptured intestines and, eventually, necrosis of the other soft tissues.

During optimal periods of growth, when larvae completed metamorphosis prior to 12 days of age, mortality was negligible. When, however, metamorphosis was delayed beyond day 12, the mortality rate increased, predominantly in larvae in the 140 to 150 μm size range, regardless of their age. In the latter group, losses often exceeded 90 %, and surviving larvae from these cultures exhibited growth retardation. Total mortality, for all affected presetting hatches, ranged from less than 5 % to over 95 %.

The disease, which appeared to be limited to presetting-stage oyster larvae, occurred at water temperatures of 20 to 30 °C and at salinities of 13 to 28 ‰ S. Hatchery records indicated its exacerbation with increasing water temperature during spring and summer, and remissions during the colder seasons. Although only 1 sample from a given larval hatch was examined from each of 3 additional hatcheries and the wild stock, all samples contained umbo larvae with signs similar to those described from acutely affected larvae (Leibovitz and co-authors, 1978).

The infestive zoospore-like bodies observed on the velar surfaces of the oyster larvae were believed to represent motile stages of an aquatic fungus. The spherical bodies from the larval stomachs appeared to be morphologically similar to *'Dermocystidium' (Perkinsus)* and to *Hyalochlorella marina*, a marine fungus of dubious affinities (Leibovitz and co-authors, 1978; Elston, 1980b). However, unlike *Perkinsus marinus*, the *Crassostrea gigas* spheres have not been observed intracellularly. Goldstein and co-authors (1965) and Goldstein and Moriber (1966) described a *'Dermocystidium* sp.' which is free-living. Poyton (1970) transferred it to a newly erected taxon, *Hyalochlorella*, "a new colourless counterpart of *Chlorella*". Although presenting morphological similarities with some stages of *P. marinus*, the fine structure of the organism is sufficiently different to allow its exclusion from close relationship (Alderman, 1976). Whether Poyton is correct in allying it to *Chlorella* (a chlorophyte known from freshwater molluscs; see section 'Agents: Protophyta'), remains to be established.

Agents: Protophyta (Algae)

Numerous associations exist between marine bivalves and algae; their nature ranges from mutualism (as in the zooxanthellae of *Tridacna*) to true parasitism (as in *Coccomyxa* living in *Placopecten magellanicus*). The Cyanophyta and Chlorophyta contain species that perforate calcareous substrata, including shells of dead and living pelecypods.

Algal burrowing is a dissolution process accomplished by the terminal cells of the endolithic filaments. The space dissolved away by an alga has the shape of a miniature calcite crystal. The directions of penetration, as well as the fine sculpture of the inner surface of the excavated tunnels, are largely controlled by the mineralogical properties of the substratum. Taxonomically distinct algae produce specific boring patterns which can be recognized in the absence of the plants (Golubic, 1969). Although direct damage resulting from algal penetration of bivalve shells is usually negligible, the burrowing plants create minute crevices, roughen the outer shell surface, and thus favour attachment and penetration by other fouling organisms. Frequently, 'shell disease' of oysters, caused by the fungus

Ostracoblabe implexa (see section 'Agents: Fungi'), is accompanied or preceded by the action of burrowing algae (Alderman and Jones, 1971a).

The mechanism of algal penetration into calcareous substrata is not yet readily understood. It may involve a combination of softening of the calcium carbonate due to the action of CO_2 or other acids produced in metabolism, and growth pressure (Ranson, 1955; Newell, 1956; Yonge, 1963).

Bornet and Flahault (1888, 1889) described blue–green algae of the genera *Hyella, Mastigocoleus* and *Plectonema*, as well as green algae of the genera *Gomontia* and *Ostreobium*, from 'coquilles marines' collected on the French Atlantic coast. Unidentified perforating algae were seen in marine bivalve shells by Kölliker (1860a, b) and Peyer (1945). Marine burrowing (endolithic) algae are ubiquitous in shallow-water carbonate substrata and are important bioerosive agents involved in the breakdown of animal-hard structures and limestone (Nadson, 1900; for literature see Peyer, 1945, and Kobluk and Risk, 1977).

Cyanophytes *Entophysalis deusta, Plectonema* spp. and *Hyella* spp., chlorophytes *Eugomontia sacculata* and *Gomontia polyrhiza*, as well as the *'Conchocelis'* phase of the rhodophyte *Porphyra purpurea*, are common inhabitants of oyster shells, giving a brown or green stain to the affected areas. Although these algae penetrate deep into the shell, they have never been observed to cause any harm to the living oyster (Prud'homme van Reine and van den Hoek, 1966; Alderman and Jones, 1971a).

Empty shells of *Cardium edule* and *Mya arenaria* from Laesø (Kattegat) were found to harbour shell-burrowing cyanophytes *Hyella balani, Mastigocoleus testarum* and *Plectonema terebrans*, as well as chlorophytes *Eugomontia sacculata, Gomontia polyrhiza, Phaeophila dendroides* (Fig. 13-62) and *P. tenuis*. The occurrence of some of these algae

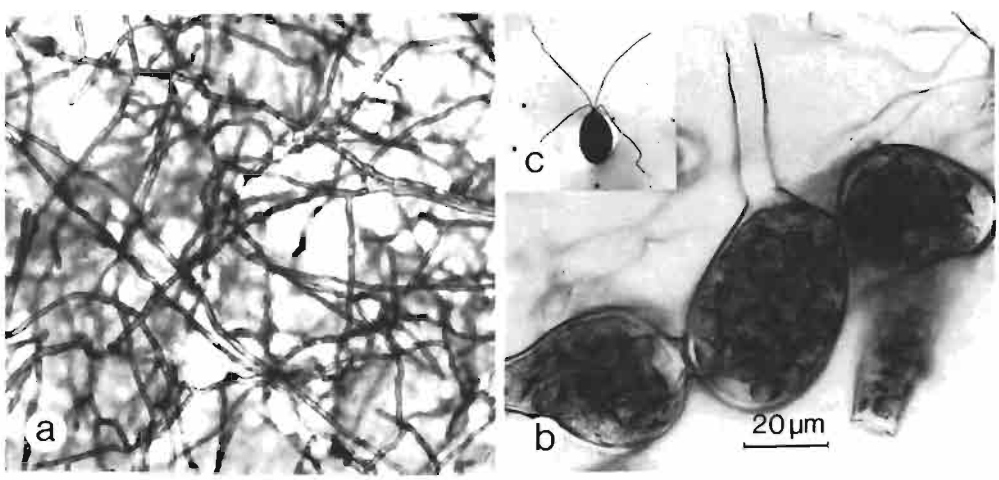


Fig. 13-62: *Phaeophila dendroides*. (a) Plants ramifying in oyster shell; (b) sporangia with zoospores; (c) liberated zoospore. (After Nielsen, 1972.)

showed a distinct correlation with wave exposure (Nielsen, 1972). *Gomontia polyrhiza*, previously named *Codiolum polyrhizum* (Figs 13-63 and 13-64), may be found in dead bivalve shells from the North Sea and European Atlantic coasts (Kornmann, 1959; Kornmann and Sahling, 1977). This alga frequently occurs together with *Eugomontia*

sacculata (Fig. 13-65) in the same shell (Kornmann, 1960). Both had been mistaken by Bornet and Flahault (1888, 1889) for stages of one and the same species. *G. polyrhiza* and *E. sacculata* have been found by the reviewer in posterior shell portions of living *Mya*

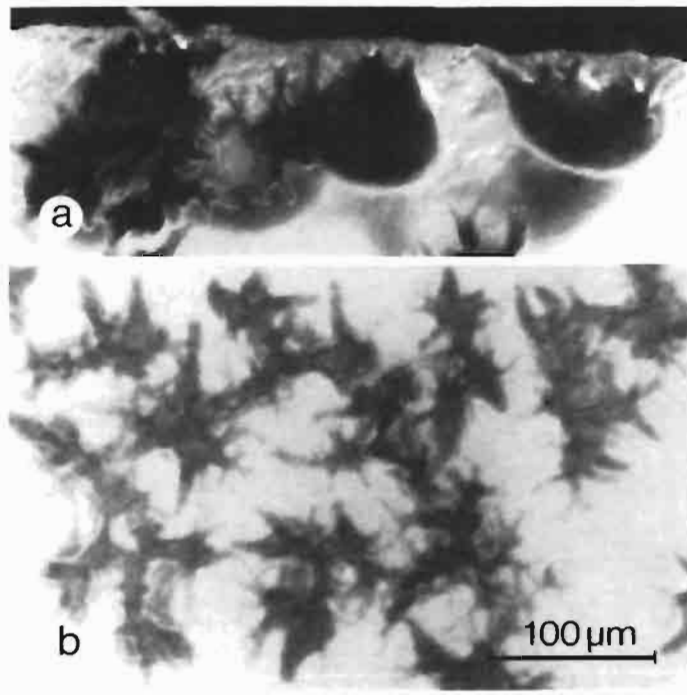


Fig. 13-63: *Gomontia polyrhiza*. Sporophytes (23-day-old) growing in thin bivalve-shell fragment. (a) Shell margin showing position of sporophytes; (b) surface view showing irregular stellate outlines of sporophytes visible through superficial translucent shell layer. (After Kornmann, 1959.)

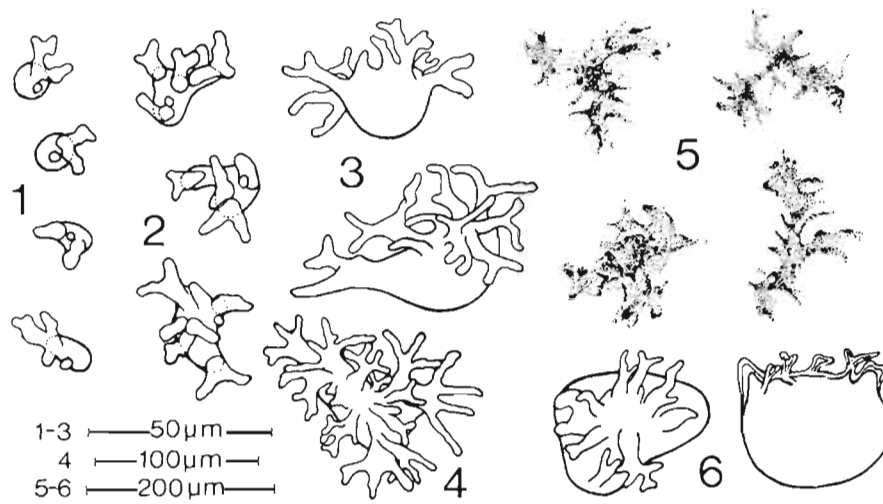


Fig. 13-64: *Gomontia polyrhiza*. Sporophytes cultivated in bivalve-shell fragment. 1–5: Living individuals (1–4: 5-, 8-, 12-, and 19-day-old; 5, 6: 8-week-old); 5: surface view; 6: same after decalcification. (After Kornmann, 1959.)

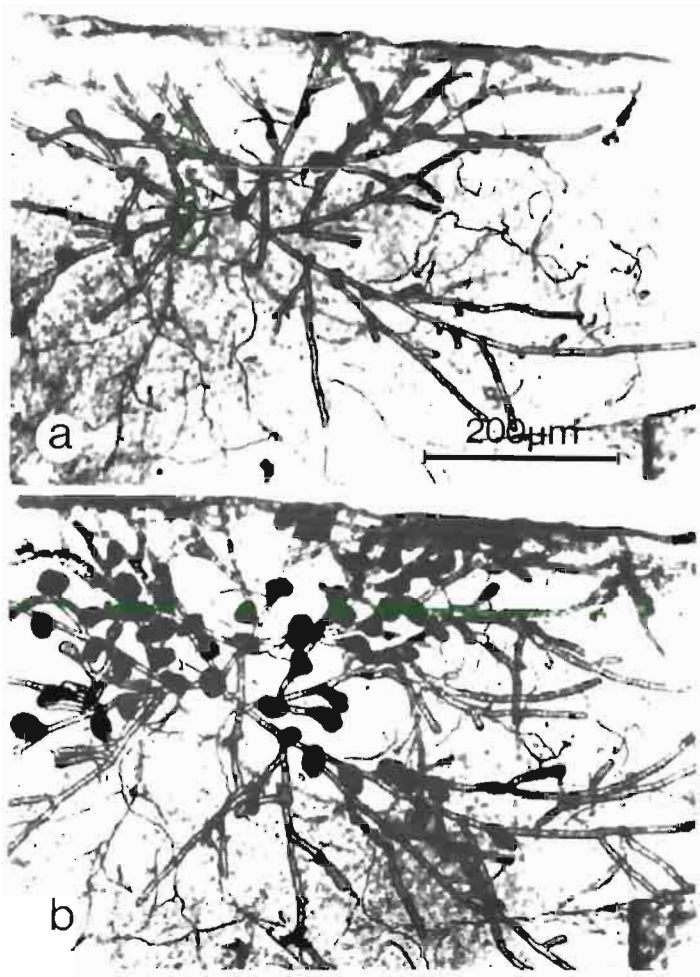


Fig. 13-65: *Eugomontia sacculata*. Sporophytes cultivated in bivalve-shell fragment. (a) 5-week-old; (b) 6-week-old. Note conspicuous dark sporangia. (After Kornmann, 1960.)

arenaria, which had partly been uncovered by strong water movement and were thus exposed to the light. In living bivalves, algal perforation is mainly restricted to shell areas in which the protecting periostracum has worn away.

Ostreobium queketti (Fig. 13-66), first described from dead molluscan shells by Bornet and Flahault (1889) and grouped with the Siphonales, was subsequently assigned to various taxa, until Kornmann and Sahling (1980) reisolated the alga from dead shells collected off Helgoland and definitely attested, by means of culture experiments and life-cycle studies, its position in the Codiales (Chlorophyta). *O. queketti* is believed to be worldwide in distribution and to burrow into a large variety of carbonate substrates (Lukas, 1974), but Kornmann and Sahling (1980) doubt the conspecificity of most of these poorly described forms.

'Conchocelis rosea', another carbonate-perforating alga isolated from molluscan shells by Batters (1892), was believed by Nadson (1900) to represent a red-coloured form

Fig. 13-66: *Ostreobium queketti.* Older thallus cultivated in bivalve-shell fragment. (After Kornmann and Sahling, 1980.)

of *Ostreobium.* Drew (1949), however, identified it as a stage in the life cycle of the red alga *Porphyra umbilicalis. Monostroma grevillei,* a green alga, has a similarly unusual heteromorphic life cycle. Its unicellular sporophyte burrows into molluscan shells, while its large gametophyte resembles *Ulva* or *Enteromorpha* in habit (Kornmann, 1962; Kornmann and Sahling, 1977).

Burrowing algae present in molluscan shells may attract limpets *Acmaea virginea,* which graze upon the plants, causing extensive erosion of the shells within the photic zone. Grazed shells may become wafer thin and highly fragmented. The faecal pellets of the gastropods contain 5- to 10-µm-sized carbonate particles, which contribute greatly to the production of fine carbonate mud in the Firth of Clyde, Scotland (Farrow and Clokie, 1979).

Some plants occurring in molluscan shells may, in fact, be the algal component of lichens, particularly of *Arthopyrenia sublitoralis, Verrucaria* spp., or related forms. *Ostracoblabe implexa* and *Lithopythium gangliiforme* (see section 'Agents: Fungi') were believed to be the fungal partners in some of these symbioses (Bornet, 1891). Santesson (1939) erroneously listed *O. implexa* as a synonym for *A. sublitoralis.* According to Johnson and Sparrow (1961), the 'marine lichen problem' is still unsolved and requires further study.

Arthopyrenia sublitoralis was found to be fairly common in shells of living *Mytilus edulis* and dead *Mya arenaria* from Isefjord (Denmark). The presence of the lichen is indicated by numerous tiny, densely spaced holes, about 150 µm in diameter, from which sometimes yellowish tissue may protrude. On first view, the infections give the impression of attack by burrowing sponges, *Cliona* spp. (Rasmussen, 1973). Santesson (1939) lists records of *A. sublitoralis* from 61 species and 6 varieties of molluscs or cirripeds. Numerous other lichens are known to grow on calcified animal shells all over the world without causing any deleterious effects (Alderman, 1976).

Algae may also invade the soft parts of bivalves. Some are definitely symbiotes

(McLaughlin and Zahl, 1966). The association of zooxanthellae with *Tridacna*, for example, is well documented (Yonge, 1944, 1957). Similarly, the presence of zooxanthellae in the gills and mantle of Indopacific heart shells *Corculum cardissa* was interpreted as mutualistic (Kawaguti, 1950). At least 1 species, *Coccomyxa parasitica*, appears to be (facultatively?) parasitic in a marine bivalve.

The algae, first seen as conspicuous colonies, 10 to over 300 μm in diameter, in *Placopecten magellanicus* from Newfoundland (Canada), were initially believed to be symbiotic zoochlorellae (Naidu and South, 1970). Subsequently, however, the relationship was recognized as being parasitic (Naidu, 1971). Infestations usually involved the peripheral mantle tissue but were also seen to extend into the distal end of the gonad and the bases of the posterior adductor muscle. While light but visible infestation apparently did not cause any easily discernible damage, severe symptoms were seen in heavy cases. The scallops developed a gradual decline in the condition of the visceral mass including the adductor muscle, i.e., the portion of the scallop that is marketed. The ability to retain free water between the valves decreased, and there was a gradual loss in total body weight. In extreme cases, the adductor muscle became atrophied and stringy, and declined significantly in weight. Affected tissues, especially the mantle, became slippery due to the presence of large amounts of mucus, which had a musty odour. Heavily infested *P. magellanicus* were unable to maintain tight shell closure. The difference between adductor muscle weights in infested and non-infested individuals was statistically highly significant. Differences between adductor muscle scar areas in both groups were significant at the 5 % level. Histopathological changes in the mantle tissue comprised encapsulation of the algal colonies by a network of connective-tissue fibres. However, there was neither evidence of impairment of normal gametogenesis nor of unusually high mortality in infested scallops. As an apparent consequence of the disturbance of the mantle-tissue metabolism, shell deformities may occur in infested scallops, which consist of the production of extra lips, i.e., newly secreted shell margins. Some individuals had grossly deformed valves. Of 183 individuals with laminated shell edges, 179 contained algae.

Of a total of 2,962 giant scallops examined, an average of 17.6 % were found infested. Percentages increased with increasing host age, reaching a maximum (44.5 to 56.4 %) in 8- to 10-year-old individuals, and diminishing gradually in older ones. Scallops 3 years old or younger never had algae. Incidences decreased slowly from May to November, suggesting a possible correlation with ambient underwater-light intensity. Scallops from deeper water were particularly free from infestation.

The causative agent, restudied by Stevenson and South (1974, 1975), was identified as a new member of the Coccomyxaceae, Chlorococcales, and named *Coccomyxa parasitica*. The unicellular organism measures from 1 to 11 μm in length (mean of 50 cells 5.5 × 3.0 μm) and is very variable in shape from spherical to elliptical, ablong or sickle-shaped. Chloroplasts occur usually singly, but up to 3 may be present. Pyrenoids are absent; the cell wall lacks cellulose. Reproduction occurs by the formation of 2, 4 or 8 (16) autospores. The alga could readily be cultivated in a variety of liquid and semi-solid media.

Phagocytosis of *Coccomyxa parasitica* cells, accomplished by agranular and granular haemocytes of different types, occurs throughout the infestive process and was also demonstrated experimentally using cultivated algae. Evidence was presented showing that the alga enters the bivalve via the normal feeding and digestion processes, the symbiote being highly resistant to digestion. Host phagocytosis contributes to the spread of the algal

cells. Greatest concentrations occur in the mantle region and appear to be related to the circulatory system (Stevenson and South, 1975).

Two other members of the genus *Coccomyxa, C. ophiurae* and *C. astericola*, have been reported as parasites of echinoderms (Mortensen and Kolderup Rosenvinge, 1910, 1933). The fact that *C. parasitica* could readily be grown on inorganic media, led Stevenson and South (1974) to assume that its relationship with *Placopecten magellanicus* is at best a case of facultative parasitism.

An algal inhabitant, silimar to *Coccomyxa parasitica*, occurs in *Clinocardium nuttalli* from Yaquina Bay, Oregon (USA). Outwardly, the siphonal tissues of infested heart cockles appear to be stained green. Distinct colonies of algal cells are discernible in lightly infested individuals, but merge to form indistinct dark green masses in heavily infested hosts, and may spread from the siphonal areas to the surrounding mantle tissues and, occasionally, to the foot. Microscopic examination reveals masses of bright green single round cells, 2 to 7 μm in diameter. Smaller, young cells have a distinct cup-shaped chloroplast, while older, larger cells have a lobate, somewhat diffuse chloroplast. Reproduction is by simple division and by the formation of autospores containing 4, 8 or, rarely, 16 daughter cells.

Electron microscopy of algal cells *in situ* disclosed that nearly every cell was surrounded by a sometimes extremely thick layer of what appeared to be a secretion product of the algal cell, made up of many thin concentric layers (Fig. 13-67). The organism grew best on medium containing filtered 'mantle fluid' (extrapallial fluid?) only, or enriched

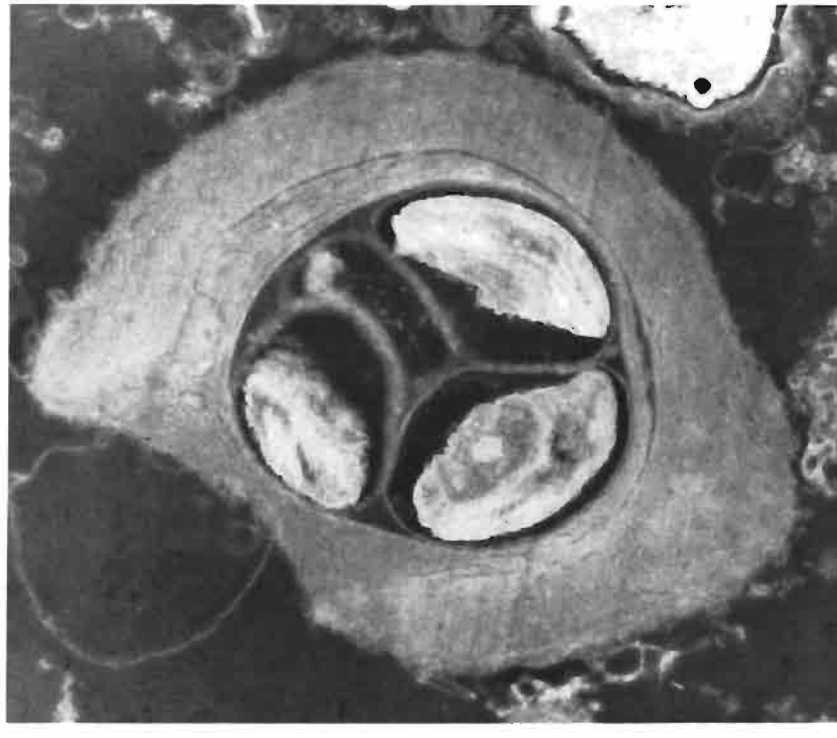


Fig. 13-67: *Chlorella* (?) sp. Dividing cell *in situ* in tissue of *Clinocardium nuttalli*. Note heavy concentric layers around cell, × 10,800. (After Hartman and Pratt, 1976.)

medium plus mantle fluid, which suggests a parasitic relationship, but cells multiplied also on inorganic medium. Experimental infestation of cockles with cultivated algal cells was successful. *In vitro* observations showed that the organisms are phagocytized by host haemocytes. Tissue invasion may result from diapedesis of phagocyte-engulfed algal cells across epithelial barriers.

Of a total of 1,290 *Clinocardium nuttalli*, collected over a 13-month period, an average of 35.0 % were found infested. Incidences were zero in cockles up to 2 years of age, but increased rapidly to reach 72.8 % in individuals 6 years or older. There was no distinct seasonality in the infestation pattern.

Although older, larger algal colonies grossly displaced the surrounding host tissue, shell closure and the ability to extend and retract the siphons were not hampered even in the most heavily infested cockles. Occasionally, colonial growth from mantle tissues into the extrapallial space was seen to stimulate the deposition of additional shell material in the infested areas. No other pathological changes have been reported.

On the basis of its morphology and an analysis of its pigments, the organism was tentatively assigned to the genus *Chlorella* (Hartman and Pratt, 1976). However, the authors were apparently unaware of the descriptions of parasitic *Coccomyxa* spp. Closer inspection and comparison might reveal the presumed *Chlorella* sp. to be a member of the latter genus. Zoochlorellae of the genus *Chlorella* are known from freshwater molluscs (Droop, 1963).

Infestation of bivalves by (pathogenic or non-pathogenic) histozoic unicellular algae appears to be a rather common phenomenon. 'Green oysters' and 'green-gilled clams' have beeen described on several occasions (Lankester, 1886; Mitchell and Barney, 1917; Ranson, 1927; Medcof, 1945; Kerswill, 1946; Wiborg, 1946). The causes of these discolourations are not always clear and may not invariably be due to tissue invasion by unicellular algae. Some of the above authors have attributed the discolouration to the absorption of pigments derived from algal food. Lankester (1886) found the gills of 'green oysters' to be covered with green-coloured granular 'secretion cells'. He later (1893) reclassified them as 'outwandered phagocytes'. Lipovsky and Chew (1972) interpreted green patches consistently seen in dying and dead *Crassostrea gigas* as a reaction to disease or an abnormal condition. Histologically, the discolouration coincided with concentrations of haemocytes just under the mantle epithelium. Boyce and Herdman (1897) reported on 'green leukocytosis' in diseased *C. virginica,* suspected to represent a degenerative process in which an increase of haemocytes was accompanied by a large deposition of copper. Ruddell (in Lipovsky and Chew, 1972) was able to experimentally produce similar conditions in *C. gigas.* Green mantles were characterized by the presence of large numbers of copper-bearing haemocytes in the process of migrating across the epithelium or already collected on the outside.

On several instances, the occurrence of green-gilled oysters has been traced to the uptake, by the oysters, of pigments derived from the digestion of diatoms *Navicula ostrearia* (Sauvageau, 1907a, b; Moreau, 1968; for bibliography see Moreau, 1970). Mitchell and Barney (1917) were able to experimentally produce green discolourations in the gills of *Crassostrea virginica* from Chesapeake Bay by maintaining oysters in water containing *N. ostrearia.*

Dinoflagellate blooms, commonly known as 'red tides', are of wide occurrence in coastal waters. 'Paralytic shellfish poison', derived from ingestion of toxic dinoflagellates

and accumulating in marine bivalves, is of considerable concern to human health. Although many invertebrates tolerate high levels of these toxins, mass mortalities due to 'red water' may occur (Coe, 1956; Stohler, 1960; Reish, 1963; Grindley and Nel, 1968; Ray, 1971; Alam, 1975; Dale and Yentsch, 1978; Schmidt and Loeblich, 1979).

Gyrodinium aureolum, one of the most common 'red-tide' dinoflagellates in northern European waters, causes a significant decrease in the filtering activity, as well as cytopathological and biochemical changes in *Mytilus edulis* (Widdows and co-authors, 1979). Dinoflagellate blooms may become an important threat to the expanding field of molluscan mariculture and should, therefore, be studied more closely in the future (Loosanoff, 1973). Paralytic shellfish poisoning and its consequences will be dealt with in greater detail in Vol. IV.

DISEASES CAUSED BY METAZOANS

Agents: Mesozoa

The phylum Mesozoa comprises a small group of multicellular endoparasites of marine invertebrates. Members of the order Dicyemida are common parasites of cephalopods while orthonectid mesozoans have been reported from representatives of various invertebrate phyla. With respect to their organization, the Mesozoa appear to occupy an intermediate position between the Protozoa and the Metazoa. Some workers, however, tend to regard them as highly modified relatives of Platyhelminthes.

Thorny jingle shells *Anomia aculeata* (*Heteranomia squamula*) from the Plymouth area (England) are hosts for orthonectid mesozoans *Rhopalura granosa* (Fig. 13-68). In the bivalve hosts, fusiform females, about 190 to 230 µm long and 55 to 75 µm wide, and

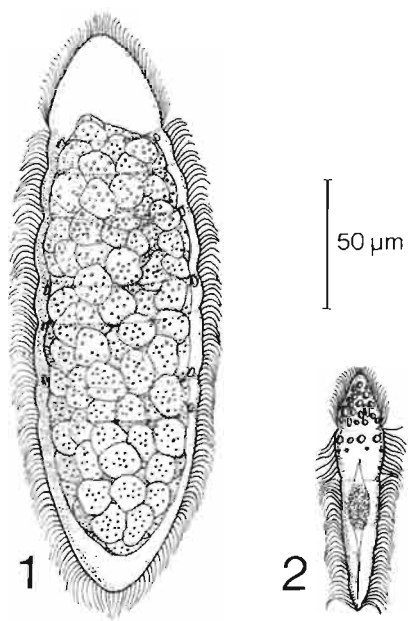


Fig. 13-68: *Rhopalura granosa* from *Anomia aculeata*. 1: Female; 2: male. (After Atkins, 1933a.)

males, 87 to 95 μm long and 20 μm wide, occupy primarily the gonads and sometimes the haemolymph spaces and vessels of the mantle and the gill filaments.

Male and female plasmodia usually inhabit separate hosts but sometimes both sexes may be found together. When this occurs, one sex generally dominates. Incidences of *Rhopalura granosa* varied from 6.6 to 16 % in field samples of *Anomia aculeata* but reached 45.4 % in individuals taken from tanks in the Plymouth Aquarium. In heavy infestations, the plasmodia containing the sexual forms of the parasite may entirely replace the gonad, rendering host sexing impossible (Atkins, 1933a).

Orthonectid mesozoans are primarily parasites of polychaete annelids and ophiuroid echinoderms. *Rhopalura philinae*, the only other orthonectid reported from molluscs (Vol. I, Chapter 12), does not castrate its host.

Agents: Porifera

Demospongiae of the family Clionidae are known for their capacity to burrow into calcareous substrates including the shells of dead and living molluscs. There are some 100 described species in the family. The genus *Cliona* with about 65 species is the best known of the 13 genera. Clionid sponges have been blamed for significant losses, particularly among oysters. Invasion by clionids is also a major controlling factor in the formation and maintenance of coral reefs and has been found to be responsible for large-scale coastal erosion (Goreau and Hartman, 1963; Neumann, 1966; Pang, 1973; Rützler, 1975; Vol. I, Chapter 6).

Although not being true parasites, burrowing sponges are serious pests of marine bivalves, particularly of oysters, which provide an ideal substratum for the activities of these destructive animals. When infested oysters reach the consumer, the sponge tissue concealed in their shells is usually dead and rotting, black and foul-smelling. This reduces market value or even makes heavily infested oysters unsaleable (Warburton, 1958c).

The taxonomic status of clionid burrowing sponges occurring along the Atlantic coast of North America has been reviewed by Old (1941), Warburton (1958a) and Wells and co-authors (1960). In ecological studies, Hopkins (1956a, 1962) demonstrated that the distribution of the shell-invading species, *Cliona celata, C. lobata, C. vastifica, C. spirilla* and *C. truitti*, in Louisiana, South Carolina and Virginia waters is governed by salinity. *C. celata* ist most abundant in high-salinity waters and does not occur in salinities below 15 ‰ S, while *C. truitti* exhibits higher prevalences in low-salinity areas. No such pattern has been found in North Carolina waters (Wells, 1959a). In Europe, *C. celata* appears to be the most abundant species affecting bivalves, particularly oysters (Giard, 1881; Korringa, 1952a; Cole, 1956a).

Osler (1826) was the first to recognize the role played by 'a kind of sponge' in the destruction of oysters at Swansea, Wales. His report appeared only a few months before Grant (1826) described *Cliona celata* occurring in oyster shells. Grant believed that his new 'zoophyte' lived in burrows excavated by worms. Hancock (1849, 1867), however, made it clear that the sponge bores its own holes. During penetration, the substratum is gradually destroyed as the sponge excavates an extensive system of confluent holes and tunnels. The cavities are filled with yellowish sponge tissue. Microscopic examination reveals typical siliceous spicules, mainly of the tylostyle type, 150 to 250 μm long, and

small skeletal elements (microscleres) of different size and shape (Nassonow, 1883; Topsent, 1887). Species identification is based on the type of cavities or galleries made by the sponge and on the shape and size of the spicules.

The manner in which clionids excavate calcareous substrates has been studied in considerable depth. Three major hypotheses on the mode of penetration have been presented: (i) Exclusively mechanical excavation of substratum; (ii) chemical dissolution of calcium carbonate by a presumed acidic etching agent; (iii) chemomechanical penetration — portions of substratum are chemically softened or loosened and subsequently removed mechanically.

Hancock's (1849, 1867) hypothesis, inferring that penetration is achieved by mechanical action of the sponge's spicules and other siliceous bodies — as well as Topsent's (1887) and Letellier's (1894) belief that it is due to traction exerted upon the host's shell material by the highly contractile sponge tissues — have been rejected by subsequent workers. Nassonow (1883) and Warburton (1958b) observed the removal of calcareous fragments by recently settled sponge larvae before the development of spicules and adhesive and contractile properties. They discussed the possible involvement, in the process, of some acid, but Topsent (1887), who observed that acid-resistant conchiolin is removed in the same way as calcium carbonate, abandoned this opinion. Cotte (1902) speculated on the combined action of acids and enzymes. Goreau and Hartman (1963) reviewed the older literature on sponge burrowing. Annotated bibliographies on marine burrowing organisms, published by Menzies (1957) and Clapp and Kenk (1963), as well as a number of symposium papers edited by Carriker and co-authors (1969), contain additional references to *Cliona*.

Today it is generally accepted that the mechanism of penetration is a combination of localized chemical dissolution coupled with mechanical dislodging of fragments or 'chips' of substratum and their subsequent discharge through the sponge's excurrent canals. Etching is accomplished by a special type of cell of probable archaeocyte origin. These etching cells, which undergo cytoplasmolysis during the process, are characterized by the presence of apical filopodia, interconnected to form a basket-like structure (Fig. 13-69, e, f). The filopodial basket penetrates the substratum by localized dissolution of calcium carbonate (W. R. Cobb, 1969, 1975; Rützler and Rieger, 1973; Pomponi, 1976, 1977; Hatch, 1980).

A number of ultrastructural similarities exist between the etching cells of sponges and the osteoclasts of vertebrates. This suggests functional similarities between the chemical mechanism of sponge boring and osteoclastic bone resorption (Pomponi, 1979a).

Etching appears to be primarily an enzymatic process. From the fact that only 2 to 3 % of the substrate broken down by the sponge goes into solution (Rützler and Rieger, 1973), it may be concluded that acids, if present, play a minor role. The chips removed are fairly uniform in size and about 40 to 60 μm in diameter (Fig. 13-69, a–d). The early observations by Nassonow (1883), Topsent (1887), Letellier (1894) and Cotte (1902), that shell material is broken down into solid chips, has notoriously been overlooked by Ginsburg (1957) and Revelle and Fairbridge (1957), who erroneously assert that all the excavated carbonate is removed by solution. Goreau and Hartman (1963) drew attention to the fact that large amounts of very fine silt are produced by clionids burrowing into coral and reef limestone. Superfluous siltation entails a variety of ecological consequences for the entire reef community. Similar siltation, although of lesser magnitude, must be

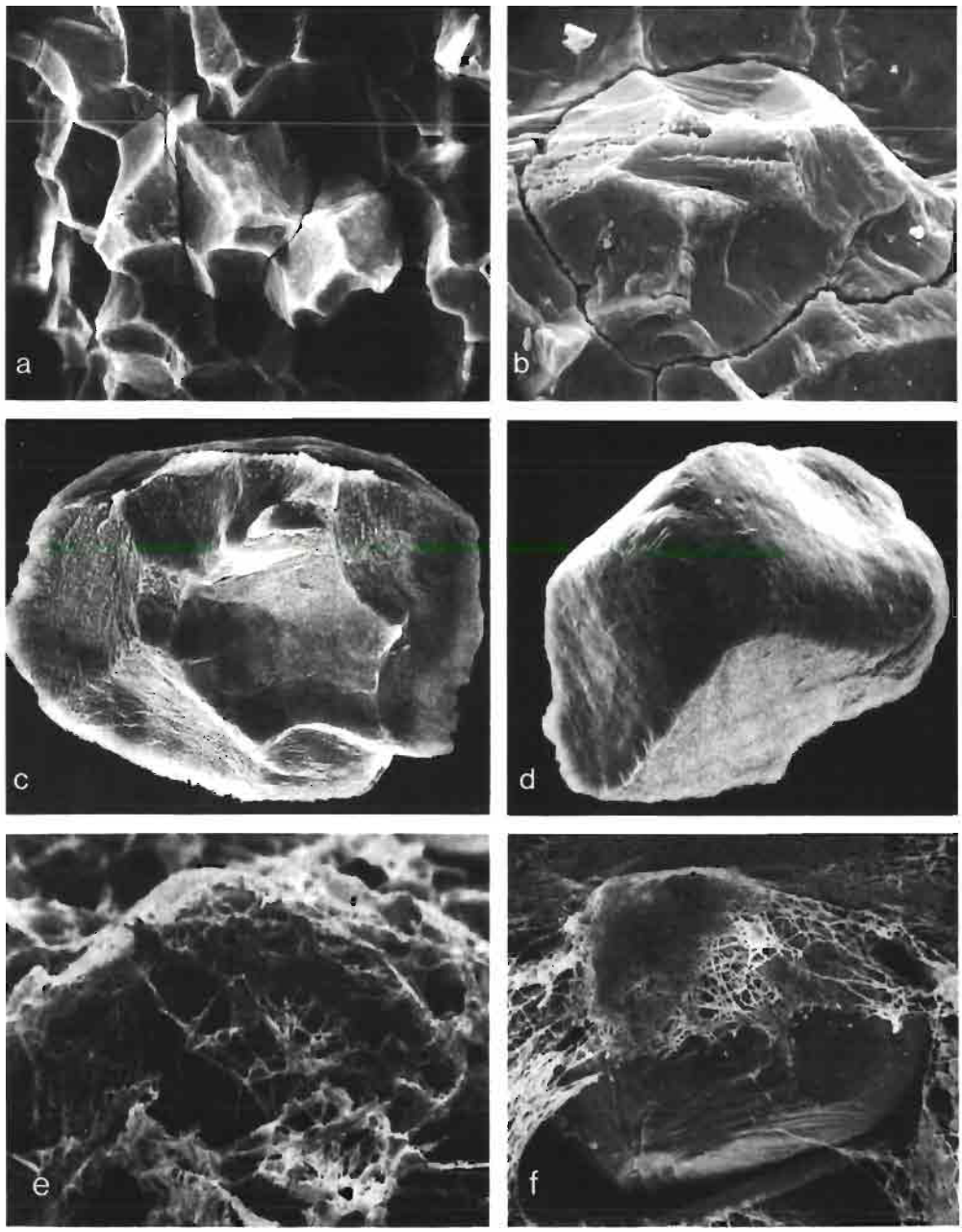


Fig. 13-69: *Cliona lampa*. SEM views of chips cored by sponge action. (a) Groups of chips etched from substrate but still in place, × 480. (b) Similar view, enlarged, × 1,280. (c), (d) Isolated chips discharged through oscular opening, × 1,200. (e) Freshly dislodged chip with adhering filopodial remains and secretory products, × 1,120. (f) Dislodged chip inside opening of exhalant canal. Part of tylostyle spicule visible at lower right, × 1,355. (After Rützler and Rieger, 1973.)

expected to result from the activity of shell-burrowing clionids, particularly on oyster beds. Apparently, the significance of this factor has not yet been studied in the field but laboratory experiments undertaken by Loosanoff and Tommers (1948) indicate that suspended anorganic silt adversely affects the filter-feeding activity of oysters. Similar negative effects of silt and other suspended matter on *Mytilus edulis* and *Cardium edule* have been demonstrated by Tammes and Dral (1955), Kristensen (1957), and others.

As stated, penetration by clionids appears to be accomplished by the localized action of enzymes. No acids have as yet been detected but Galtsoff and Pertzoff (1926), who titrated suspensions of *Cliona* and *Microciona*, found that the former bound less acid and more base than the latter. Enzymes involved in clionid etching are acid phosphatase and carbonic anhydrase. Acid phosphatase activity has been demonstrated in etching cells. It is most intense on the outer surfaces of the membranes of the filopodial processes where the enzyme is released, as well as in the extracellular channels between etching cell processes (Pomponi, 1979b). Acid phosphatase is recognized as a reliable marker for lysosomes when demonstrated by electron microscopic cytochemistry (Novikoff, 1963). The lysosomes represent a group of cytoplasmic vesicles, which occur in almost all cells, particularly in phagocytes, and synthesize a large number of enzymes capable of breaking down most intra- and extracellular (macro-)molecules (Dingle, 1973; Hers and van Hoof, 1973). From Pomponi's (1979b) study it would appear, therefore, that the etching cell is capable of enzymatic substrate digestion, both intracellularly and extracellularly, via the lysosomal system and the membranes of the filopodial extensions. The exact significance of the implication, in this process, of acid phosphatase and its possible interaction with other enzymes, are not known.

The fact that calcium carbonate is the primary mineral excavated by clionids may suggest a mechanism involving a localized modification of the $CaCO_3$ solubility equilibrium. An enzyme participating in shifting solubility equilibria by catalyzing the reaction $CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons 2\ H^+ + CO_3^{2-}$ is carbonic anhydrase. It is known to influence calcium carbonate deposition in the molluscan shell (Wilbur and Jodrey, 1955; Istin and Girard, 1970; Carter, 1972), and to aid in the dissolution of carbonate as well (Chétail and Binot, 1967; Chétail and co-authors, 1968; Rosenberg and co-authors, 1968; Chétail and Fournié, 1969; Smarsh and co-authors, 1969; Carriker and Chauncey, 1974; see also section 'Agents: Gastropoda'). Measureable amounts of carbonic anhydrase activity have been detected in the mitochondrial-sized fraction of *Cliona celata* homogenates. The enzyme appeared to be contained within, or associated with, the membrane of vesicles within the filopodial basket. There is a positive correlation between the excavating activity of the sponge (mg of $CaCO_3$ removed per day) and the concentration of carbonic anhydrase in sponge tissues. Sodium acetazolamide is capable of specifically inhibiting the activity of clionid carbonic anhydrase *in vitro* and has been shown to markedly reduce the ability of *C. celata* to remove calcium carbonate from the valves of *Mercenaria mercenaria* under *in vivo* conditions, with no apparent inhibition of the overall metabolism of the sponge. However, as the direct breakdown of $CaCO_3$ by carbonic anhydrase has been ruled out (Carriker and Chauncey, 1974), the exact manner in which the enzyme might mediate the finely controlled dissolution of the substratum by clionid etching cells is unclear (Hatch, 1980).

The periostracum of molluscan shells is penetrated by clionids in much the same manner as calcium carbonate, and chips similar to those of the inorganic shell material are

formed. Topsent (1887), who first observed the process, believed it to be due to purely mechanical action brought about by the contractibility of the sponge cells but Cotte (1902), Nassonow (1924) and Vosmaer (1933) suggested that an enzyme might be secreted which attacks the organic shell components. Although, according to Nassonow (1924) and Warburton (1958b), conchiolin is penetrated less readily than $CaCO_3$, burrowing sponges may inflict severe damage on the conchiolinous hinge ligament of oysters under field conditions. The mechanism of deterioration of the periostracum and organic shell matrix, which probably involves the action of a proteolytic enzyme (or enzymes), has not yet been studied biochemically.

Incidence of *Cliona* spp. invasion of bivalves may vary with locality and probably reflects the salinity regime of the sampling stations. The overall infestation of 2,706 *Crassostrea virginica* from Newport River, North Carolina, was found to be as low as 10.4 % (Wells, 1959a), while Hartman (1958) reported burrowing-sponge invasion in 65 to 80 % of oyster shells from Long Island Sound and Hopkins (1956a) in 72 % of oyster shells from South Carolina. *C. celata* comprised about two-thirds of the infestations in all of these cases. In Pacific waters, *C. celata* is less abundant. In British Columbia, for instance, it occurs only sporadically and attacks only clam and oyster shells of 10 years and older (Quayle, 1969).

Ecological niche differentiation has been found to govern the vertical ranges and relative abundance of 9 sympatric clionid sponges from the Adriatic Sea. A gradation in the sizes of oscular papillae and ostia within the 9 species (which include the shell-invading *Cliona celata, C. vastifica* and *C. viridis*) suggests that food differences may exist which are significant to the co-existence of the species (Volz, 1939; Hartman, 1957).

Susceptibility and severity of *Cliona* attack vary with the bivalve species involved and may reflect differences in the internal microstructure of the shell (Kobayashi, 1969). Thus, *Ostrea equestris* appears to be more prone to burrowing-sponge invasion than *Crassostrea virginica*. In Newport River, North Carolina, horse-oyster shells were commonly extensively eroded, while Eastern oysters to which the *O. equestris* were attached, exhibited much less severe infestation (Wells, 1959a). *C. gigas* appears to be completely immune to invasion by burrowing sponges, which led to reflections on the usefulness and possibility of selection and hybridization of introduced species with native forms (Menzel, 1972).

The burrowing activity of clionids is most pronounced in subtidal areas. In South Carolina, damage from *Cliona celata* and *C. vastifica* is locally so serious that it was considered to prevent the cultivation of oysters below low-water mark except in a few limited places (Lunz, 1943). Wells (1959a), however, pointed out that oysters less than 30 mm long are rarely infested.

Burrowing sponges are the most common animals associated with oysters. Small round holes on the surface of the valves indicate their presence (Fig. 13-70, top). The diameter of the holes varies with the species involved. Perforations made by *Cliona celata*, which are most conspicuous, are 1.5 to 3.0 mm wide for oscular openings and 0.8 to 1.1 mm wide for pore openings. Those produced in oyster shells by *C. lobata* are small — 0.4 to 0.6 mm for oscular and 0.15 to 0.3 mm for incurrent openings. *C. vastifica, C. truitti* and *C. spirilla* perforations are intermediate in size, ranging from 0.4 to 1.6 mm in diameter (Wells and co-authors, 1960).

Sponge galleries approaching the inner valve surface are normally sealed off by the deposition of layers of conchiolin. In most cases, the oyster's protective measures prevent

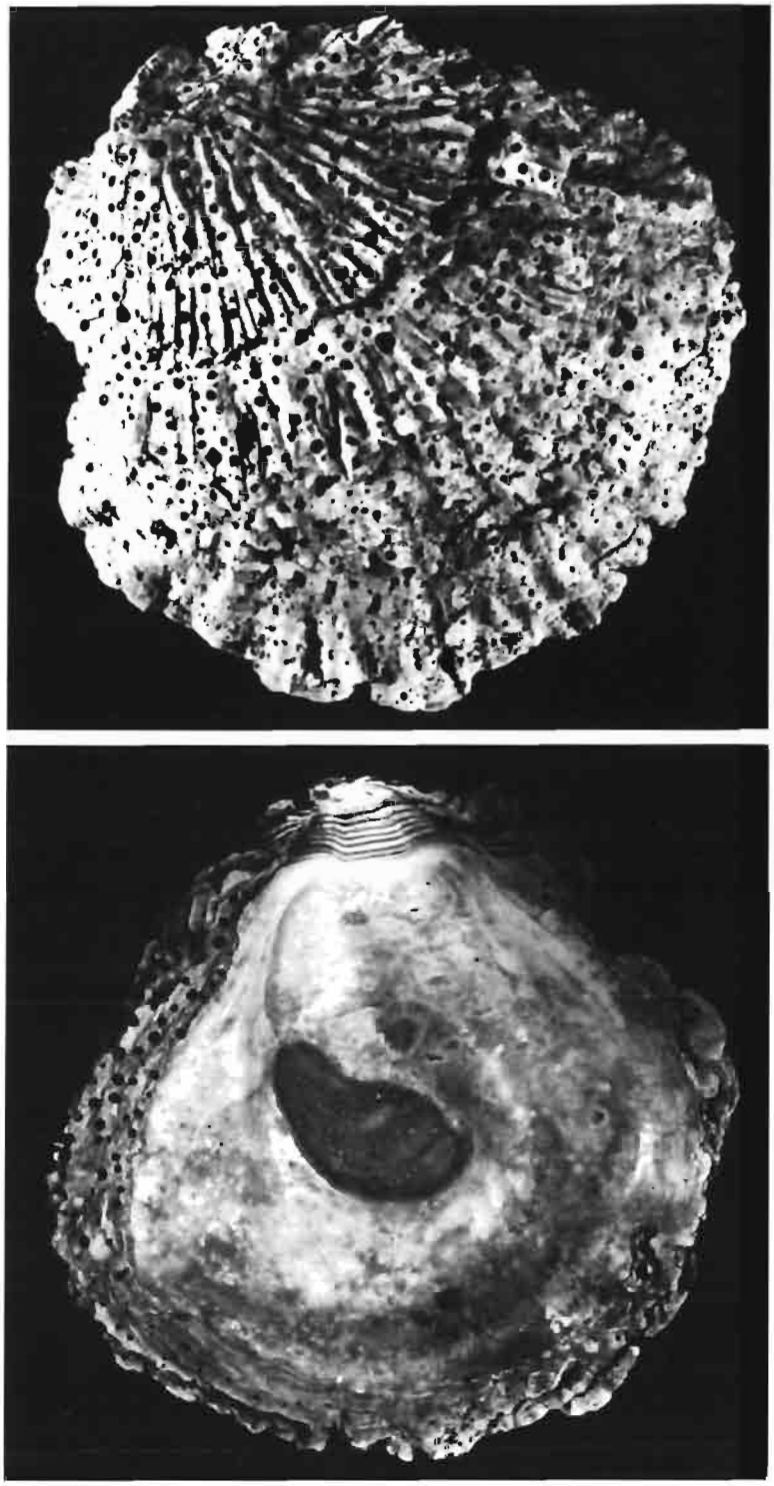


Fig. 13-70: *Cliona* sp. Sponge perforations in shell of *Ostrea edulis*. (Photographs courtesy E. Ziegelmeier.)

direct contact between the sponge and the mantle. However, physical exhaustion, resulting from increased secretion of shell material stimulated by the burrowing organisms, may reduce the bivalves' natural resistance to pathogenic organisms and environmental stress. Should the deposition of shell material be delayed by adverse conditions, the resultant direct contact between the sponge and the oyster's mantle produces lysis of the epithelium and underlying connective tissue. As a consequence, dark pigmented pustules form exactly opposite the bore holes in the shell. The tissue of such oysters is flabby, and the mantle can be easily detached from the shell surface. Sponge perforations under the adductor muscle are especially difficult to repair and may cause injury leading to imperfect valve closure, which, in turn, entails a variety of dangers. Total perforation of the shell and resultant affection of the mantle frequently leads to death. Penetration of the hinge ligament may occur also, its total destruction invariably resulting in death of the host. Substantial losses, particularly among oysters, have been traceable to abundant sponge infestations (Needler, 1941; Old, 1941; de Laubenfels, 1947; Korringa, 1951a, 1952a; Warburton, 1958c; Galtsoff, 1964). *Cliona lampa* has been found responsible for heavy mortality among jewel boxes, *Chama macerophylla,* populations at Bermuda. The sponge may excavate the bivalves' shells to the extent that the adductor muscle breaks loose (Rützler, pers. comm.).

None of the burrowing sponges appear to be true parasites. It is not known whether they utilize the organic shell matrix (which appears unlikely), but it is obvious that they do not draw their nutrients from the tissues of the oyster (Galtsoff, 1964). Their penetration, however, weakens the shells sufficiently to make the oysters less desirable for the market. By stimulating the production of extra layers of shell material, burrowing sponges divert energy of oysters that otherwise might be utilized in the production of edible meats. The condition index of heavily sponge-infested *Crassostrea virginica* was found to be lowered significantly (Warburton, 1958c). *Cliona celata* sometimes riddles oyster shells to such an extent that they become extremely 'honeycombed' and easily break apart, a condition termed 'maladie du pain d'épices' ('spice bread disease') by French oystergrowers (Giard, 1881). It has been estimated that, due to the erosive sponge action, the shells of *C. virginica* heavily infested with *C. celata* or *Cliona* sp., respectively, lose up to 40 % of their weight (Warburton, 1958c; Perera and Arudpragasam, 1966). Shucking of such oysters for marketing purposes is difficult.

Repeated artificially produced shell damage was found to affect gametogenesis and sex ratio in *Crassostrea virginica.* Samples of oysters with filed shell margins exhibited an unusual predominance of males (Bahr and Hillman, 1967; Davis and Hillman, 1971). Whether shell damage produced by burrowing sponges may have a similar 'masculinizing' effect has, apparently, not yet been studied.

Cliona celata can be quite a serious pest in areas with high salinities and has been claimed to destroy entire oyster beds (Giard, 1881). Old (1941) refers to the ruin of the oyster business in 1934 in the Little Choptank region of Chesapeake Bay. In British waters, the sponge occurs abundantly in various substrates and frequently invades *Ostrea edulis.* Although it is rare to find affected brood oysters, large old individuals are very commonly infested. Such 'rotten-back' oysters, as they are called, have shells, the interior of which is discoloured and spotted (Fig. 13-70, bottom). Heavily infested oysters are unmarketable, except in the cheapest grade. Where, however, a regular system of cultivation is followed and oysters are marketed as soon as they reach a saleable condition, the trouble is never too serious (Cole, 1956a).

In addition to oysters, clionid sponges may attack a variety of other bivalve species. Evans (1969) studied the extent of shell destruction by *Cliona vastifica* in *Placopecten magellanicus* from Newfoundland (Canada) waters. In radiographs of affected valves, the cavities excavated by the sponge "look like a malignant cancer", with long, branching, root-like extensions growing out through the shell. With time, neighbouring branches join and interconnect. Eventually, nearly all of the shell material between the outer and inner surfaces is destroyed, leaving only isolated supporting pillars. *C. vastifica* makes no effort to avoid neighbouring sponge galleries or polychaete burrows. However, it does not indiscriminately destroy either the inner or outer surface of the scallop shell. The shell of *P. magellanicus* may also be invaded by *C. celata* which, besides burrowing into the shell, often forms large, irregular masses 20 to 25 cm high over the surface of the upper valve (Merrill, 1961). One would normally assume that epizoic growths of such magnitude would impair mobility and thus reduce the scallops' ability to escape from predators (Wells and co-authors, 1964; Sindermann, 1971).

Clionid invasion of the shell may also affect the soft parts of scallops and cause a condition termed 'dark-meat'. While normal scallop meats are creamy-white in colour and have a firm 'short' texture, those of affected individuals have a grayish brown tinge, are flaccid and stringy in texture and usually small for the size of the shell from which they were taken. The meat yield of lightly infested scallops is only about 59 %, that of heavily infested ones only 46 % of the yield of healthy individuals. Among scallops dredged off Digby, New Scotland (Canada), only those of about 8 to 9 years or older were found to be affected by clionids. Sponge invasion starts in the oldest and most worn parts of the lower valve, near the hinge, and spreads from there until the entire shell is honey-combed. As a result of attempted shell repair, the valves may reach 2 to 3 times their normal thickness

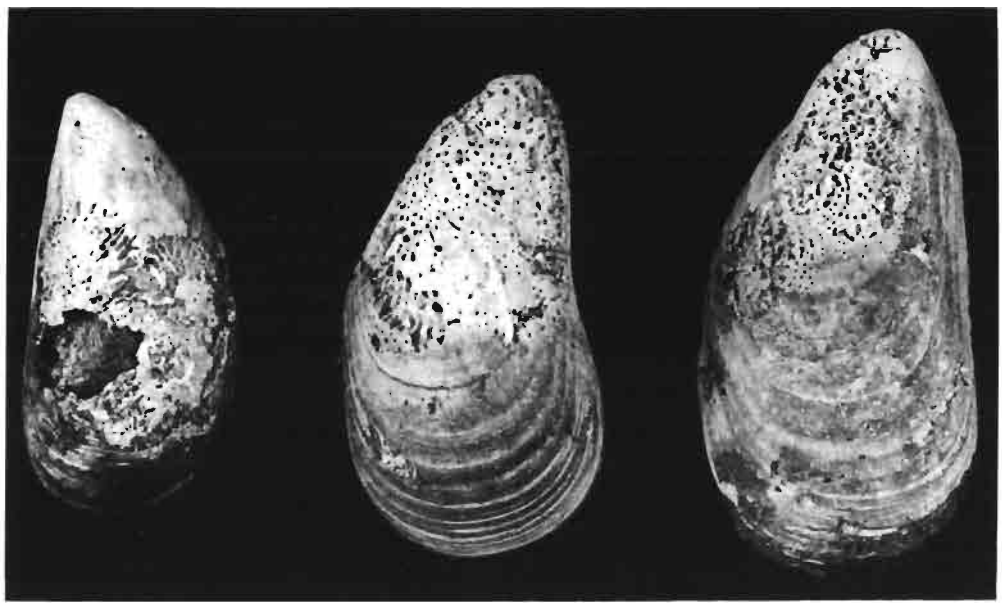


Fig. 13-71: *Cliona* sp. boring into *Mytilus edulis* shells. Shell lengths 60, 70 and 80 mm, respectively. (After Rasmussen, 1973.)

and assume 'the brittle texture of a soda biscuit' — a tough outer and inner surface but very little between. Badly affected scallops probably die (Medcof, 1949).

In Isefjord (Denmark), *Mytilus edulis* sometimes suffers severe attacks by clionids, presumably *Cliona lobata* (Fig. 13-71). Boring usually starts in the thickest part of the shell, i.e., the oldest portion in which the protecting periostracum, which is very tough in intact mussels, has been worn off. A rather high percentage of larger mussels can often be found with completely perforated shells. Affected *M. edulis* respond by secreting a thin sheet over the perforated area (Fig. 13-71, left), which, however, often breaks, so that the mussel may soon die as a result of attacks by crabs or other predators. Although clionids are seemingly capable of penetrating a bivalve's intact periostracum (see above), such penetration has never been observed in *Mytilus edulis* from Isefjord (Rasmussen, 1973).

Individuals of *Pinctada margaritifera* from the Gambier Islands, South Pacific, and the Gulf of Mannar, Ceylon, may be heavily attacked by *Cliona margaritiferae*. Pearl oysters with shells rendered rotten by the burrows of the sponge fall easier prey to molluscivorous fish. Rays *Aetobatus* (= *Myliobatis*) *narinari* showed a feeding preference for such affected oysters (Herdman, 1905, 1906; Seurat, 1906a). Clionid infestation of the shells may considerably detract from the market value of *P. maxima* from the Arafura Sea, Japan (Takemura and Okutani, 1956).

Kölliker (1860a,b) found a shell of *Ostrea edulis*, which was heavily attacked by *Cliona* sp., to harbour simultaneously enormous numbers of fungal hyphae. Whether there are factors favouring this co-occurrence has not been investigated, but probably the openings of the sponge burrows form the entrance ports for the infective zoospores of shell-inhabiting fungi, such as *Ostracoblabe implexa* (see section 'Agents: Fungi').

Inspite of the destructiveness of burrowing clionids, control measures effective against sponge affection of commercially important bivalve stocks have not yet been developed. De Laubenfels (1947) and Warburton (1958) discussed the possibility of dipping infested oysters in freshwater in order to kill the sponges. Shearer and MacKenzie (1961) claim to have obtained positive results by 3- to 5-min exposure of sponge-infested *Crassostrea virginica* to saturated or partially saturated 'rock salt' solutions, followed by drying for 1 hour. Korringa (1952a) recommended prolonged bathing in freshwater or in seawater containing a disinfectant. But, since both the incurrent and excurrent papillae of clionids are strongly contractile and enable the sponge to insulate itself from unfavourable environmental conditions (Emson, 1966), little benefit is expected to result from such treatment. Churchill (1920, p. 40) made the pessimistic statement: "No means of protection against the sponge can be suggested." Some improvement, however, may be gained from proper adjustment of oyster culture methods to the peculiarities of sponge biology.

Not all species of *Cliona* invariably exhibit the characteristic burrowing behaviour. Some may grow from a burrowing (or alpha) stage into an encrusting (or beta) form and, eventually, into a final (or gamma) stage, now showing up as large, upright massive sponge bodies. *C. celata* can be found burrowing into calcareous shells, in an encrusting state, or in a massive, free-living form showing little trace of its burrowing nature (Wells and co-authors, 1960). As an oyster infested with *C. celata* dies, the sponge continues to grow on the shell, reaches its encrusting stage after some time and, eventually, forms large, irregular masses 60 cm or more wide and several cm thick (Galtsoff, 1964). It is obvious that such large sponges will produce high numbers of eggs and larvae and, hence, favour increased sponge invasion in oysters. Removal of dead shells from oyster beds and disuse

of oyster valves as cultch for brood stocks is, therefore, to be recommended. Planting of oysters into low-salinity habitats may also considerably reduce *Cliona* attack.

In addition to *Cliona* spp., a number of epizoic sponges utilize oysters and other bivalves as substrate. A well-known example is the 'oyster sponge' *Microciona prolifera* (de Laubenfels, 1947). A mutualistic relationship exists between the crumb-of-bread sponge *Halichondria panicea* and scallops *Chlamys* (*Pecten*) *varia*. The majority of scallops from oyster banks off Galway on the Atlantic coast of Ireland were found to be covered by a substantial growth of *H. panicea*. The bivalves are protected against predatory sea stars by the sponge which interferes with the attachment of tube feet and makes the vulnerable byssal opening and valve margins inaccessible to the predator's gastric membranes. The sponge, in turn, benefits from an increased food supply made available by the inhalant current of the scallop (Forester, 1979). A similar relationship exists between scallops *C. hastata hericia* and *C. rubida* and encrusting sponges *Myxilla incrustans* and *Mycale adherens* from the Washington (USA) coast (Bloom, 1975). *Chlamys dieffenbachi* from New Zealand, initially apparently used merely as substrate for attachment by an undetermined sponge species, soon becomes entirely enclosed by the sponge mass. Scallops living in these sponges grow larger than any others and apparently benefit, in some unknown way, from the association (Beu, 1965).

Agents: Cnidaria

Many hydroids associate with marine bivalves, ranging from species classifiable as inquilines to much more intimate associates (W. J. Rees, 1967). Most of the polyps attach to the outer surfaces of the molluscs' valves, but several species have been reported from within the mantle cavity.

Some of the former kind of association may be termed mutualistic because the cnidarian partner, who gains advantage from the filter-feeding currents of the bivalve, simultaneously provides some protection for the mollusc by means of its cnidocysts. Polyps of *Obelia dichotoma, O. longissima, Hydrallmania falcata* and other species live on the shells of mussels, scallops and oysters. *Ostrea edulis* shells may also support colonies of *Tubularia larynx, Laomedea gelatinosa* and *Gonothyraea loveni* (Korringa, 1951a), as well as the scyphistomae of *Aurelia aurita*. The latter are known to devour quantities of oyster larvae (W. J. Rees, 1967). Dense fringes formed by the polyps around the shell margins, as reported for *Neoturris pileata* living on *Nucula sulcata* shells (Edwards, 1965), may impair the mobility of the pelecypod or even check shell growth, as is the case in the *Hydractinia echinata – Placopecten magellanicus* association. Normally, this colonial hydrozoan lives as an epiphoront on gastropod shells occupied by hermit crabs *Eupagurus bernhardus*. Accelerated colonial growth along the shell edge causes the formation of a new margin composed of a thick periderm layer which continuously extends. Rarely, *H. echinata* also incrusts the valves of live pelecypods. Extreme shell deformity caused by this hydrozoan has been observed in the sea scallop *P. magellanicus*. Extension of the periderm beyond the valve edge causes the mantle epithelium to retract. The scallop responds to the stimulus by secreting an entirely new shell margin. In some individuals the process of mantle retraction and successful new growth was repeated several times, resulting in grossly abnormal shells. Lack of correlation, however, indicated that *H.*

echinata was not the cause of the observed heavy natural mortality of *P. magellanicus*. On the other hand, mortality possibly attributable to this hydroid was noticed in pelecypods *Anomia aculeata* which were found attached to shells of *P. magellanicus* (Merrill, 1964, 1967).

Hydroids of *Neoturris pileata* were found attached abundantly to the posterior and ventral shell margins of *Nucula sulcata* from the Clyde Sea area. Individuals of *Nucula turgida* were less heavily and those of *N. nucleus* only sparingly infested. *N. pileata* was neither found on sympatric individuals of *Nuculana minuta* nor on any other invertebrate species. It seems that the hydroid has specialized habits adapted to the peculiar burrowing mode of life of the above protobranch hosts. In contrast, hydroids of *Leuckartiara octona* were found on *Nucula* spp., as well as on *Nuculana minuta*. Individuals of *N. sulcata* sometimes supported colonies of both *N. pileata* and *L. octona* (Edwards, 1965).

Monobrachium parasitum, a one-tentacled athecate hydroid, lives attached to the posterior shell margins or around the siphons of bivalves of the genera *Tellina* and *Macoma* in the high Arctic. Originally described and named by Mereschkowsky (1877), subsequent workers (listed in Hand, 1957) tend to spell it *M. parasiticum*. Hand reported what appears to be the same species from bivalves of the genera *Axinopsis* and *Aligena* in Canadian Arctic Atlantic and Pacific waters. Another species, *M. drachi* (Fig. 13-72), has been found in association with small (2.5 mm shell length) bivalves *Cuna gambiensis* from Gorée, Senegal (Marche-Marchad, 1963). The highly specialized medusae of *Monobrachium* (Fig. 13-72, 2) are non-pelagic.

Since the bivalves to which *Monobrachium* spp. attach are usually very small, considerable impairment of their vital functions is expected to result from dense hydroid

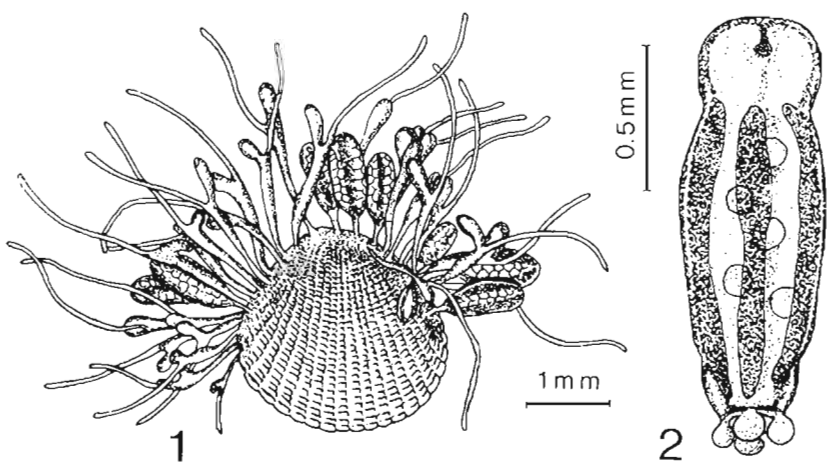


Fig. 13-72: *Monobrachium drachi*. 1: Colony of polyps and gonothecae on *Cuna gambiensis* shell; 2: non-pelagic medusa. (After Marche-Marchad, 1963.)

colony upgrowth. In fact, Høpner Petersen (1978) found the average soft-part weight of *Macoma calcarea* from Godhavn Havn, West Greenland, to drop from 0.355 g (individuals without *Monobrachium* upgrowth) to 0.146 g (individuals with *Monobrachium* on shells). Clams with hydroids had reduced shell lengths and their gonad development was

retarded. Individuals of *Macoma baltica* were constantly devoid of hydroids, probably because this species burrows deeper.

Other cnidarians are known to attach to the soft tissues of marine bivalves. W. J. Rees (1967) found unidentified campanularians on the siphons of *Mya arenaria* and *Saxicava* (*Hiatella*) *arctica*. Small solitary athecate hydroids of the genera *Eugymnanthea* and *Eutima* (Fig. 13-73) are found, attached by a basal disc, within the mantle cavity of marine

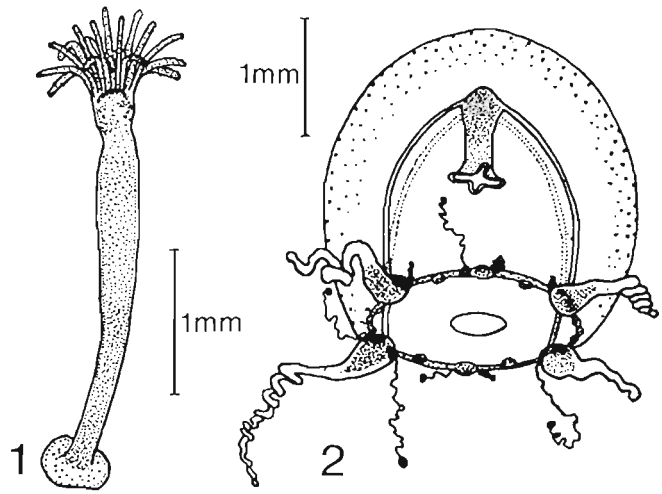


Fig. 13-73: *Eutima* (*Eugymnanthea*) *cirrhifera*. 1: Polyp with conspicuous basal disc; 2: medusa, 5 days after liberation. (After Kakinuma, 1964.)

bivalves. *Eugymnanthea inquilina* has been described from *Tapes decussatus* at Naples, Italy (Palombi, 1936). *E. polimantii* — originally described by Cerruti (1941b) as *Mytilhydra polimantii* — inhabits *Mytilus galloprovincialis* at Taranto (Italy), and *E. ostrearum* occurs in *Crassostrea rhizophorae* from Puerto Rico (Mattox and Crowell, 1951).

Crowell (1957), who restudied the *Eugymnanthea* occurring in the Gulf of Naples, assumed that they are not as host-specific as previously believed, and that *E. inquilina* and *E. polimantii* are identical. On the other hand, the hydroid polyps reported from the various Mediterranean bivalves may well represent several species, as Uchida (1964) described medusae, taken in plankton hauls off Naples, as a new species, *E. minuta*.

In Japan, Yamada (1950) described *Ostreohydra japonica*, found attached to the gills of *Crassostrea gigas*. Crowell (1957) transferred it to the genus *Eugynmanthea* as *E. japonica*. *E. cirrhifera*, first described from *Mytilus edulis* in Japan by Kakinuma (1964), was redescribed as *Eutima cirrhifera* by Kubota (1978). *Mytilus coruscus* from Japan harbours *E. cirrhifera*, as well as polyps believed to represent a subspecies of Palombi's *Eugymnanthea inquilina* (Kubota, 1979).

Other *Eutima* polyps occurring in the mantle cavity of marine bivalves include *E. commensalis*, found in wood-boring bivalves *Nausitora hedleyi, Teredo furcifera* and *Martesia striata* in India (Santhakumari and Balakrishnan Nair, 1969; Santhakumari, 1970), as well as *E. sapinhoa*, associating with *Tivela mactroides* in Brazil (Narchi and Hebling, 1975). Judging from the large known number of planktonic mature medusae of

the genus *Eutima* (Kramp, 1961, 1968), numerous further bivalve–cnidarian associations may be expected to occur in various parts of the world ocean.

Within the bivalves' mantle cavity, the polyps occupy different microhabitats which may vary from species to species. *Eugymnanthea inquilina* (*Mytilhydra polimantii*) was found almost exclusively on the mantle epithelium of Mediterranean *Mytilus galloprovincialis* and only rarely on the body wall, foot or palps, but never on the gills (Cerruti, 1941b; Crowell, 1957). More than 50 % of the individuals of the Japanese subspecies, *E. inquilina japonica*, occurred on the mantle of *M. coruscus* but the remainder were found attached to other body surfaces including the gills (Kubota, 1979). *Eugymnanthea* (*Ostreohydra*) *japonica* attach (exclusively?) to the gills of *Crassostrea gigas* (Yamada, 1950). Individual polyps measure, on the average, 1 to 3 mm in length but may become as large as 5 mm.

Incidence and intensity of 'infestation' with polyps may be very high and may vary with locality and season. A 4.5-cm long *Crassostrea rhizophorae* was found to harbour 210 *Eugymnanthea ostrearum*. Of 138 mangrove oysters from Puerto Rico, 12 % contained polyps (Mattox and Crowell, 1951). Two small individuals of *Mytilus coruscus* from Japan, 3.4 and 4.4 cm in shell length, had respectively 669 and 574 polyps of *E. inquilina japonica*. Curiously, one mussel liberated exclusively female medusae, the other only male ones. Three of 252 mussels were found to harbour polyps (Kubota, 1979). Crowell (1957) recorded hydroids of *Eugymnanthea* sp. (or spp.) from 29 % of *Mytilus galloprovincialis*, 16 % of *Tapes decussatus*, 50 % of *Cardium edule* and 7 % of *Ostrea* sp. from Lago Fusaro, a shallow brackish-water lagoon adjacent to the Gulf of Naples. In the open gulf, infestation incidences were much lower, being only 8 % in *M. galloprovincialis* and 4 % in *Cardium tuberculatum*. Large *M. galloprovincialis* sometimes harboured many hundred polyps. From October to January, *Tivela mactroides* from Itagua Beach, Ubatuba (Brazil), were 100 % infested with *Eutima sapinhoa*, whereas in the other months of the year the incidence was only 10 to 20 % (Narchi and Hebling, 1975). An interesting case of host-sex related differential attack has been reported by Kakinuma (1964). Individuals of *Eutima cirrhifera* occurred only in one- or more-year-old *M. edulis* from Hachinohe, Aomori Prefecture (Japan), which had developed gonads. Polyps were found in 20 to 30 % of the male and in 70 to 80 % of the female mussels. Between 30 and over 200 hydroids were recovered from individual mussels.

Little is known about the relationships between bivalves and mantle-cavity inhabiting hydroids. The absence of both a perisarc and stolons in these highly specialized hydrozoans may be considered as adaptive to commensalism (Mattox and Crowell, 1951; Crowell, 1957). Cerruti (1941b) observed loss of cilia from epithelial surfaces of the mantle of *Mytilus galloprovincialis* to which *Eugymnanthea polimantii* had been attached, as well as the appearance of eosinophilic granules in the cytoplasm of cells underlying the attachment site of the hydroids' pedal disc. No such pathological alterations were seen in *Crassostrea rhizophorae* harbouring *E. ostrearum* (Mattox and Crowell, 1951). The distribution of *Eugymnanthea* spp. and *Eutima* spp. in their bivalve hosts is strongly overdispersed (clustered). This suggests initial invasion of the mantle cavity by only one dispersal stage, probably the planula larva (Cerruti, 1941b), which then produces a clone asexually by budding. Vegetative multiplication by budding has been observed in most of the species. The polyps, which attach to the epithelial surfaces by gentle suction of their slightly concave basal disc, are capable of slow locomotion. The disc does not perforate the host's epithelium.

Although there are no indications for a parasitic way of life in *Eugymnanthea* spp. and *Eutima* spp., heavy invasion by these hydroids presumably interferes with the normal filtering activity of their respective hosts, but experimental evidence substantiating such impairment is as yet lacking. One may speculate on a possible interference of hydroid infestation in bivalve molluscs with human health. The toxin of the cnidarians is probably heat-labile and may be inactivated by cooking or during processing but may become a problem if, for example, heavily infested oysters are consumed raw. In Holland there is a popular belief that consumption of *Mytilus edulis* harbouring pea crabs *Pinnotheres pisum* causes 'nettle rash'. There is, beyond doubt, no correlation between the presence of these crabs and the allergic reaction some individuals show after eating mussels, but one may hypothesize about the possible occurrence of mantle-cavity hydroids in allergenic bivalves.

Agents: Turbellaria

While Turbellaria are predominantly free-living predators or scavengers, members of the orders Rhabdocoela and Alloeocoela associate more intimately with marine molluscs. While several alloeocoelans inhabit the mantle cavity of bivalves, rhabdocoelans of the family Graffillidae live in the alimentary tract of these hosts. Their exact status has not been defined but Jennings (1971, 1973), who reviewed host-symbiote relationships in the Turbellaria, considers the species of *Paravortex* reported from lamellibranch guts "to be midway between being entocommensal and being parasitic".

Unidentified turbellarians, first seen in the alimentary tract of *Scrobicularia* (= *Abra*) *tenuis* from France by Villot (1879), were named *Paravortex scrobiculariae* by Wahl (1906), who refound them in Mediterranean *Scrobicularia piperata* (= *S. plana*) and *Tapes decussatus*. The other European species, *P. cardii*, occurs in the stomach and intestine of *Cardium edule*. It has been named and studied in great detail by Hallez (1908, 1909). Lebour (1905) and Nicoll (1906a), who gave fairly good descriptions and illustrations of the adult containing daughter individuals (Fig. 13-74), mistook it for a 'ciliated trematode sporocyst with eye spots'. Atkins (1933a), Leigh-Sharpe (1933) and Chrenko (1934) erroneously recorded it as *Graffilla gemellipara*. 'Ciliated sporocysts with 2 eyes', found in *Macoma baltica* by Lebour (1906), as well as *Paravortex* spp. occurring in other bivalves (von Graff, 1913), may either be referable to the above forms or represent additional species.

In American waters, *Paravortex gemellipara* (originally described as *Graffilla gemellipara* by Linton, 1910) inhabits the mussels *Geukensia demissa* ('*Modiolus plicatus*'), *Ischadium* (*Brachidontes*) *recurvum* and *Mytilopsis* (*Congeria*) *leucopheata*. Contrary to the findings of Linton (1910), Patterson (1912) and Ball (1916), who described *P. gemellipara* from the gill and the kidney, respectively, Wardle (1980a) made it clear that the flatworm, like its European counterparts, inhabits the digestive tract of the mussels.

Locally, incidence and intensity of infestation of bivalves with *Paravortex* spp. may be high. Wahl (1906) recovered 290 *P. scrobiculariae* from 65 of 125 *Scrobicularia plana* from Trieste, Adriatic Sea, and 207 individuals from 36 of 50 *Tapes decussatus* from Naples, Tyrrhenian Sea. Maximum numbers of worms found in single individuals of the 2 host species were 22 and 45, respectively. Although Freeman (1957) found *P. scrobiculariae* to be rare in clams from Whitstable, Kent (England), 70.4 % of the *S. plana* examined

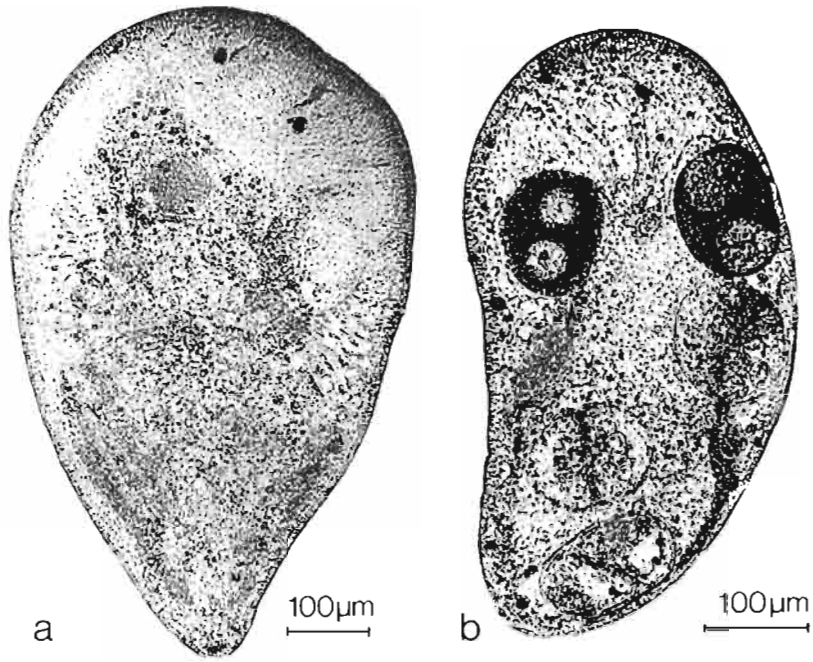


Fig. 13-74: *Paravortex cardii* from intestine of *Cardium edule*. (a) Immature individual, flattened under cover-glass pressure; (b) individual with embryos in various stages of development. Note conspicuous eye-spots. (Original.)

by Jennings and Phillips (1978) in the River Esk, Whitby, Yorkshire (England) harboured the turbellarian. The mean infestation rate was 6.1 rhabdocoels per host with a range of 0 to 77.

Similar prevalences — within the range of some 50 to over 80 % — have been recorded for *Paravortex cardii* in *Cardium edule* from British waters (Atkins, 1934; Hancock and Urquhart, 1965; Jennings and Phillips, 1978). Wardle (1980a) found up to 100 % *P. gemellipara* infestations in *Ischadium recurvum* and *Geukensia demissa* from Chesapeake Bay, Virginia, but prevalences of only 12.5 to 21.4 % in hosts from the Gulf of Mexico. He inspected additional 13 bivalve species from Chesapeake Bay and 30 species from Galveston Bay, Texas, but found no flatworms, which indicates that *P. gemellipara* may be specific to bivalves of the family Mytilidae. The graffillid reported by Uzmann (in Cheng, 1967) from *Mya arenaria* possibly represents another species.

In spite of heavy *Paravortex cardii* burden, individuals of *Cardium edule* from Plymouth, England, appeared to be healthy (Atkins, 1934). Dissection of host guts harbouring *P. gemellipara* revealed no damage to the intestinal epithelium (Wardle, 1980a). Studies on feeding, digestion and the physiology of *Paravortex* spp. indicate that these flatworms are well adapted to live in the intestine of their bivalve hosts (Jennings, 1971, 1973; Jennings and Phillips, 1978; Phillips, 1978; Jennings and LeFlore, 1979).

Alloeocoel turbellarians of the genus *Urastoma* occur in the mantle cavity and on the gills of *Mytilus edulis*, *Modiolus modiolus*, *Lima hians* and other marine bivalves (Dörler, 1900; von Graff, 1903; Westblad, 1926, 1955a,b). Burt and Drinnan (1968) reported

Urastoma (probably *cyprinae*) as occurring in large numbers on the gills of *Crassostrea virginica* from Prince Edward Island, Canada. Although these 'oyster gill worms', as they are called, may cause an unsightly appearance, they have to be regarded as harmless facultative commensals (Jennings, 1971, 1973). Clams *Hiatella byssifera* from West Greenland were found to be heavily infested with an unidentified, cigar-shaped, pink coloured turbellarian, 0.2 to 0.6 mm in length. The infestation showed a seasonal pattern, with a maximum in spring and summer (Høpner Petersen, 1978).

Acoelans *Convoluta japonica* have been blamed for the destruction of *Tapes* (*Venerupis, Amygdalum*) *philippinarum* in Chiba Prefecture, Japan (Kato, 1951). The way in which the turbellarian achieves this, has not been explained in the English summary of the paper.

Polycladid turbellarians of the genera *Stylochus, Pseudostylochus* and other genera are known as predators of oysters and other marine bivalves. Some species of 'oyster leeches' are regarded as true pests (Pearse, 1938). Among oyster brood on beds at Rovinj (Adriatic Sea), *S. pilidium* was said to have caused losses of up to 87 % (Bytinski-Salz, 1935). *S. ellipticus* was held responsible for large-scale mortalities among young *Crassostrea virginica* in New England and Chesapeake Bay waters (Loosanoff, 1956; Provenzano, 1961; Webster and Medford, 1961). As demonstrated by laboratory observations, the worms have little difficulty in entering and killing oyster spat, even those measuring more than 6 mm in length. In one experiment, when 10 worms were placed in the same dish with 30 healthy spat averaging 1.7 cm in length, *S. ellipticus* killed 21 spat in less than 1 month. *Stylochus* spp. and other flatworms with direct life cycles may completely eliminate young stages of cultivated bivalves if accidentally introduced into rearing ponds. Similar 100 % losses of seed oysters have been witnessed in South Korea oyster growing areas where an overwhelming flatworm population existed (Sindermann, 1973).

Stylochus frontalis feeds primarily on oysters (Hyman, 1940), but Pearse and Wharton (1938) were unable to identify this polyclad as the primary cause of oyster mortalities in Apalachicola Bay, Florida. They suggested that *S. frontalis* merely attacks and kills oysters weakened by some other unknown cause and thus unable to close their valves. *Pseudostylochus ostreophagus* is definitely a predator, attacking *Ostrea lurida, Crassostrea virginica* and *C. gigas*, and causing heavy mortalities among cultivated oysters along the North American Pacific coast (Woelke, 1957). Galleni and co-authors (1980) have assembled a list of polyclads and their (mainly molluscan) prey organisms.

Agents: Trematoda

Bivalve molluscs, freshwater as well as marine, are hosts for digenetic trematodes. A considerable number of these bivalves are of economic importance. Larval flukes have been reported from virtually every marine bivalve species, and it is certainly no overstatement to consider the digeneans as the most important metazoan parasites of these molluscs. The host-parasite relationship between Mollusca and Digenea is of long standing. There can be little doubt that orginally the flukes were parasites of molluscs, and that additional intermediate and definite (vertebrate) hosts have been adopted subsequently (Stunkard, 1957, 1959a, b, 1967, 1970b; Wright, 1960, 1966).

Larval digenetic trematodes are mainly described on the basis of the morphology of

their cercariae. The designation 'cercaria' has even been raised to generic rank. The systematics, taxonomy and nomenclature of the Trematoda are in constant flux. Inherent problems have found ample attention in the scientific literature (Looss, 1902; Stunkard, 1937, 1957, 1960b, 1963; LaRue, 1957; and others). Consideration of morphological and developmental data leads to the conviction that, in the Digenea, the higher taxonomic units have little phylogenetic or systematic significance (Stunkard, 1946). Therefore, no reference will be made in this treatise to the validity and justification of higher taxonomic categories employed in this parasite group.

The main drawback met with by the trematodologist is the lack of exhaustive knowledge of digenean life histories. Compared with the vast number of cercariae and adults described, relatively few complete life cycles have been worked out experimentally. The importance and practical implications of such studies have been emphasized repeatedly. Thus, Stunkard (1940, p. 15) has long-sightedly stated:

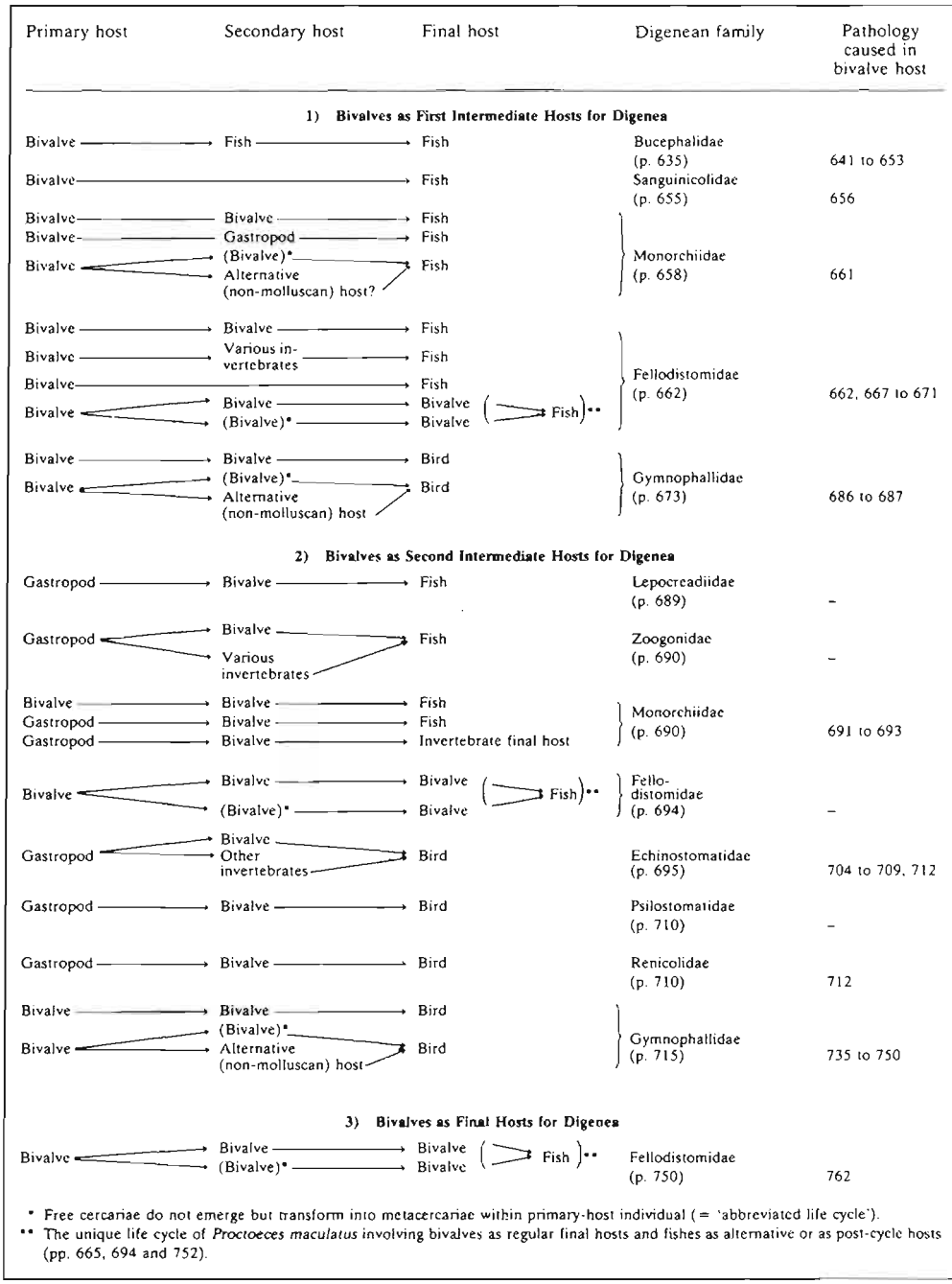
> "More complete knowledge of life cycles will permit more intelligent and more effective regulatory methods, the reduction of morbidity, the advancement of health, and the conservation of natural resources."

Most publications referring to marine bivalve-digenean associations merely comprise taxonomic descriptions, host-parasite lists, and locality records. Studies on physiological and biochemical interrelationships are rare. Many life cycles have tentatively been inferred — often quite unjustified and including gross errors — purely on the basis of observed morphological similarities between larval and adult stages. Much confusion has arisen from this practice.

As a consequence of the barren status of trematode biology and taxonomy, many synonymies exist, and misidentifications at the specific, generic, and even higher taxonomic levels are the rule rather than exception. Therefore, the specific — and sometimes also generic or familial — designations used below are not necessarily those employed by the initial workers. On the other hand, the original names have been retained in those cases in which the correct identification of the stages described is impossible or uncertain.

With the recent introduction of chaetotaxy, a powerful tool has become available for the detection and characterization of intraspecific variations and interspecific differences among digeneans. Promising results have been obtained with respect to the systematics and phylogenetic relationships of higher taxonomic categories (Richard, 1971, 1976, 1977; Bayssade-Dufour, 1973, 1974, 1977; Bayssade-Dufour and Lang, 1973; Bartoli, 1974a; Bayssade-Dufour and Maillard, 1974, 1975; Richard and Bartoli, 1974; Richard and Prévot, 1974; Combes and co-authors, 1976).

Digenetic trematodes may utilize bivalves as primary (first intermediate) or secondary (second intermediate) hosts, or — exceptionally — as definite hosts. The basic life-cycle pathways of the Digenea parasitizing marine Bivalvia may be outlined as follows:



* Free cercariae do not emerge but transform into metacercariae within primary-host individual (= 'abbreviated life cycle').

** The unique life cycle of *Proctoeces maculatus* involving bivalves as regular final hosts and fishes as alternative or as post-cycle hosts (pp. 665, 694 and 752).

Taking into account the ubiquity of digenean parasites in coastal waters, an intimate knowledge of trematode life cycles must be considered as a basic prerequisite for a sound understanding of population dynamics and general ecology in the marine environment (R. M. Anderson, 1979; Anderson and co-authors, 1979). An attempt will be made here

to revise and update the immense body of literature on digenean parasites of marine bivalves. Admittedly, the result is of considerable long-windedness; but it must be so necessarily! Readers merely interested in, or concerned with, the pathology produced by digeneans are referred to the pages indicated in the right column of the above scheme.

The pathology produced by rediae or sporocysts in gastropods is fairly well documented in the pertinent literature (Vol. I, Chapter 12). In contrast, the metacercariae in the secondary host are usually regarded as 'resting stages' inflicting little if any harm on the intermediate host. It has been argued time and again that it cannot be to the 'interest' of the parasite to harm or debilitate its host excessively. However, this viewpoint is totally wrong and the opposite is true: Parasite-induced behavioural changes or debilitation of intermediate hosts carrying the stage (i.e., the metacercaria) infestive for the definite host may be quite well to the benefit of a parasite which aims at completing its life cycle.

Evidence will be presented here that trematode metacercariae are capable of eliciting a great variety of physiological, biochemical, morphological and behavioural responses in their respective hosts.

Bivalves as First Intermediate Hosts for Digenea

While digeneans parasitizing gastropods have either sporocysts or rediae, or both, the first intramolluscan stage occurring in bivalves are sporocysts.

Digenea utilizing fishes as final hosts: family Bucephalidae

Trematodes of the suborder Gasterostomata — members of the solitary family Bucephalidae — have fairly uniform life-cycle patterns. Their sporocysts and cercariae invariably occur in bivalves; the metacercariae live in cysts in various parts of the central and peripheral nervous system or in internal organs and the musculature of teleost fishes; the adults inhabit the alimentary tract of predatory fishes. Yamaguti (1958) recognized some 145 species of adult bucephalids parasitizing marine and freshwater fishes in various parts of the world, and an additional number of species have since been described.

Knowledge of the life cycles and ecology of the Bucephalidae is of great importance since their larvae affect numerous commercially important marine invertebrates, such as mussels and oysters, and food fishes, such as flatfishes and gadoids. Infestation of bivalves with bucephalid sporocysts and cercariae causes castration and debilitation. The metacercariae of some species of gasterostomes may invade fish in numbers often sufficient to cause harm or to render the fish unpalatable (Matthews, 1973b). Liston and co-authors (1960), for example, reported considerable economic losses caused to the Pacific rockfish industry by larval gasterostome trematodes. For reviews of the family Bucephalidae see Eckmann (1932), Kniskern (1952), Hopkins (1954a) and Stunkard (1974a).

In contrast to the fairly large number of adult bucephalids described thus far, comparatively few cercariae and metacercariae have been reported, and experimental life-cycle studies on members of this family are scarce. Instead, numerous gasterostome life histories have been pieced together more or less arbitrarily, and merely on the basis of morphological similarities between the various stages. In general, bucephalid cercariae do not even show any of the features which are used to distinguish genera. Hence, the assignment of a given cercaria to a known adult worm is not possible until the life cycle has

been worked out by means of experimental infestation (Hopkins, 1954a; Stunkard, 1974a, 1976). Once the cycle is known, specific identification of larval forms parasitizing bivalves is facilitated by the fact that bucephalids are markedly specific with respect to the choice of their first intermediate hosts — a circumstance disregarded by most workers.

The confusion started right away with the discovery of the first marine bucephalid larva. Lacaze-Duthiers (1854) described *Bucephalus haimeanus* from *Ostrea edulis* taken at Mahón (Balearic Islands, Spain) and *Cardium rusticum* (= *Cardium glaucum*) collected from brackish lagoons near Sète (French Mediterranean coast). The cercariae from these 2 bivalve species represent, beyond doubt, 2 distinct species of gasterostomes. Regarding the host specificity of digenetic trematodes, particularly in the primary host, it seems impossible that one and the same cercaria occurs in 2 such unrelated hosts. Both Lebour (1912) and Maillard (1976) provided evidence for the existence of 2 separate species. Although these authors recorded gasterostome cercariae in *C. edule* and *C. glaucum*, respectively, they never found larval bucephalids in oysters living side by side with the infested cockles. Since *O. edulis* is the type host for *B. haimeanus*, the specific name *haimeanus* has to be reserved for the larva from the oyster. A cercaria from *C. glaucum* — beyond doubt identical with the one described from the same host and locality by Lacaze-Duthiers (1854) — has been identified as the larva of *Labratrema* (*Bucephalus*) *minimus* (Stossich, 1887) by Maillard (1975a, 1976). Peneda (1965) misidentified larval *L. minimus* parasitizing *C. edule* in the Ría de Aveiro (Portugal) as *'Bucephalus mytili'* (= *Prosorhynchus squamatus*), a larval bucephalid parasite of *Mytilus edulis* (see below). This error led her to conclude that this digenean is not host-specific.

Although, by the end of the nineteenth century, 10 distinct species of gasterostomes had been described from adult worms parasitizing marine fishes in European marine waters, the bucephalid cercariae from at least 8 different bivalve species were all referred to *Bucephalus haimeanus*. Thus, Huet (1888a, 1893), Johnstone (1905a, b, 1922), Pelseneer (1906), Lebour (1907a, 1912), James and Bowers (1967a, b, c), Bowers (1969), Deltreil and His (1970) and Matthews (1973b) reported this cercaria from *Cardium edule*. Whether all these forms represent a single species, and whether this is identical with *Labratrema minimus* found in *C. glaucum* by Maillard (1975a, 1976), remains to be established. Matthews (1973b) maintains that his larva from *C. edule* develops into *B. minimus* (Stossich, 1887). Maillard (1976), on the other hand, notes differences between the excretory formulae of his *L. minimus* from French Mediterranean lagoons and Matthews' (1973b) worm from British *C. edule*, the former having 72 and the latter 60 flame cells. Moreover, Maillard (1976) found, in *C. edule* from the coast of Brittany (France), a bucephalid larva believed to represent *L. minimus* but having a number of flame cells intermediary between those of the above-reported forms. Maillard attributed these differences to possible intraspecific geographic variation (!). Whether these larvae actually represent 1, 2 or, possibly, 3 species of bucephalids, is uncertain. It is generally accepted that the excretory formula (i.e., the number and arrangement of flame cells) is important in trematode classification. In the Bucephalidae, however, there is, thus far, no agreement between accepted generic features and the excretory system (Hopkins, 1956c; Stunkard, 1974a).

Bucephalid cercariae from Mediterranean *Tapes aureus* were found to develop experimentally into a previously undescribed adult which was designated *Bucephalus baeri* (Maillard, 1976). Larval *'B. haimeanus'*, reported from *T. aureus, T. decussatus* and *T.*

pullastra by Vaullegeard (1894), Pelseneer, (1906), Palombi (1934a) and Andréu (1949), are possibly all referable to *B. baeri*. Sinitsin's (1911) *Cercaria hydriformis* from Black Sea *Tapes rugatus* might be an additional record of *B. baeri*. '*B. haimeanus*'-type cercariae, described from *Spisula* (*Mactra*) *solida* by Huet (1893), from *Donax trunculus* by Giard (1897a, footnote p. 955) and from *Abra* (*Syndosmya*) *alba* by Pelseneer (1906), represent still another — or, more probably, 3 other — bucephalid species. The most confusing example of misidentification it that of Tennent (1905, 1906, 1909), who described a larval gasterostome from North American *Crassostrea virginica* as *B. haimeanus*, thereby disregarding McCrady's (1874) valid description of the parasite of the American oyster under the name *B. cuculus*. Tennent (1906) furthermore claimed that his '*B. haimeanus*' develops into *Gasterostomum* (= *Prosorhynchoides*) *gracilescens* in *Tylosurus marinus* (= *Strongylura marina*).

The life cycle of *Prosorhynchoides gracilescens* has been pieced together by Matthews (1974), who found the cercaria in *Abra alba*. The metacercaria is specific to gadoid fishes and the adult occurs in the angler, *Lophius piscatorius*. In the literature, *P. gracilescens* is generally listed under the generic names *Bucephalopsis* Nicoll, 1914, or *Bucephaloides* Hopkins, 1954a. Stunkard (1976), however, declared the latter a junior synonym of *Prosorhynchoides* Dollfus, 1929. Hence, the correct name for Matthews' (1974) cercaria from *Abra alba* is *Prosorhynchoides gracilescens*.

Lebour (1912) erroneously linked the cercaria of *Labratrema minimus* (misnamed *Bucephalus haimeanus*) from *Cardium edule* with the then known stages in the life cycle of *Prosorhynchoides gracilescens*. In the same paper she described *Bucephalus syndosmyae* from *Syndosmya* (= *Abra*) *alba*. According to Matthews (1974), *B. syndosymae* differs from *P. gracilescens* in the morphology of both the cercaria and the sporocyst. Both species have been obtained from *A. alba* taken off Millport in the Firth of Clyde, Scotland. Whether the bucephalid found in the same host in France by Pelseneer (1906) is identical with either of these species, remains unknown.

Sporocysts and cercariae of *Prosorhynchus squamatus* and *P.* (= *Rudolphinus*) *crucibulum* occur in *Mytilus edulis*. The larvae of both species are easily distinguishable on the basis of gross morphology, the former having a trilobed and the latter a bilobed tail stem (Matthews, 1966, 1973a; Figs 13-75 and 13-76). The cercaria of *P. squamatus* had previously been described from mussels in England as *Bucephalus mytili* by Cole (1935) and was recorded from White Sea *M. edulis* by Chubrik (1952a, 1966), who also pieced together the other stages in the life cycle of this species. Whether Selikman's (1966) identification of *P. squamatus* in White Sea mussels *Musculus laevigatus* is correct remains to be established. '*P. squamatus*', reported from *Natica clausa* in the same paper, beyond doubt represents a distinct species. '*B. mytili*' found in *M. edulis* from Aveiro, Portugal (Días and Serrano, 1972), is probably referable to *P. crucibulum*. A gasterostome cercaria, recovered from the mytilid *Modiolaria* (= *Musculus*) *discors* at Egedesminde (Greenland) by Levinsen (1881) and named *Bucephalus crux*, is possibly also identical with *P. squamatus*, as already suggested by Odhner (1906). Stunkard (1974a) demonstrated that *P. squamatus* and *P. crucibulum* are not congeneric. He erected the new genus *Rudolphinus* with *R. crucibulum* as type species. Peneda (1965) reported *'B. mytili'* from both *M. edulis* and *Cardium edule* from the Ría de Aveiro and concluded that the parasite is not host-specific. However, her *'B. mytili'* in the mussel actually represents *R. crucibulum*, not *P. squamatus*, and the one in the cockle *Labratrema minimus*.

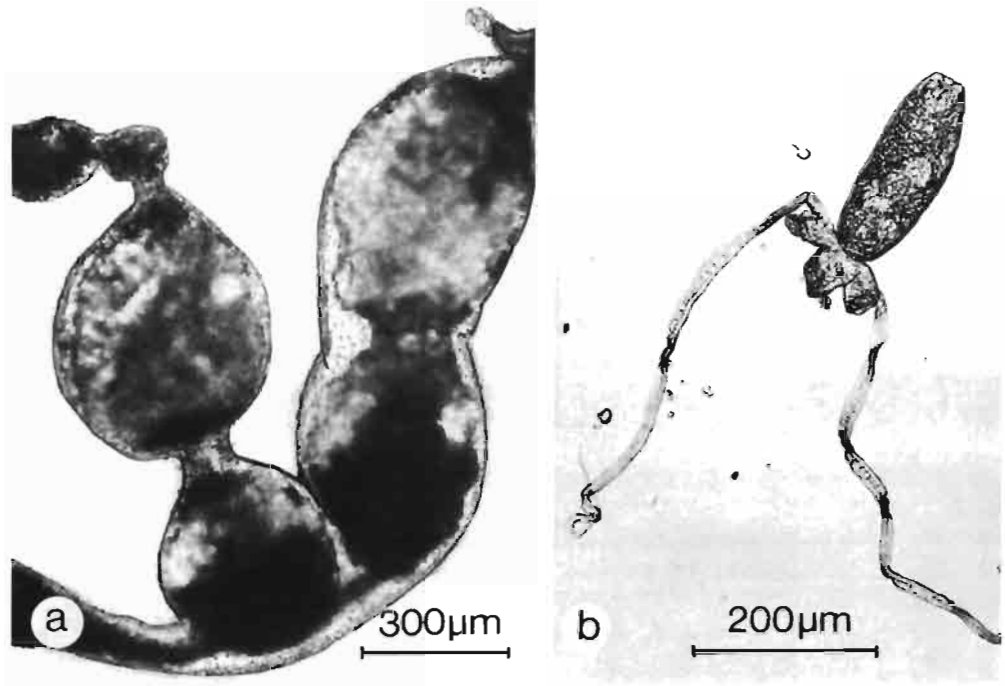


Fig. 13-75: *Prosorhynchus squamatus* from *Mytilus edulis*. (a) Dichotomously branched sporocyst dilated into chambers containing developing cercariae. (b) Fully developed cercaria. Note characteristic trilobate tail stem. (Original.)

The first marine gasterostome trematode reported from American waters is *Bucephalus cuculus*, parasitic in *Crassostrea virginica* (McCrady, 1874). Its cercaria has been misidentified by Tennent (1905, 1906, 1909) as *B. haimeanus*, the bucephalid of European oysters *Ostrea edulis*. In Tennent's experimental work, life-cycle stages of several other bucephalids have been confused with *B. cuculus*, and, although its life history is still unknown, Hopkins (1954a) suspected that the larva from *C. virginica* might develop into an adult of the genus *Rhipidocotyle*.

McCrady's (1874) larval *Bucephalus cuculus* was found in *Crassostrea virginica* from Charleston, South Carolina. Cercariae believed to represent the same species were subsequently reported from Louisiana oysters by Glaser (1904), Hopkins (1954a) and Menzel and Hopkins (1955), from Maryland oysters by Sprague (1964) and from various other localities along the North American Atlantic and Gulf coasts by Hopkins (1957b). Bucephalid cercariae were also recorded from oysters in New Jersey (Nelson, 1890, 1903, 1915; Feng and Canzonier, 1970; Feng and co-authors, 1970), in Delaware (Tripp, 1973) and in Rhode Island waters (Cheng, 1965a; Cheng and Burton, 1965a, 1966). Whether all of these represent a single species, i.e., *Bucephalus cuculus*, remains to be established. The *Bucephalus* sp. reported by the latter authors was found to be morphologically similar to *B. cuculus*. Cheng and Burton (1965a), however, noted certain discrepancies between the primary site of infestation of their *Bucephalus* sp. and that of *B. cuculus*, as reported by other workers (and confirmed by them). *B. cuculus* sporocysts are mainly (and in light infestations exclusively) confined to the oyster's gonads, whereas the primary site of

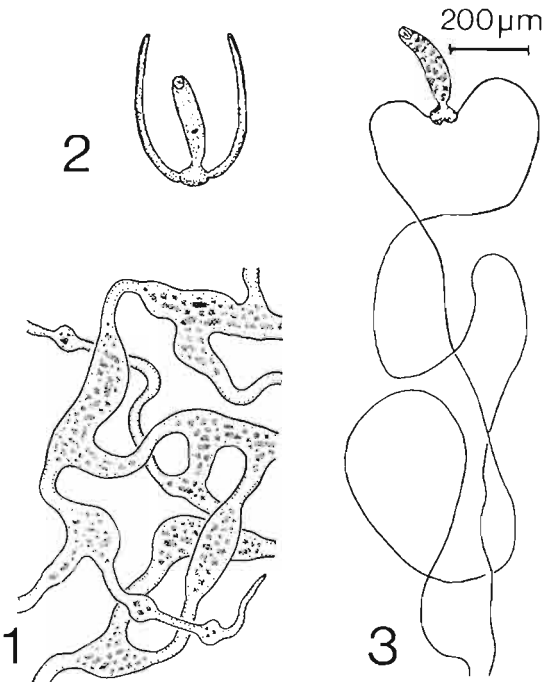


Fig. 13-76: *Rudolphinus crucibulum*. 1: Portion of dichotomously branched sporocyst; 2: cercaria, furcae retracted; 3: cercaria, furcae fully extended. (After Matthews, 1973a.)

infestation by *Bucephalus* sp. are the spaces between the digestive diverticula. According to Hopkins (1954a) there is no reason why 2 or more species of bucephalids could not occur in *C. virginica*, but so far there is no evidence of specific differences between the gasterostome cercariae in oysters described by different American workers.

Further records of larval bucephalids from the North American Atlantic and Gulf coasts and the Caribbean include *Rhipidocotyle transversale* and *R. lintoni*. Both species employ the same second intermediate and definite hosts. Cercariae found in paper shells *Lyonsia hyalina* cannot be assigned with certainty to either species because identifying morphological features are not yet developed, but probably both *R. transversale* and *R. lintoni* occur in *L. hyalina* (Stunkard, 1974a, 1976). Another larval bucephalid, first seen by Loesch (1957) and later described and provisionally named *Bucephalus loeschi* by Hopkins (1958a), occurs in *Donax variabilis* from Texas. *Ostrea equestris* from the same area harbour another unidentified bucephalid (Hopkins, 1951). Further American gasterostome cercariae have been reported as *Cercaria N* from *Pinna cornea* in the Dry Tortugas (Miller, 1925), as *Cercaria caribbea XLII* from Caribbean *Donax denticulatus*, *Tellina lintea* and *T. martinicensis* (Cable, 1956, 1963; Wade, 1967), and as *Cercaria apalachiensis* from *Mulinia lateralis* in Biscayne Bay, Florida (Holliman, 1961). An unnamed larval gasterostome infests *Tagelus divisus*, also in Biscayne Bay (Fraser, 1967). From the North American Pacific coast, *Cercaria noblei* has been described as a parasite of *Mytilus californianus*. Giles (1962) discussed its relationship with *Prosorhynchus squamatus* ('*Bucephalus mytili*') and *Bucephalus crux* reported from Europe and Greenland. The complete life cycles of all of these gasterostomes are as yet unknown.

Records of larval marine bucephalids from the southern hemisphere include *Bucephalus* (*urophyci*?) and *Prosorhynchus* (*australis*?) from mussels *Brachidontes rodriguezi* obtained from Puerto Quequén, Argentina, and *Bucephalus* (*chilensis*?) from *Semimytilus algosus* taken near Valdivia, Chile (Szidat, 1963). *Bucephalopsis modiolae* has been described from South African *Modiola capensis* (Faust, 1928). In Australia, Roughley (1933) and Sanders (1966) reported parasitization of Australian *Saccostrea cucullata* and *Pecten alba* by unidentified larval bucephalids. In New Zealand, *Bucephalus* sp. and *B. longicornutus* occur in *Mytilus latus* and in *Ostrea lutaria*, respectively (Haswell, 1903; Millar, 1963; Howell, 1966, 1967).

In Japan, pearl oysters *Pinctada martensi* were found infested by a gasterostome cercaria which was named *Bucephalus margaritae*. The parasite was found to have serious effects on the formation of pearls (Ozaki and Ishibashi, 1934). Unidentified larval *Bucephalus* spp. have been recorded from estuarine bivalves *Musculista arcuatula*, green mussels *Mytilus viridis* and Indian backwater oysters *Crassostrea madrasensis* in India (George and Balakrishnan Nair, 1974; Stephen and Joseph, 1977; Joseph, 1978), as well as from *Ostrea belcheri* in Pakistan and from *C. gigas* in Taiwan (Laird, 1961; Sindermann and Rosenfield, 1967).

In spite of the great number of known larval and adult gasterostomes, only a few complete life histories have been worked out experimentally. Tennent's (1905, 1906) early account of the bucephalid(s) from *Crassostrea virginica* are highly equivocal. The author erroneously assumed that the larvae found by him in the oyster are identical with those described by McCrady (1874) as *Bucephalus cuculus* and represent, in fact, those of *B. haimeanus*. On the basis of dubious experiments he furthermore concluded that the larval '*B. haimeanus*' develops into *Gasterostomum* (= *Prosorhynchoides*) *gracilescens* in gar *Tylosurus marinus*, silversides *Menidia menidia* acting as second intermediate hosts. Later (1909), Tennent claimed to have obtained experimental infestations in oysters by injecting suspensions of faeces from *Lepisosteus osseus* harbouring adult '*Gasterostomum gracilescens*' between the valves of uninfested oysters placed in a wire box in the open water. As stated earlier, Tennent has confounded stages of several species of bucephalids.

The first entire life cycle of a marine representative of the Bucephalidae traced reliably by artificial infestation of intermediate and final hosts was that of *Bucephalus longicornutus* in New Zealand. In a pioneering study, Howell (1966, 1967) demonstrated that the gasterostome cercaria previously reported from *Ostrea lutaria* by Millar (1963) would attack and encyst in fishes of the genus *Tripterygion* and in *Acanthoclinus quadridactylus*. Metacercariae developed into adults in *Scorpaena cardinalis* and in *Kathetostoma giganteum*, the natural definite host of the parasite in Foveaux Strait. The worms were identified as *Bucephalus longicornutus*, originally described as *Alcicornis longicornutus* from the same fish host in Cook Strait by Manter (1954). However, experimental infestation of *O. lutaria* with ova obtained from adult *B. longicornutus* was unsuccessful.

In a long series of studies, Sakaguchi (1962, 1964, 1965, 1966a, b, 1967, 1968) traced the life cycle of a bucephalid of the pearl oyster *Pinctada martensi* in Japan, previously described as *Bucephalus margaritae* by Ozaki and Ishibashi (1934). Metacercariae developed in a number of small fish species including *Rudarius ercodes*, which was used to experimentally infest horse mackerels *Caranx* spp. Adult worms recovered from the intestine of the definite hosts were identified as *Bucephalus varicus* Manter, 1940.

Successfully employing 0-group turbot *Scophthalmus maximus* and dab *Limanda*

limanda, as well as adult five-bearded rocklings *Onos* (= *Ciliata*) *mustela* and common gobies *Pomatoschistus minutus* as second intermediate hosts, Matthews (1973a) experimentally linked the cercaria from *Mytilus edulis*, described as *Bucephalus mytili* by Cole (1935), with the adult stage found in *Conger conger*. The adult has long been known as *Monostomum crucibulum* Rudolphi, 1819, and has now, after repeated rediscription, become *Rudolphinus crucibulum* (Odhner, 1906) Stunkard, 1974.

Maillard (1975a) traced the life cycle of *Labratrema minimus* by experimental infestation of the intermediate and final hosts. Its sporocysts and cercariae develop in *Cardium glaucum*; the metacercariae occur in gobies *Pomatoschistus microps* and smelt *Atherina mouchon*; and the adult parasitizes in the intestine of bass *Dicentrarchus labrax*. Free-swimming miracidia were not obtained and experimental infestation of cockles with ova taken from dissected adult worms was unsuccessful.

The last complete marine bucephalid life cycle worked out experimentally is that of *Bucephalus baeri*. Cercariae from *Tapes aureus* develop into metacercariae in *Pomatoschistus microps* and further into adults in *Dicentrarchus labrax* (Maillard, 1976). As was the case with *Labratrema minimus*, experimental infestation of the first intermediate host with ova taken from mature *B. baeri* was unsuccessful. Other bucephalid life histories have merely been pieced together; they will not be considered here further.

Infestation of marine bivalves with larval gasterostomes varies greatly with locality and season, as well as with the host and parasite species involved. Incidences well over 30 % have been recorded from oyster and clam populations in various parts of the world (Table 13-10). Szidat (1963) even found a 100 % infestation in *Semimytilus algosus* (an unexploited species, however) from Valdivia, Chile. The impact of such levels of parasitization on commercially exploited molluscan populations is obvious.

Larval bucephalids are probably the most deleterious metazoan parasites of marine bivalves. Their conspicuous dichotomously branching sporocysts (Fig. 13-76, 1), which form a densely interwoven network, infiltrate practically every organ except the foot. Usually the gonad is most heavily invaded. Terminal growth into the haemocoelic spaces leads to a gradual destruction and replacement of the molluscan tissue by the sporocyst mass. Infiltration of the gonad results in partial or, mostly, complete parasitic castration. Eventually all of the molluscan tissues become depleted and host survival is doubtful.

Usually both sexes of the bivalve host are affected and sterilized by bucephalids to a similar extent. A deviation from this general rule has been observed in *Pecten alba*. Normally this scallop is hermaphroditic. In Port Phillip Bay (Victoria, Australia), however, up to 67 % of the scallop population were found to consist of sterile females. Their proportion increased from 4 % in size class 71-75 mm to 81 % in size class 96-100 mm. The absence of sterile females below 70 mm shell length suggested that they arise from formerly hermaphroditic individuals. Closer examination of sectioned material revealed the presence of unidentified larval bucephalids in the gonads of all sterile females, and their absence in the hermaphrodites (Sanders, 1966).

The opposite, i.e., hermaphroditism produced by bucephalid infestation, has been observed in *Crassostrea virginica*. In 3 males infested with *Bucephalus* sp. (the species which invades the digestive gland rather than the gonad), clusters of eggs or single eggs were found in the midst of well-developed testes. This phenomenon did not occur in uninfested oysters, and sperm was never seen in the ovaries of *Bucephalus*-infested females. Sporocyst branches were found near, but not touching or penetrating, the gonad,

Table 13-10
Infestation of marine bivalves with larval Bucephalidae
(Compiled from the sources indicated)

Host species	Parasite	Host-sample size	Infestation incidence (%)	Locality	Source
Crassostrea virginica	*Bucephalus (cuculus?)*	several 100	up to 25	Beaufort, North Carolina	Tennent (1906)
Crassostrea virginica	*Bucephalus (cuculus?)*	n.i.[1])	more than 33	Various localities on North American Atlantic and Gulf coasts	Hopkins (1957b)
Crassostrea virginica	*Bucephalus* sp.	436	5.3	Ninigret Pond, Rhode Island	Cheng and Burton (1965a)
Crassostrea virginica	*Bucephalus* sp.	n.i.	approx. 30 to 60	Navesink River, New Jersey	Feng and Canzonier (1970)
Crassostrea virginica	*Bucephalus* sp.	274	13.9	Mispillion River, Delaware	Tripp (1973)
Donax variabilis	*Bucephalus loeschi*	1,017/100	0.29/2.0	Mustang Island, Texas	Hopkins (1958a)
Mulinia lateralis	*Cercaria apalachiensis*	446	0.22	Live Oak Point, Florida	Holliman (1961)
Lyonsia hyalina	*Rhipidocotyle transversale/R. lintoni*	n.i.	varying, 5 % or less	Woods Hole, Massachusetts	Stunkard (1976)
Mytilus californianus	*Cercaria noblei*	2,116	0.42	Dillon Beach, California	Giles (1962)
Mytilus edulis	*Prosorhynchus squamatus*	approx. 1,000	2 individuals infested	Conway, Wales	Cole (1935)
Mytilus edulis	*Prosorhynchus squamatus*	n.i.	up to 42	Coutances, French coast of English Channel	Breton (1970)
Mytilus edulis	*Prosorhynchus squamatus*	6,907	0.06	Cardigan Bay, Wales	Matthews (1973a)
Mytilus edulis	*Prosorhynchus squamatus*	63	1.59	Grimsey Island, Iceland	Sannia and James (1977a)
Mytilus edulis	*Rudolphinus crucibulum*	6,907	0.26	Cardigan Bay, Wales	Matthews (1973a)
Mytilus edulis	*Rudolphinus crucibulum*	665	2 to 60	Baltic Sea (GDR)	Reimer (1970)
Mytilus viridis	*Bucephalus* sp.	n.i.	n.i.	Someshwar, South Kanara, India	Stephen and Joseph (1977)
Musculus laevigatus	*Prosorhynchus squamatus*	77	19.5	White Sea	Selikman (1966)
Cardium edule	*Labratrema minimus*	n.i.	approx. 2 to approx. 10	Fenham Flats, Northumberland/ Emsworth, Hampshire	Lebour (1912)
Cardium edule	*Labratrema minimus*	420	26.4	Plymouth, England	Hutton (1952)

Table 13-10 (continued)

Cardium edule	*Labratrema minimus*	n.i.	approx. 2	North Wales	Cole (1956a)
Cardium edule	*Labratrema minimus*	50	18	Burry estuary, Wales	Hancock and Urquhart (1965)
Cardium edule	*Labratrema minimus*	10,500	9.5	Burry estuary, Wales	James and Bowers (1967c)
Cardium edule	*Labratrema minimus*	n.i.	40.7	Arcachon, France	Deltreil and His (1970)
Tapes aureus	*Bucephalus baeri* (?)	n.i.	more than 50	Gulf of Naples, Italy	Palombi (1934a)
Tapes decussatus	*Bucephalus baeri* (?)	several 1,000	2 individuals infested	Gulf of Naples, Italy	Palombi (1934a)
Tapes decussatus	*Bucephalus baeri* (?)	n.i.	approx. 7	Luc-sur-Mer, France	Vaullegeard (1894)
Tapes pullastra	*Bucephalus baeri* (?)	n.i.	approx. 7	Luc-sur-Mer, France	Vaullegeard (1894)
Tapes rugatus	*Cercaria hydriformis*	103	3	Sebastopol, Black Sea	Sinitsin (1911)
Abra alba	*Bucephalus syndosmyae*	5	20 (= 1 of 5)	Millport, Scotland	Lebour (1912)
Abra alba	*Prosorhynchoides gracilescens*	300	approx. 15 (up to 50 % in 4-year-old *A. alba*)	Firth of Clyde, Scotland	Matthews (1974)
Ostrea lutaria	*Bucephalus longicornutus*	22/72	13.6/52.8	Foveaux Strait, New Zealand	Millar (1963)
Ostrea lutaria	*Bucephalus longicornutus*	17/100	47/18	Foveaux Strait, New Zealand	Howell (1967)
Pecten alba	unidentified bucephalid	n.i.	67	Port Phillip Bay, Victoria, Austr.	Sanders (1966)
Pecten alba	same	1,250	31	Port Phillip Bay	Sanders and Lester (1981)
Ostrea belcheri	*Bucephalus* sp.	20	15	Karachi, India	Laird (1961)
Musculista arcuatula	*Bucephalus* sp.	30	6.7	Neendakara Barmouth, India	George and Balakrishnan Nair (1974)
Crassostrea madrasensis	*Bucephalus* sp.	n.i.	3.5	Mulki estuary, South Kanara, India	Joseph (1978)
Brachidontes rodriguezi	*Bucephalus* (*urophyci* ?)	n.i.	2.9	Puerto Quequén, Argentina	Szidat (1963)
Brachidontes rodriguezi	*Prosorhynchus* (*australis* ?)	n.i	12.5	Puerto Quequén, Argentina	Szidat (1963)
Semimytilus algosus	*Bucephalus* (*chilensis* ?)	n.i.	up to 100	Valdivia, Chile	Szidat (1963)

[1]) n.i. – Sample size not indicated

which supports the notion of physiological rather than mechanical disturbance of gonad development in the case of *Bucephalus* sp. Protandry is the rule in *C. virginica*, and hermaphroditism has been reported only rarely in functional males. It was suspected that

the association of bucephalid infestation with oyster bisexuality might be due to (i) premature 'ageing' of foci in the gonad, (ii) deprivation of the gonadal tissue, by the sporocysts, of a metabolite (glycogen?) essential for testes maturation, or (iii) damage to the cerebral and/or visceral ganglia by the developing parasite resulting in disruption of normal neurosecretory activity required for testicular development (Tripp, 1973).

Infestation by larval bucephalids significantly reduces the host's overall resistance to environmental stress and may affect the bivalve in a variety of ways. Thus, dying cockles *Cardium edule*, washed ashore on Llanrhidian Sands, South Wales, had an unusually high incidence of infestation with '*Cercaria bucephalopsis haimeana*' (= *Labratrema minimus*). It was assumed that the parasite, in weakening the cockle, makes it more susceptible to adverse changes in environmental conditions (Bowers, 1969). Similarly, Deltreil and His (1970) found *L. minimus*-infested cockles from Arcachon (France) to be in a very bad physiological condition and almost in a dying state. Such individuals were unable to perform their normal digging movements essential for burrowing in the sediment. Howell (1967) observed weakening of the adductor muscle and resultant gaping of the valves in New Zealand mud oysters *Ostrea lutaria* infested with *Bucephalus longicornutus*. Muscle weakening and gaping is also typical of *Mytilus edulis* harbouring sporocysts and cercariae of *Prosorhynchus squamatus*. Infested individuals could easily be separated from healthy ones by selecting gapers from batches of mussels kept in air for several hours (Lauckner, unpublished). Gaping and resultant reduced survival of such animals may pose serious problems in shipping and marketing mussels from populations with high incidences of *P. squamatus* infestation.

Over the 6-month period during which the sporocysts of a specifically unidentified bucephalid parasitizing *Pecten alba* in Port Phillip Bay, Victoria (Australia), produced cercariae, the average weight of the scallops' muscle (i.e., the portion which is marketed) fell below that of unparasitized individuals, presumably reflecting a strain on the energy resources of the host (Sanders and Lester, 1981).

Japanese pearl oysters *Pinctada martensi* harbouring sporocysts and cercariae of *Bucephalas varicus* showed a marked decline in condition. A high percentage of infested individuals died after insertion of pearl cores and the remainder produced pearls of low quality (Sakaguchi, 1964, 1965).

Survival of *Ostrea lutaria,* infested with *Bucephalus longicornutus* and kept in an aquarium for 6 months, was considerably reduced as compared to that of healthy oysters (Millar, 1963):

	Number parasitized	Number not parasitized	Infestation incidence (%)
Oysters that died	35	17	67.3
Oysters that remained alive	3	17	15.0

The difference between both groups (Chi-square = 13.83) is statistically highly significant ($p = 0.2 \times 10^{-3}$). Similar experimental results have been obtained by Howell (1967) who, from extensive field studies, concluded that *B. longicornutus* may be responsible, at least in part, for the decline in catch per effort on the Foveaux Strait (New Zealand) oyster beds. Huet (1888a), Tennent (1906), Menzel and Hopkins (1955), Hopkins (1957b), Cheng and Burton (1965a), Howell (1967), Bowers (1969) and Deltreil and His (1970) have also suggested that bucephalids are capable of killing or at least debilitating their first intermediate host.

The incidence of infestation of bivalves with larval bucephalids usually increases with

age. Such an increase is to be expected since, by chance alone, more individuals must become infested the longer the host is exposed to the invasive stage, i. e., the miracidium. In samples of *Abra alba* harbouring *Prosorhynchoides gracilescens*, 10, 26 and 50 % infestations were recorded in groups of 2-, 3- and 4-year-old individuals, respectively (Matthews, 1974). Selikman (1966) found bucephalids, believed to represent *Prosorhynchus squamatus*, in 5.4 % of 74 *Musculus laevigatus* 3 to 10 mm long and in 19.5 % of 77 hosts 14 to 22 mm in length. Hancock and Urquhart (1965) recorded considerable annual variation in the *Labratrema minimus* incidence in 2 age groups of *Cardium edule* from Llanrhidian Sands (Burry Inlet, Wales) (Table 13-11). A drastic decline in percentage infestation in cockles over 4 years of age, as observed by Bowers (1969; Fig. 13-77, b) suggests an increase in mortality of infested individuals together with a possible increase in resistance to infestation with host age. In a given year class, larger cockles are more susceptible to infestation than smaller ones (Fig. 13-77, c). Susceptibility to bucephalid invasion may also be correlated with the breeding cycle of the host. Thus, *L. minimus* only grows in sexually mature but spent *C. edule* over 18 mm in length and 1 year old (Fig. 13-77, a).

Table 13-11
Cardium edule. Infestation with larval *Labratrema minimus* as a function of time and host age (After Hancock and Urquhart, 1965)

Month	Second-year cockles			Older cockles			Mean %
	Number examined	Number infested	% infested	Number examined	Number infested	% infested	
May 1959 to May 1960	123	19	15	108	8	7	12
May 1960 to May 1961	66	1	2	83	3	4	3
May 1961 to May 1962	38	0	0	50	9	18	12
Total	227	20	9	241	20	6	8

In 37 out of 40 infested cockles, inspected by Hancock und Urquhart (1965), no traces of sex products were seen. The remaining 3 contained either ova or sperm in addition to sporocysts. In contrast, Deltreil and His (1970) observed an abundant presence of sex products in 90 % of *Labratrema minimus*-infested *Cardium edule* from Arcachon (France). Parasitic castration was virtually absent from all cockle populations inspected by them, in spite of the high incidence of up to 40 % recorded at some of the sampling stations. Furthermore, the authors noted that, in the Arcachon region, *L. minimus* appears in *C. edule* in early spring und vanishes by the beginning of the winter. From March to October, infestation incidences were fairly constant. The authors did not indicate, however, whether the disappearance of the parasite is due to death of the sporocysts and subsequent regeneration of the molluscan tissues (which is very improbable) or due to the death of infested hosts (which is more likely). Deltreil and His' (1970) observation is in direct contrast to the conditions governing the abundance of *L. minimus* in *C. edule* on Llanrhidian Sands, as reported by James (1969; Fig. 13-77, a). Detailed ecological studies conducive to explain these discrepancies are lacking.

Growth acceleration due to parasitization, sometimes termed 'gigantism', has been observed or suspected to occur in a number of host-parasite systems (Vol. I, Chapter 12;

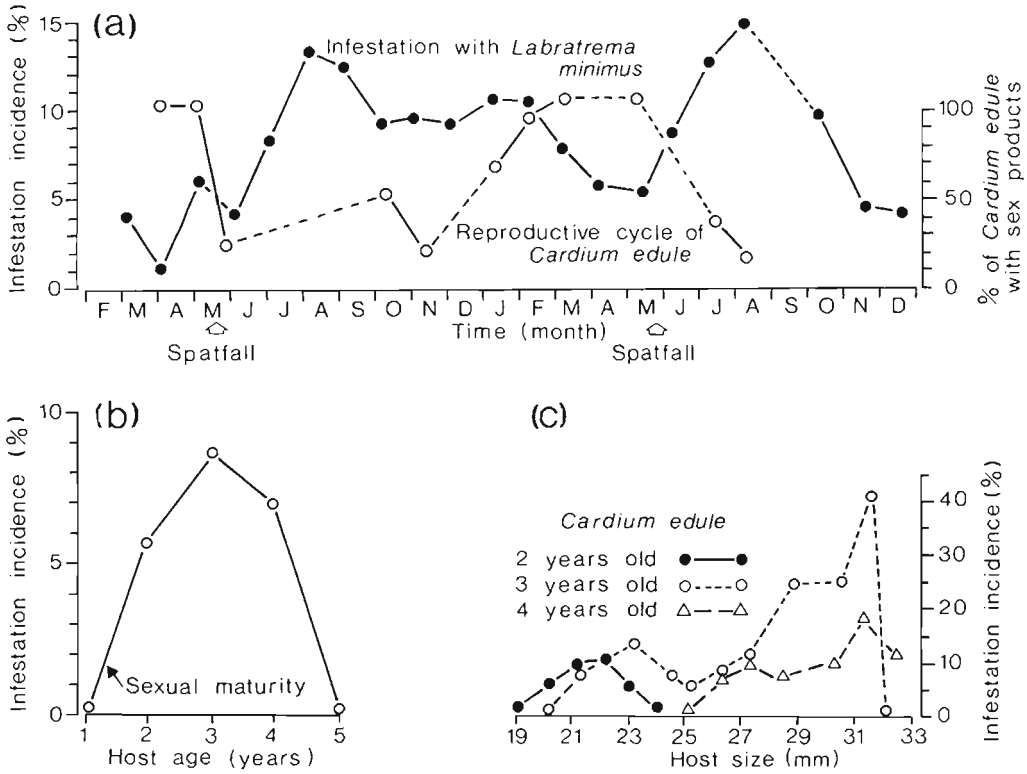


Fig. 13-77: *Cardium edule* infested with *Labratrema minimus*. (a) Seasonal variation in percentage infestation and in the reproductive cycle. (b) Variation in infestation incidence with host age. (c) Variation in infestation incidence with host size. (After Bowers, 1969.)

for review and literature consult Cheng, 1971). Similar conditions have been reported from bivalves infested with larval bucephalids. In *Cardium edule* parasitized by *Labratrema minimus* sporocysts and cercariae, the total flesh yield*) was significantly higher than that of unparasitized specimens (Fig. 13-78, a).

In order to determine whether this improvement was caused by the bulk of the parasite itself, the mass of sporocysts was carefully dissected away and the flesh yield of the remaining host tissue recalculated. The resulting decline in the flesh yield (Fig. 13-78, b) was not sufficient to reduce it to the level of uninfested cockles. It was concluded that the parasite was responsible for the increase in growth of the soft parts of the cockle. Concomitant growth enhancement was not observed in the shell of *L. minimus*-infested *C. edule*. On the contrary, the mean shell increment of 3-year-old cockles during their third year of life was 4.50 mm in parasitized cockles as opposed by 5.27 mm in healthy individuals (Bowers, 1969).

Similar effects — i.e., decrease in shell growth but improvement in flesh yield — have been observed in *Crassostrea virginica* parasitized by *Bucephalus cuculus* (Menzel and

*) Yield (%) $= \frac{\text{Meat volume}}{\text{Whole volume} - \text{shell volume}} \times 100$

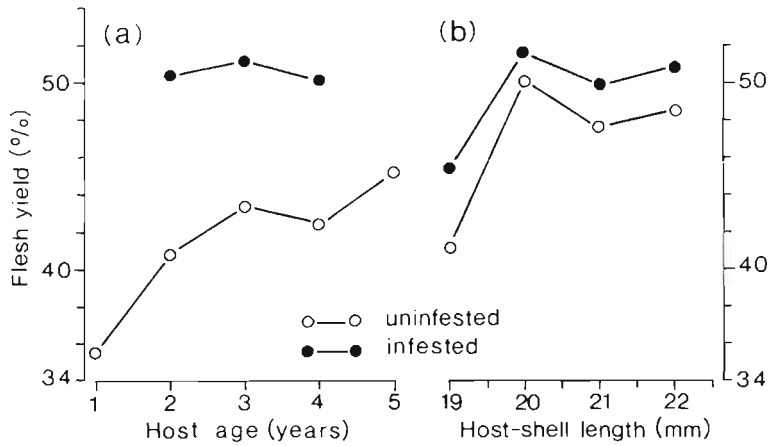


Fig. 13-78: *Cardium edule*. Effect of *Labratrema minimus* infestation on flesh yield. (a) Entire parasitized and unparasitized specimens of different year classes. (b) Flesh yield of 2-year-old cockles after removal of parasite tissue. (After Bowers, 1969.)

Hopkins, 1955). Sannia and James (1977a) report a fourfold increase in the total tissue volume of *Mytilus edulis* infested with *Prosorhynchus squamatus*. In mussels of a given year class, Breton (1970) found larger individuals to be more frequently parasitized by '*Bucephalus mytili*' (= *P. squamatus*) than smaller ones, which he interpreted as parasite-induced growth acceleration. No experimental data substantiating this assumption are presented, and Breton's interpretation is probably incorrect. Larger mussels simply screen larger volumes of water (Winter, 1969) and, hence, have a greater 'chance' of ingesting a miracidium than smaller individuals.

With respect to the oyster parasite, Menzel and Hopkins (1955) came to a curious conclusion. They stated (p. 341):

> "*Bucephalus cuculus* is actually beneficial from the gastronomic standpoint. In southern waters, normal oysters spawn so heavily and so long that they become emaciated and nearly tasteless by late summer, but *Bucephalus*-infected oysters, being castrated or 'caponized', remain fat and retain an excellent flavour all summer. Personal tests have proved that the *Bucephalus* sporocysts taste even better than the oyster."

Breton (1970), studying the infestation of *Mytilus edulis* with *Prosorhynchus squamatus*, came to exactly the same conclusion. As pointed out by Cheng (1965b, 1967; see below), consumption of trematode-infested molluscs may be hazardous due to the accumulation of toxic metabolites in the tissues of such animals.

Although parasitization of bivalves by larval bucephalids may be somewhat 'beneficial' in the early stages by improving the flesh yield, there can be little doubt that the parasite becomes more harmful in advanced infestations and, eventually, lethal in the terminal stage. After having destroyed the gonads and caused castration, the sporocysts invade and gradually break down virtually all essential organs of the bivalve host. In *Mytilus edulis* heavily parasitized by *Prosorhynchus squamatus*, for example, eventually so much host tissue is destroyed, crushed or distorted that "it is difficult to understand how such affected animals remained alive" (Sannia and James, 1977a).

Histopathological effects of bucephalid infestation have been studied in detail in oysters and cockles. In New Zealand *Ostrea lutaria*, moderately parasitized by *Bucephalus longicornutus*, connective tissue in the immediate vicinity of a sporocyst showed signs of necrosis with deformation of cells and some condensation of chromatin into small, irregularly shaped masses within the nuclei. In the gonad, hermaphrodite follicles adjacent to sporocyst tubules were partially or completely collapsed, and contained either a few degenerating eggs and sperm or none. In heavily invaded oysters, the visceral mass was reduced in size and of gelatinous appearance. Host cells were grossly deformed and in place of readily recognizable large, individual, vacuolated cells, clearly set off from one another, the appearance was that of a syncytium with continuous cytoplasm and scattered nuclei, some of the latter showing marked necrotic changes in the condensation of chromatin. There were also scattered chromatin particles in the cytoplasm. An increase in the number of haemocytes was common in the degenerating tissue. The haemocytes contained chromatin inclusions, presumably representing debris of connective-tissue nuclei, in their cytoplasm (Howell, 1967).

In digestive-gland cells of *Cardium edule* parasitized by *Labratrema minimus* (termed *Cercaria bucephalopsis haimeana* by James and Bowers, 1967a), 2 major effects are apparent: (i) Digestive cells cut off from their energy supply undergo degeneration. The initial disappearance of food vacuoles from such cells indicates that mechanical pressure exerted by the growing sporocysts may have crushed the digestive ducts, closing the lumen and blocking the passage of food from the stomach to the digestive tubules. The ensuing process may thus be regarded as starvation autolysis (James, 1965; Vol. I, Chapter 12). It is quite different from the histolysis of the digestive gland described from other mollusc-trematode associations. Thus, G. Rees (1934) and Cheng and Snyder (1962) interpret the histolysis observed by them as the result of a liberation of toxic excretory products of parasite origin into the host tissues. In *C. edule* infested with *L. minimus*, such a mechanism is not effective since the parasite's excretory products accumulate within the sporocyst lumen and do not pass into the host's haemocoel. (ii) Digestive cells not so cut off do not undergo starvation autolysis but their metabolism is, nevertheless, disturbed by the parasite. In these cells, glucose, glycogen, glycoproteins, phospholipids and proteolipid food storage globules, as well as acid mucopolysaccharides are reduced. Neutral lipids, fatty acids, alkaline phosphatase, acid phosphatase and non-specific esterase are increased, and there is also a compensatory increase in the number of food vacuoles. In the visceral haemocoel, in the neighbourhood of the sporocysts, glucose, fatty acids, neutral lipids and acid mucopolysaccharides are increased and glycogen is reduced (James and Bowers, 1967a). Corresponding studies on the distribution of carbohydrates, lipids and enzymes in the sporocyst of *L. minimus* have been conducted by James and Bowers (1967b). James and Bowers (1967c) and James and co-authors (1966) described the ultrastructure and reproduction in the sporocyst.

A series of detailed investigations have been conducted to determine the biochemical and histological changes in the tissues of *Crassostrea virginica* associated with *Bucephalus* sp. infestation. As already stated, this species differs from other larval bucephalids — and in particular from *B. cuculus* infesting the same host species — in that it primarily invades the intertubular spaces of the digestive gland, which are normally occupied by connective-tissue (Leydig tissue-)cells, rather than the oyster's gonad. The assumption that *Bucephalus* sp. invasion initiates in, and spreads from, the digestive gland is further substantiated by

the observation that sporocyst branches in the digestive gland are statistically significantly larger in cross section area than those in the gonad (Cheng and Burton, 1965a). In contrast, McCrady (1874), Tennent (1906) and Hopkins (1954a) have all pointed out that, in their studies on *Bucephalus cuculus,* the sporocysts primarily invade, and are concentrated in, the gonads — a statement confirmed by Cheng and Burton (1965a). There can be little doubt that *Crassostrea virginica* is host for at least 2 species of bucephalids, *B. cuculus* representing an entity of more southerly distribution, while *Bucephalus* sp. appears to be a more northerly form.

In light infestations, the *Bucephalus* sp. sporocysts merely exert mechanical pressure on the digestive diverticula, pushing these toward the periphery and against the gonadal tissues. A few sporocyst branches may also be found among the Leydig cells surrounding the gut. As sporocyst growth advances, tissue destruction resulting from both mechanical pressure and physiological decomposition becomes apparent. In older and heavy infestations, sporocyt branches can be found in every major organ and structure in the oyster except the heart. The resulting pathology is severe. The degenerate appearance and smaller size of the ova in heavily affected individuals suggest that some physiological pressure is being exerted by the parasite. Ova from infested oysters measure, on the average, 28 ×21 μm, and those from healthy individuals 53 × 30 μm. The difference is statistically highly significant (Cheng and Burton, 1965a).

In spite of the severe pathology associated with *Bucephalus* sp. infestations, there is

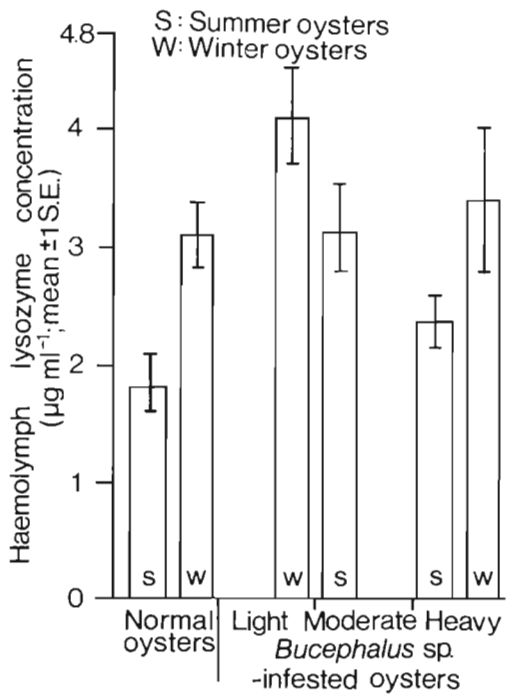


Fig. 13-79: *Crassostrea virginica.* Variation in oyster haemolymph lysozyme activities associated with season and *Bucephalus* sp. infestation. See also Fig. 13-38. (After Feng and Canzonier, 1970.)

usually little host response to the parasite. Douglass (1976) reported on a case in which young sporocysts in presumably new infestations elicited an intense cellular response in 3 of 7 oysters with similar infestation intensities. In one oyster, phagocytes were observed in the sporocyst proper. This response may be due to previous experience with the parasite. Normally, the number of haemocytes is not increased in the digestive gland or gonad of *Bucephalus* sp.-infested *C. virginica,* and is only slightly increased in the connective tissues of the palps, gills and mantle, although no phagocytosis or encapsulation occur (Cheng and Burton, 1965a). On the other hand, there may be massive biochemical alterations.

In *Crassostrea virginica* infested with *Bucephalus* sp., haemolymph lysozyme activities were significantly increased (Fig. 13-79). Total haemolymph protein was not greatly altered although a tendency toward a decline in concentration was apparent. Polyacrylamide gel electrophoresis of haemolymph from diseased and healthy oysters revealed one major fast-migrating anodal fraction (I) and 3 slow-moving cathodal fractions (II, III, IV).

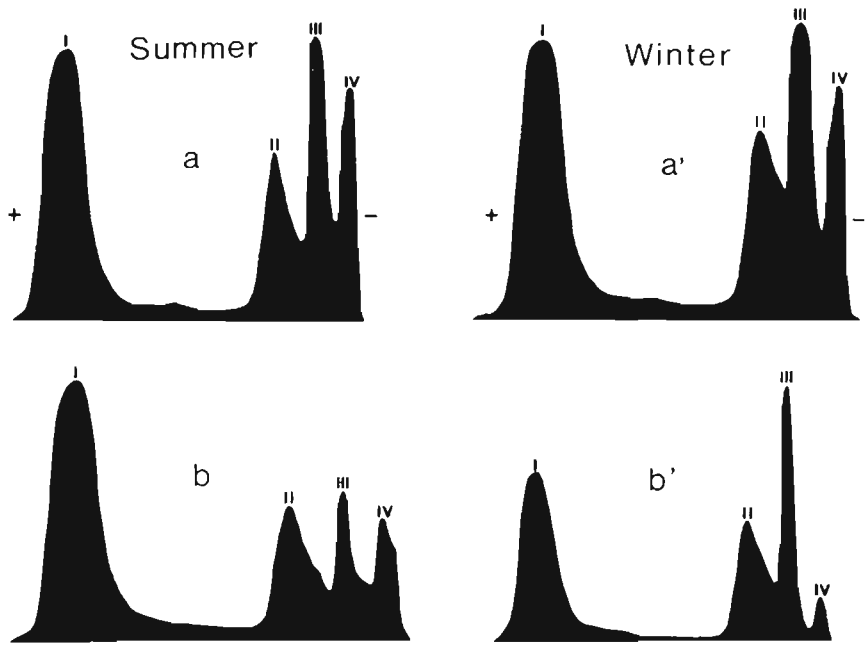


Fig. 13-80: *Crassostrea virginica.* Polyacrylamide gel electropherograms showing effect of *Bucephalus* sp. infestation on haemolymph proteins. (a) Pool of 8 summer-uninfested oysters; (a') pool of 8 winter-uninfested individuals; (b) pool of 4 summer-*Bucephalus*-infested oysters; (b') pool of 2 winter-*Bucephalus*-infested individuals (Anode on the left, cathode on the right; see also Fig. 13-39). (After Feng and Canzonier, 1970.)

Infested oysters displayed a significant increase in Fraction II with a concurrent diminution in Fraction IV (Figs 13-80 and 13-81). The quantitative change could be correlated with the severity of the infestation and was interpreted as evidence of host-humoural response to the presence of the parasite. However, the functional significance of the rise and the fall in haemolymph lysozyme activities and protein Fractions II and IV relative to protection of the host against the parasite has yet to be determined (Feng and Canzonier, 1970).

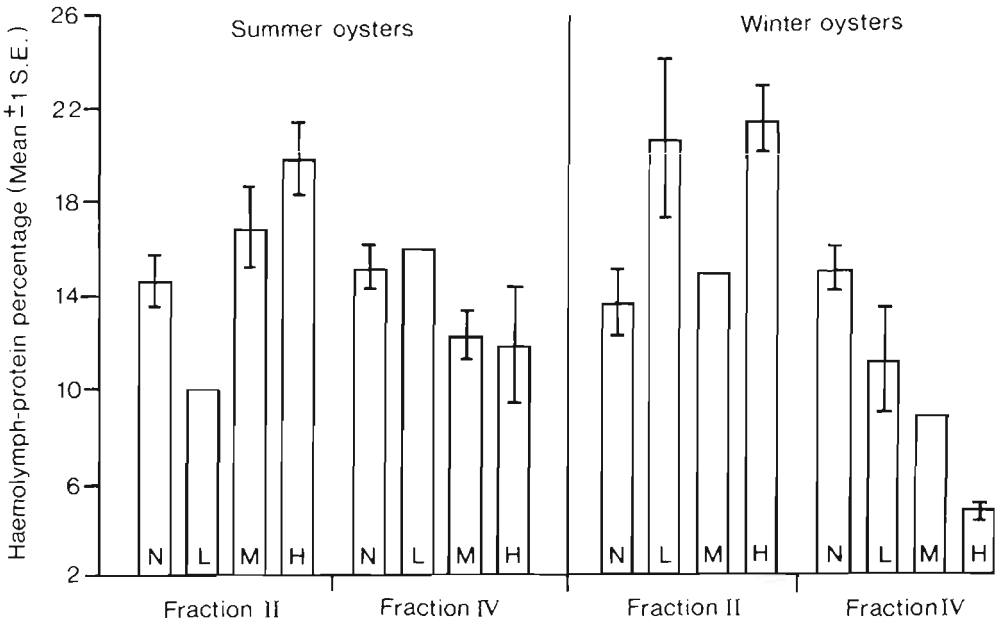


Fig. 13-81: *Crassostrea virginica*. Effect of *Bucephalus* sp. infestation on haemolymph protein fractions II and IV of normal oysters (N) and individuals with light (L), moderate (M) and heavy (H) infestations in relation to season. See also Fig. 13-40. (After Feng and Canzonier, 1970.)

Total non-protein nitrogenous compounds in the haemolymph exhibited a pronounced decrease in *Bucephalus* sp.-infested oysters while individual free amino acids (FAA) — mainly taurine, phosphoserine and aspartic acid — generally increased significantly (Table 13-12; Fig. 13-82). Differences in the concentration of certain FAA in oysters from the 2 sampling stations can, to a large extent, be ascribed to the salinity regime of the 2 areas, the salinity being higher at Cape Shore. It is known, for example, that changes in adductor muscle taurine and alanine concentrations are correlated with variations in the ambient salinity. The differences in total FAA between normal and infested oysters reach a minimum in the winter. This probably reflects the relative quiescence of the parasite. Lowering of ambient temperatures may conceivably affect the rate of transport and utilization of host-FAA by the *Bucephalus* sporocysts (Feng and co-authors, 1970).

Changes in the carbohydrate and lipid composition of oysters parasitized by *Bucephalus* sp. have been observed by Cheng (1965a) and Cheng and Burton (1966). There is a marked reduction of glycogen in the digestive-diverticular cells, in the connective-tissue cells located between the diverticula, in the matrices of the palps, mantle and gills, in the walls of blood vessels and in the ova. In healthy individuals, glucose deposits are present in the same Leydig cells in which glycogen is found. In infested oysters, the reduction of glucose at these sites is almost complete. In contrast, acid mucopolysaccharides remain unaltered.

In infested oysters there is also a dramatic change in the total fat picture. A complete or almost complete reduction of neutral fats, acidic fats and fatty acids in the Leydig cells is accompanied by a concomitant increase of these components in the digestive-diverticular

Table 13-12

Crassostrea virginica. Effect of infestation with *Bucephalus* sp. or concurrent infestation with *Bucephalus* sp. and *Haplosporidium nelsoni* on concentration (nanomoles ml^{-1}) of free amino acids and related compounds in the haemolymph of oysters from two different sampling stations and in relation to season. See also Table 13-5 (After Feng and co-authors, 1970; modified)

	Summer					Winter				
	Cape Shore (Delaware Bay)			Monmouth Beach (New Jersey)		Cape Shore (Delaware Bay)			Monmouth Beach (New Jersey)	
Amino acid or compound	NOR	BUC	HAP-BUC	NOR	BUC	NOR	BUC	HAP-BUC	NOR	BUC
Urea	725	488	714	1980	624	–	440	352	1030	318
Ammonia	520	356	368	512	449	299	344	85	399	128
Taurine	250	58	261*	152	324*	112	354*	314*	68	376*
Alanine	229	56	171	104	70	330	119	89	263	261
Glycine	124	46	56	106	85	109	39	61	136	118
Serine	120	14	16	53	26	87	74	20	339	182
PE (footnote)	72	19	75	57	46	434	191	136	416	167
β-alanine	71	60	28	43	14	104	35	27	68	55
Ornithine	69	25	43	14	19*	111	54	71	97	44
PS (footnote)	62	200*	21	76	112*	142	5	72	101	131*
Arginine	27	8	–	17	–	24	16	–	24	21
Lysine	20	+	+	7	–	17	9	11	23	19
Glutamic acid	12	8	103*	32	22	114	46	30	57	59
Leucine	8	5	–	7	6	5	–	–	+	+
Isoleucine	3	+	–	+	5*	6	+	–	+	+
GABA (footnote)	3	+	21*	2	+	11	46*	9	33	19
Aspartic acid	+	11*	37*	+	15*	7	32*	35*	3	7*
Threonine	+	5*	6*	+	5*	+	–	+	1	+
Methionine	–	+	–	+	8*	+	–	–	–	–
Total amino acids	1072	519	839	674	758	1615	1021	876	1631	1460
No. of oysters	3	2	3	13	7	3	3	4	11	5

NOR normal; BUC *Bucephalus*-infested; HAP-BUC infested with both *Haplosporidium nelsoni* and *Bucephalus* sp.; GABA γ-aminobutyric acid; PE phosphoethanolamine; PS phosphoserine; + trace quantities or less than 1 nanomole ml^{-1} haemolymph. In calculating total amino acids, + is counted as 1 nanomole; * denotes increases in amino acid and amine concentrations of infested oysters as contrasted with their appropriate controls (NOR). Histidine, proline, cystine, valine, tyrosine and phenylanaline not detected in samples.

cells. The observed continuous increase in fatty acids in diverticular cells could be explained by enzymatic hydrolysis of neutral fats, with the lipase detected in host tissues in the neighbourhood of sporocysts being of parasite origin. The increase of neutral fats in the diverticular cells, on the other hand, appeared to be stimulated, by some unknown mechanism, by the presence of the sporocysts (Cheng, 1965a).

Considering the profound depletion of body reserves in *Crassostrea virginica* infested with *Bucephalus* sp., as reported by Cheng (1965a) and Cheng and Burton (1966), it is difficult to understand Menzel and Hopkins' (1955) and Hopkins' (1957b) contention that oysters parasitized by *B. cuculus* are "fat-looking and full of glycogen". The only possible explanation would be that both bucephalids affect the oyster in quite a different way.

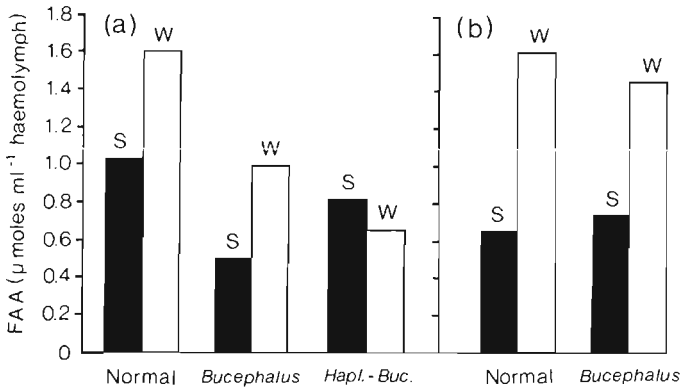


Fig. 13-82: *Crassostrea virginica*. Effect of *Bucephalus* sp. infestation or concurrent *Bucephalus* sp. and *Haplosporidium nelsoni* infestation on total free amino acid (FAA) pool of oysters from Cape Shore, Delaware Bay (a) and Monmouth Beach, New Jersey (b). S: Summer concentrations, W: winter concentrations. See also Fig. 13-41. (After Feng and co-authors, 1970.)

Cheng (1965b, 1967) called attention to the possibility that the accumulation, in the tissues, of butyric and other toxic short-chained fatty acids resulting from the degradation of the host's neutral fats by parasite-secreted enzymes may render trematode-infested molluscs unsuitable for human consumption.

Several cases of hyperparasitism by sporozoans have been recorded in marine bivalves infested with bucephalids. Microsporans *Nosema dollfusi**) invade *Bucephalus cuculus* parasitic in *Crassostrea virginica* from Chesapeake Bay. Spherical bodies, 1.5 to 2 μm in diameter, were tentatively identified as schizonts, and certain other bodies, 2 to 2.5 μm in diameter and intermediate in appearance between the vegetative stages and the spores, were interpreted as sporonts or sporoblasts. These elongate and transform directly into ovoid spores, which measure about 1.7 × 3 μm. Some of the spores were seen inside the cells of the sporocyst wall. Affected cells showed great hypertrophy of the cytoplasm, but the nuclei did not appear to be involved. Most of the spores occurred in the lumina of the sporocysts, either free or in cell fragments. The spores and cellular debris sometimes filled the entire sporocyst lumen. Normally developed cercariae were not seen in affected sporocysts.

One of the hyperparasitized oysters from Chesapeake Bay, which was dead at the time of inspection, harboured, in addition to *Nosema dollfusi*, an unidentified haplosporidian**). In this oyster there was a necrotic area in the visceral mass containing both sporozoans, which apparently had been liberated from a ruptured *Bucephalus cuculus* sporocyst. It was believed that the escape of the sporozoan spores into the host tissues could have contributed to the death of the oyster (Sprague, 1964, 1970a). Similarly, Mackin and Loesch (1955) described the destruction of *B. cuculus* sporocysts by an unidentified haplosporidian hyperparasite. Where the sporocyst wall had broken down, there was an intense cellular host reaction. The spores of this haplosporidian were ovoid and measured about 3 to 5 × 5 to 7 μm. Haplosporidians with plasmodial stages like those

*) compare comment on p. 732

**) It should be remembered that the classical 'haplosporidians' are now in order Balanosporida (p. 553, Table 13-3).

described by Mackin and Loesch (1955) have also been found in *Bucephalus*-infested oysters by Sprague (1970a). The plasmodia, which measured up to 30 × 50 μm, occurred most frequently in the sporocyst lumen, but were also seen intracellularly in the sporocyst wall. No spores were seen. Embryonic cercariae in affected sporocysts appeared degenerate.

Another haplosporidian hyperparasite, *Urosporidium constantae*, invades the sporocysts and cercariae of *Bucephalus longicornutus* in *Ostrea lutaria* from New Zealand. Some of the affected sporocysts were rich brown in colour and dilated to approximately twice their normal diameter. Embryonic cercariae within the sporocysts were degenerating or had entirely been replaced by spores or stages in sporogony of the hyperparasite. The characteristic drop-shaped, operculate spores were 4 to 5 μm in diameter and had a 10 to 12 μm long tail filament. In the oysters inspected, all cercariae were destroyed. Although schizogony was not clearly recognized, it was assumed that schizogonic stages were responsible for this fatal infestation. The nuclei of degenerated embryos showed marked necrotic changes and had undergone pyknosis and vacuolation. The sporocyst wall remained intact, but necrotic changes were apparent. No developmental stages of *U. constantae* occurred in the sporocyst wall (Howell, 1967). The author discussed the usability of *U. constantae* as a possible means of biologically controlling *B. longicornutus* infestation in *O. lutaria* on the heavily (> 40 %) affected Foveaux Strait oyster beds. Eventually, however, he came to the conclusion that ecological conditions, as well as the difficulty of collecting sufficiently large numbers of infestive spores, would preclude an effective utilization of the hyperparasite in the control of *B. longicornutus*.

Although the deleterious effects of bucephalid infestation on the bivalve first intermediate host are fairly well documented, much remains to be learned about the impact of metacercarial invasion on the second intermediate host, particularly on juvenile fishes. Matthews (1973a) obtained experimental development of *Rudolphinus crucibulum* in 0-group individuals of commercially important flatfishes *Scophthalmus maximus*. The metacercariae have also been reported from *Gadus morrhua*, as well as from a number of non-commercial fish species. 0-group *Pleuronectes platessa* are highly susceptible to invasion by *Labratrema minimus* ('*Bucephalus haimeanus*') during early summer, and 100 % natural infestation with as many as 200 metacercariae was recorded in plaice from Cardigan Bay, Wales. In young fishes, the liver is extensively damaged by migrating metacercariae and, in experiments, exposure to 50 or more *L. minimus* cercariae killed larval plaice within 2 days (Matthews, 1973b). The metacercariae of *Prosorhynchoides gracilescens* invade the nervous tissues of several species of commercially important gadoid fishes (Maddox, 1867; Dawes, 1947; Crofton and Fraser, 1955). Although the behaviour of moderately infested *Ciliata* (*Onos*) *mustela* in aquaria appeared unimpaired, heavy metacercarial burden might be expected to affect the motor and sensory systems of the fish host (Matthews, 1974). Szidat (1963) observed high incidences and intensities of *Prosorhynchus* sp. infestation in juvenile Argentine soles *Oncopterus darwini*, and Stunkard (1974b) identified from 10 to 40 % of silverside minnows *Menidia menidia* from Woods Hole as carriers of *Rhipidocotyle transversale* and *R. lintoni* metacercariae.

Pacific ocean perch *Sebastodes alutus* commonly harbour *Prosorhynchus* sp. metacercariae. The cysts appear as conspicuous whitish to black spots, 2 to 5 mm in length, in the muscle tissue. Of 805 *S. alutus* fillets examined, 39 % were found to be positive. Although the parasite does not infest man, fillets containing metacercariae present a serious aesthetic

problem. A related species of rockfish, *S. melanops,* is reputed to be so heavily parasitized by metacercariae that plant operators refuse to buy it except for mink food (Liston and co-authors, 1960).

Studies of the behaviour of bucephalid cercariae revealed that they do not perform active swimming movements but rely upon water turbulence to remain suspended (Hopkins, 1954a; Holliman, 1961; Howell, 1966; Matthews, 1974; Stunkard, 1976). Floating, dispersal and attachment to the second intermediate host are aided by the long, extendable and retractable furcae of the cercariae (Figs 13-75, b and 13-76, 3). Although the cercariae, often several hundred at a time, are forcibly discharged through the bivalve's exhalant siphon, they concentrate at or immediately above the bottom, thereby presenting a health hazard mainly for benthic fishes. Thus, for example, the greatest risk of *Labratrema minimus* infestation in *Pleuronectes platessa* follows their metamorphosis when the young flatfish become fully adapted for life on the bottom during the spring (Matthews, 1973b).

The number of cercariae shed by bucephalid-infested bivalves may be enormous. Thus, the total number of larvae liberated by 11 *Bucephalus longicornutus*-infested New Zealand *Ostrea lutaria* varied from 9,605 to as many as 126,545 within a period of 53 days, with peaks of up to 10,000 cercariae oyster^{-1} day^{-1} (Howell, 1966, 1967). By multiplying this figure by the incidence index in the first intermediate host — up to 47 % in *O. lutaria* —, one gains an impression of the possible — and probable — impact of bucephalids on marine fish populations. The influence of this factor on, say, 'natural mortality' in exploited fish species has never been analyzed in the field. There is an urgent need for such ecological studies which, however, are embarrassed by the gap in our present knowledge of gasterostome life cycles. With respect to the first 2 larval bucephalids reported from *Ostrea edulis* and *Crassostrea virginica*, respectively, Stunkard (1976, p. 313) has characterized the 'state of the art':

> "At present, a challenging problem in the family Bucephalidae is the discovery of the life cycles, secondary and definite hosts of *B. haimeanus* from the European oyster and of *B. cuculus* from the American oyster. It is distressing to realize that this situation has remained unresolved for more than a century."

Digenea utilizing fishes as final hosts: family Sanguinicolidae

The Sanguinicolidae comprise another family of fish-pathogenic digenetic trematodes whose cercariae occur in sporocysts in marine bivalves or in either sporocysts or rediae in polychaete annelids. There is no metacercarial stage, the cercaria penetrating the fish (final) host directly and developing into the adult worm in the blood vascular system or, rarely, the body cavity. Most of the larval and adult sanguinicolids described from marine hosts are listed as members of the family Aporocotylidae. Van der Land (1967), however, pointed out that Aporocotylidae Odhner, 1912, is a junior synonym of Sanguinicolidae von Graff, 1907. Hence, the latter family name is correct. Information pertaining to the Sanguinicolidae has been reviewed by J. W. Smith (1972).

Six marine sanguinicolid cercariae have, thus far, been found in pelecypods and 4 in annelids, but no complete life history has as yet been elucidated experimentally*).

*) Note added in proof: Very recently, Køie (1982) experimentally traced the entire life cycle of *Aporocotyle simplex*, a sanguinicolid which has a polychaete annelid, *Artacama proboscidea*, as sole intermediate host.

Unidentified and unnamed larval sanguinicolids have been reported from *Argopecten irradians* in the Woods Hole region (Massachusetts) and in *Tagelus divisus* in Biscayne Bay, Florida (Linton, 1915b; Fraser, 1967). Eight of 1,763 *Donax variabilis* from the Apalachee Bay area (Florida) were found to harbour *Cercaria asymmetrica*, and *Chione cancellata* from the same area are host for *Cercaria cristulata*, 1 of 120 clams containing sporocysts and cercariae (Holliman, 1958, 1961). *Cercaria mercenariae* parasitizes southern quahaugs *Mercenaria campechiensis* at Galveston Island (Texas), 3 of 5 and 5 of 23 clams from 2 sites having been found infested (Wardle, 1979).

All of the above-mentioned cercariae are typical sanguinicolid larvae in being apharyngeate, longicercous, brevifurcous, and provided with an anterior penetration organ (Fig. 13-83). Martin (1944b) described *Cercaria solemyae*, an aberrant brevicercous larva, believed to represent a member of the Sanguinicolidae, from *Solemya velum* collected in the vicinity of Woods Hole.

Apart from the 'usual' parasitic castration caused by the developing sporocysts, nothing else is reported about the effects of larval sanguinicolids on their molluscan hosts. Judging from the limited amount of information, one may conclude that the Sanguinicolidae are of rare occurrence in bivalves. The highest infestation incidences recorded are those reported for *Mercenaria campechiensis* and *Tagelus divisus* (see above). Of 664 *T. divisus* examined by Fraser (1967), 112 (16.7 %) had parasites, but this figure includes

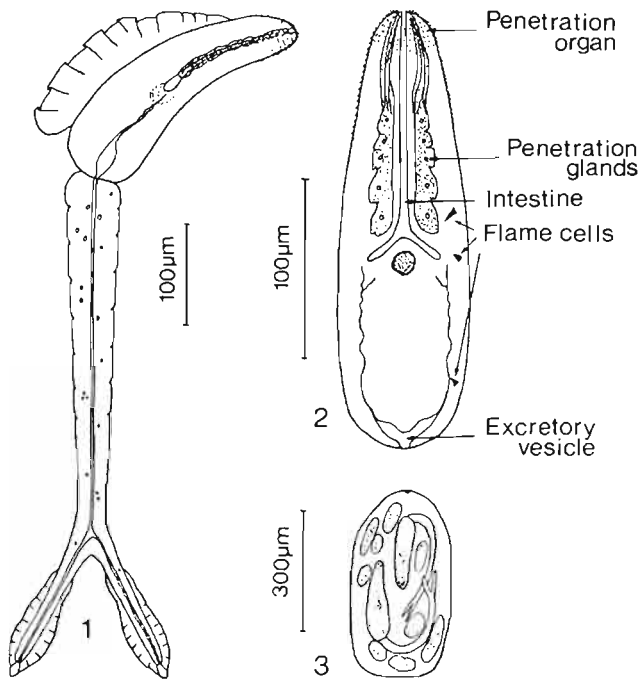


Fig. 13-83: Sanguinicolidae. *Cercaria mercenariae* from *Mercenaria campechiensis*. 1: Cercaria, body in lateral view, caudal furcae in dorso-ventral view; 2: cercarial body, ventral aspect; 3: sporocyst. (After Wardle, 1979.)

bucephalid infestations. The proportion of clams carrying sanguinicolids has not been stated. Sanguinicolids infesting marine annelids appear to be of similar low prevalence. Incidences, reported for 2 of the 4 known species, range from 0.8 and 3.3 % for *Cercaria loossi* occurring in *Hydroides dianthus* to 8.3 % for *C. hartmanae* parasitizing *Lanicides vayssieri* (= *L. bilobata*) (Linton, 1915a; Stunkard, 1929; Martin, 1944a, 1952; Rankin, 1946; Oglesby, 1961).

The apparent low prevalence of sanguinicolid trematodes in the molluscan and polychaete intermediate hosts is markedly contrasted by a generally high prevalence of the adults worms in fishes. Odhner (1900a) reported *Aporocotyle simplex* in 15 (11.5 %) of 140 *Limanda limanda* from Gullmar Fjord on the west coast of Sweden. He later (Odhner, 1911a) identified *Drepanopsetta* (= *Hippoglossoides*) *platessoides* as the main host for *A. simplex*. Thulin (1980), who restudied the parasite, recorded it in 55 (98 %) of 56 *H. platessoides*, in 27 (47 %) of 57 *L. limanda* and in 7 (13 %) of 53 *Pleuronectes platessa* from the same (i.e. type-) locality. Mean numbers (range in parantheses) of *A. simplex* in these host species were 11.1 (1-49), 4.3 (1-12) and 8.9 (2-19), respectively. Of 1,090 hake *Merluccius merluccius* caught off the British Isles, 323 (29.6 %) harboured up to 60 individuals of *A. spinosicanalis* (J. W. Smith and Williams, 1967). Holmes (1971a, b) found 5 species of scorpaenid rockfishes of the genus *Sebastes* from the North American Pacific coast to be infested with *A. macfarlani*. From 1 to 184 (mean 27) worms were dissected from 75 (72.1 %) of 104 hosts. Another sanguinicolid, *Psettarium sebastodorum*, was recovered in numbers ranging from 1 to 36 (mean 6) from 63 (47.7 %) of 132 rockfishes representing 11 species. Overstreet (1969) isolated sanguinicolids *Deontacylix ovalis* from the body cavity of all of 6 Bermuda chub *Kyphosus sectatrix*.

These few examples are sufficient to provide distinct evidence for the common and abundant occurrence of sanguinicolid digeneans in marine teleosts. Representatives of the family also occur in elasmobranchs and holocephalans. But, nevertheless, records of Sanguinicolidae have been omitted from most pertinent parasite lists. This circumstance is largely due to the fact that these peculiar digeneans are commonly overlooked by trematode taxonomists who do not usually examine the heart and blood vessels of the fish. Closer inspection of the blood-vascular system will no doubt lead to the discovery of additional species. The pathological effects of sanguinicolid blood flukes in marine fish are severe but have been analyzed or estimated in only a few cases (Martin, 1960; Thulin, 1975, 1980). Thus far, the Sanguinicolidae have mostly been studied in commercially exploited fish species, and these have frequently been found to be heavily infested. Therefore, the consideration of marine bivalves as intermediate hosts of fish blood flukes is a matter of economic importance.

As is the case in the Bucephalidae, the taxonomy of the Sanguinicolidae is in a truly barren state. For one example, numerous worms from a variety of quite unrelated hosts from various parts of the world ocean have, in the past, been listed as *Aporocotyle simplex*. Most of these probably represent new species and should, therefore, be left undetermined as '*Aporocotyle* sp.' until further material becomes available (J. W. Smith, 1967). Furthermore, none of the marine sanguinicolid cercariae known at present have been assigned to any of the adult forms*). It is hoped that this review might help to stimulate future research on this important group of helminth parasites.

*) See footnote on p. 655.

Digenea utilizing fishes as final hosts: family Monorchiidae

Several marine bivalve species have been reported as hosts for the sporocysts and cercariae of monorchiid trematodes. The metacercariae occur in the same or other pelecypods, and the adults live in the intestine of teleost fishes. The complete life cycles are known of only a few monorchiids.

The distome, ocellate cercariae of *Monorcheides cumingiae* occur in sac-shaped sporocysts in the digestive gland of clams *Cumingia tellinoides, Tellina tenera* and *Macoma tenta* from the Woods Hole region. In heavy infestations, most of the host's visceral region may become filled with sporocysts which gradually replace the digestive-gland tissue. This process appears to be a mechanical rather than a biochemical one. The resultant pathology is severe. The metacercariae of *M. cumingiae* (see following subchapter) encyst in the same bivalve species, and adult worms occur in eels and most frequently in the winter flounder *Pseudopleuronectes americanus* (Martin, 1938, 1940; Stunkard, 1970b, 1974c). Larvae similar to those of *M. cumingiae* have been described as *Cercaria caribbea LXIII* and *C. caribbea LXIV* from Jamaican *Tellina martinicensis* and *Codakia pectinella*, respectively. Their further life-cycle stages are not known (Cable, 1963).

Paratimonia gobii has developmental stages similar to those of *Monorcheides cumingiae*. Its sporocysts and cercariae occur in the gonad of *Abra ovata* from the French Mediterranean coast; the metacercariae encyst in *A. ovata* and *Cardium glaucum* (for effect on second intermediate host see following subchapter); the adult parasitizes in the intestine of gobies *Pomatoschistus microps* (Prévot and Bartoli, 1967; Maillard, 1975b, 1976). The cercaria of *P. gobii* is possibly identical with *Cercaria myocerca*, superficially described from *Scrobicularia* (= *Abra*) *tenuis* from the French Atlantic coast by Villot (1879). Two other monorchiid cercariae, *C. myocercoides* and *C. nigrotincta*, occur in *Syndosmya* (= *Abra*) *alba* from the same area (Pelseneer, 1906). *C. myocercoides* is distinguished from *C. myocerca* by its lack of eyespots.

Venus fasciata from the Gulf of Marseille is host for *Cercaria longicaudata*, which develops in colourless, immobile sporocysts in the clam's visceral mass, causing parasitic castration. Experiments conducted to obtain encysted metacercariae and to identify the adult stage have failed thus far (Bartoli, 1966a). *C. longicaudata* is very similar to (if not identical with) *C. ophicerca*, occurring in *Tapes aureus* and *Tapes decussatus* from the Gulf of Naples and from Bahía de Santander, Spanish Atlantic coast (Palombi, 1934a; Andréu, 1949). The further life history of *C. ophicerca* is unknown, but Palombi reported the finding of a single metacercaria in an individual of *T. decussatus*. Another similar larva, *Cercaria caribbea XXXV*, occurs in Puerto Rican *Macoma cerina*. Its metacercariae encyst in the same host species; the adult is unknown (Cable, 1956).

Bean clams *Donax gouldi* from California are first and second intermediate hosts for *Postmonorchis donacis*. When present in large numbers, the sporocysts cause extensive destruction of the clam's visceral mass. The adult of *P. donacis* is found in a number of teleosts, mainly surf perches (Embiotocidae) (Young, 1953).

All of the above-mentioned monorchiid cercariae from marine bivalves have well-developed tails identifying them as dispersal stages capable of swimming. There is evidence that encystment only ensues after at least a brief free-swimming period. Another group of monorchiid cercariae is characterized by the presence of a rudimentary tail quite unsuitable for locomotion in the water column. Those with appendages reduced to what may be

taken for an adhesive organ probably can reach their second intermediate host by creeping along the sediment surface, while those with tails reduced to a knob-like protuberance may have abbreviated life cycles, with the cercariae transforming into metacercariae without leaving the primary host.

One such cercaria, *Cercaria choanura*, was recorded in 26 of 1,017 coquina clams *Donax variabilis* from Mustang Island, Texas (Hopkins, 1958a). This is the larva provisionally named '*Cercaria A*' by Loesch (1957), who found it to be the most common cercaria in coquinas from Mustang Island. It develops in orange-pigmented sporocysts in the gonad and causes parasitic castration. *C. pocillator*, recovered from 5 of 1,763 *D. variabilis* at Alligator Harbor, Florida, by Holliman (1961), is similar to but probably specifically different from *C. choanura*. Clams *Transennella tantilla* from the North American Pacific coast are host for the sporocysts and brevifurcate cercariae of *Telolecithus pugetensis*. Its metacercariae encyst in a wide variety of bivalves and prosobranch snails (p. 691), and the adults parasitize in the intestine of embiotocid fishes. The sporocysts have severe detrimental effects on the clam host in suppressing gametogenesis and greatly reducing the visceral mass. Uninfested *T. tantilla* lived longer in the laboratory than infested ones (DeMartini and Pratt, 1964). Further monorchiids with stump-tailed cercariae include *C. caribbea XXXVI* parasitizing *Gemma purpurea* and *Chione cancellata* in Puerto Rico and Curaçao (Cable, 1956, 1963; Holliman, 1961). The entire (probably abbreviated) life cycles of these trematodes are unknown.

The monorchiid *Cercaria cerastodermae I*, which occurs in *Cardium edule*, has a peculiar seasonal life cycle. Cockles become infested in March to May and release cercariae until the end of September. Cercariae in cockles not eaten by the final host

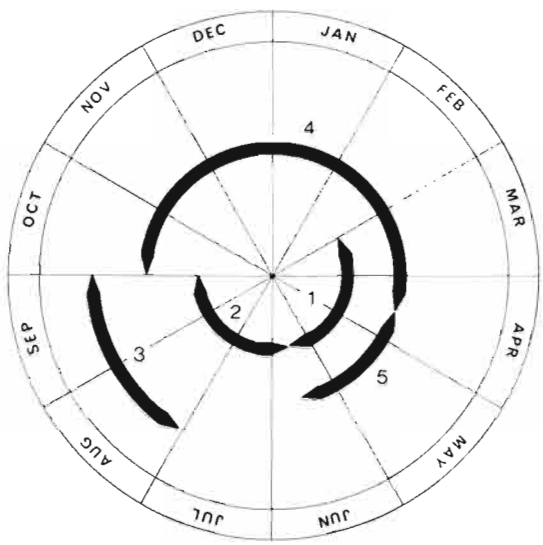


Fig. 13-84: *Cercaria cerastodermae I.* Postulated seasonal life cycle. 1: Infestation of primary host, *Cardium edule;* 2: production and liberation of cercariae; 3: infestation of (unknown) secondary host by free-swimming cercariae; 4: transformation of unshed cercariae into metacercariae within sporocysts in primary host; 5: death of cockles infested during previous year. (After Sannia and James, 1978.)

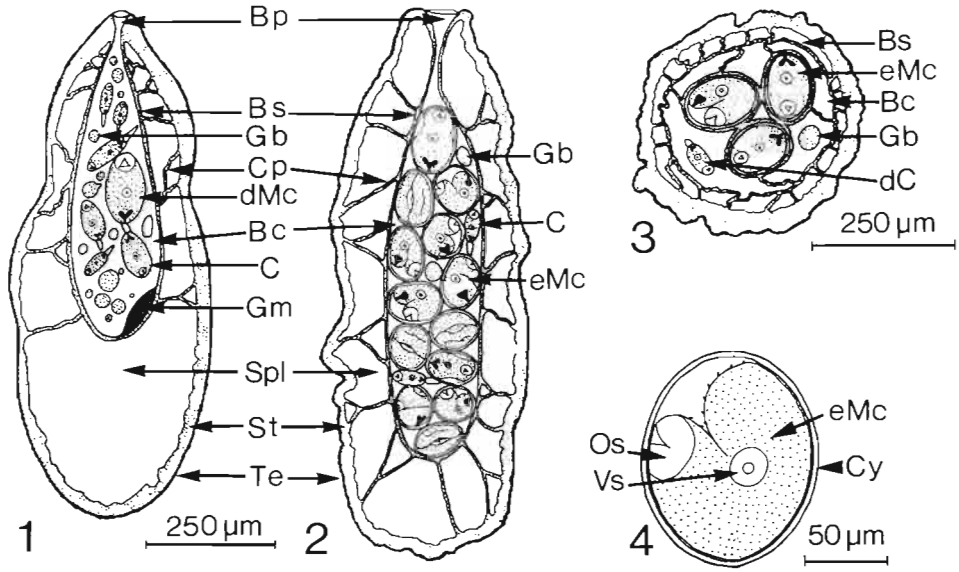


Fig. 13-85: *Cercaria cerastodermae I.* 1: Young daughter sporocyst containing mainly germinal balls and cercariae; 2: fully-formed daughter sporocyst containing mainly encysted metacercariae; 3: sporocyst, transverse section through mid-body; 4: encysted metacercaria. Bc brood chamber, Bs brood sac, Bp birth pore, C fully-formed cercaria, Cy cyst wall, dC developing cercaria, free in brood sac, eMc encysted fully-formed metacercaria, Gb germinal ball, Gm germinal mass, dMc developing unencysted metacercaria, Os oral sucker, Spl daughter sporocyst lumen, Cp cytoplasmic projection of subtegument, St subtegument, Te tegument, Vs ventral sucker. (After Sannia and co-authors, 1978.)

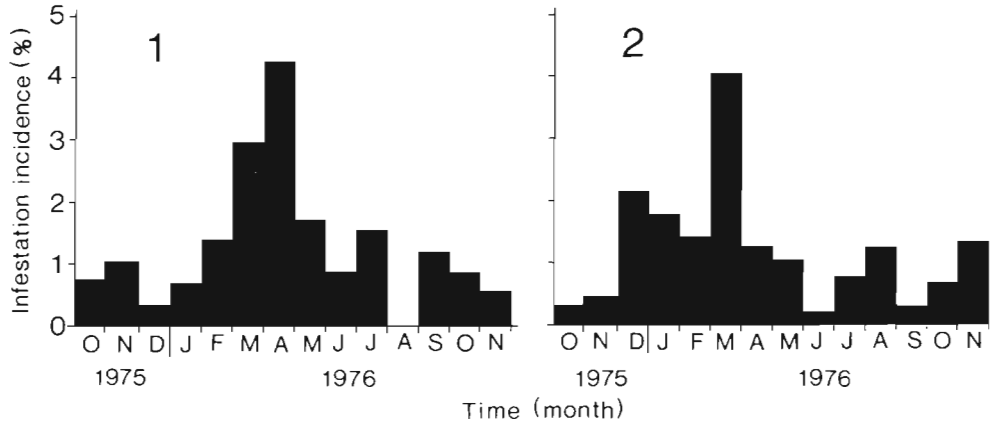


Fig. 13-86: *Cardium edule.* Seasonal incidence of infestation with *Cercaria cerastodermae I* in cockles from Thames estuary. (1) Chalkwell Ooze, (2) Maplin Sands. (After Sannia and James, 1978.)

transform into metacercariae within the sporocyst, and from October to May only cockles with metacercariae are found (Fig. 13-84). The daughter sporocysts of *C. cerastodermae I* are unusual in having a central brood sac connected to the sporocyst wall by anucleate cytoplasmic extensions of parenchyma cells in the subtegument (Fig. 13-85). Fully-formed

cercariae are equipped with 4 pairs of well-developed penetration glands, suggesting that they actively penetrate a — yet unknown — second intermediate host. In mature metacercariae dissected from their cysts enclosed by the brood sac in *C. edule*, penetration glands are no longer discernible. The transformation of cercariae into metacercariae, as well as the pronounced seasonality of *C. cerastodermae I* (Fig. 13-86), are probably triggered by changes in the ambient temperature or by the reproductive cycle of the primary host (Bowers, 1965a; Sannia and James, 1977b, 1978; James and co-authors, 1977; Sannia and co-authors, 1978).

Cercaria cerastodermae I invades the haemocoel of the digestive gland, the gonad and the foot of *Cardium edule*. The damage to the host's tissues is severe: During late spring, cockles with old mature infestations die due to the excessive destruction of tissues in the visceral mass. The assumption that the parasite kills the cockle within a year is further substantiated by the distinct seasonality in the occurrence of the parasite (Fig. 13-86). Cockles of all size and age groups are equally susceptible to *C. cerastodermae I*. Infested individuals exhibit growth reduction, the difference in shell length of healthy and diseased cockles being statistically significant.

Infestation by *Cercaria cerastodermae I* may furthermore increase the susceptibility of cockles to other digenean species. Of 10,406 individuals examined, 1.18 % had sporocysts of *C. cerastodermae I*, 0.34 % sporocysts of *Labratrema minimus* and 0.15 % sporocysts of *Gymnophallus choledochus*. One (0.01 %) harboured a double infestation of *L. minimus* and *C. cerastodermae I*. The expected incidence of double infestation is, hence,

$$\frac{1.18 \times 0.34}{100} = 0.004\ \%,$$

i.e., much less (statistically significantly less, as computed by the reviewer) than the observed incidence (Sannia and James, 1978).

The development of *Cercaria cerastodermae I* suggests that it has an alternative life cycle similar to that of *Gymnophallus choledochus*, as reported by Loos-Frank (1969a, b; see below): In the summer, free-swimming cercariae leave the first intermediate host and seek a second host. In the fall, unshed cercariae metamorphose directly into metacercariae within the primary (and, in this case, sole intermediate) host. The final host acquires the parasite by either ingesting infested cockles or individuals of the 'normal' secondary host which is, however, unknown in the present case. *C. cerastodermae I* appears to be a rare parasite of the cockle. Sannia and James (1978) reported it from 123 (1.18 %) of 10,406 *Cardium edule* from the Thames estuary; Bowers (1965a) found it in 1 of 200 cockles from the Kyle of Tongue, North Scotland; and Boyden (1969) recorded its presence in England in 18 of 318 cockles from Southend, in 2 of 87 from Maplin Sands, and in 2 of 2,696 from the River Crouch estuary. In addition, Boyden (1969) found the sporocysts and cercariae in 3 of 2,401 and 1 of 41 *C. glaucum* (= *C. lamarcki*) from the River Crouch and River Roach estuaries, respectively. Lebour (1906, 1907b), who first saw *C. cerastodermae I*, described it very briefly as *Distomum* sp., and later (Lebour, 1908c, 1912) assigned it, without experimental evidence, to *Lepodora rachiaea* (= *Lepidapedon rachion*), the adult of which occurs in the intestine of the haddock, *Melanogrammus aeglefinus*. However, *L. rachion* is a member of the Lepocreadiidae, but *C. cerastodermae I* is a monorchiid, probably a species of *Monorchis*. The adult is as yet unknown (Sannia and James, 1977b; Sannia and co-authors, 1978).

Digenea utilizing fishes as final hosts: family Fellodistomidae

The Fellodistomidae comprise another family of digenean trematodes which utilize marine pelecypods as primary hosts and fishes as definite hosts. Invertebrates of various other phyla may act as secondary hosts. In some cases the metacercarial stage is omitted, the cercaria being directly infestive to the fish final host.

Carpet-shells *Tapes decussatus* from Arcachon (French Atlantic coast) were found to be parasitized by sporocysts and trichocercous cercariae, named *Cercaria lata* by Lespès (1857b). Huet (1891) described a morphologically similar larva from *Donax anatinum* (= *D. vittatus*) as *C. pectinata*. Giard (1897a) named what appears to be the same cercaria from the same host species *C. lutea*, and claimed to have found it also in *Barnea candida* from Boulogne-sur-Mer (France). Subsequently, the name '*C. pectinata*' was adopted for trichocercous (setigerous) cercariae from a variety of bivalves in various waters. Cercariae of this type have been reported in Europe from *Tapes pullastra* (Jobert, 1894; Pelseneer, 1906; Arvy, 1972), *T. decussatus* (Jobert, 1894; Dollfus, 1925; Palombi, 1928, 1933a, b, 1934a, b; Arvy, 1972) and *T. aureus* (Palombi, 1933a, b, 1934a, b); from *Donax vittatus* (Pelseneer, 1906, 1928; Dollfus, 1911, 1925; Arvy, 1972; Matricon-Gondran, 1965, 1966, 1969, 1971a, b; James and co-authors, 1977); and from *Barnea candida* (Pelseneer, 1906). *C. setifera*, occurring in *Abra tenuis* in France (Villot, 1879), is also a representative of the *C. pectinata*-type of fellodistomid larvae (later, however, the specific name '*setifera*' has been applied to larval lepocreadiids; see Vol. I, Chapter 12).

Cercaria pectinata is a serious parasite. Heavy sporocyst infiltration causes complete castration and a depletion of body reserves to such an extent that the tissues of the host assume a flaccid consistency. In the commercially important *Tapes* spp. it has been blamed to produce "une mortalité telle qu'elle a pu causer de véritables préjudices pécuniaires aux importateurs" (Jobert, 1894, p. 519). On the French Atlantic and Italian Mediterranean coasts, *C. pectinata* was held responsible for large-scale fluctuations in the abundance of *Tapes* spp. and *Donax vittatus* populations. Instead of being normally burrowed, infested clams frequently lie, exposed to predators, on the surface of the sediment. In *D. vittatus*, the deleterious effects of the trematode were found to be at times superimposed by those produced by a haplosporidian hyperparasite (see p. 671) (Pelseneer, 1928; Palombi, 1934b).

'*Cercaria pectinata* Huet, 1891', occurring in the gonads of Mediterranean *Tapes decussatus, T. pullastra* and *T. aureus*, was found by Palombi (1933a, b, 1934a, b) to develop into adult *Bacciger bacciger* in smelt *Atherina hepsetus*. Infestations, which were heaviest (up to 70 %) in *T. decussatus* from the Gulf of Naples, caused complete castration of the host. Amphipods *Erichthonius difformis* were identified as second intermediate hosts. Palombi believed that his '*C. pectinata*' from *Tapes* spp. is identical with *C. lata* Lespès, 1857, *C. lutea* Giard, 1897, and *C. pectinata* Huet, 1891. He later (Palombi, 1940) also attributed to *B. bacciger* a cercaria found in *Tellina exigua* (= *T. tenuis*) from Naples, and Dolgikh (1968a) reported larval '*B. bacciger*' from Black Sea *Chione* (*Venus*) *gallina*.

Outside Europe, '*Cercaria pectinata*' has been recorded in *Chione stutchburyi* from Otago Habour, New Zealand (Chilton, 1905), in *Tapes philippinarum* and a number of other marine bivalves from Japan (Fujita, 1906, 1907; Kobayashi, 1922; Ito, 1964), and in *Meretrix lusoria* from Korea (Chun and Lee, 1976; Bae and co-authors, 1977). Of 2,639 *M. lusoria* from Busan, 13.2 % were found to harbour from 2,000 to 5,000 sporocysts

each. Infestation incidences were highest (29.6 %) in July and lowest (2.0 %) in March. Heavy mortalities occurred in clams kept in the laboratory. All of 60 infested *M. lusoria* died within 35 days. The parasite appeared to be host-specific to *M. lusoria* since it was not found in *M. petachialis*. Beyond doubt the East Asian '*C. pectinata*' have nothing to do with the European form. Bae and co-authors (1977) regard the cercaria from *M. lusoria* as the larva of *Bacciger harengulae*.

There can also be little doubt that the European forms synonymized with *Cercaria pectinata* Huet, 1891, represent, in fact, 3 or more species of larval fellodistomids. The reported hosts are members of 4 families of 3 distinct superfamilies, i.e., the Veneracea, the Tellinacea and the Pholadacea. Taking into account the host specificity of digenetic trematodes, which is usually most strict in the first intermediate host, it is difficult to believe that one and the same cercaria occurs in such a great number of unrelated hosts. That host specificity displayed by larval fellodistomids can be very pronounced, may be illustrated by the following example: *Cercaria caribbea XXXIX*, which is very similar to *Bacciger bacciger* (*Cercaria pectinata sensu* Palombi), occurs only in Caribbean *Tellina pauperata*, whereas *C. caribbea XL, C. caribbea LIII, C. caribbea LIV* and *C. caribbea LV* (which, with the exception of *C. caribbea XL*, are also very similar to *B. bacciger*) parasitize only *T. martinicensis* (Cable, 1956, 1963).

If the European '*Cercaria pectinata*' would comprise several larval *Bacciger* spp., more than one adult should occur in fishes from European Atlantic and Mediterranean waters. Palombi (1934b) believed that '*B. bacciger*', described from sand smelt *Atherina presbyter* from the English Channel by Nicoll (1914), is different from the Mediterranean form, and consequently named it *Bacciger nicolli*. Margolis and Ching (1965) agreed with Nicoll (1914) in separating a northern (Atlantic) from a southern (Mediterranean) species of *Bacciger*, but Bray and Gibson (1980) disagree and maintain that a single species of the genus *Bacciger, B. bacciger*, occurs in the area under consideration. It is possible that some of the described '*C. pectinata*'-type cercariae are larvae of members of the genus *Pronoprymna* (= *Pentagramma*), which is closely related to *Bacciger*.

Further non-ocellate, distome, trichocercous fellodistomid cercariae have been described as *C. pennata* from *Tapes rugatus* and as *C. plumosa* from *Abra* (*Syndosmya*) *alba* in the Black Sea (Sinitsin, 1911). Bartoli (1974a) identified *C. plumosa*, reported from *A. ovata* in the Mediterranean by Bayssade-Dufour and Maillard (1975), as a member of the Cryptogonimidae. The life cycles of all of these cercariae are unknown and their systematic position is sometimes dubious. Chaetotaxy, as employed by Bayssade-Dufour and Maillard (1975), might prove a useful tool in identifying and characterizing *C. pectinata*-type and similar fellodistomid cercariae.

About 10 % of *Mytilus latus* from New Zealand were found to harbour sporocysts and furcocercous cercariae, misidentified as a species of *Echinostomum* (Haswell, 1903). Odhner (1911c) recognized it as a member of the fellodistomid genus *Tergestia,* and Dollfus (1927) named it *T. haswelli*. Angel (1960) re-examined Haswell's material and discussed its position in the genus *Tergestia*. The further life cycle of *T. haswelli* is unknown. It might be identical with *T. agnostomi,* whose unencysted metacercaria occurs in ctenophores *Pleurobrachia pileus* (Boyle, 1966; Vol. I, Chapter 7), and the adult of which parasitizes in the intestine of yellow-eyed mullets *Agnostomus forsteri* in New Zealand waters (Manter, 1954; Jones, 1978). Other cercariae of the genus *Tergestia* — *Cercaria dichotoma* (p. 681), *C. kenti* and *C. mathiasi* — have been found free in the

plankton (La Valette St. George, 1855; Kent, 1871; Dollfus, 1927; Dubois and co-authors, 1952). Their further life-cycle stages remain unknown, but Odhner (1911b) attributed *'C. dichotoma* La Valette, 1855' to *T. laticollis*, an intestinal trematode of carangid fishes (Bray and Gibson, 1980). Angel (1960), however, argues that *C. dichotoma* La Valette does not possess any of the characters typical of *Tergestia* cercariae and cannot be regarded as a larva of this genus. Another larva, *C. dichotoma* Pelseneer, 1906, which is usually referred to in the literature as a gymnophallid cercaria (p. 681), is possibly a member of the genus *Haplocladus* (= *Monascus*) (p. 665; Odhner, 1911c).

Morton's egg cockles *Laevicardium mortoni* from Woods Hole, Massachusetts, are primary hosts for *Lintonium vibex*. Its unencysted metacercariae occur in ctenophores *Mnemiopsis leidyi* (Vol. I, Chapter 7), and the adults parasitize in the intestine of northern puffers *Spheroides maculatus*. Infestations were common in cockles from the Woods Hole area, and in certain collections about one-half of the bivalves had sporocysts (Stunkard, 1978). The cercaria of *L. vibex* was first described as *Cercaria laevicardium* by Martin (1945), who also postulated the life cycle. Cable (1954) recorded *L. vibex* infestations in 25 to 33 % of large *L. mortoni* from Martha's Vineyard, Massachusetts. Nothing has been reported on the pathology of *L. vibex* in *L. mortoni*, but almost certainly parasitic castration ensues in heavy infestations.

Nut shells — bivalves of the genera *Nucula* and *Nuculana* (*Leda*) — from various parts of the North Atlantic and adjacent seas are hosts for several larval fellodistomids. Sporocysts and furcocercous cercariae of *Fellodistomum fellis* have been recovered from 17 (5.7 %) of 300 *Nucula tenuis* in the Barents and White Seas, from 5 (0.24 %) of 2,110 in Disco Bay, western Greenland, and from 1 of 10 in Gullmar Fjord, west coast of Sweden. The metacercariae occur in ophiuroids and the adults in the gall bladder of flatfishes and catfish *Anarhichas lupus* (Chubrik 1952b, 1966; Køie, 1980). The species was first described as *Steringophorus ovacutus* from *Hippoglossoides platessoides* in Britain (Lebour, 1908a).

Nucula nucleus and *N. nitidosa* are primary (and sole intermediate)hosts for *Steringotrema pagelli*. About 2 % of *N. nitidosa* from western Kattegat and 5 (6 %) of 84 *N. nucleus* from Gullmar Fjord were found infested (Køie, 1980). The cercaria, which superficially resembles a larval bucephalid due to its bifid tail consisting of 2 long, contractile furcae, has previously been recorded but not identified in about 2 % of 800 *N. nucleus* dredged off Plymouth, England (Jones and Rothschild, 1932). In infested hosts, the gonad is generally completely destroyed. The free-swimming cercaria directly invades flatfishes; the metacercarial stage is entirely omitted. Adult *S. pagelli* have been identified as intestinal parasites of lemon soles *Microstomus kitt* and dab *Limanda limanda*.

Monascus (*Haplocladus*) *filiformis* has its larval stage in *Nucula nitidosa* and *N. nucleus*. The furcocercous cercariae, which occur in thick-walled red-brown sporocysts in the digestive gland and gonad, are unusually large, the body of living individuals measuring, on the average, 970 × 340 μm, the tail 1.0 to 1.1 mm and the furcae 0.5 to 0.8 mm in length. W. J. Rees (1947) found 12 out of 16 *N. nucleus*, dredged off Plymouth, England, to be infested with this species. Køie (1979) recorded it in 30 % of about 200 *N. nitidosa* from western Kattegat off Frederikshavn, Denmark. Mature adults of *M. filiformis*, most of which have been listed under the synonyms *M. typicus* or *M. minor*, occur in flatfishes and carangids (Køie, 1979). Odhner (1911b) briefly mentioned a cercaria of the genus

Haplocladus (*Monascus*), found free-swimming in an aquarium at Kristineberg, Sweden, containing *N. nucleus* and *Abra alba*. Which of either bivalve species was the host, has not been determined. The larva was tentatively referred to *M. minor* (= *M. filiformis*), but it is distinctly smaller (body length 0.35 mm, length including tail 1.15 to 1.25 mm) than the cercaria of the latter and probably represents another species.

A furcocercous larva, *Cercaria dichotoma* Pelseneer, 1906, develops in short, nodose, colourless and transparent sporocysts in *Tellina solidula* (= *Macoma baltica*) from deep water off Boulogne-sur-Mer, French Atlantic coast (Pelseneer, 1906). In the pertinent literature this cercaria is frequently referred to as a gymnophallid larva with which it shares superficial resemblance (p. 681). Odhner (1911c), however, regards it — probably rightly — as a member of the genus *Haplocladus* (= *Monascus*). Pelseneer's brief description and his inadequate drawings do not permit a definite allocation of this larva.

The large leptocercous cercaria of *Steringophorus furciger* develops in orange-brown sporocysts, up to 5 mm long and containing up to 60 cercariae, in *Nuculana* (*Leda*) *minuta*. The cercarial body measures, on the average, 415 × 220 μm, and the tail varies in length between 1.3 and 3.0 mm. *S. furciger* has been recorded in 5 (2.5 %) of 200 *N. minuta* in Øresund and in 1 of 92 hosts taken off Godhavn, western Greenland (Køie, 1979). Chubrik (1966) found 0.4 % of *N. pernula* in the Barents and White Seas to be infested with this digenean. According to Køie (1979), however, the Russian author did not distinguish between *N. minuta* and *N. pernula*; she probably also dealt with *N. minuta*. None of several hundred *N. pernula* from Øresund had larvae of *S. furciger*. Adults of this trematode have been reported from a wide variety of teleosts, particularly flatfishes (Dawes, 1947). In the Barents Sea, 87.5 % of *Limanda limanda*, 60 % of *Pleuronectes platessa*, 60 % of *Hippoglossoides platessoides* and 6.25 % of *Platichthys flesus* have been found to harbour *S. furciger* (Chubrik, 1966).

Another huge-tailed fellodistomid larva, *Cercaria megalocerca*, has been described from 10 % of *Portlandia arctica* in the Barents and White Seas. In living individuals the cercarial body measures 0.6 to 0.9 mm and the tail 1.3 to 1.5 mm in length (Chubrik, 1966). The life cycle of *C. megalocerca* has not been traced experimentally, but according to Køie (1980) it may be the larva of *Steringophorus* (= *Fellodistomum*) *agnotus*, the only fellodistomid from that area with an unknown life cycle.

In the abbreviated life cycles of *Steringotrema pagelli*, *Monascus filiformis* and *Steringophorus furciger* the metacercarial stage is omitted, the free-swimming cercaria being directly infestive to the final host, which is a fish in all 3 cases. Curiously, adult *S. furciger* have also been reported from the intestinal tract of whelks *Buccinum undatum* (Køie, 1969; Vol. I, Chapter 12). As demonstrated experimentally, the prosobranchs are accidental hosts in which *S. furciger* attains the same size as in fishes but does not mature. The whelks acquire the worms by feeding on dead fish only (Køie, 1979). In contrast, fellodistomids of the genus *Proctoeces* utilize gastropods (Vol. I, Chapter 12) and bivalves (see below and subchapter after next) as regular definite hosts.

Proctoeces maculatus is capable of completing its entire life cycle in *Mytilus edulis*. The cercaria, metacercaria and adult of this cosmopolitan species have been described under a number of names from different hosts in various parts of the world. Dubois (1907b) provided the first brief description of the developmental stages of *P. maculatus*, from ovum to metacercaria, all occurring in *M. galloprovincialis* from the Rhône estuary (French Mediterranean coast). He erroneously believed the stages seen in the mussel to

belong to the 'pearl-inducing' gymnophallid (p. 721). Dubois' confusing report fell into oblivion. As *'Cercaria tenuans'* infesting *M. edulis* from Conway, Wales, *P. maculatus* was identified as the causative agent of a condition in mussels termed 'orange sickness'. Diseased hosts were easily recognized macroscopically, as the mantle was a vivid marigold to blood orange in colour due to the presence of enormous numbers of saccate, motile, orange-pigmented sporocysts (Fig. 13-87, 1). These were found to infiltrate all the tissues of the body except the foot and were most numerous in the blood sinuses and interfollicular lymph spaces of the mussel's mantle gonad which, in infested individuals, was of transparent thinness (Cole, 1935).

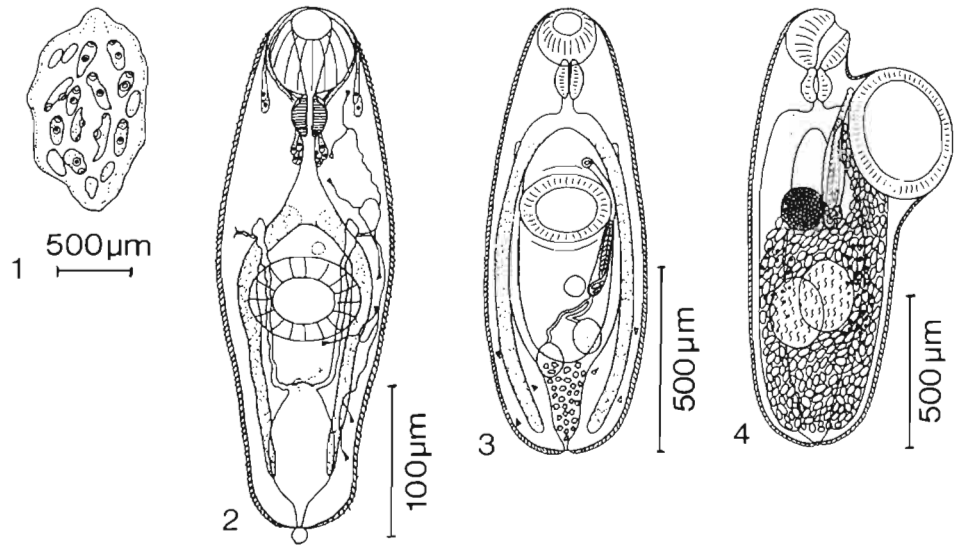


Fig. 13-87: *Proctoeces maculatus.* Sporocyst (1), cercaria (2) and metacercaria (3) from *Ischadium recurvum;* adult (4) from *Archosargus probatocephalus.* (After Wardle, 1980b.)

Uzmann (1953) reported the occurrence of sporocysts and microcercous cercariae (Fig. 13-87, 2) in *Mytilus edulis* from New York and Connecticut waters. Although making reference to Cole's (1935) description of *Cercaria tenuans*, he failed to recognize the specific identity of both forms and renamed the larva *C. milfordensis.* He also found, in some of the mussels bearing *C. milfordensis* infestations, several individuals of "unencysted progenetic larvae referable to the genus *Proctoeces*". These actually represent young adults, which were later identified as *P. maculatus* by Stunkard and Uzmann (1959), who ascertained that the entire life cycle of the species can unfold in *M. edulis.* This was, at that time, an astonishing discovery since adult *P. maculatus* had previously been described only from the hind-gut of Mediterranean teleosts of the genera *Labrus, Crenilabrus* and *Blennius* (Looss, 1901; Odhner, 1911b). Peneda (1965) reported *'C. tenuans'* from *M. edulis* in the Ría de Aveiro, Portugal. Some of the infested mussels harboured concurrent *'Bucephalus mytili'* (= *Prosorhynchus squamatus*) infestations. Dubois (1907b), Prévot (1965a), Dupouy and Martinez (1973), Martinez (1973) and Machkevsky and Parukhin (1981) identified *M. galloprovincialis* as intermediate host of *P. maculatus* in the Mediterranean and Black Seas, respectively. Palombi (1926) reported the advanced

metacercaria as *'Cercaria megalophallos'* from *M. galloprovincialis* in Mar Piccolo, Italy, but failed to discover the immature cercarial stage. Unaware of the specific identity, Cerruti (1948) described the latter as *'Cercaria dubia'*.

Hooked mussels *Ischadium recurvum* (syn. *Brachidontes recurvus, Mytilus recurvus, M. hamatus*) from Barataria Bay (Louisiana) are hosts for *Cercaria brachidontis* (Hopkins, 1954b). Although pointing out similarities between this larva and *C. milfordensis*, the author considered several features sufficient to separate both forms specifically. It remained to Wardle (1980b) to exemplify that *C. tenuans, C. milfordensis* and *C. brachidontis* are actually conspecific. Unidentified sporocysts and microcercous cercariae, similar to those of *P. maculatus* and named *C. adranocerca*, have been found in 2 of some 300 *Gemma gemma* from Boothbay Harbor, Maine (Stunkard and Uzmann, 1959).

Most of the previous confusion concerning the specific identity of *Cercaria tenuans* and *C. brachidontis* results from the misinterpretion of tailless *Proctoeces maculatus* metacercariae as 'fully developed cercariae'. In fact, the young cercariae have a bulbous vestigial tail, while mature metacercariae have none (Fig. 13-87, 2, 3). Cole (1935) described *C. tenuans* as tailless, but Canzonier (1972) observed knob-like protuberances on about half of the larvae inspected by him. Apparently, the knob-like appendage is but weakly attached to the body and may either be lost while the cercariae are still confined to the sporocyst, or retained for several days after emergence (Uzmann, 1953). In *C. brachidontis*, a tail is said to be present in immature but not in 'mature cercariae' (Hopkins, 1954b). The latter actually represent fully developed metacercariae.

The prevalence of *Proctoeces maculatus* infestation varies greatly with locality and season, as well as with the host species involved (Table 13-13). In mussels from Shark River (Belmar, New Jersey), peak incidences of up to 46.2 % occurred during August and September, and lows with 6.7 % and 4.7 %, respectively, from December to March (Lang and Dennis, 1976; Fig. 13-139). The percentage incidence of sporocysts was found to increase with host size — from less than 10 % in mussels 20 to 25 mm in length to 43.1 % and 60.0 %, respectively, in mussels of the upper size classes (Fig. 13-88). Hence, a considerable proportion of the mussels is eliminated from the breeding population due to parasitic castration. Hosts less than 15 mm long (and probably immature) were only rarely found infested. It may thus be concluded that the susceptibility of *Mytilus edulis* to *P. maculatus* is somehow correlated with the onset of gonad development in young hosts. In adult *M. edulis*, invasion commences at the time when the mussels are sexually spent, i.e., in late July to early August. As a consequence, sporocysts are most common in late summer (Uzmann, 1953; Martinez, 1973; Lang and Dennis, 1976).

The pathogenicity of *Proctoeces maculatus* appears to vary with host species and possibly also with external factors, such as water temperature. 'Orange sickness' appeared to be a serious disease condition in *Mytilus edulis* at the Conway (Wales) mussel purification plant since "it was possible to obtain specimens by inspecting the few dead mussels which are picked out of the tanks during the normal process of purification" (Dodgson in Cole, 1935, p. 276). Similarly, Canzonier (1972) found 2 mussels, heavily infested with sporocysts and cercariae, to be rather weak and partially gaping. Detrimental effects on the host appear to be caused mainly by a 'blocking layer' of sporocysts developing in the mantle veins and leading to a serious reduction in the efficiency of the entire circulatory system. The motile sporocysts infiltrating the gill-blood vessels were seen to cause temporary enlargement of affected filaments "as they wormed their way along".

Table 13-13
Proctoeces maculatus. Infestation incidence in relation to host species and locality (Compiled from the sources indicated)

Proctoeces maculatus reported as	Host species	Locality	Infestation incidence	Source
Not named	*Mytilus edulis*	Padstow, England	235 (2.2 %) of 10,866	Atkins (1931a)
Cercaria tenuans	*M. edulis*	Conway, Wales	6 of 300 to 400	Cole (1935)
C. tenuans	*M. edulis*	Ría de Arosa, Spain	4 (4.2 %) of 95	Canzonier (1972)
C. tenuans with unspecified proportion of *Prosorhynchoides gracilescens*	*M. edulis*	Ría de Aveiro, Portugal	1 to 15 %	Días and Serrano (1972)
C. milfordensis	*M. edulis*	Long Island, New York	4 (4.3 %) of 93	Uzmann (1953)
C. milfordensis	*M. edulis*	Milford, Connecticut	30 (6.6 %) of 454	Uzmann (1953)
C. milfordensis	*M. edulis*	Bridgeport, Connecticut	44 (7.7 %) of 567	Uzmann (1953)
Proctoeces maculatus	*M. edulis*	Woods Hole, Massachusetts	0.5 %	Stunkard and Uzmann (1959)
P. maculatus	*M. edulis*	Shark River, Belmar, New Jersey	81 (16.4 %) of 493 106 (19.9 %) of 534	Lang and Dennis (1976)
P. maculatus	*M. galloprovincialis*	Gulf of Marseille, France	2.5 %	Prévot (1965a)
P. maculatus	*M. galloprovincialis*	Gulf of Marseille, France	91 (6.0 %) of 1,523	Martinez (1973)
C. brachidontis	*Ischadium recurvum*	Barataria Bay, Louisiana	5 (12.5 %) of 40	Hopkins (1954b)
P. maculatus	*I. recurvum*	Hanna's Reef, Galveston Bay, Texas	1 (5.8 %) of 17	Wardle (1980b)

The parasites sometimes occluded the lumen and distended the gill filaments to such an extent that it was surprising that they did not burst through the epithelium. Sporocysts crowding in the pallial vessels caused them to assume a deep orange colour against the creamy white of the mantle. Parasites in the gonads of *M. edulis* caused suppression of gametogenesis or, in heavy infestations, complete atrophy of the reproductive organs. Similarly, infested *Ischadium recurvum* had the gonads completely destroyed and replaced by masses of orange-pigmented sporocysts, which resembled and moved like active fly maggots. Sporocysts also occupied part of the mantle tissue and had spread into other organs to some extent (Atkins, 1931a; Uzmann, 1953; Hopkins, 1954b).

Proctoeces maculatus infestation was believed to be lethal under temporary or sustained periods of ecological conditions unfavourable to the mussel. Of 30 infested *Mytilus edulis*, kept in running sea water in the laboratory for several months, all but 2 died. Although controls were not kept, it was assumed that the mortality was abnormally high and possibly attributable to the presence of *P. maculatus* sporocysts (Uzmann, 1953).

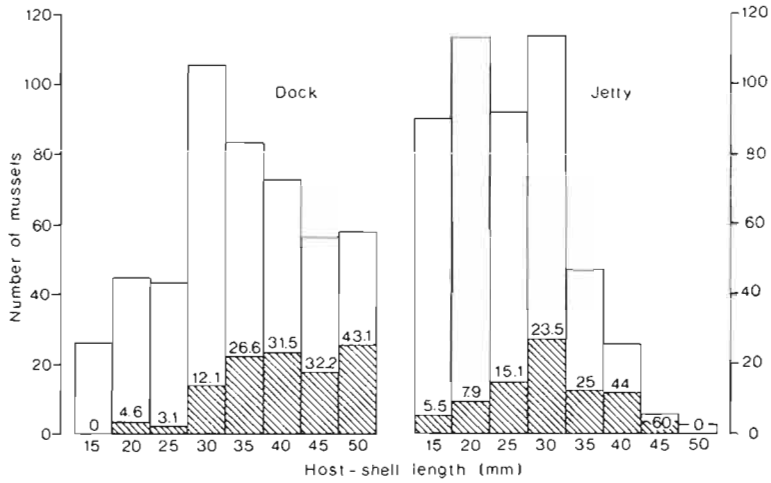


Fig. 13-88: *Mytilus edulis*. Size-class distribution and percentage infestation (= numbers above columns) with *Proctoeces maculatus* sporocysts at 2 sites ('Dock', 'Jetty') in Shark River (Belmar, New Jersey). (After Lang and Dennis, 1976.)

Hancock and Urquhart (1965) even blame *Cercaria tenuans* for causing 'extreme wasting conditions' in *M. edulis* in British waters, and *C. milfordensis* is said to cause mass mortalities of *M. galloprovincialis* in the Black Sea (Machkevsky and Parukhin, 1981). In contrast, Canzonier (1972) reported minimum pathology of *C. tenuans* in *M. edulis* imported into Italy from Ría de Arosa (northwest coast of Spain). At the tissue level, no overt response to healthy sporocysts was noted, the most apparent effects being the blocking and distension of vessels and ducts resulting in compression and distortion of adjacent tissues. Although portions of the gonad actually occupied by sporocysts exhibited reduced reproductive capacity, there was no evidence of general castration, gonad tissue not infiltrated by sporocysts being normal in appearance. Similarly, *M. galloprovincialis* from Sète (French Mediterranean coast) appeared to be only moderately affected by *P. maculatus*. The proliferation of the parasite was found to be intimately correlated with the gonad cycle of the host, but complete parasitic castration was rarely observed (Dupouy and Martinez, 1973; Martinez, 1973).

Biochemical and histochemical studies revealed a complex picture of the delicate balance governing the host-parasite interrelationship between *Mytilus edulis* and *Proctoeces maculatus*. It is well known that molluscs infested with larval trematodes usually display increased phosphatase activity (Cheng, 1964; James and Bowers, 1967a; Reader, 1971; Michelson and Dubois, 1973; Marshall and co-authors, 1974a, b; see also p. 648 and Vol. I, p. 337). The non-specific phosphomonoesterases are believed to be involved in the metabolism of carbohydrates and the active transport of metabolites (Erasmus, 1957a, b, 1958; Cheng, 1964; von Brand, 1966; Threadgold, 1968). Normally, an increase in the phosphatase activity in trematode-infested hosts is correlated with a concomitant breakdown of glycogen by the parasite (Cheng and Snyder, 1962). Biochemical and histochemical studies of the phosphatase activity in the tissues of *Mytilus edulis* revealed that the acid phosphatase (AcPase) concentration in the mantle and digestive gland remained unaltered by infestation with *Proctoeces maculatus* sporocysts. The presence of AcPase-positive parasites in these and other tissues, however, reflected greater enzyme activity at these

sites than at the respective sites of uninfested individuals. Similarly, the luminal border of epithelial cells of the kidney was light for AcPase activity except when adult *P. maculatus* were present in the kidney lumen; then cells bordering the parasites exhibited increased activity. High AcPase concentrations were detected in the tegument and parenchymal cells of the sporocysts. No differences were observed in the alkaline phosphatase (AlkPase) activity of tissues of sporocyst-infested and uninfested mussels, but haemolymph AlkPase increased significantly in mussels harbouring adult *P. maculatus*. The concentration of haemolymph AlkPase of uninfested hosts and those carrying sporocysts was only 0.17 mg 100 g^{-1}, while that of mussels harbouring adult parasites was 1.51 mg 100 g^{-1}. In *P. maculatus*, only the excretory system of the adult stage showed intense AlkPase activity,the other tissues and life-cycle stages being negative (Dennis and co-authors, 1974).

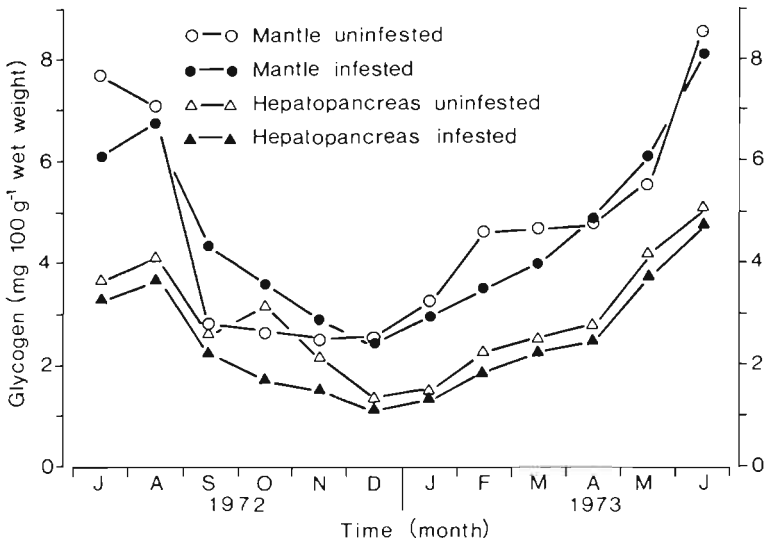


Fig. 13-89: *Mytilus edulis*. Seasonal variation in glycogen concentrations in mantle and hepatopancreas of uninfested and *Proctoeces maculatus*-infested individuals. (After Dennis and co-authors, 1974.)

Infestation with larval trematodes generally depletes or severely reduces the carbohydrate reserves of the host (Cheng and Snyder, 1962; Cheng, 1963, 1967; James, 1965; Wright, 1966; James and Bowers, 1967a; see also pp. 648 and 651, and Vol. I, pp. 331–334 and 341). In contrast, studies of the distribution and concentration of carbohydrate reserves in the tissues of *Mytilus edulis* demonstrated that the annual glycogen cycle of *Proctoeces maculatus*-infested mussels is similar to that of uninfested individuals. Glycogen levels may be higher in infested than in uninfested mantle tissue or *vice versa*, no constant pattern being apparent. In digestive-gland tissue, concentrations were constantly higher in uninfested than in infested mussels, but except for assays done in October, the difference was statistically and perhaps functionally insignificant (Fig. 13-89). In contrast, starvation of mussels collected in February and kept at a temperature conducive to high metabolic activity of the parasite (8 °C) resulted in a rapid decline in the glycogen content of infested individuals by the first week of starvation, whereas a stable glycogen concentra-

tion was maintained for 4 weeks by starved uninfested mussels (Fig. 13-90). It may thus be concluded that, under normal ecological conditions, *P. maculatus* does not significantly alter the carbohydrate reserves of its host. Glycogen depletion occurs only during severe stress, such as imposed starvation, and at temperatures favourable for the metabolism of the parasite. Obviously, the life cycle and changes in nutritive requirements of *P. maculatus* are adapted, in a delicate balance, to the annual pattern of metabolic changes in *M. edulis* (Dennis and co-authors, 1974). Whether the nutritional demands of the parasite have a significant effect on host protein and lipid metabolism remains to be studied.

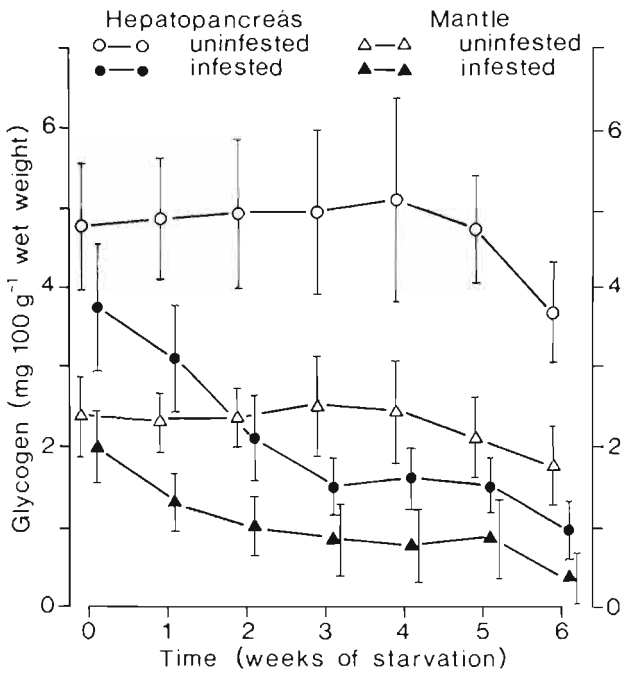


Fig. 13-90: *Mytilus edulis*. Glycogen concentrations in mantle and hepatopancreas of uninfested and *Proctoeces maculatus*-infested individuals collected in February and starved for 6 weeks at 8 °C. Vertical bars: standard errors. (After Dennis and co-authors, 1974.)

Hyperparasitization of larval fellodistomids has been observed in several instances. Pelseneer (1895) described and figured clusters of dark brownish spores within sporocysts of *Cercaria pectinata*, parasitic in the gonad of *Donax trunculus* (= *Donax vittatus*) from Wimereux, France, which he interpreted as stages in the development of the trematode. He did not realize that he actually dealt with sporocysts hyperparasitized by a haplosporidian (Fig. 13-91). The sporozoan was restudied by Caullery and Chappellier (1906), who failed to observe the delicate 'tails' on the spores (Vol. I, Fig. 10-7), and consequently named it *Anurosporidium pelseneeri*. Cépède (1911) contributed a further study on the life cycle of the hyperparasite. Finally, Dollfus (1925, 1946a) found tails on the spores and renamed the species *Urosporidium pelseneeri*.

Intracellular schizogonic stages of the haplosporidian were seen within the sporocyst wall of *Cercaria pectinata*, while advanced developmental stages and spores accumulate, in enormous numbers, in the sporocyst lumen (Fig. 13-91, 4). In recently infested sporocysts,

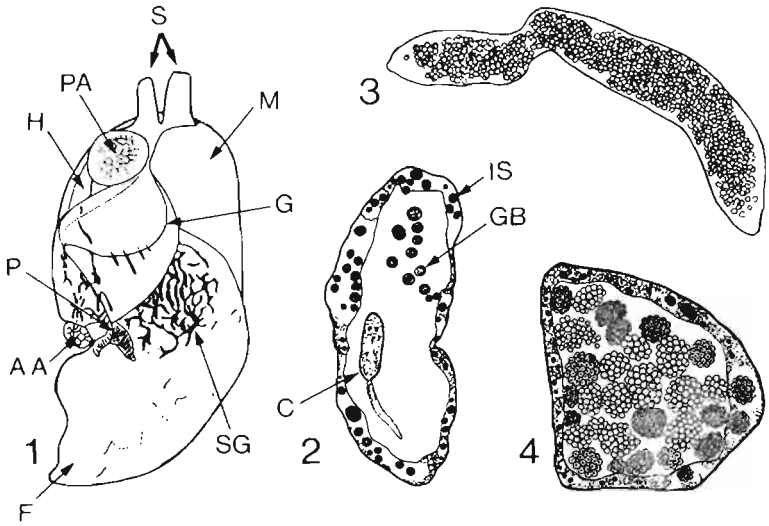


Fig. 13-91: *Donax vittatus* parasitized by *Cercaria pectinata* and hyperparasitized by *Urosporidium pelseneeri*. 1: Soft body of clam with sporocysts in gonad (× 1.9); 2: *C. pectinata* sporocyst with intracellular schizogonic stages of *U. pelseneeri* within wall and infested germinal masses, as well as uninfested cercaria within lumen (× 83); 3: sporocyst entirely filled with *U. pelseneeri* spores (× 38); 4: sporocyst with intracellular schizogonic stages and differentiating spores of *U. pelseneeri* within wall and clusters of mature and immature spores within lumen (transverse section, × 248). AA anterior adductor muscle, C cercaria, F foot, G gill, GB germinal ball, H heart, IS intracellular schizogonic stages, M mantle, P palps, PA posterior adductor muscle, S siphons, SG sporocysts in gonad. (After Pelseneer, 1895.)

unaffected germ balls and cercariae in various stages of development occur side by side with infested ones. Germ balls appear to be more susceptible to invasion by the sporozoan than young cercariae, and mature cercariae never become infested. It appears likely, however, that the latter, although unaffected by the haplosporidian itself, would secondarily undergo starvation and, eventually, lysis due to the reduced capacity of the affected sporocyst to supply the necessary nutrients. The single remaining fully-grown cercaria within an infested sporocyst figured by Pelseneer (1895) appears to be degenerating (Fig. 13-91, 2). In advanced infestations, the *C. pectinata* sporocysts contain nothing but *Urosporidium pelseneeri* spores (Fig. 13-91, 3, 4), which measure about 4.5 to 5.5 μm in diameter and possess a variable number of protoplasmic processes ('tails'). If an infestation occurs, all of the sporocysts in a particular clam are simultaneously affected. It was assumed that the establishment of the haplosporidian occurs at a very early stage in the life history of the trematode, possibly in the miracidium. All developmental stages of *U. pelseneeri* are strictly confined to the sporocysts of *C. pectinata,* the tissues of the molluscan host, *Donax vittatus,* never being attacked (Caullery and Chappellier, 1906).

A haplosporidian hyperparasite, similar to or identical with the sporozoan in *Donax vittatus,* occurred in *Barnea candida* from Wimereux, French coast of English Channel (Guyénot, 1943; Vol. I, Fig. 10-8). The trematode host could not be identified because its sporocysts were so heavily affected by the haplosporidian that cercariae did not develop. It was believed to be identical with the one described from the same host and area as *Cercaria lutea* by Giard (1897a). Dollfus (1946a), who restudied the *B. candida* parasite,

identified the sporocysts as *C. pectinata* and concluded that the sporozoan is very probably *Urosporidium pelseneeri*.

Since the sporocysts of *Cercaria pectinata* are entirely destroyed by *Urosporidium pelseneeri*, the hyperparasite constitutes a natural means of 'biological control' of the trematode and is, therefore, actually beneficial to *Donax vittatus*. How effective such 'biological control' can be in the field has been observed by Pelseneer (1928). *C. pectinata* infestations had become abundant in *D. vittatus* from the French Atlantic coast by the end of the last century. As the hyperparasite commenced to flourish, the abundance of *C. pectinata* receded and, for some time, the clams were less infested. Eventually, however, the incidence of *C. pectinata* increased again to such an extent that the *D. vittatus* populations suffered heavy losses and did not recover until 1925 to 1926.

The population fluctuations of *Donax vittatus* in France, as recorded by Pelseneer (1928), remind one of similar fluctuations in abundance, observed in American *Donax* species (p. 494). It appears worth mentioning, in this context, that Mackin and Loesch (1955) briefly reported an unidentified haplosporidian hyperparasite in *D. variabilis* from Texas (USA), which they felt was neither the same as that in *Bucephalus cuculus* (p. 653) nor *U. pelseneeri* found in *D. vittatus* in France. The study of the interrelationships between host, parasite and hyperparasite and their possible bearing on host population fluctuations is an untouched ground.

Digenea utilizing birds as final hosts: family Gymnophallidae

In addition to the fish trematodes discussed above, a number of bird trematodes — members of the exclusively marine family Gymnophallidae — utilize bivalves as first (and, partially, also as second) intermediate hosts. Most gymnophallids are only known by their metacercarial stage, although particularly the early authors failed to distinguish between cercariae and metacercariae and described the latter as 'tailless cercariae' or 'cercariaea'. Due to the morphological uniformity within this parasite group there is an extreme confusion regarding the taxonomy, synonymies and life histories of the Gymnophallidae. Many life histories have been pieced together arbitrarily and merely on the basis of morphological similarities between larval and adult stages, but only a few have been worked out completely by means of experimental infestation of intermediate and final hosts. Although Cable (1953), Stunkard and Uzmann (1958), Stunkard (1959c) and James (1964) have published reviews of the Gymnophallidae, "the situation is chaotic and one of utter confusion" (Stunkard and Uzmann, 1958, p. 285).

Typically, gymnophallid cercariae, which develop in sporocysts in the haemocoel of the digestive gland and gonad of marine bivalves, have forked tails (Fig. 13-92). As early as 1906, Pelseneer suspected that the 'young immature distome' (actually a gymnophallid metacercaria), found in several bivalve species between the mantle and shell, might be an advanced stage of his *Cercaria syndosmyae*, a furcocercous larva from *Abra (Syndosmya) alba*. But subsequent workers failed to recognize this relationship, and Lebour (1908a, p. 33) even states that "no tailed forms of the cercariae of *Gymnophallus* have ever been seen". Several other workers misinterpreted flame cells in the anterior portion of gymnophallid metacercariae as 'eye-spots'. Fujita (1925) figured 'ocelli' in *Gymnophalloides tokiensis* somewhat resembling bivalve statocysts (!). The true nature of these structures remains obscure. Hutton (1952) believed the lack of eye-spots to be a feature distinguish-

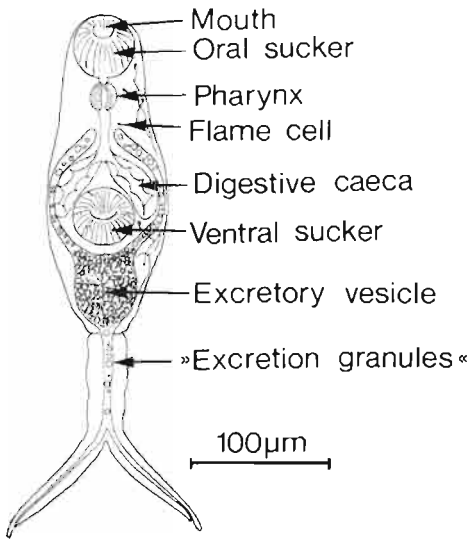


Fig. 13-92: *Gymnophallus choledochus*. Typical fork-tailed gymnophallid cercaria (ventral view). (After Loos-Frank, 1969a.)

ing his *'Cercaria fulbrighti'* (i.e., the cercaria of *G. choledochus*) in *Cardium edule* from others.

Unfortunately, this bulk of errors has found its way into the major part of the papers and reviews on the Gymnophallidae (i.e., G. Rees, 1939; Cable, 1953; Stunkard and Uzmann, 1958; Stunkard, 1959c; James, 1964). As a consequence, Cheng (1967), in his book on 'Marine Molluscs as Hosts for Symbioses', stated (p. 212):

> "The cercariae possess eye-spots which may be reduced or wanting. Furthermore, they are usually without a tail by the time they escape from the sporocyst; however, a small forked tail does develop in certain species, thus suggesting their relationship to the furcocercous cercariae, but the tail is usually lost prior to emergence."

None of these diagnostic statements is correct. It must be emphasized that (i) gymnophallid cercariae invariably possess bifid tails and (ii) invariably lack eye-spots, and that (iii) 'tailless gymnophallid cercariae' or 'cercariaea' invariably are metacercariae, be they enclosed in sporocysts or not.

Gymnophallid cercariae have repeatedly been confounded with larvae of the Fellodistomidae and other families, or *vice versa*. Young (1936) compared an unidentified (distinctly gymnophallid) cercaria from the Bering Sea with 8 types of marine furcocercous cercariae. Of these, however, only 2 were gymnophallids, the others representing 4 different unrelated families, namely the Bucephalidae, Schistosomatidae, Sanguinicolidae and Fellodistomidae (!).

Another source of confusion is the fact that most workers did not take into account the apparent strict host specificity of gymnophallid cercariae. Although several species have been incriminated to parasitize more than one bivalve species, a critical evaluation of the published information indicates that this is not the case. A different picture arises with respect to the *meta*cercariae. As stated above, 'tailless cercariae', reported from different

molluscan hosts and claimed to be specifically identical, are, in fact, *meta*cercariae. With a few exceptions, the host specificity of gymnophallid *meta*cercariae is weak. Many occur in a number of unrelated bivalve species. Finally, gymnophallid metacercariae enveloped by (second intermediate-) host tissue have sometimes been mistaken for 'tailless cercariae inside sporocysts'. One gains an impression of the possible amount of confusion if one takes into consideration that, on the German North Sea tidal flats, almost every adult individual of *Cardium edule* is simultaneously parasitized by at least 3 and sometimes even by 4 species of larval gymnophallids (Lauckner, 1971):

Parasite	Stage in bivalve host	In the literature commonly referred to as
Meiogymnophallus minutus	Metacercariae enveloped by host tissue	'Tailless cercariae inside sporocysts'
Meiogymnophallus sp.	Metacercariae between mantle and shell	'Tailless cercariae'
Gymnophallus gibberosus	Metacercariae in host tissues around adductor muscle	'Tailless cercariae', 'pearl trematode'
Gymnophallus choledochus	sporocysts with metacercariae	'Tailless cercariae'

Although occupying different microhabitats in the cockle, these parasites have frequently been confounded. With some imaginative faculty it is even possible to construct a 'developmental sequence' leading from the 'sporocyst-enclosed tailless young cercaria' (i.e., the metacercaria of *Meiogymnophallus minutus*) to the 'fully grown cercaria escaped from the sporocyst' (i.e., the metacercaria of *Gymnophallus gibberosus*). This has occurred more than one time in the literature.

North Sea *Scrobicularia plana* are host for the sporocysts and cercariae of *Meiogymnophallus minutus*. This species has a long and fascinating history. Its adult is known since 1859, the metacercaria — described under various names — since the turn of the century (see following subsection). Although almost every cockle on North Sea tidal flats is infested with the metacercariae, the cercaria remained hidden until 1977, when James and co-authors reported — without giving a valid description — the first larval stage from individuals of *S. plana* collected in Burry Inlet (Wales). The low incidence of sporocyst infestations — 0.3 % in clams collected during the period 1967-74 — may have accounted for this late discovery. Loos-Frank (1971b) found unidentified sporocysts and cercariae (probably *M. minutus*) in 2 of 251 *S. plana* from the German North Sea coast.

From statements in the paper of James and co-authors (1977), the impression arises that the authors regard *Meiogymnophallus minutus* to be specifically identical with *Gymnophallus fossarum*, which parasitizes *Scrobicularia plana* in the Mediterranean, and whose metacercariae occur in a great number of bivalves (Table 13-21; Bartoli, 1974a). However, *M. minutus* cercariae possess 2 pairs of penetration glands, which are lacking in *G. fossarum*.

The life history and ecology of *Gymnophallus fossarum* have been studied in great detail (Bartoli, 1965a, 1972, 1973a, b, 1974a, 1976, 1981a, b). Its cercaria is morphologi-

Table 13–14
Gymnophallidae. Variations in life-cylce pattern (Compiled from the sources indicated)

Species	Life-cycle pattern*)	First intermediate host	Second intermediate host	Main (natural) final host. [ ] = experimental host	Geographic area	Main references
Gymnophallus strigatus	I	*Tellina tenuis*	*Donax, Ensis, Spisula, Solen,* and others	unknown	Mediterranean, Britain	*3, 23, 24*
G. fossarum	I	*Scrobicularia plana*	*Cardium, Tapes, Spisula, Solen, Ensis,* and others	*Haematopus ostralegus, [Larus argentatus]*	French Mediterranean coast (Camargue)	*1, 2, 3*
G. rostratus	I	*Loripes lacteus*	*L. lacteus, Tapes, Donax, Abra, Tellina,* and others	unknown	Camargue	*3*
G. rebecqui	I	*Abra ovata* (probably)	*A. ovata, Cardium glaucum*	*Aythya* spp., *Anas clypeata*	Camargue	*3*
G. choledochus	III	*Cardium edule*	(C. edule)†), *Nereis diversicolor*	*Larus* spp., *Tadorna tadorna,* Anatidae, Limicolidae	North Sea	*12, 15, 16*
G. nereicola	III	*Abra ovata*	*(A. ovata)*†), *N. diversicolor*	*H. ostralegus, Charadrius* spp., *Calidris* spp., *Larus* spp.	Camargue	*2, 3*
Gymnophallus sp. *(deliciosus?)*	III	*Cardium lamarcki*	*(C. lamarcki)*†), *N. diversicolor*	unknown	Baltic Sea	*12*
G. (Parvatrema?) australis	IV	*Mytilus platensis*	*M. platensis*	unknown	South American Atlantic coast	*30, 31*
Parvatrema borinqueñae	I	*Gemma purpurea*	*Cerithidea costata* (Gastr.)	*[Gallus domesticus]*	Puerto Rico	*4, 5*
P. borealis	I(?)	*Gemma gemma*	*Gemma gemma, Nereis* spp.	*[Somateria mollissima]*	North American Atlantic and Pacific coasts	*21, 27, 28, 29*

P. donacis	I	*Donax variabilis*	*D. variabilis*	unknown	Gulf of Mexico	*10, 14*
P. affinis	II	*Macoma baltica*	*M. baltica*	*H. ostralegus, S. mollissima, Melanitta* spp., *Sterna* spp.	Baltic, North and White Seas	*11, 18, 19, 25*
P. isostoma	I	*Abra ovata*	*A. ovata, C. edule (glaucum?)*	Charadriiformes	Black Sea	*8*
P. obscurus	I	*Transennella tantilla*	*T. tantilla*	unknown	North American Pacific coast	*20*
Lacunovermis macomae	I	*Macoma baltica*	*M. baltica*	*Melanitta nigra, S. mollissima*	Baltic and North Seas	*11, 17, 18, 19*
L. conspicuus	I(?)	*Macoma inconspicua*	*M. inconspicua*	*Aythya marila, Melanitta nigra*	Vancouver (Canada)	*6a, 6b*
Meiogymnophallus minutus	I	*Scrobicularia plana*	*Cardium edule*	*Haematopus ostralegus*	North Sea	*12, 32*
Meiogymnophallus multigemmulus	II	*Macoma inconspicua*	*M. inconspicua*	*Melanitta* spp.	Vancouver (Canada)	*6a, 6b*
'Gymnophallus margaritarum'	II	*Mytilus galloprovincialis*	*M. galloprovincialis*	unknown	Gulf of Naples (Italy)	*22*
'Cercaria turtonii'	II	*Turtonia minuta*	*T. minuta*	unknown	Barents and White Seas	*7*
'Cercaria discursata'	II	*Abra (Syndosmya) alba*	*A. alba*	unknown	Black Sea	*26*
'Cercaria granosa'	II	*Mulinia lateralis*	*M. lateralis*	unknown	Florida (USA)	*9*
'Cercaria fragosa'	I(?)	*Donax variabilis*	*D. variabilis*	unknown	Florida	*9*

*) *Life-cycle patterns* — I: *normal*, II: *abbreviated*, III: *alternative*, IV: *aberrant*; *for explanation see text.*
†) *Cercariae do not leave first host but transform into metacercariae therein.*

References: 1 Bartoli (1965a), *2* Bartoli (1972), *3* Bartoli (1974a), *4* Cable (1953), *5* Cable (1956), *6a* Ching (1965), *6b* Ching (1973a), *7* Chubrik (1966), *8* Dolgikh (1968b), *9* Holliman (1961), *10* Hopkins (1958), *11* Jameson and Nicoll (1913), *12* Lauckner (1971), *13* Lebour (1908a), *14* Loesch (1957), *15* Loos-Frank (1969a), *16* Loos-Frank (1969b), *17* Loos-Frank (1970), *18* Loos-Frank (1971b), *19* Markowski (1936), *20* Obreski (1968), *21* Oglesby (1965), *22* Palombi (1924), *23* Palombi (1934), *24* G. Rees (1939), *25* Selikman (1953), *26* Sinitsin (1911), *27* Stunkard (1962), *28* Stunkard (1970a), *29* Stunkard and Uzmann (1958), *30* Szidat (1962), *31* Yamaguti (1975), *32* James and co-authors (1977).

cally almost identical with that of *G. nereicola*, the only distinguishing feature being the presence of 2 pairs of penetration glands in the latter which are absent in *G. fossarum*. Chaetotaxy, however, revealed differences, at the specific level, in the number and arrangement of sensory papillae (Richard and Bartoli, 1974). It appears likely that chaetotaxy might prove a suitable means for the characterization and identification of other larval trematodes, particularly of the morphologically very similar gymnophallid cercariae.

Meiogymnophallus minutus and *Gymnophallus fossarum* have a 'normal' life-cycle (here designated 'Type I'-) pattern in that they utilize a secondary host which is specifically different from the primary host. In the Gymnophallidae, however, this kind of pattern is the exception rather than the rule. Of the 23 species of gymnophallids listed in Table 13-14, at least 6 have abbreviated (Type II-) and at least 3 have alternative (Type III-) life cycles. In the remaining 13 species (Type I), the cercariae leave the first intermediate host and penetrate a second (bivalve) intermediate host which, however, may be another individual of the same host species in 9 of the 13 trematodes considered here.

Gymnophallus strigatus is another species with a 'normal' life cycle. In the Mediterranean, *Tellina tenuis* is the first intermediate host, and the metacercariae develop in a great number of bivalves, curiously with the exception of *T. tenuis* (Bartoli, 1974a). If Bartoli's specific identification is correct, the life-cycle pattern of *G. strigatus* displays geographic variation. Lebour (1908a) originally described the metacercaria as a 'tailless cercaria' from *Tellina tenuis* (i.e., the secondary host which is refractory to *G. strigatus* in the Mediterranean) and *Donax vittatus* from Alnmouth, Northumberland. Sporocysts and cercariae were found in neither of these bivalves. G. Rees (1939) redescribed the metacercaria from *T. tenuis* and claimed to have identified *Cardium edule* as first intermediate host. Unfortunately, Miss Rees mistook sporocysts and tailless metacercariae of *G. choledochus* occurring in the cockle for stages in the life history of *G. strigatus*.

Cercaria baltica, found in 18 of 300 *Macoma baltica* from the Polish Baltic Sea coast (Markowski, 1936), is probably the larval stage of *Lacunovermis macomae* (Loos-Frank, 1970, 1971b). A related and morphologically similar species, *L. conspicuus*, parasitizes *Macoma inconspicua* on the North American Pacific coast (Ching, 1965). Further gymnophallids exhibiting normal life-cycle patterns are listed in Table 13-14. Of these, *Parvatrema borinqueñae* is somewhat exceptional in utilizing gastropods *Cerithidea costata* rather than bivalves as second intermediate host (Cable, 1953).

A second group (Type II) of gymnophallids is characterized by an abbreviation of the life cycle: The cercariae develop into metacercariae inside the sporocyst without leaving the first intermediate host. In some species, the cercarial stage may be transitional and of short duration. Although the maturation of the sporocyst-enclosed metacercariae is accompanied by a considerable increase in size, this process has been termed 'degeneration' by some workers. *Parvatrema affinis* parasitizes *Macoma baltica* from the North, Baltic and White Seas (Selikman, 1953; Loos-Frank, 1971b). The sporocyst-enclosed metacercaria (Fig. 13-93) has first been described as *Metacercaria morula* by Markowski (1936). *Meiogymnophallus multigemmulus* develops similarly from cercaria to metacercaria *in situ* in *Macoma inconspicua* from the North American Pacific coast (Ching, 1965). Further gymnophallids with abbreviated life cycles are listed in Table 13-14. All of these are only known in their sporocyst-enclosed metacercarial stage.

The third group (Type III) of gymnophallids includes species which display host

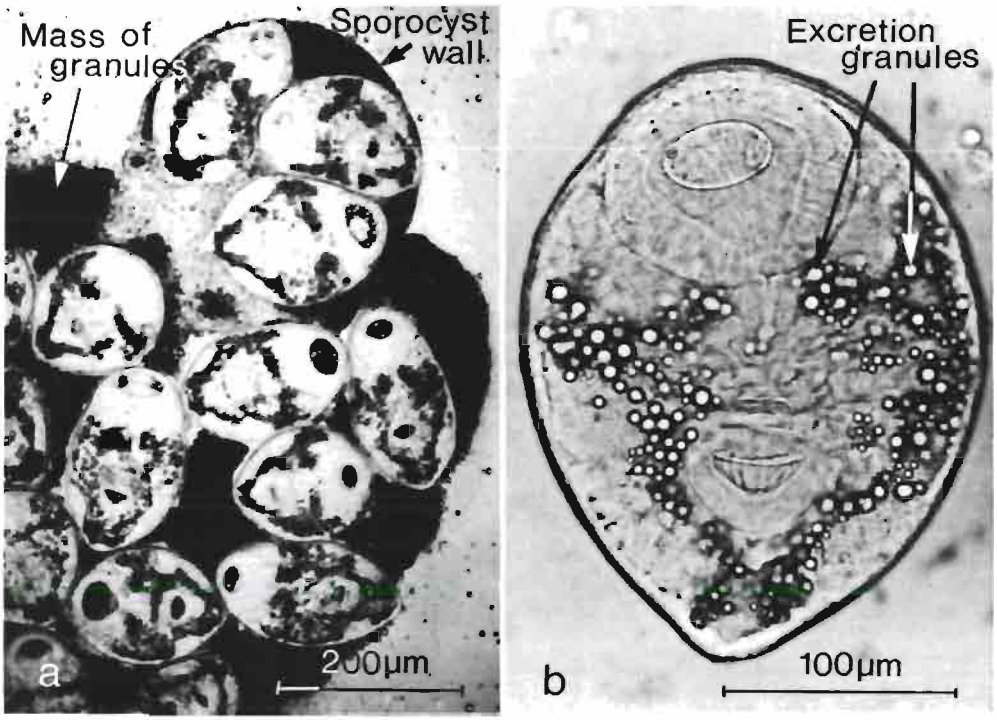


Fig. 13-93: *Parvatrema affinis* from *Macoma baltica*. (a) Disrupted sporocyst liberating numerous metacercariae and dense masses of granules appearing dark and refringent in transmitted light and whitish in reflected light; (b) metacercaria (under slight cover-glass pressure) showing conspicuous 'excretion granules'. (Original.)

alternation: At certain times, usually during the warm season, fork-tailed cercariae emerge from the first intermediate host and penetrate a second intermediate host which, as far as is known, is invariably a polychaete annelid. At other times, usually during the cold season, the cercariae remain in the first intermediate host and develop into metacercariae inside the sporocyst. Hence, the Type-III gymnophallid life-cycle pattern may be regarded as a combination of the Types I and II. Experiments have shown conclusively that cercariae of gymnophallids with Type-III life cycles are not capable of surviving and developing in molluscs. Whether polychaete annelids are the only compatible second intermediate hosts is not known, since the susceptibility of representatives of other invertebrate groups to cercariae of this kind has not yet been tested. In this context it is interesting to note that Paine (1962) found unidentified gymnophallid metacercariae in brachiopods *Glottidia pyramidata* from intertidal waters on the Gulf coast of Florida. The author suspected that the larvae might be related to *Cercaria pusilla*, described from *Chione cancellata* by Holliman (1961). Clams of this species were found to aggregate in the vegetation covering the brachiopods (Vol. I, Chapter 8).

The first alternative digenean life cycle has been described for *Gymnophallus choledochus*. Its motile sporocysts, which develop in the gonads of North Sea *Cardium edule*, give birth to fork-tailed cercariae during the summer months. These emerge from the cockle and penetrate polychaetes *Nereis diversicolor, Nephtys hombergi* and

Arenicola marina, in which they develop into tailless, unencysted metacercariae. Cercariae remaining inside the sporocysts until winter grow therein, shed their tails and transform directly into infestive metacercariae without leaving the sporocysts (Prévot, 1965b; Loos-Frank, 1969a, b; Figs 13-92, 13-94 and 13-95). Cercariae emerging from sporocysts and re-entering cockles or other bivalves do not develop into metacercariae. Hence, larvae from between the mantle and shell of *Mytilus edulis*, identified as *G. choledochus* by Selikman (1962), cannot be attributed to this species. The adults of *G. choledochus* mature in the gall-bladder of gulls, ducks and wading birds (Odhner, 1900b, 1906).

The sporocysts, cercariae and sporocyst-enclosed metacercariae of *Gymnophallus choledochus* have repeatedly been described in the literature under various names. The first account stems from Huet (1888b), who gave a fairly good description of the stages occurring in *Cardium edule*. He saw sporocysts (which are actually daughter sporocysts) in various stages of development. Some contained young third-generation sporocysts side by side with fully-developed furcocercous cercariae; others contained cercariae and tailless metacercariae (which he, however, regarded as cercariae). He also observed a metacercaria which was accidentally ejected through the birth pore of the sporocyst. This made him believe that (p. 151) "c'est à cet état que les cercaires . . . sortent du sporocyste qui les contient". This error, i.e., the belief that the 'tailless cercariae' leave the sporocyst and seek a second intermediate host, has persisted in the literature until the last decade.

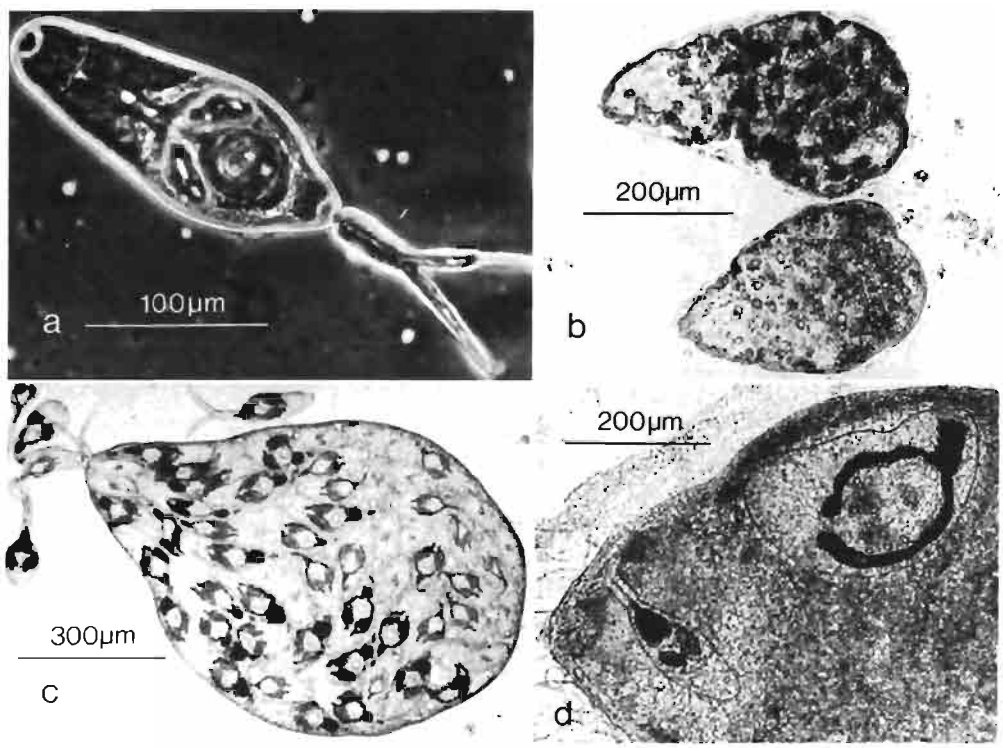


Fig. 13-94: *Gymnophallus choledochus* from *Cardium edule*. (a) Furcocercous cercaria (phase contrast); (b) immature 'spring' sporocysts with germinal balls; (c) mature 'summer' sporocyst with cercariae, some leaving sporocyst through birth pore; (d) portion of 'autumn' sporocyst with large tailless metacercaria and smaller tailed cercaria. (Original.)

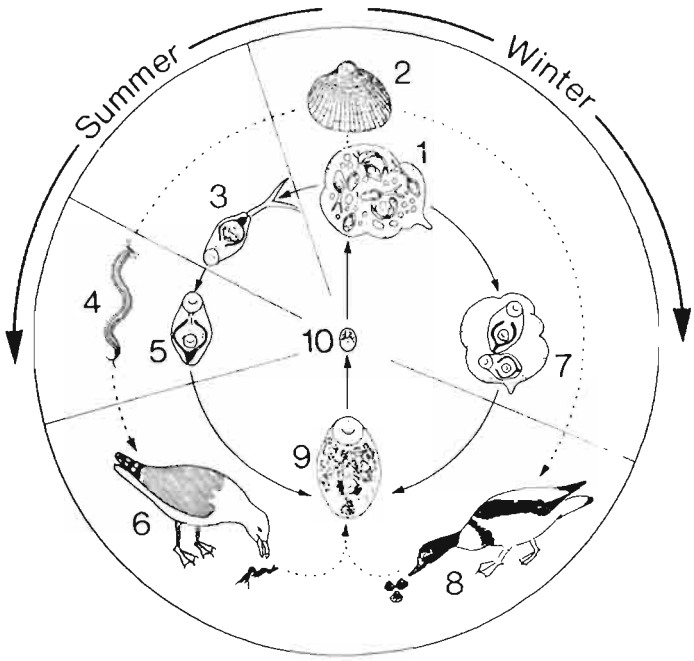


Fig. 13-95: *Gymnophallus choledochus* from *Cardium edule*. Alternative life cycles. Summer cycle: Sporocysts (1) in first intermediate host (2) produce cercariae (3) which penetrate polychaete second intermediate host (4) and develop into metacercariae (5) infestive for worm-eating final host (6). Winter cycle: Cercariae within sporocyst (1) shed tails and develop into infestive metacercariae (7) which are ingested, together with intermediate host, by mollusc-eating final host (8). Adult worm (9) in gall-bladder of bird final host produces eggs (10) eaten by first intermediate host (2) and developing into sporocyst (1). (After Loos-Frank, 1969a.)

Johnstone (1905a), obviously unaware of Huet's (1888b) description, referred to *Gymnophallus choledochus* as '*Cercaria fissicauda* La Val.'. However, the larva originally described by La Valette St. George (1855) under that name originates from freshwater pulmonate snails *Lymnaea stagnalis*. Villot (1879) described a furcocercous gymnophallid cercaria, likewise misnamed *C. fissicauda*, from *Abra* (*Scrobicularia*) *tenuis* in France, and Pelseneer (1906) reported a similar larva from *Tellina solidula* (= *Macoma baltica*). Pelseneer introduced another serious error in claiming that *C. fissicauda* should be named *C. dichotoma* Müller. The latter larva, however, has been found free-swimming in the Mediterranean off Nice. According to Odhner (1911b, c) and W. J. Rees (1947), it is in all probability the cercaria of the fellodistomid *Tergestia laticollis*. Hence, neither *C. fissicauda* La Valette St. George, 1855, nor *C. dichotoma* Müller, 1850, have anything to do with *G. choledochus* from *Cardium edule*. Lebour (1908a, 1912) made the confusion complete in figuring '*C. dichotoma* Müller' from *Abra tenuis*, but maintaining that the same cercaria occurs in *C. edule*. '*C. dichotoma*' (*sensu* Chubrik, 1966) from White Sea *Turtonia minuta* represents yet another entity. Lebour (1908a) reported 'tailless cercariae' (obviously metacercariae of *G. choledochus*) inside sporocysts in *C. edule*, but she erroneously regarded these as the 'younger stage' of *Cercaria strigata* (i.e., the metacercaria of *G. strigatus*) found by her in *Tellina tenuis*.

Hutton (1952), who became aware of the discrepancies and errors in the literature, restudied the parasite of the cockle and renamed it *Cercaria fulbrighti*. He also noted distinct morphological affinities between the metacercaria from *Cardium edule* and the adult of *Gymnophallus choledochus*. From the occurrence, side by side, of tailed cercariae and tailless metacercariae within the sporocyst he concluded that 2 types of life histories may exist. However, Hutton followed Huet (1888b) in erroneously regarding the tailless metacercariae as 'fully developed cercariae'. In spite of Hutton's (1952) clear delineation, Bowers (1965a), James and Bowers (1967c), Pascoe and co-authors (1968) and Richards and co-authors (1970) fell back into the previous errors and named the stages in *C. edule* '*Cercaria dichotoma* Lebour, 1911 (non Müller)' (in addition, the reference to 'Lebour, 1911' is incorrect since the 1911 volume of *Parasitology* in which the article appeared was published in 1912). Cheng (1967) further contributed to the confusion concerning *G. choledochus*. He did not recognize the specific identity of Hutton's (1952) *Cercaria fulbrighti* and Huet's (1888b) unnamed larva from *C. edule* and designated it '*C. hueti*'. He furthermore believed that '*C. hueti*' loses its tail prior to escaping from the sporocyst.

The abundance of *Gymnophallus choledochus* appears to vary geographically. It has been recorded in 5 of 420 *Cardium edule* at Plymouth, England, and in 32 of 10,500 and 1 of 1,059 cockles on Llanrhidian Sands, Gower Peninsula, Wales (Hutton, 1952; James and Bowers, 1967c; Richards and co-authors, 1970). Somewhat higher incidences — up to 7 % — have been observed in 3,650 cockles from German North Sea tidal flats (Loos-Frank, 1969a). Provided that the identification of a gymnophallid cercaria encountered in Mediterranean cockles is correct, *G. choledochus* also occurs — in low abundance — in *C. glaucum* from Camargue (France). Metacercariae were found in *Nereis diversicolor* but not within the sporocysts in *C. glaucum* (Bartoli, 1974a, b). Perhaps the parasite does not display host alternation in the warmer coastal waters of the Mediterranean.

A yet unidentified *Gymnophallus* sp., with a cercaria similar to but smaller than that of *G. choledochus*, parasitizes *Cardium lamarcki* from the Baltic Sea. Its metacercariae occur in *Nereis diversicolor* but occupy other microhabitats than *G. choledochus* (Lauckner, 1971). *G. nereicola* from Camargue resembles both species in utilizing the same secondary host (Timon-David and Rebecq, 1958; Rebecq and Prévot, 1962; Bartoli, 1972), but its sporocysts and cercariae occur in *Abra ovata*. On one occasion, *G. nereicola* metacercariae have been found side by side with furcocercous cercariae within the same sporocysts in *A. ovata*. It seems, therefore, that *G. nereicola* can utilize — like *G. choledochus* — its primary host as alternative secondary host, at least under certain environmental conditions. *G. nereicola* is the most abundant of all larval gymnophallids in Camargue. It infests up to 40 % of the *A. ovata* at certain sampling stations (Bartoli, 1974a).

The metacercarial and adult stages of the other gymnophallid cercariae reported from bivalves of the genus *Abra* — *Cercaria fissicauda* Villot, 1879, and *C. dichotoma* Müller *sensu* Lebour, 1908a, from *A. tenuis*, and *C. syndosmyae* Pelseneer, 1906, from *A. alba* — are unknown. They may be specifically identical and perhaps synonymous with *Gymnophallus nereicola*. *Parvatrema isostoma*, whose cercariae parasitize Black Sea *A. ovata*, appears to be a separate species because its metacercariae invade *Cardium* (*edule*?) and *A. ovata* but do not occur in annelids (Dolgikh, 1968b). *C. discursata* develops from cercarial to metacercarial stage within sporocysts in Black Sea *A. alba* and *A. ovata* (Sinitsin, 1911; Nechaeva, 1964) and, hence, has an abbreviated life cycle. It cannot be ruled out,

however, that *C. discursata*, as well as other gymnophallids listed in Table 13-14 as having abbreviated life cycles, exhibit in fact an alternation of hosts. In these species, the cercarial stage may be transitional and so brief that the chance to encounter fork-tailed cercariae is small.

Gymnophallus australis, developing in *Mytilus platensis* from Puerto Quequén, Argentina, has an aberrant (Type-IV) life-cycle pattern. Tailless 'cercariaea' develop in 'rediae' from which they apparently escape into the mussel's tissues. The initially free 'metacercariae' then become 'encysted' for some time. Upon subsequent dissolution (or bacterial destruction?) of the cyst wall, the 'metacercariae' undergo a curious process of 'parthenogenetic multiplication' in producing a new generation of 'cercariaea' within their parenchyme. These 'cercariaea', which carry an ephemeral vestigial tail, are an exact 'miniature edition' of the 'metacercaria' from which they originate (Szidat, 1962; Fig. 13-96). The above observations were made on preserved material, and Szidat hesitatingly and with all reserves linked the various parasite stages found in *M. platensis* with the life cycle of *G. australis*. If his conclusions are correct, the development of *G. australis* in the mussel somewhat resembles that of *Parvatrema homoeotecnum* in gastropods *Littorina saxatilis tenebrosa* from Wales (James, 1960, 1964, 1965, 1968c; Vol. I, Chapter 12), of *Cercaria*

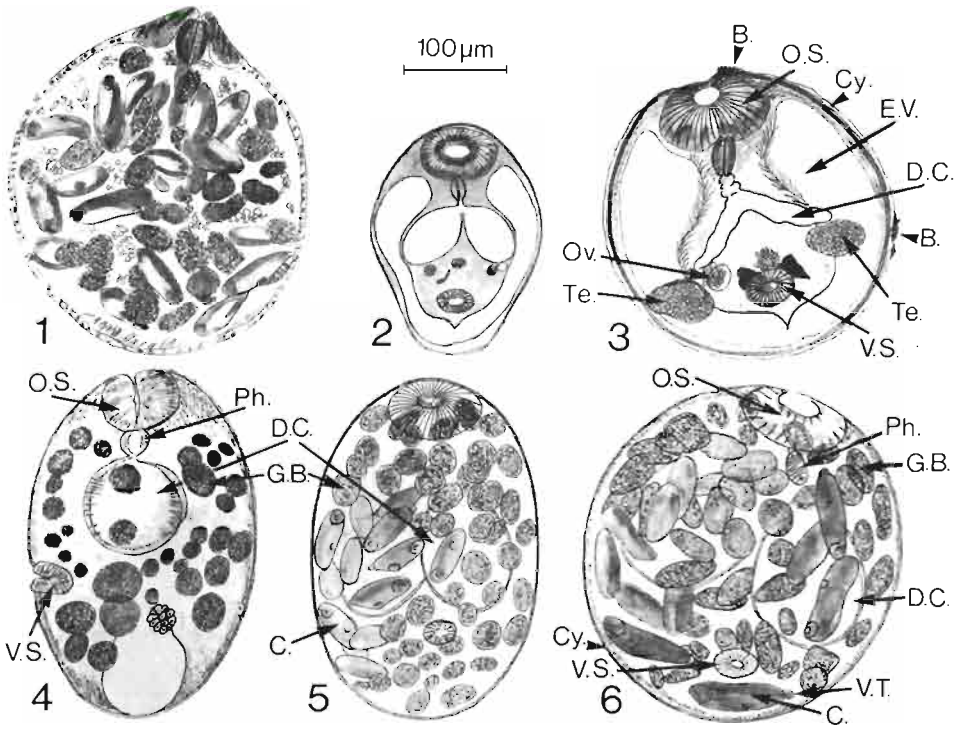


Fig. 13-96: *Gymnophallus (Parvatrema?) australis*. Developmental stages in *Mytilus platensis*. 1: Germinal sac ('redia') containing 'cercariaea', germ balls and food particles; 2: free metacercaria; 3: 'encysted metacercaria'; 4: liberated 'metacercaria' (daughter germinal sac?) producing germ balls in parenchyme; 5: advanced stage containing germ balls and young 'cercariaea'; 6: final stage containing germ balls and mature 'cercariaea'. B. bacteria, C. 'cercariaea', Cy. cyst, D.C. digestive caeca, E.V. excretory vesicle, G.B. germ balls, O.S. oral sucker, Ov. ovary, Ph. pharynx, Te. testes, V.S. ventral sucker, V.T. vestigal tail. (After Szidat, 1962.)

Table 13-15
Gymnophallidae. Some cercariae with unknown life cycles
(Compiled from the sources indicated)

Primary host	Parasite/Name adopted	Prevalence in primary host	Source	Locality	Remarks
Mya arenaria	*Cercaria myae*	2 of 143 3 of 910	Uzmann (1952)	Newburyport, Massachusetts (USA)	Motile sporocysts and cercariae similar to *Gymnophallus choledochus*. Believed to be cercaria of Brachylaemidae. Severe pathology in primary host. Cercaria also seen but not named by Stafford (1912).
Saxicava (Hiatella) arctica	*Cercaria reesi*	12 of 200	Hutton (1953)	Plymouth, England	Similar to *G. choledochus*. Sporocysts ovoid to sausage-shaped. Nothing on pathology.
Saxicava (Hiatella) striata	*Cercaria reesi*	15 of 100	Hutton (1953)	Plymouth, England	
Macoma baltica	*Cercaria duoglandulosa*	11 of 416	Reimer (1962)	Baltic Sea	Believed to be different from *Cercaria baltica* Markowski, 1936 (= *Parvatrema affinis*). See also Table 13–14. Nothing on pathology.
Macoma baltica	*Cercaria trioglandulosa*	1 of 416	Reimer (1962)	Baltic Sea	
Scrobicularia plana	unnamed	2 of 251	Loos-Frank (1971b)	North Sea	Nothing on pathology.
Mulinia lateralis	*Cercaria imbecilla*	30 of 446	Holliman (1961)	Wakulla County, Florida (USA)	
Donax variabilis	*Cercaria fragosa*	18 of 1,763	Holliman (1961)	Franklin County, Florida (USA)	Listed as fellodistomid cercariae. Nothing on pathology.
Chione cancellata	*Cercaria pusilla*	3 of 120	Holliman (1961)	Franklin County, Florida (USA)	
Semele proficua	*Cercaria fimbriata*	7 of 9	Holliman (1961)	Wakulla County, Florida (USA)	
Turtonia minuta	*Cercaria dichotoma*	8 of 1,269	Chubrik (1966)	Barents and White Seas	Misnamed '*Cercaria dichotoma* Pelseneer, 1906'.
Abra tenuis	*Cercaria dichotoma*	1 of 50	Lebour (1908a, 1912)	Northumberland, England	Misnamed '*Cercaria dichotoma* Müller'.
Tapes decussatus	unnamed	n.i.	Jameson (1902)	Billiers, French Atlantic coast	Author reports experimental infestation of *Mytilus edulis* with 'cercariae' from *Tapes decussatus*. Doubtful report. 'Tailless cercariae' = metacercariae? Possibly gymnophallids with abbreviated life cycles.
Cardium edule	unnamed	n.i.	Jameson (1902)	Billiers, French Atlantic coast	
Tapes pullastra	unnamed	2 of 516	Johannessen (1973)	Espegrend, Norway	'Split-tailed' cercariae in ovaries of 2 female clams. Partial parasitic castration. Possibly not a gymnophallid but a fellodistomid.

Table 13-16
Gymnophallus spp. Number of cercariae shed by infested bivalve hosts (After Bartoli, 1974a; modified)

Host species	Parasite species	Host-shell length (cm)	Observation period (days)	Number of cercariae shed during this period	Mean cercarial count day^{-1}
Abra ovata	*Gymnophallus nereicola*	1.23	30	94,040	3,135
Scrobicularia plana	*G. fossarum*	4.08	58	2 127,300	36,678
		3.75	46	1 238,650	26,927
		4.41	23	1 682,750	73,163
		3.61	27	1 664,320	61,641
Loripes lacteus	*G. rostratus*	1.14	23	329,500	14,326

quadriramis in gastropods *Onoba aculeus, Margarites helicina, Epheria divaricata, Littorina saxatilis, L. obtusata, Buccinum groenlandicum* and *Lacuna neriteidea* from the Barents and White Seas (Chubrik, 1966), and of *Parvatrema rebunense* in abalones *Haliotis discus hannai* from Rebun Island, Hokkaido, Japan (Shimazu, 1975). Szidat's (1962) 'redia' probably corresponds to James' (1964) 'primary germinal sac', the enclosed 'cercariaea' and 'encysted metacercariae' being daughter germinal sacs. Yamaguti (1975) tentatively transferred *G. australis* to the genus *Parvatrema*.

Thus far, only a few gymnophallid life cycles have been worked out experimentally; in other cases the specific identity of larval and adult stages has merely been deduced on the basis of morphological similarities. Several forms are only known in their cercarial and metacercarial stages (Table 13-14). In addition, a number of gymnophallid cercariae have been described whose specific identity and life-cycle pattern are unknown (Table 13-15).

The number of cercariae shed by bivalves infested with gymnophallid sporocysts may be enormous (Table 13-16). The figures shown in the table probably represent a conservative estimate of the number of larvae produced under field conditions. Due to unfavourable laboratory conditions, the infested bivalves died before having liberated their entire bulk of parasites. For example, the 4.41-cm individual of *Scrobicularia plana* infested with *Gymnophallus fossarum* harboured, at the time of its death, about 58,000 sporocysts. Some of these contained up to 115 cercariae. Considering an average of only 50 larvae per sporocyst, the dead host harboured at least 50 × 58,000 = 2 900,000 of unshed cercariae. Adding the number of larvae already shed (= 1 682,750), one obtains a total of 4 582,750 cercariae, produced in a single, medium-sized clam. Usually, periods of high cercarial emission alternate with periods of quiescence. The time intervals of maximum emergence of *G. fossarum* cercariae from individuals of *S. plana* rarely exceeded 2 h, and the highest number of larvae shed by a host of 3.6 cm shell length during a 2-h observation period was 110,790 (Fig. 13-97). Cercariae emerged by day and night; no distinct circadian pattern became apparent, but there was some indication of a lunar periodicity, with peak emergences occurring at full and at new moon (Bartoli, 1974a).

In spite of the astronomical numbers of cercariae emerging from infested bivalves, free-swimming individuals have rarely been encountered. Young (1936) reported on an unidentified gymnophallid cercaria detected among plankton organisms taken in vertical

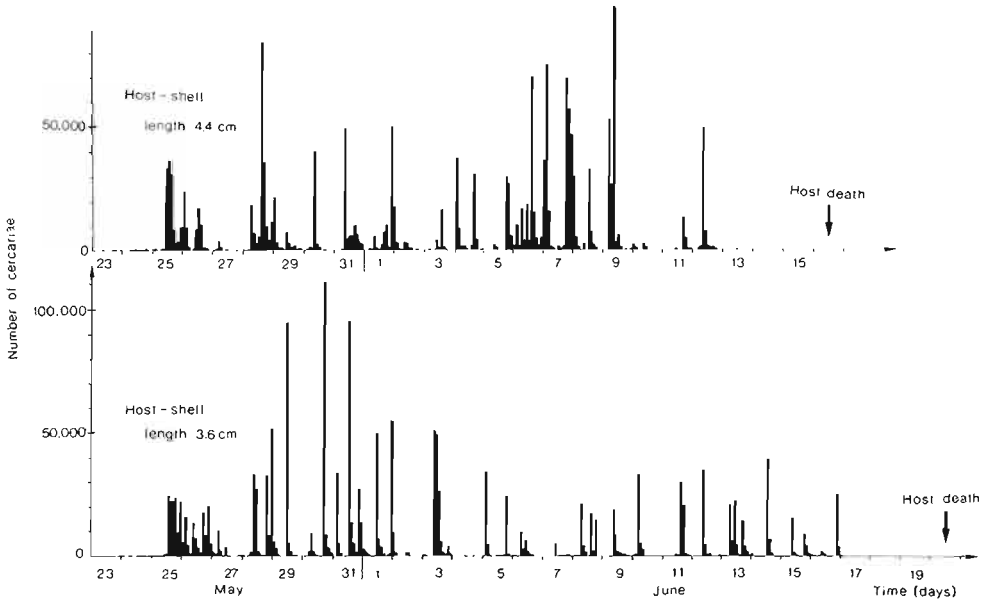


Fig. 13-97: *Scrobicularia plana.* Time sequence of cercarial emission by two individuals infested with *Gymnophallus fossarum* (2-h intervals). (After Bartoli, 1974a.)

tow net hauls near St. Paul's Island, Bering Sea. Thirty-three specimens, evidently belonging to the same species, were found in 11 out of 102 collections examined from that area.

Little has been reported on the pathology of gymnophallids in their primary hosts, but it must be expected to be similar to that reported for other larval trematodes. Strangely, Richards and co-authors (1970) stated that in an individual of *Cardium edule,* infested with *Gymnophallus choledochus* ('*Cercaria dichotoma*'), the host tissue appeared to be in a healthy condition. This might have been due to the fact that the infestation was very light. In heavy *G. choledochus* infestations, the viscera and foot of *C. edule* are generally heavily bloated by the mass of sporocysts, the tissues appearing translucent, softened and watery. Affected cockles cannot be sexed due to the disappearance of gonadal tissues; they become emaciated and sluggish and are no longer capable of burrowing in the sediment. Resistance to thermal and osmotic stress and to desiccation is considerably reduced. Infested cockles, transported in air to the laboratory, soon commence to gape and are thereby readily separable from healthy ones. *C. lamarcki* infested with *Gymnophallus* sp. exhibit similar signs of emaciation and disease (Lauckner, unpublished).

Stafford (1912) found the visceral sac of *Mya arenaria* from Gaspé Bay (Canada), infested with gymnophallid sporocysts and cercariae (probably *Cercaria myae* Uzmann, 1952, which he, however, mistook for larval bucephalids), to be distended, soft, translucent and pale greenish-yellow in colour. This condition is known by clam diggers as 'waterbelly' (Stunkard and Uzmann, 1958). Severe atrophy of the gonad was observed in *M. arenaria* infested with *Cercaria myae.* There was histological evidence for partial to complete parasitic castration. In one case the affection was so extensive that it was considered doubtful that similarly infested animals could survive abnormal conditions

(Uzmann, 1952). Of 516 individuals of *Tapes pullastra* from Norway, 2 females had their ovaries filled with sporocysts containing specifically unidentified split-tailed (gymnophallid or fellodistomid?) cercariae. The affected clams had only a few very small ova compared with uninfested ones (Johannessen, 1973). Hopkins (1958a) observed parasitic castration in *Donax variabilis* from Mustang Island, Texas (USA), parasitized by *Parvatrema donacis*.

Parasitic castration due to the infiltration of the gonad and the digestive gland by larval gymnophallids was also commonly observed in *Abra ovata, Scrobicularia plana, Tellina tenuis* and *Loripes lacteus* from Camargue. In no case have remnants of degenerating sporocysts and regenerating host tissues been seen, which would indicate that a previously parasitized bivalve recovers from a gymnophallid infestation. Tissue destruction is generally so extensive that, in the long run, affected hosts succumb to their injuries (Bartoli, 1974a).

A curious 'mutualistic' interrelationship appears to exist between *Donax variabilis* from Mustang Island, Texas, and two organisms growing on the posterior portion of the valves of some clams — an alga (*Enteromorpha flexuosa*) and an undetermined hydroid. Of 498 *D. variabilis* devoid of these organisms, 0.6 % and 1.6 %, respectively, were infested with sporocysts and cercariae of *Bucephalus loeschi* and *Parvatrema donacis*. No trematode infestations were detected in 332 clams of the same size groups, carrying either hydroids or algae. A third larval trematode, *Cercaria choanura*, however, was found to invade clams of all three groups (Loesch, 1957).

Several cases of double infestation involving gymnophallids have been observed. Of 420 *Cardium edule* collected near Plymouth (England), 111 (26.4 %) harboured the larval stage of *Labratrema minimus* ('*Bucephalus haimeanus*'), and 5 (1.19 %) were infested with *Gymnophallus choledochus* ('*Cercaria fulbrighti*'). Four (!) of the 5 cockles carrying *G. choledochus* sporocysts simultaneously had *L. minimus* infestations (Hutton, 1952). The author did not further discuss this remarkable finding. The theoretically expected frequency of double infestations (for computation see Vol. I, p. 352-353) is $E = 1.06$ and $\chi^2 = 5.61$, which yields a $p < 0.025$. In plaintext: Double infestations involving *G. choledochus* and *L. minimus* occur statistically significantly more frequently than expected to occur by chance alone. Hutton (1952) also reported 2 cases of triple infestation in *Cardium edule* involving '*Cercaria cambrensis*' in addition to the 2 above-mentioned trematodes. But the latter name is a synonym for *Meiogymnophallus minutus* (see following subchapter), and Hutton actually mistook metacercariae of *M. minutus* enclosed by host tissue for 'tailless cercariae within sporocysts'. Therefore, Hutton's findings should not be interpreted as cases of triple infestation.

Of 176 *Abra ovata* harbouring *Gymnophallus nereicola*, 6 were concurrently infested with sporocysts of the cryptogonimid *Cercaria plumosa* Sinitsin, 1911, and 3 had sporocysts of the monorchiid *Paratimonia gobii*. In all of the 9 cases, cercariae of both species were shed concomitantly, which implies that the parasites did not adversely affect each other within the host (Bartoli, 1974a).

Gymnophallus nereicola is frequently hyperparasitized by a haplosporidian, *Urosporidium jiroveci*. Its spores (Fig. 13-98) have a dark brown endospore. This gives the infested trematode sporocysts an abnormal blackish colour with reflected light, by which the hyperparasite is easily detected under a dissecting microscope. The spores are fairly uniform in size, with an endospore diameter of about 7 µm. The number of affected

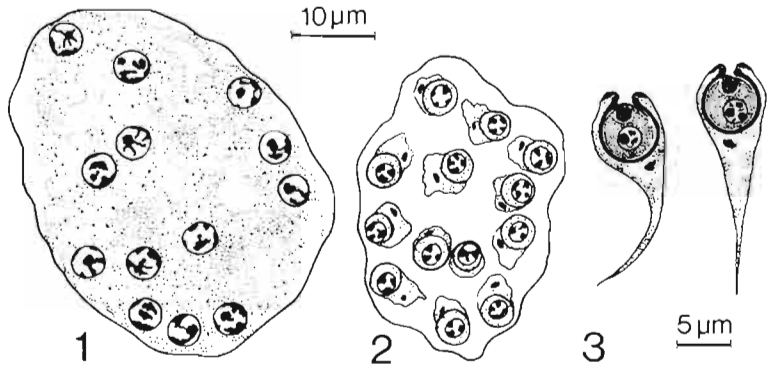


Fig. 13-98: *Urosporidium jiroveci.* Stages in development. 1: Plasmodium presumed to be early stage in sporogenesis sequence; 2: sporoblasts after separation, with envelopes developing around each sporoplasm; 3: spores. (After Ormières and co-authors, 1973.)

sporocysts varies; in heavy infestations, virtually all of the larval trematodes in a particular host are involved. Developing germ balls are more easily invaded than mature cercariae, but as the haplosporidian spreads, the cercariae vanish, leaving a sporocyst completely filled with *U. jiroveci* spores. The hyperparasite is restricted to *G. nereicola*; it never attacks the tissues of *A. ovata*. *U. jiroveci* appears to be fairly host-specific. It was never seen in larval *G. fossarum* parasitizing *Scrobicularia plana,* which lives side by side with the hyperparasitized *A. ovata*. On the other hand, *U. jiroveci* was found in 2 of 5 *A. ovata* infested with *Paratimonia gobii,* but in none of 2 clams harbouring sporocysts of *Cercaria plumosa* (misidentified as the larva of *Bacciger bacciger* by Ormières and co-authors, 1973). In a single *A. ovata,* infested with both *G. nereicola* and *C. plumosa,* many sporocysts of the former were hyperparasitized by *U. jiroveci,* whereas those of the latter were uninfested. The haplosporidian shows a distinct seasonal distribution, with a peak abundance of 75 % in September and a sharp decline in the winter and early summer (Vol. I, Fig. 10-9*). The peak of the *U. jiroveci* incidence coincides with the maximum of *G. nereicola* infestations in *A. ovata*. It is believed that hyperparasitization of such a magnitude might have a moderating effect on the excessive dissemination of the trematode. It appears noteworthy that *G. nereicola* is the only hyperparasitized species among 8 larval gymnophallids occurring in Camargue (Ormières and co-authors, 1973; Bartoli, 1974a).

Bivalves as Second Intermediate Hosts for Digenea

Marine bivalves function as secondary hosts for a vast number of digeneans. Several families of fish trematodes — mainly the Monorchiidae, Lepocreadiidae and Zoogonidae — have life-cycle stages in pelecypods. Their metacercariae mostly encyst, but lepocreadiids have also been reported to occur free in the tissues of molluscan hosts. Occasionally, unencysted fellodistomid metacercariae of the genus *Proctoeces* have been reported from marine bivalves.

*) Caption of Fig. 10-9 contains a misprint. It must read: '*Gymnophallus nereicola* from *Abra ovata*'.

Metacercariae of digeneans maturing in birds represent the overwhelming majority of species, occur in greatest numbers, and cause the most serious pathology in bivalves. Larvae of the Echinostomatidae, Renicolidae and Psilostomatidae encyst in virtually every organ or tissue of the molluscan host, while those of the Gymnophallidae occur, unencysted, mostly in the extrapallial space. Some species exhibit a more or less pronounced preference for certain sites, while others are restricted to well-defined, special microhabitats.

Metacercariae do not normally cause 'parasitic castration' and are generally considered to cause little if any detriment to their host, "even when present in large numbers" (Lebour, 1907c). Evidence will be presented below that this view is entirely wrong. Among the effects produced by metacercariae the following may be diagnosed:

General debilitation,
shell gaping,
shell deformities,
chemical erosion of shells,
production of pearls and chalky concretions,
hypertrophy or atrophy of affected host tissues,
alterations of host behaviour favouring detection and predation by final host,
increase in oxygen uptake and metabolic rate,
changes in host response to environmental stress,
autotomy of body parts (siphons),
and, in heavy infestations, host death.

Digenea utilizing fishes as final hosts: family Lepocreadiidae

A number of fish trematodes — mainly representatives of the Monorchiidae, Lepocreadiidae and Zoogonidae — utilize marine bivalves as second intermediate hosts. *Tapes aureus* and *T. decussatus* from the Gulf of Naples have been identified as metacercarial hosts of *Lepocreadium album*. Its rediae and cercariae occur in *Nassarius mutabilis* and *N. corniculus,* and the adult parasitizes in the intestine of *Blennius gattorugine, Oblata melanura* and *Spondyliosoma cantharus*. The metacercariae, which measure about 300 μm in diameter, are found either encysted in the mantle or unencysted between mantle and shell. Up to 6.5 % of the *T. decussatus* from Naples were found infested (Palombi, 1934a, 1937; Vol. I, Chapter 12).

Donax semistriatus, Venus (*Chione*) *gallina, Spisula subtruncata, Mactra corallina* and *Cardium tuberculatum* from the French Mediterranean coast are natural hosts for the metacercariae of *Lepocreadium pegorchis*. The unencysted larvae inhabit small cavities in the tissues of the palps, gills, mantle, foot and wall of the visceral mass. No pathological reaction of host tissue being in contact with the metacercariae was observed. *Cardita sulcata, Venus fasciata, V. verrucosa* and *Psammobia tellinella* were successfully infested experimentally with *L. pegorchis,* but *Modiolus barbatus, Arca noae, A. barbata, A. tetragona, Astarte fusca, Quadrans serratus* and *Cardita calyculata* proved to be refractory. *Nassarius mutabilis* has been identified as first intermediate host of this trematode; the adult is a common intestinal parasite of *Maena smaris, Pagellus erythrinus, P. mormyrus* and *Pomatoschistus* (*Gobius*) *microps* (Bartoli, 1966b, 1967; Vol. I, Chapter 12).

Metacercariae of the lepocreadiid *Holorchis pycnoporus* have been found encysted in

the musculature of the body wall of *Parvicardium papillosum* from the French Mediterranean coast. About 60 % of the bivalves were infested with up to 4 metacercariae each. Cysts of *H. pycnoporus* also occurred in the first intermediate host, the small rissoid snail *Barleeia rubra*, but only in individuals harbouring the rediae of that species. Attempts to experimentally infest individuals of *B. rubra* devoid of rediae met with failure. Similarly, bivalves other than *P. papillosum* — i.e., *Abra ovata* and *Venus verrucosa* — proved to be refractory to the cercariae (Bartoli and Prévot, 1978). The adult of *H. pycnoporus* occurs in the intestine of *Diplodus vulgaris, D. annularis* and *Pagellus erythrinus* (Orecchia and Paggi, 1974; Paggi and Orecchia, 1974).

Gemma gemma from the New England coast have been reported as experimental second intermediate hosts for the lepocreadiid *Homalometron pallidum*. Its metacercarial cysts are oval, 80 to 100 μm in length and 75 to 80 μm in width, and have a very thin wall. Little growth occurs in the bivalve host. The rediae and cercariae of *H. pallidum* develop in *Hydrobia minuta* (Stunkard, 1964a, 1970a). The adult occurs in *Fundulus heteroclitus* and other teleosts (Stafford, 1904; Linton, 1940).

Digenea utilizing fishes as final hosts: family Zoogonidae

Single metacercarial cysts, about 300 μm in diameter, have been dissected from 2 of 5 *Lima hians* from Millport, Scotland. The larvae, which were named *Cercaria limae*, were believed to represent a stage in the life cycle of a fellodistomid, either *Steringophorus* or *Fellodistomum* (Nicoll and Small, 1909). James and co-authors (1977) refound the worm in *Macoma baltica* from Burry Inlet, South Wales. While Bray and Gibson (1980) agree with Nicoll and Small (1909) in assigning *C. limae* to the Fellodistomidae, and to *F. fellis* in particular, Køie (1980) doubts this relationship. In all probability, *C. limae* is the second larval stage of *Zoogonoides viviparus*. Its sporocysts and cercariae occur in *Buccinum undatum* (Vol. I, p. 374), and the adult parasitizes in the intestine of marine flatfishes. The metacercariae, first described from ophiuroids as *Cercaria capriciosa* by Cuénot (1892), have been recorded from a wide variety of invertebrates including gastropods, bivalves, polychaetes and echinoderms (Lauckner, 1973; Orrhage, 1973; Køie, 1976). In the Øresund (Baltic Sea), nearly all *Nuculana pernula* longer than 20 mm were found to harbour the metacercariae of *Z. viviparus*. These mostly occur encysted in the mantle margin but may also be found in the gills. The cysts are frequently empty or contain dead larvae or remnants of metacercariae. Larvae excised from intact cysts show little activity (Køie, 1976). This suggests that bivalves are not regular intermediate hosts for *Z. viviparus*.

Digenea utilizing fishes as final hosts: family Monorchiidae

Monorchiids typically encyst in bivalves, sometimes in the same host individual by which the cercariae have been shed. *Cumingia tellinoides, Tellina tenera* and *Macoma tenta* from Woods Hole, Massachusetts, are the first and second intermediate hosts for *Monorcheides cumingiae* (p. 658). After at least a brief free-swimming period, the cercariae encyst in the mantle, foot, gills and, most frequently, the siphons. In heavy experimental infestations, the distal end, particularly of the inhalant siphon, may become so filled with

metacercariae that it presents a frayed appearance. Frequently the heavily infested siphons are autotomized. The detached pieces may retain their ability to move, simulating the motion of annelids, and thereby attracting the attention of fishes. Cercariae encysting in the gills cause considerable distortion of affected filaments (Martin, 1938, 1940).

Clams *Tellina salmonea* and *Macoma nasuta* from the North American Pacific coast may be heavily infested with metacercariae of *Telolecithus pugetensis*. In the laboratory, the larvae also encysted in individuals of *Transennella tantilla*, the first intermediate host of the species (p. 659), as well as in *Clinocardium nuttalli* and *Tresus* (*Schizothaerus*) *nuttalli*. Limpets *Acmaea digitalis* and snails *Littorina planaxis*, exposed to *T. pugetensis* cercariae, became likewise infested, but the highest numbers of metacercariae were always recovered from *T. salmonea* and *M. nasuta* (DeMartini and Pratt, 1964). The authors believed that this might indicate a certain degree of physiological specificity of the metacercaria, but another explanation seems more likely: The brevifurcate cercariae of *T. pugetensis* are unable to swim and, hence, can only reach their second intermediate host by crawling along the bottom. The tellinid clams *T. salmonea* and *M. nasuta*, which are deposit feeders, collect the cercariae along with their food from the sediment surface, and thereby become more heavily infested than the other clam species which are filter feeders.

Metacercariae of *Postmonorchis donacis* occur encysted, mainly at the base of the siphons, but also in the gills and the mantle margin of *Donax gouldi* from the Californian coast. Of 400 bean clams examined, only 5 were devoid of *P. donacis* metacercariae (Young, 1953). P. T. Johnson (1968b) found all samples of *D. gouldi*, collected from 4 populations in southern California, to be infested to a varying degree with larval *P. donacis*, which "caused castration in otherwise healthy individuals". This sounds much like "lung cancer has been detected in otherwise healthy humans". Trematode-infested bivalves can in no way be regarded as 'otherwise healthy', and this is particularly true of the bean clam, which lives in one of the harshest, most unstable of marine environments. Even the slightest parasite-induced debilitation could be fatal for this clam. In spite of its small size, *D. gouldi* has been one of the commercially most important bivalves of the North American Pacific coast. At times it has occurred in tremendous numbers on the sandy beaches of southern California and northern Baja California, where it has repeatedly undergone more or less gradual population declines and subsequent resurgences, or sudden and devastating 'population crashes', bringing the related industry to a stand-still in the period of a few weeks (Johnson, 1968b). Various reasons possibly responsible for the rise and fall of the Californian *D. gouldi* populations have been discussed by Coe (1953, 1955, 1956), who held that, among other factors, trematodes (probably *P. donacis*) were important in controlling population size in bean clams. Gradual attrition of numbers over several months and eventual disappearance of populations of young *D. gouldi*, as reported by Johnson (1968b), is very much like that observed in juvenile, trematode-infested *Cardium edule* from North Sea tidal flats (see below).

Donax variabilis and *D. tumida* from the Gulf of Mexico are hosts for the metacercariae of the monorchiid *Cercaria choanura* (Hopkins, 1958a). Individuals of *D. variabilis* from Mustang Island, Texas, of *D. tumida* from Grand Isle, Louisiana, and of clams of both species examined at Port Aransas, Texas, all harboured this larval trematode (Loesch, 1957). The adult is unknown, and effects of the metacercariae on the clams have not been determined. Further records of larval monorchiids from American waters include *Cercaria caribbea XXXV* from Puerto Rican *Macoma cerina*, and *C. caribbea LXIII* from

Jamaican *Tellina martinicensis*. Both encyst in the siphons and foot of individuals of the same host species after an obligate swimming period (Cable, 1956, 1963).

Metacercariae of *Lasiotocus longicystis* occur in *Tapes aureus* and *T. decussatus* from the French Mediterranean coast. Up to 53 % of individuals of the former and 47 % of the latter species have been found infested with up to 62 cysts. The larvae were mostly located at the base of the inhalant and less often in the exhalant siphon, foot and mantle margin. The metacercariae were also found, but much less frequently, in *Solen marginatus, Ensis siliqua, Mactra corallina, Spisula subtruncata, Scrobicularia plana* and *Chione* (*Venus*) *gallina*. In these hosts they never occurred in the siphons. *Mytilus edulis, Cardium glaucum* and *Tellina* (*Macoma*) *tenuis* from the same localities appear to be refractory to *L. longicystis*. Inside the second intermediate host the larvae grow considerably, and marked development of internal organs occurs. Advanced metacercariae measure from 576 to 840 µm in length and from 280 to 416 µm in width (Bartoli, 1965c). Although the metacercaria was studied in great detail, the adult remained unknown. Postlarvae, obtained from experimental infestations of eels *Anguilla anguilla*, showed little further development of adult structures. *Lasiotocus longicystis* metacercariae are morphologically very similar to, and possibly identical with, *Metacercaria acherusiae*, described by Palombi (1934a) from *Tapes decussatus* in Lago di Fusaro, a brackish-water lagoon in the Gulf of Naples.

Abra ovata from the Gulf of Marseille is host for the sporocysts and cercariae of *Paratimonia gobii*. Its metacercariae, which are about 150 µm in diameter, encyst in the inhalant siphon of *A. ovata* and in the mantle margin of *Cardium glaucum, Parvicardium exiguum, Mytilus galloprovincialis* and other bivalves including the primary host. Heavy metacercarial invasion causes the siphons of *A. ovata* to autotomize. The detached, worm-like siphons are then easily accessible to the final host, *Pomatoschistus microps* (Maillard, 1975b, 1976).

Metacercariae of *Asymphylodora demeli* (Fig 13-99, b) occur — singly or in low

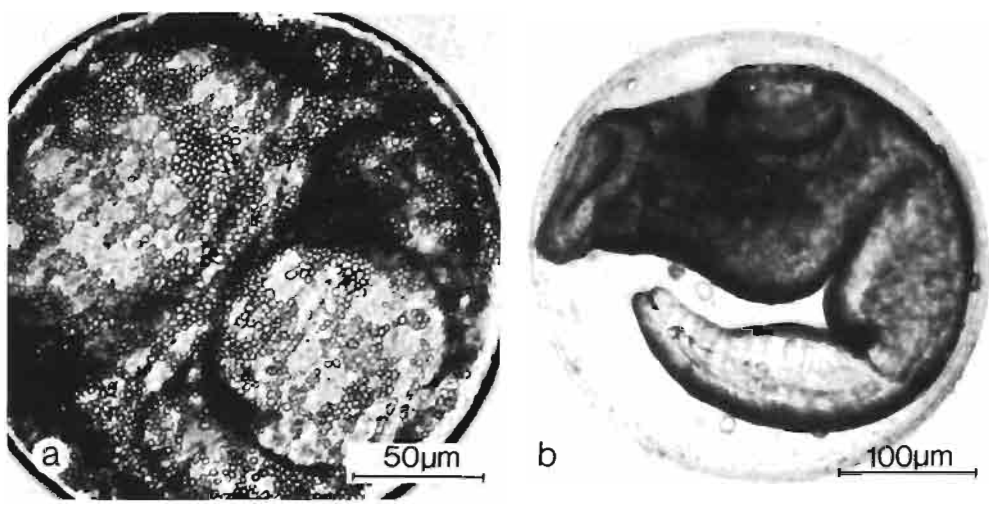


Fig. 13-99: Metacercariae from *Cardium lamarcki*. (a) *Psilostomum brevicolle*. Note characteristic diffuse, granular excretory system; (b) *Asymphylodora demeli*. (Original.)

numbers — encysted in the kidney of *Cardium lamarcki* (misidentified by some workers as *C. edule*) from the Baltic Sea (Markowski, 1936; Reimer, 1970; Lauckner, 1971). Vaes (1974) recorded it in *C. glaucum* from a brackish-water lagoon in Belgium. The rediae and tailless cercariae ('cercariaea') of *A. demeli* have been described under the name *Cercariaeum hydrobiae ventrosae* from *Hydrobia ventrosa* at Hel, Poland, by Markowski (1936). In the same paper that author reported the metacercariae from '*C. edule*' (= *C. lamarcki*) but did not recognize their specific identity with the cercariae occurring in *H. ventrosa*. Ankel (1962) even mistook advanced *A. demeli* metacercariae encysted in *H. ventrosa* and *H. ulvae* for 'echinostome(!) adolescariae', although she correctly identified the rediae and cercariae as belonging to the genus *Asymphylodora*.

Of 200 *Cardium lamarcki* from Hel, Poland, 69 were found to be infested with 1 to 12 metacercariae of *Asymphylodora demeli*. Aged cysts were frequently petrified to a varying extent. During the process of calcification, the initially transparent cysts assume a progressively darkening brownish colouration and become brittle. Larvae excised from such calcified cysts may still be alive (Markowski, 1936). *A. demeli* grows considerably within the secondary host — from a cyst diameter of about 160 μm to almost 380 μm, which corresponds to a volume increase of 13.4 times. Even when present singly or in low numbers, *A. demeli* metacercariae debilitate small bivalve hosts to such an extent that their resistance to thermal, osmotic and starvation stress is significantly reduced (Lauckner, unpubl.).

Trematodes of the genus *Asymphylodora* are generally considered as parasites of fishes. Adult *A. demeli* have been reported from *Gobius niger* and *Pomatoschistus* spp. in the North and Baltic Seas (Markowski, 1935; Koter, 1962; Reimer, 1970; Fonds, 1973), but Reimer (1973) and Vaes (1974) found adult worms in the intestine of annelids *Nereis diversicolor*. None of 42 *P. minutus* examined by Vaes (1974) were infested, while up to 40 % of the polychaetes had adult *A. demeli*. Reimer (1973) recorded sexually mature worms from 58 of 321 *N. diversicolor*, collected from various localities along the Baltic Sea coast. The high abundance of adult trematodes in the polychaetes and their infrequent occurrence in fishes suggests that *N. diversicolor* is the natural definite host of *A. demeli*, and that the gobies act as alternate or merely as post-cycle hosts. Still more abridged life cycles have been reported for other species of *Asymphylodora*. 'Progenetic' metacercariae of *A. progenetica*, *A. dollfusi* and *A. amnicolae* parasitizing freshwater snails (Serkova and Bychovskii, 1940; Biguet and co-authors, 1956; Stunkard, 1959a, b) are actually sexually mature adults. That this is true has been shown experimentally by Stunkard. Eggs from 'progenetic metacercariae' of *A. amnicolae*, fed to laboratory-raised *Amnicola limosa*, produced infestations in these molluscs. Hence, a vertebrate host is not essential for completion of the life cycle. Experiments involving *A. progenetica* from *Bithynia tentaculata* are suggestive of a similar life-cycle pattern in this species, but the results are not quite convincing because Serkova and Bychovskii (1940) did not maintain proper controls.

Monorchiid cercariae normally develop in sporocysts in bivalves. Members of the genus *Asymphylodora*, on the other hand, possess rediae which develop in gastropods. According to Cable (1956), the status of *Asymphylodora* as a genus in the Monorchiidae is open to serious question.

Digenea utilizing fishes as final hosts: family Fellodistomidae

Fellodistomid metacercariae of the genus *Proctoeces* (Fig. 13-87) have been reported from a number of marine bivalves. The various aspects of the life cycle of *P. maculatus* are exemplified elsewhere (pp. 665 ff. and 752 ff.). In addition to the bivalves identified as first and simultaneous second intermediate hosts of this species — *Mytilus edulis, M. galloprovincialis* and *Ischadium recurvum* —, its metacercariae have been found in 2 of 10 platform mussels *Mytilopsis* (*Congeria*) *leucopheata* from Galveston Bay, Texas (Wardle, 1980b). In the Mediterranean Sea, *P. maculatus* metacercariae also occur in a number of quite unrelated invertebrates, i.e., in polyplacophorans *Acanthochites discrepens*, in archaegastropods *Patella coerulea*, in errant polychaete annelids *Nereis caudata* and *Leptonereis glauca*, and in sedentary polychaetes *Hydroides norvegica* (Prévot, 1965a; Dupouy and Martinez, 1973; Martinez, 1973). Experiments with Black Sea molluscs (*Rissoa splendida, Hydrobia arenarum, Nana donovani, Bittium reticulatum*) and crustaceans (*Palaemon elegans, Gammarus olivia, Idotea baltica basteri, Chaetogaster marmoratus*) revealed that *P. maculatus* metacercariae develop in *R. splendida*, but in none of the other invertebrate species listed. The latter, however, were believed to exert population control over *P. maculatus* in the study area by feeding extensively on the cercariae (Machkevsky and Parukhin, 1981).

The variation in life-cycle pattern, as displayed by *Proctoeces maculatus* in different geographical areas, is indeed puzzling. In northern temperate waters, all stages occur in *Mytilus edulis*. In addition, bivalves *Scrobicularia plana* and gastropods *Buccinum undatum* may function as final hosts, but fishes from areas where these molluscs are heavily parasitized, never harbour adult worms. Quite a different picture arises in more southerly, warmer waters. In Galveston Bay, Texas, *P. maculatus* metacercariae occurring in *Ischadium recurvum* and *Mytilopsis leucopheata* never exhibit signs of maturity or progenesis, and ovigerous adults have only been recovered from the rectum of teleosts *Archosargus probatocephalus* (Wardle, 1980b). In the Mediterranean, *P. maculatus* has a normal 3-host cycle. Its larvae have been found in *M. galloprovincialis* from the Rhône estuary. From 2.5 to 6.0 % of the mussels from that area were found to be infested with sporocysts and cercariae, and from 6 to 20 % harboured 1 to 12 metacercariae. These, however, exhibited incomplete progenesis, with only a few abnormal eggs present. No adult *P. maculatus* were recovered from *M. galloprovincialis*, but natural infestations with mature worms were found in labrid fishes of the genera *Labrus* and *Crenilabrus*, while *C. griseus* (= *C. cinereus*) was successfully infested experimentally (Dubois, 1907b; Prévot, 1965a, Martinez, 1973).

This situation is markedly contrasted by the conditions governing the development of *Proctoeces maculatus* in Massachusetts and Connecticut waters, as reported by Uzmann (1953) and Stunkard and Uzmann (1959). Prévot (1965a) has, consequently, put forward the hypothesis that the environmental temperature might be the factor responsible for the observed discrepancies. He concluded that in the colder northerly waters the entire life cycle of *P. maculatus* can unfold in *Mytilus edulis*, while in the warmer southerly waters the parasite employs fishes as regular (obligate) final hosts. This hypothesis is, nevertheless, disproven by Dolgikh's (1967) finding of ovigerous and obviously sexually mature *P. maculatus* and *'P. major'* in Black Sea *Rissoa splendida*, and Dollfus' (1965, 1966) discovery of sexually mature *Proctoeces progeneticus* in gastropods *Gibbula umbilicalis* on

the North African Atlantic coast (Vol. I, p. 386). According to Bray and Gibson (1980), *P. progeneticus* is specifically identical with *P. maculatus*. Martinez (1973) argues that the geographical variations in the developmental pattern of the species could be due to the fact that we are actually dealing with 2 or more very closely related, morphologically as yet indistinguishable species of *Proctoeces*.

Prévot's (1965a) statement that *Proctoeces maculatus* has a normal 3-host cycle in the Mediterranean has been confirmed by Martinez (1973), who identified *Mytilus galloprovincialis* as cercarial, annelids *Leptonereis glauca* as metacercarial, and *Crenilabrus* spp. as final hosts involved in natural infestations. However, attempts to experimentally infest *M. galloprovincialis, Scrobicularia plana, L. glauca* and *Nereis diversicolor,* exposed to mature cercariae at 6°, 18° and 24 °C for periods varying between 9 and 22 days, met with failure, although the cercariae in the experimental containers survived outside the host for up to 2 months. It was concluded that the physical properties of the experimental milieu, as well as the condition of the cercariae, which were obtained from laboratory-held mussels, might have been responsible for this failure.

In Japan, ovigerous *Proctoeces* individuals, obtained from the gonad of *Crassostrea gigas* from Lake Hamana, were regarded as progenetic metacercariae of a separate species and named *P. ostreae* (Fujita, 1925). Non-ovigerous *Proctoeces* metacercariae have been recovered from mussels *Brachidontes senhausi* and pearl oysters *Pinctada martensi* (Yamaguti, 1938; Sakaguchi and co-authors, 1970a, b).

Proctoeces sp. Sakaguchi, Hoshina et Minami, 1970, was found more frequently and in higher numbers in *Pinctada martensi* living under field conditions than in cultivated pearl oysters. Adult worms were not seen in the oyster, but metacercariae fed to black porgies, *Mylio macrocephalus,* attained sexual maturity in the fish after about 2 weeks. No further growth of the worms occurred in the teleost's intestine, and the number of parasites recovered declined rapidly with time, indicating that *M. macrocephalus* (and probably fish hosts in general) are not well suited as final hosts. Similar reduced longevity has been observed in *P. maculatus* experimentally introduced into teleosts (Freeman, 1963a). The metacercariae from *P. martensi,* which also occurred in almost all bivalves and gastropods on and around the pearl-oyster beds, were later ascribed to *P. ostreae* Fujita, 1925. Sexually mature worms, morphologically identical with those obtained experimentally, were recovered from the hind-gut of 1 of 24 *M. macrocephalus* in Tanabe Bay, Japan (Sakaguchi, 1972). *P. ostreae* is presumably identical with *Xenopera* (*Proctoeces*) *insolita* (Bray and Gibson, 1980).

Digenea utilizing birds as final hosts: family Echinostomatidae

In addition to fish trematodes, a considerable number of digeneans maturing in the intestine, gall bladder or renal tubes of shore birds utilize marine Bivalvia as second intermediate hosts. Larval echinostomatids, renicolids and psilostomatids encyst in various tissues, while gymnophallids occur free within the tissues or the extrapallial space of the molluscan hosts.

Among the larval digeneans encysting in bivalves, members of the family Echinostomatidae constitute a group of major importance. Their cercariae develop in rediae in gastropods, and the adults parasitize in the intestine of shore birds (Vol. I, Chapter 12). The metacercarial cysts recovered from marine bivalves have frequently been misiden-

tified and assigned — mostly arbitrarily and without experimental proof — to various species, mainly of the genus *Himasthla*. Confusion is, consequently, the rule rather than the exception.

No attempt will be made here to present a review of the genus *Himasthla*. The reader is referred to Dietz (1909, 1910), Stunkard (1939, 1960a) and Loos-Frank (1967). An evaluation of previous records of adult and, in particular, of larval stages of these digeneans is difficult since

> "the solution of the problem . . . is not to be obtained by the reexamination of old material. It is rather to be sought in the application of the experimental method for the measurement of specific variation" (Stunkard, 1939, p. 721).

This statement is still valid.

However, a few clarifying remarks concerning the specific identification of echinostome metacercariae in marine bivalves will be made. The reasons for this are: (i) The primary hosts of *Himasthla* spp. are invariably common gastropod species occurring in sometimes considerable population densities in the littoral zone. Incidences of *Himasthla* infestation in these molluscs are frequently high. (ii) The metacercariae of *Himasthla* spp. exhibit weak host specificity; they invade common littoral bivalves in sometimes vast numbers, causing a variety of pathological responses; and they may exert population control over their hosts by killing young spat. (iii) In enzootic areas, parasitization by *Himasthla* metacercariae may interfere with cultivation and exploitation of commercially important bivalve species. (iv) Larval echinostomes, including *Himasthla,* have been implicated in human gastrointestinal disturbances following the consumption of raw bivalves.

Life cycle, prevalence and pathogenicity of *Himasthla elongata* in the gastropod first intermediate host have been discussed in detail in Vol. I, Chapter 12. The metacercariae of this species (Figs 13-100 and 13-104) encyst primarily in the foot of *Cardium edule, Mytilus edulis* and, more rarely, of other bivalves from the North Sea and adjacent areas. The cysts were first recorded from *C. edule* by Lebour (1905) and named *Echinostomum secundum* by Nicoll (1906a, b), who also reported the adult from the intestine of the oystercatcher *Haematopus ostralegus*. Both the generic and the specific designation have to be suppressed in favour of *Himasthla elongata* (Mehlis, 1831) Dietz, 1909. However, the name *'H. secunda'* has falsely persisted in the English marine biological literature until today (Lebour, 1907a, c, 1912, 1914; W. J. Rees, 1935, 1936; Rothschild, 1936; Chapman and Wilson, 1970, 1973; Chapman, 1973; Vol. I, Chapter 12). Other British workers (James, 1968b; Bowers, 1969; Robson and Williams, 1970, 1971a, b; Watts, 1970, 1971; Marshall and co-authors, 1974b; J. S. Thomas, 1974; Williams and Ellis, 1976; Moore and Halton, 1977; and others) have misidentified larval stages of *H. elongata* as those of *H. leptosoma* (Creplin, 1829). Previously, Palombi (1934a) had suggested the possible identity of *H. leptosoma* and *'H. secunda',* whereas Sprehn (1932), Cheng (1967), and Dawes (1968) list *'H. secunda'* and *H. militaris,* respectively, as synonyms of *H. leptosoma.* As shown by Loos-Frank (1967), both *H. militaris* and *H. leptosoma* are valid species, which are clearly distinguishable from *'H. secunda'* (= *H. elongata*). The intermediate hosts of *H. leptosoma* are unknown. Provided that the identification by Timon-David and Rebecq (1958) and Rebecq (1961, 1964a, b) is correct, the intermediate hosts of *H. militaris* are *Hydrobia ventrosa* and *Nereis diversicolor.*

The cercaria of *Himasthla elongata* from *Littorina littorea* (Vol. I, Fig. 12-6) has first

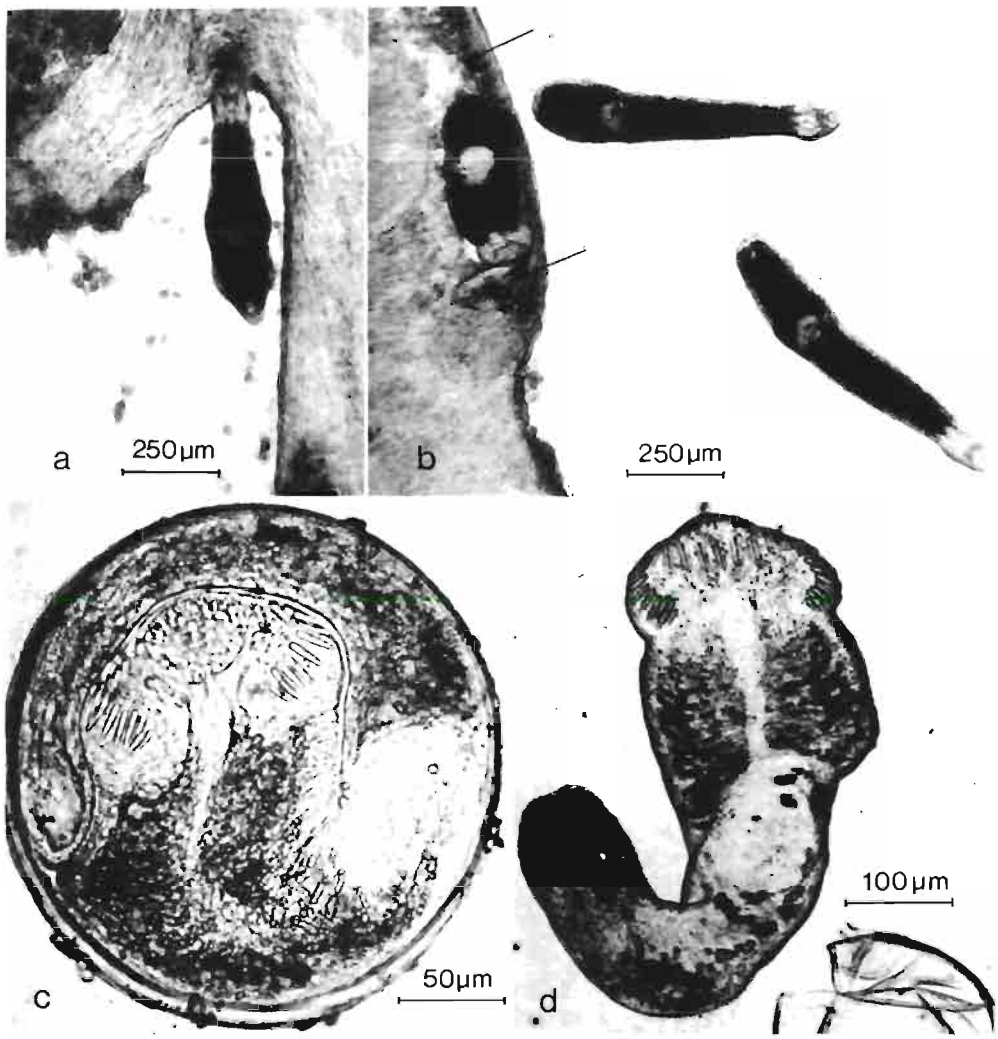


Fig. 13-100: *Himasthla elongata.* (a) Cercaria, with tail already shed, in process of penetrating foot of *Cardium edule;* (b) completion of penetration, cercaria initiating encystment. Note epithelial destruction at site of penetration (lower arrow) and tissue lysis (upper arrow) caused by cercarial enzymes. Two other cercariae, with shed tails, have lost contact to host due to vigorous defensive movements of host foot; (c) encysted metacercaria; (d) metacercaria excised from cyst. Note conspicuous row of head spines. (Original.)

been described by Lespès (1857b) as *Cercaria proxima.* Lebour (1905), who reported the encysted metacercariae from *Cardium edule,* mistook rhabdocoel turbellarians (p. 630) in the cockle's intestine for the sporocysts of that species. Upon discovery of the rediae and cercariae occurring in periwinkles, she corrected her error (Lebour, 1906), and later (1908b) obtained experimental metacercarial infestations in *Mytilus edulis.* Werding (1969) completed the life cycle. In the North Sea, *H. elongata* is the only echinostome parasitizing *L. littorea* (Lauckner, 1981).

Although *Cardium edule* and *Mytilus edulis* are the favourite second intermediate

hosts of *Himasthla elongata*, its cercariae also invade, to a lesser extent, *C. lamarcki, Mya arenaria* and *Macoma baltica* (Lebour, 1905, 1906, 1908b, 1912; Nicoll, 1906a; Loos-Frank, 1967; Werding, 1969; Lauckner, 1971; Dethlefsen, 1972). Whether metacercariae reported from *Mactra stultorum* (= *M. corallina*) and *Tapes pullastra* are also referable to *H. elongata*, remains to be demonstrated. Palombi's (1925) record of '*Echinostomum secundum*' in *Mytilus galloprovincialis* from the Gulf of Naples is certainly a false report, since the first intermediate host, *Littorina littorea*, does not occur in that area.

The metacercariae of *Himasthla elongata* have frequently been confounded with those of *H. continua* and *H. interrupta*. Both utilize *Hydrobia ulvae* as first intermediate host (Loos-Frank, 1967), but their metacercariae may be found side by side with those of *H. elongata* in the same second intermediate-host individual (Lauckner, 1971). The cysts of the 3 species are easily distinguishable by their diameter; they also prefer different microhabitats within the second intermediate host (p. 702, Fig. 13-101, Table 13-17). In spite of their frequent occurrence, *H. interrupta* and *H. continua* have not been recognized as distinct species until their validation and separation from *H. elongata* by Loos-Frank (1967). Therefore, the older accounts of the adult as well as metacercarial stages of '*H. secunda*' may refer to either of the 3 above species. Nicoll (1906a) gave the cyst diameters of '*H. secunda*' from the foot of *Cardium edule* as ranging from 210 to 250 μm. He also reported metacercariae from the mantle margin of the cockle (which, in all probability, are attributable to *H. interrupta*), but did not recognize their considerably smaller size. Markowski (1936) found cysts, measuring from 196 × 200 μm to 200 × 215 μm in diameter and attributed to '*H. secunda*', in the foot of *C. edule* (probably *C. lamarcki*) from Hel (Polish coast of Baltic Sea). This is clearly a misidentification because *H. elongata* does not penetrate into the Baltic proper (Lauckner, 1971). The size of the cysts, as well as their occurrence in the foot of the cockle, are suggestive of *H. continua*.

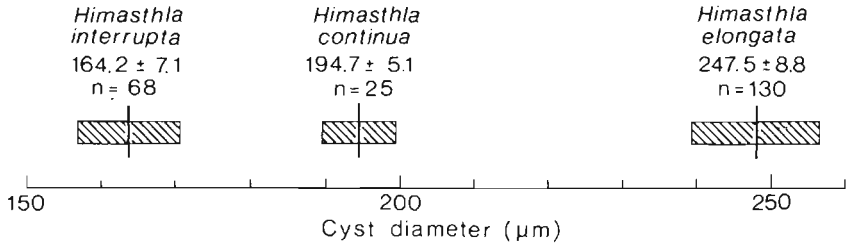


Fig. 13-101: *Himasthla* spp. Cyst diameters (vertical lines: means; horizontal hatched bars: standard deviations) of different species of metacercariae from North Sea bivalves. (Based on data presented by Lauckner, 1971.)

Villot (1875, 1879) described metacercarial cysts, 110 to 120 μm in diameter and believed to belong to *Himasthla leptosoma*, from the foot and siphons of *Scrobicularia* (= *Abra*) *tenuis* in France. The cysts, which are distinctly smaller than the above forms, cannot be attributed to either of these species. Villot's identification may be correct, but the cercaria described under that name from *Paludestrina stagnalis* (= *Hydrobia ventrosa*) in England by Lebour (1907a, 1912) belongs, beyond doubt, to *H. continua*. Apart from this, the latter cannot be mistaken for the larva of *H. militaris*, which occurs in the same host species but is distinctly larger (Timon-David and Rebecq, 1958; Rebecq, 1961,

Table 13-17
Cardium edule. Microhabitat selection (percentage distribution) of 3 larval trematodes in various body parts of juvenile cockles (Original)

Trematode species	Microhabitat					Total number of metacercariae	Total number of hosts	Number (percentage) of hosts infested	Mean number of metacercariae infested host^{-1}
	Foot	Paips	Gilis	Mantle margin	Visceral mass				
Himasthla elongata									
Percentage of cysts									
in summer spat	96.7	0	0	3.3	0	4,527	23	23 (100 %)	196.8
in autumn spat	97.0	0	0	3.0	0	33	24	9 (37.5 %)	3.7
Himasthla interrupta									
Percentage of cysts									
in summer spat	0.5	0	0	99.5	0	186	23	22 (95.7 %)	8.5
in autumn spat	14.1	5.1	1.0	75.8	4.0	99	24	17 (70.8 %)	5.8
Renicola roscovita									
Percentage of cysts									
in summer spat	0.6	71.7	2.1	10.5	15.1	3,879	23	23 (100 %)	168.7
in autumn spat	1.1	18.2	3.9	41.4	35.4	181	24	22 (91.7 %)	8.2

1964a, b). Nor can the difference in the selection of the second intermediate host be taken as a distinguishing feature. Timon-David and Rebecq found the cysts of *H. militaris*, which measure 200 to 275 µm in diameter, in annelids *Nereis diversicolor*, whereas metacercariae attributed to *H. leptosoma* have been reported from *Arenicola marina* and holothurians *Synapta inhaerens* (Cuénot, 1892, 1912, 1927; Caullery and Mesnil, 1900). The cysts found by Cuénot were 160 to 240 µm in diameter and, hence, considerably larger than those reported from *Abra tenuis* by Villot (1879). They were also said to possess '31 or 32' head spines, whereas *H. leptosoma* has only 29. Experimental encystment of *H. elongata* in *N. diversicolor* has been accomplished by Lauckner (unpublished) and Reimer (1971), and worms from the German North Sea coast were found to harbour natural infestations. Unidentified echinostome metacercariae have also been observed in *N. diversicolor* and *N. succinea* from the Azov Sea and from Scotland (Latysheva, 1939; Burt, 1962; Nechaeva, 1964). Thus, the occurrence of echinostome metacercariae is not restricted to bivalves. Their host spectrum may be broader than expected.

Frequently, cysts of more than one species of echinostome occur in a single host. Loos-Frank (1967) was unable to distinguish *Himasthla elongata, H. continua* and *H. interrupta* metacercariae present in the same host individual but, as shown by Lauckner (1971), the cysts can be separated into 3 discrete groups, with little if any overlap, by means of exact morphometric analysis. Timon-David and Rebecq (1958) found the diameter of *H. militaris* metacercariae to vary between 200 and 275 µm. Worms excised from cysts measured from 410 to 865 µm in length, which is an unusually wide range. The authors assumed that growth occurs within the cysts, but, as revealed by experimental and biometrical studies, *Himasthla* spp. do not increase in size within the second intermediate host (Lauckner, 1971). Hence, it must be concluded that Timon-David and Rebecq (1958) dealt with material consisting of more than one species of *Himasthla*.

From this summarizing account it becomes apparent that the situation regarding specific determinations in the genus *Himasthla* is chaotic, particularly with respect to the larval forms. In northern Europe, 6 distinct species of adult *Himasthla* are recognized (Loos-Frank, 1967). Of 4 of these — *H. elongata, H. continua, H. interrupta* and *H. militaris* — the entire life cycles are known, whereas *H. leptosoma* and *H. avosettae* are only known in the adult stage. This picture is contrasted by the record of at least 4 additional, specifically unidentified European *Himasthla* cercariae, i.e., *C. littorinae obtusatae* from *Littorina obtusata* (*L. littoralis*) (Lebour, 1912; Chubrik, 1966; James, 1968a), *C. himasthla* spec. I and *C. himasthla* spec. II from *Hydrobia ulvae* (Loos-Frank, 1967), and an unnamed larva from *Nassarius reticulatus* (Tallmark and Norrgren, 1976).

In America, the situation is much less chaotic. The life cycles of 3 species of *Himasthla* involving marine bivalves as second intermediate hosts are completely known. The rediae and cercariae of *H. quissetensis* develop in *Nassarius obsoletus* from Woods Hole, Massachusetts, and the metacercariae, 140 to 190 μm in diameter, occur in the mantle, gills and foot of *Mya arenaria, Mytilus edulis, Modiolus modiolus, Cumingia tellinoides, Argopecten irradians* and *Ensis directus* (Miller and Northup, 1926; Stunkard, 1938). *M. arenaria* also serves as second intermediate host for *H. compacta*, whose rediae and cercariae have been reported from *Hydrobia minuta* from Boothbay Harbor, Maine, and Woods Hole, Massachusetts (Stunkard, 1960a, 1970a). *M. edulis* is the favourite second intermediate host for *H. littorinae*, whose metacercarial cysts measure about 260 μm in diameter. The cercariae have been found in *Littorina saxatilis* and *L. obtusata* from Woods Hole (USA) and Dale, Wales (Stunkard, 1966; James, 1968a).

All of the above-mentioned *Himasthla* cercariae and metacercariae have 29 head spines, with the exception of *H. quissetensis*, which has 31. Unidentified himasthline metacercariae, with 31 head spines and about 220 μm in diameter, have also been found in *Cardium edule* from Sylt, German North Sea coast (Lauckner, 1971), although adult worms with a corresponding number of cephalic spines have not been recorded from birds in that area (Loos-Frank, 1967). Another species, thus far only known in the metacercarial stage, is *H. ambigua*. It was found encysted in the gill filaments of *Tapes decussatus* in the Gulf of Naples and claimed to have 32 instead of 31 head spines (Palombi, 1934a).

Incidence and intensity of infestation of bivalves with echinostome metacercariae vary greatly and are closely correlated with the abundance and infestation rate of the first intermediate hosts. Uzmann (1951) reported incidences of *Himasthla quissetensis* metacercariae in *Mya arenaria* from Merrimack Bay, Plum Island Sound and Annisquam River (Massachusetts, USA), ranging from 43 to 100 %. Intensities varied between 1 and 99 larvae per clam, with larger individuals showing a higher degree and frequency of infestation.

In *Mytilus edulis* and *Cardium edule* from the North Sea, incidences and intensities of echinostome infestation range from negligible to extremly high. They are lowest in mussels from rocky shores and cockles from wide, strongly exposed tidal flats, and highest in bivalves from sheltered areas. Hosts from intertidal habitats are consistently more heavily infested than individuals occurring subtidally (Figs 13-102 and 13-103). Both incidence and intensity of infestation closely parallel the degree of parasitization of the first intermediate host, *Littorina littorea* (Vol. I, Table 12-1). On the sheltered tidal flats of Sylt (German North Sea coast) it is virtually impossible to find uninfested cockles or mussels.

Himasthla metacercariae usually display little host specificity, although experimental

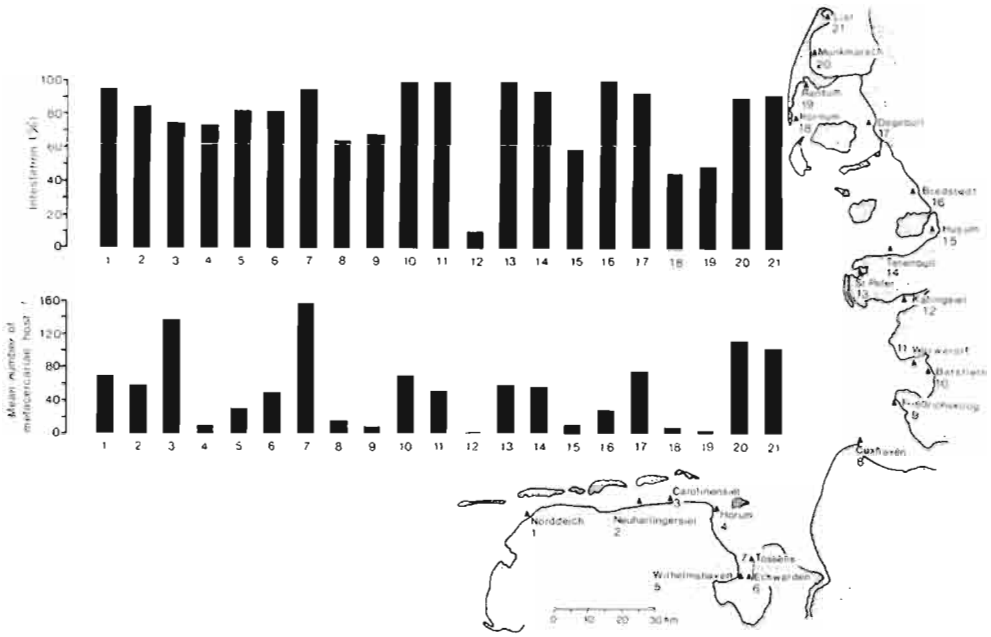


Fig. 13-102: *Mytilus edulis.* Incidence and intensity of infestation with metacercariae in mussels from intertidal sampling stations (records of unspecified metacercariae include *Himasthla* spp., *Renicola roscovita* and *Psilostomum brevicolle,* according to Lauckner, 1971). (After Dethlefsen, 1972.)

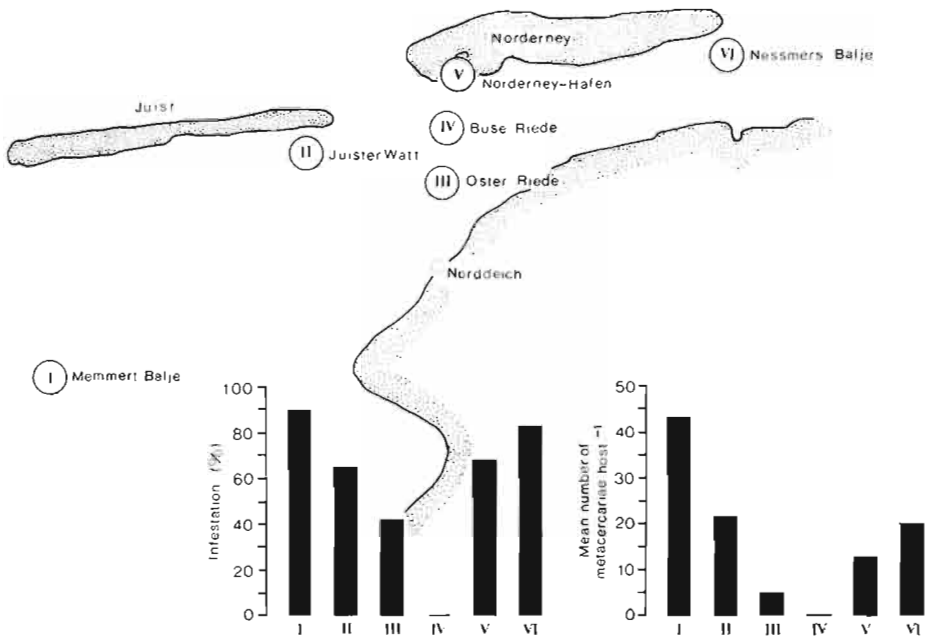


Fig. 13-103: *Mytilus edulis.* Incidence and intensity of infestation with metacercariae in mussels from subtidal sampling stations. See also Fig. 13-102. (After Dethlefsen, 1972.)

as well as field observations indicate varying degrees of host preference. Of 8 species of bivalves experimentally challenged with cercariae of *H. quissetensis*, *Mya arenaria*, *Mytilus edulis* and *Ensis directus* became most heavily infested, while *Crassostrea virginica* and *C. gigas* were refractory to the larvae, except for a few encysted metacercariae found in blood vessels but not in the tissues of *C. virginica* (Table 13-18; Cheng and co-authors, 1966a; Lauckner, 1974). The low overall recovery rate (max. 10 % of 150 cercariae) may be due to the experimental procedure employed, i.e., application of cercariae through holes bored into the hosts' valves instead of exposure of undisturbed test bivalves to free-swimming cercariae. The observed differences in host preference, exhibited by *H. quissetensis* cercariae, remain largely unexplained. Addition of plasma and tissue extracts of 7 of the bivalves listed in Table 13-18 was found to stimulate cercarial encystment, but the rapidity of cyst formation varied with the types of sera (Cheng and co-authors, 1966b). A number of other substrates similarly induced encystation, and it was concluded that this process is a relatively unselective response (Laurie, 1974). In fact, *H. quissetensis* cercariae, kept in sea water for 24 h or more, may readily encyst in the container or on a glass slide (Miller and Northup, 1926; Stunkard, 1938). In *H. elongata, H. interrupta* and *H. continua*, free encystation has been induced by slightly lowering the salinity of the medium (Lauckner, 1971).

Table 13-18

Himasthla quissetensis. Proportion of metacercariae recovered from 8 species of bivalves after experimental exposure to cercariae. Each mollusc challenged with 150 cercariae (Recalculated from Cheng and co-authors, 1966a)

Host species	Number of hosts challenged	Number of meta-cercariae recovered (Mean ± S.D.)	Percentage of (n = 150) metacercariae recovered
Mya arenaria	15	15.4 ± 5.8	10.3
Mytilus edulis	5	10.0 ± 2.7	6.7
Ensis directus	10	9.7 ± 3.4	6.5
Tapes philippinarum	10	4.8 ± 2.0	3.2
Modiolus demissus (= *Geukensia demissa*)	6	4.0 ± 2.0	2.7
Mercenaria mercenaria	10	2.1 ± 2.0	1.4
Crassostrea virginica	10	0.4 ± 0.1	0.3
Crassostrea gigas	10	0	0

Most *Himasthla* metacercariae exhibit a definite preference for certain organs or tissues of the intermediate host. The preferred sites can, however, differ between host species involved. Thus, the predominant foci of *H. quissetensis* invasion in *Mya arenaria* are the palps and gills; few metacercariae occur in other organs (Uzmann, 1951). In *Ensis directus*, on the other hand, *H. quissetensis* cysts occur invariably in the foot musculature whereas, in *Mytilus edulis*, they occupy both the foot and the digestive gland (Cheng and co-authors, 1966a). Similarly, *H. elongata, H. interrupta* and *H. continua* display distinct microhabitat segregation in their second intermediate hosts, *Cardium edule* and *Mytilus edulis*. *H. elongata* encysts almost exclusively in the distal portions of the foot musculature (Fig. 13-104); *H. continua* prefers the proximal part of the foot, while *H. interrupta* is

almost exlusively confined to the mantle margin (Lauckner, 1971; Table 13-17). This microhabitat segregation within the second intermediate host is apparently correlated with the behaviour of the cercariae. Those of *H. interrupta* and *H. continua* exhibit positive phototaxis (Loos-Frank, 1967), while *H. elongata* is strongly negative (Lauckner, personal observation). Chapman and Wilson (1970), however, failed to detect a sensitivity to light

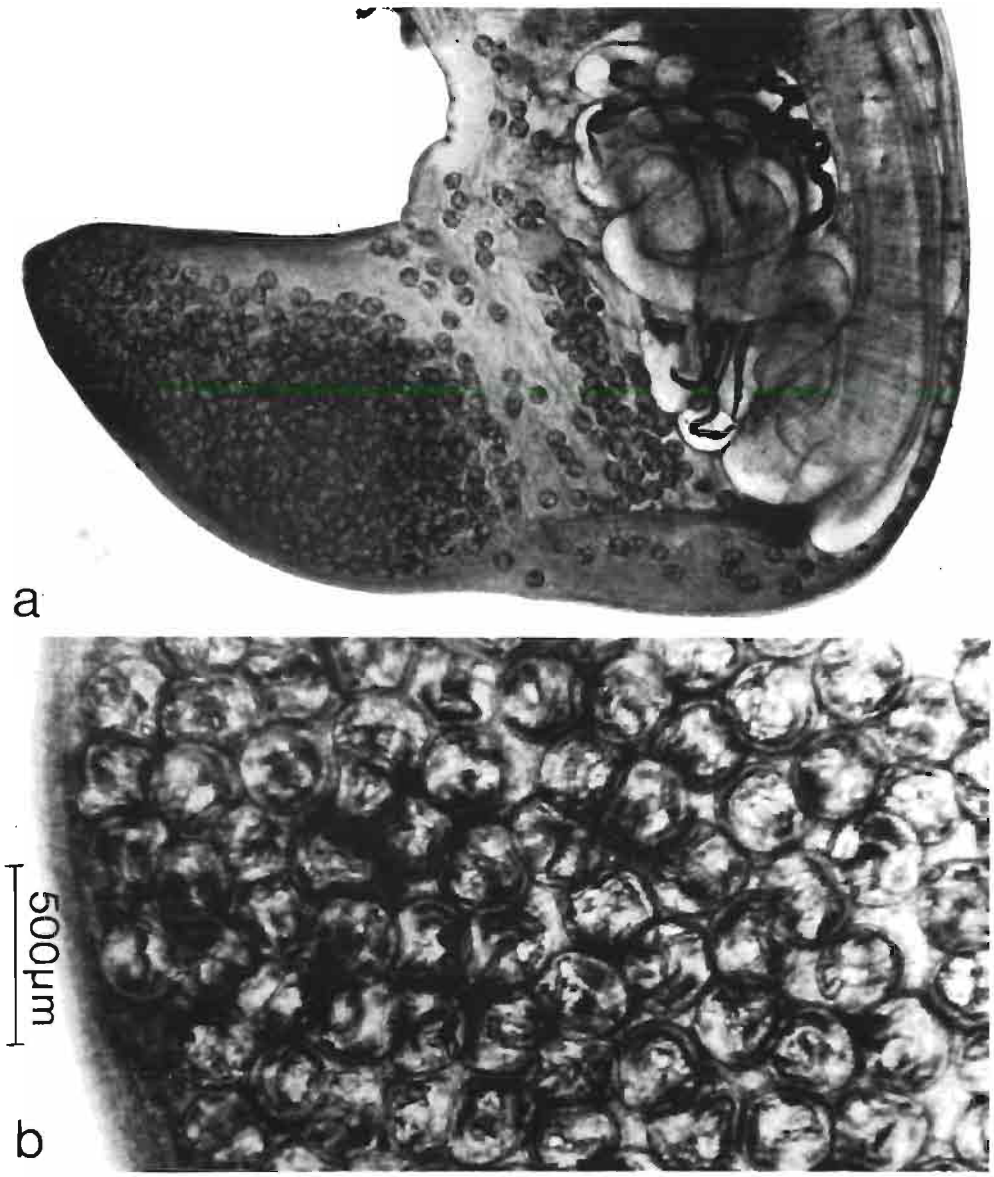


Fig. 13-104: *Cardium edule*. (a) Foot of juvenile individual (15.2 mm shell length) with extremely heavy natural *Himasthla elongata* infestation showing distribution of 195 cysts; (b) enlarged portion of (a). Metacercariae, so densely packed that cysts deform each other, have almost entirely replaced host-muscular tissue. (Original.)

and Werding (1969) even reported positive phototaxis in cercariae of *H. elongata*. The reviewer, on the other hand, never observed a deviation from distinct negative light response in thousands of cercariae of this species, regardless of the experimental conditions.

The pathology caused by *Himasthla* metacercariae may be intense. Host response includes the accumulation of haemocytes and fibrous tissue around the parasite-secreted inner cyst wall. Heavy metacercarial invasion may result in distortion of cells and ducts and hypertrophy of affected organs (Lebour, 1905, 1907c; Palombi, 1925; Cheng and co-authors, 1966a; Fig. 13-104). *H. elongata* seriously interferes with the burrowing activity of *Cardium edule*. When placed on the sediment surface, juvenile cockles with moderate experimental infestations remained uncovered significantly longer than healthy individuals (Fig. 13-105). The median time after which 50 % of 50 infested cockles had taken up their normal burrowed position was 290 min, as opposed by only 12.5 min for the control (for the graphical representation of data of this kind on probability paper see Cassie, 1954, and Henning and Wartmann, 1958). In order to test whether the effect exerted by the metacercariae is mechanical or physiological, or both, another sample of cockles was experimentally infested with *Renicola roscovita,* whose metacercariae invade the palps, mantle and viscera but not the foot. Since in this group there was no deviation of the median burrowing time from that of the control (Fig. 13-105), the effect of *H. elongata* was interpreted as mainly mechanical. The ability of rapid burrowing, as well as the maintenance of a proper position in the sediment, are considered to be of vital importance for cockles living in tidal habitats where they are often washed out of the sediment and carried away by strong currents. Moreover, cockles lying free on the sand surface are more easily detected and eaten by wading birds (Lauckner, 1972). The burrowing behaviour of *C. edule* has previously been studied by Hecht and Matern (1930), Trueman and co-

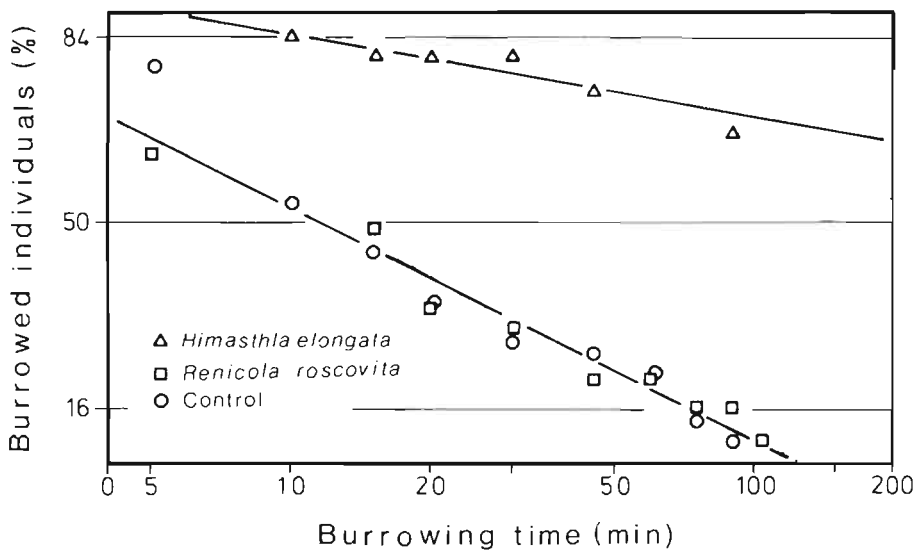


Fig. 13-105: *Cardium edule*. Burrowing activity of healthy and trematode-infested juvenile individuals. Plotted on logarithmic probability paper. (After Lauckner, 1972.)

authors (1966) and Breum (1970), but possible effects of trematode infestation on the digging performance of their test animals have not been considered by these authors.

Mytilus edulis and *Cardium edule* exhibit a distinct response to penetrating *Himasthla elongata* cercariae. In attempts to get rid of the parasites adhering to the body surface, the foot is stripped off between the edges of the valves, which are held closely sealed. This behaviour is frequently successful. Cercariae that had gained access to the mantle cavity sometimes become entangled in mucus and are forcibly expelled through the exhalant siphon. When normally burrowed *C. edule* are exposed to large numbers of *H. elongata* cercariae, they immediately dig their way to the sediment surface where they perform erratic movements in order to cast off the parasites. Lebour (1908b) assumed that the cercariae enter the mussel or cockle by the mouth and bore their way into the soft parts of the host's body. She believed it to be unlikely that the larvae are capable of penetrating the foot directly because the epidermis is very tough. However, as shown experimentally, penetration occurs via the foot epidermis (Lauckner, 1971; Fig. 13-100).

As far as the reviewer was able to determine, no data concerning the longevity of echinostome metacercariae are available in the literature. Living and apparently intact cysts of *Himasthla elongata* have been recovered from the foot musculature of individuals of *Cardium edule* kept in the laboratory for more than 400 days at 4 °C. Most of these metacercariae were found to be surrounded by a brittle brownish 'envelope' consisting of partially calcified conchiolinous material (Fig. 13-106). It is difficult to unterstand as to

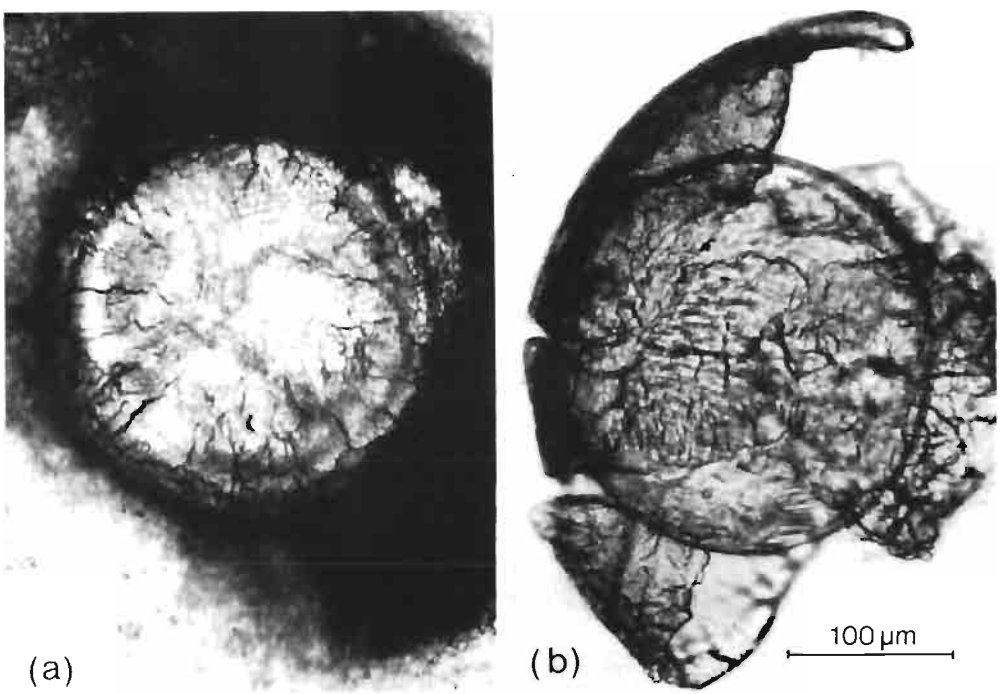


Fig. 13-106: *Himasthla elongata* metacercariae from foot of 4-year-old *Cardium edule*. (a) Brittle 'envelope' of partially calcified conchiolinous material enclosing aged, living cyst; (b) ruptured 'envelope' liberating intact metacercaria. (Original.)

how the enclosed larvae were able to maintain their oxygen and nutrient supply across this massive barrier. Probably, the low temperature and the resultant low metabolic rate were favourable for the long-term survival of the metacercariae. Whether such aged larvae retain their infestivity to the final host has not been determined.

Cardium edule more than 2 years of age sometimes harbour dead *Himasthla elongata* metacercariae. These are most numerous in cockles collected freshly in spring. Preliminary experiments have shown that larval trematodes are generally less resistant to freezing than their molluscan hosts. Probably, the metacercariae had died when the cockle's tissues were partially frozen during cold spells in the preceding winter. Low environmental temperatures may thus remove part of the parasite burden from a host population. Whether the cockles can rid themselves of dead *H. elongata* cysts is not known. Resorption processes

Table 13-19
Mytilus edulis. Effect of parasitization by *Himasthla elongata* metacercariae on byssal thread production in juvenile mussels (Original)

(A) Relation between byssus-thread production and cyst number

Test group	Number of cysts (mean ± standard deviation; n = number of test individuals)	Number of byssus threads produced (n = number of experiments) after 22.5 h	after 77.5 h
(1) Control	0 (n=16)	31.1 ± 16.7 (n=86)	61.2 ± 26.1 (n=19)
(2) Moderate infestation	71.0 ± 45.8 (n=12)	29.9 ± 17.8 (n=108)	62.6 ± 31.7 (n=24)
(3) Heavy infestation	116.1 ± 66.4 (n=14)	21.9 ± 13.6 (n=126)	40.4 ± 20.6 (n=28)

Comparison	after 22.5 h		after 77.5 h	
	t	p	t	p
1—2	not significant		not significant	
2—3	3.93	0.11×10^{-3}	3.04	0.38×10^{-2}
1—3	4.42	0.16×10^{-4}	3.04	0.39×10^{-2}

(B) Relation between byssus-thread production and time
Regression: y = a + bx, where y = number of byssus threads and x = log time

Test group	a	b	n	r
(1) Control	−50.04	60.63	126	0.6588
(2) Moderate infestation	−64.45	70.34	161	0.6301
(3) Heavy infestation	−32.34	46.26	223	0.5317

Comparison of regression coefficients:

	t	p
b_1—b_2	not significant	
b_2—b_3	3.89	0.12×10^{-3}
b_1—b_3	2.76	0.61×10^{-2}

comparable to those characteristic of *Renicola roscovita* cysts (see below) have not been observed. Although the response of littoral marine invertebrates to freezing has been studied repeatedly (Kanwisher, 1955, 1959, 1966; Sömme, 1967; and others), parasitization by larval trematodes has never been considered as a factor possibly affecting their survival.

Once established in the foot musculature of *Cardium edule* in sufficient numbers, *Himasthla elongata* metacercariae may considerably affect the rheotactic behaviour of the cockle. Healthy individuals normally burrow in such a manner that the ingestion siphon is directed toward the incoming water current. As can be deduced from hydrodynamic laws, such a position should maximize the water flow through the mantle cavity and concomitantly minimize the energy required for food collection. In tidal areas with an approximate 180° shift of the current direction in the course of a full tide cycle, cockles should be expected to follow this shift. Field observations indicate that healthy *C. edule* do so, while individuals infested with *H. elongata* are apparently unable to move to any great extent and remain, during the entire tide cycle, with their ingestion siphon directed against the ebb-tide current (Lauckner, 1972).

In *Mytilus edulis*, metacercarial infestation of the foot musculature impairs byssal thread secretion. The production of byssus material has been studied in juvenile mussels (10.5 mm mean shell length), experimentally infested with *Himasthla elongata*. There was a distinct negative correlation between the number of metacercarial cysts in the foot musculature and the number of byssal threads produced. Heavily parasitized mussels secreted highly significantly fewer threads during time intervals of 22.5 and 77.5 h than uninfested controls (Table 13-19; Lauckner, 1978). Under natural conditions, infested mussels having fewer threads than healthy ones may easily become dislodged by surf action or predators. It is also possible that bank formation, which is typical of *M. edulis* living on tidal flats (Maas Geesteranus, 1942), may be impaired to some extent by heavy *H. elongata* infestation.

Byssal thread production by *Mytilus edulis* has been studied repeatedly (Glaus, 1968; Reish and Ayers, 1968; Mahéo, 1970; Van Winkle, 1970; Martella, 1974; Martin and co-authors, 1975; Allen and co-authors, 1976; Carr and Reish, 1978; Price, 1980; and others). Possible effects of metacercarial infestation on byssal activity have not been taken into consideration by these authors.

Young *Mytilus edulis* display a characteristic shell-cleaning behaviour involving the strongly extensible foot as a 'brush' (Theisen, 1972). The presence of large numbers of metacercarial cysts in the musculature considerably reduces the extensibility of the foot, as well as the general locomotory activity of the mussel. Hence, it must be expected that *Himasthla elongata* infestation may impair shell cleaning and thus favour the attachment of fouling organisms to the valves.

Metacercarial infestation may alter the response of bivalves to osmotic and thermal stress. While the thermal resistance of *Cardium edule* infested with *Himasthla elongata* and *Renicola roscovita* is significantly reduced (Fig. 13-107; see below), a more complex picture arises with respect to changes in the osmotic resistance. Juvenile *C. edule* (5 to 9 mm shell length), experimentally infested with *H. elongata* metacercariae, were kept unfed in sea water of 30 ‰ S and 15 ‰ S at 22 °C. In 30 ‰ S, survival of the experimentally infested cockles (Fig. 13-108; in the range of the median ± 1 standard deviation = 50 ± 34 %) was reduced as compared with the uninfested controls, but

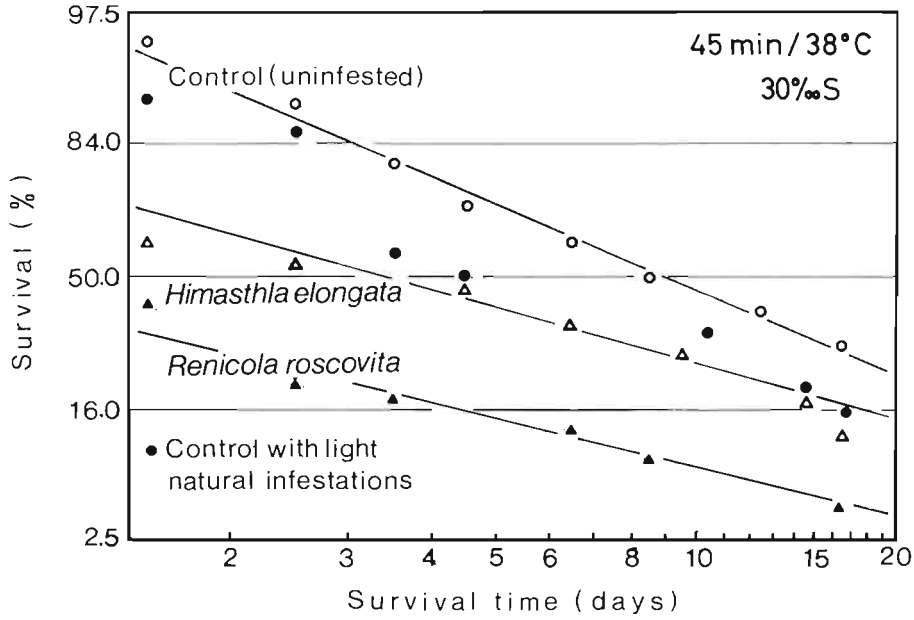


Fig. 13-107: *Cardium edule*. Survival of healthy and trematode-infested juvenile individuals after exposure to thermal stress. Plotted on logarithmic probability paper. (After Lauckner, 1972.)

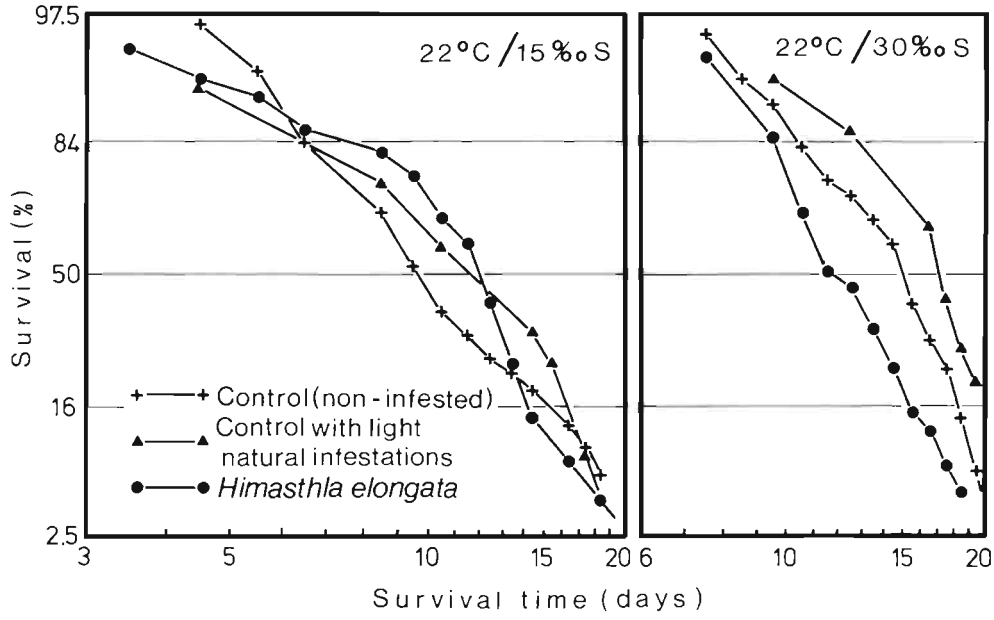


Fig. 13-108: *Cardium edule*. Survival of healthy and trematode-infested juvenile individuals under different temperature-salinity conditions. Plotted on logarithmic probability paper. (After Lauckner, 1972.)

individuals carrying light natural infestations (1 to 4 metacercariae of *H. elongata* and *R. roscovita* per cockle) lived even slightly longer than the uninfested controls. In 15 ‰ S, the difference was not as pronounced, but the experimentally as well as the naturally infested individuals lived slightly longer than the controls. The results are difficult to interpret. It appears likely that 2 antagonistic mechanisms are acting simultaneously: Massive metacercarial invasion causing displacement of organs, disruption of tissues, occlusion of ducts and blood sinuses, etc. is clearly detrimental and reduces host survival. Light metacercarial infestation, on the other hand, appears to improve the cockle's resistance to osmotic stress, possibly through the action of some unknown biochemical mechanism.

Improvement in resistance to environmental stress, caused by larval trematodes, has been reported for mud-flat snails *Nassarius obsoletus* (Riel, 1975; Vol. I, Chapter 12). Such improvement may be due to an increase in the level of tissue-ionic calcium (Schlieper and Kowalski, 1956a, b). Increased levels of ionic calcium have been detected in snails *Nitocris dilatatus* parasitized by larval trematodes *Prosthodendrium* (*Acanthatrium*) *anaplocami*. Each gram of soft tissue of 7 infested snails contained significantly more calcium (0.570 ± 0.170 mg) than that of 45 healthy snails (0.409 ± 0.242 mg) (Cheng and co-authors, 1966c). Metacercariae of *Renicola roscovita* — and, to a lesser extent, those of *Himasthla elongata* — concentrate calcium in their excretory system. Whether this accumulation has any effect on the calcium concentration in the tissues of the host remains unknown.

As stated, oysters have been found to be refractory to infestation by *Himasthla* spp. Another echinostome, *Acanthoparyphium spinulosum,* may occasionally encyst in these bivalves. Fifty individuals of *Crassostrea virginica* from Port Isabel, Texas (USA), inspected for larval trematodes, were found to be nearly 100 % infested with an average of 45 larvae per oyster embedded along the mantle margin. The adult of *A. spinulosum*, which parasitizes in the intestine of shore birds, has been reported from California, Japan and Australia. Apparently, the infestivity of the metacercariae to the final host decreased with time since the recovery rate of fully developed worms obtained from experimentally infested day-old chicks dropped from 26.2 % for freshly collected oysters to 4.5 % for oysters kept in the laboratory for 2 months. Dead and deteriorating metacercariae were seen along with healthy larvae in the mantle of *C. virginica* (Little and co-authors, 1966, 1969). This is the first report of a larval echinostome in oysters and, apparently, the extreme southern tip of Texas is the only place in the United States where such infestations occur. The source of infestation has not been determined, but in California *Cerithidea californica*, in Australia *Pyrazus* (*Velacumantus*) *australis,* and in Japan *Batillaria multiformis* have been identified as first and second intermediate hosts of *Acanthoparyphium* sp. (Yamaguti, 1934; Bearup, 1960; Martin and Adams, 1961).

The rapid decrease in infestivity of metacercariae encysted in laboratory-held oysters may indicate that *Crassostrea virginica* is not a normal intermediate host for *Acanthoparyphium spinulosum*. However, the possible importance of such infestations for shellfish consumers must be taken into consideration. Preliminary experiments, conducted by Little and co-authors (1966), indicate that *A. spinulosum* can survive in laboratory rats. Echinostomes generally are infestive to a wide range of bird and mammalian hosts, and human infestations are possibly limited only by the rarity with which bivalves harbouring metacercariae are eaten raw. At least one case of gastrointestinal disturbance in a human patient, caused by the ingestion of (probably infested) raw quahaugs *Mercenaria mer-*

cenaria, has been documented in the literature. Sexually mature worms, obtained from the stool of that patient, were identified as a new species, *Himasthla muehlensi* (Vogel, 1933). Stunkard (1938) considered it to be possibly identical with *H. quissetensis*. Similar to *Bucephalus* sp. in *Crassostrea virginica* (p. 653), infestation of *M. mercenaria* with *H. quissetensis* may cause an accumulation of toxic short-chained fatty acids within the host's tissues. Consumption of large numbers of such contaminated quahaugs may, therefore, produce temporary gastroenteritic disturbances in humans (Cheng, 1965b). Loos-Frank (1967), in an attempt to test the infestivity of *H. continua* and *H. interrupta* to humans, swallowed several portions of raw *Mytilus edulis* and *Cardium edule* containing metacercariae. No ill effects were noted and the examination of stools for worms was negative.

Digenea utilizing birds as final hosts: families Psilostomatidae and Renicolidae

Mytilus edulis, Mya arenaria, Cardium edule and several other bivalves have been reported as second intermediate hosts for trematodes of the families Psilostomatidae and Renicolidae. The metacercariae of *Psilostomum brevicolle* (Fig. 13-99, a), which measure from 200 to 230 μm in diameter, encyst in the digestive gland of *M. edulis, C. edule* and *C. lamarcki* from the North and Baltic Seas. Lebour (1912), who first described it as *Cercaria mytili*, found it to be common in mussels and cockles from Northumberland and Scotland, and Reimer (1964) reported it from 4 of 501 *M. edulis* and 20 of 486 *'C. edule'* (probably *C. lamarcki*) from Hiddensee (Baltic Sea). The life cycle of *P. brevicolle* includes *Hydrobia ulvae* as first intermediate and various shore birds as final hosts (Loos-Frank, 1968). Reimer (1964) mistook gigantocercous cercariae from Baltic Sea *H. ventrosa* for those of *P. brevicolle*.

Metacercariae of *Renicola thaidus* occur in the gills, walls of the suprabranchial chambers, mantle and foot of *Mytilus edulis, Argopecten irradians* and *Gemma gemma* from Woods Hole (Massachusetts, USA). The first intermediate host is *Thais lapillus*, and the adult parasitizes in the kidneys of birds. The metacercariae, which are 120 to 160 μm in diameter, do not grow in the second intermediate host. *Mya arenaria* and *Crassostrea virginica* have been found to be refractory to attack by this species (Stunkard, 1964b). *Littorina* spp. from Woods Hole harbour larval renicolids *Cercaria parvicaudata* (Stunkard and Shaw, 1931; Stunkard, 1950, 1964b, 1970a). The species is morphologically indistinguishable (but probably specifically different) from *Renicola roscovita*, reported from the same first intermediate hosts in Europe (Stunkard, 1932, 1950; James, 1968a; Werding, 1969). Preliminary experiments (which require further confirmation) indicate that metacercariae of *C. parvicaudata* encyst mainly in the foot and less frequently in other organs of *M. edulis* (Lauckner, unpublished), whereas *R. roscovita* prefers (in descending order) the palps, mantle margin and visceral mass of *M. edulis, C. edule, C. lamarcki* and *Mya arenaria* as primary site for encystment, and only rarely invades the foot (Fig. 13-109; Table 13-17; Lauckner, 1971). Whether this apparent microhabitat segregation of *R. roscovita* and *Cercaria parvicaudata* in the second intermediate host might be indicative of differences at the species level, remains to be studied. Werding (1969) achieved experimental development of *R. roscovita* in the renal tubules of gulls *Larus argentatus*, and natural infestations with adults of this species are found in almost every gull from the North Frisian coast (Lauckner, unpublished). Stunkard (1971), on the other hand, fed

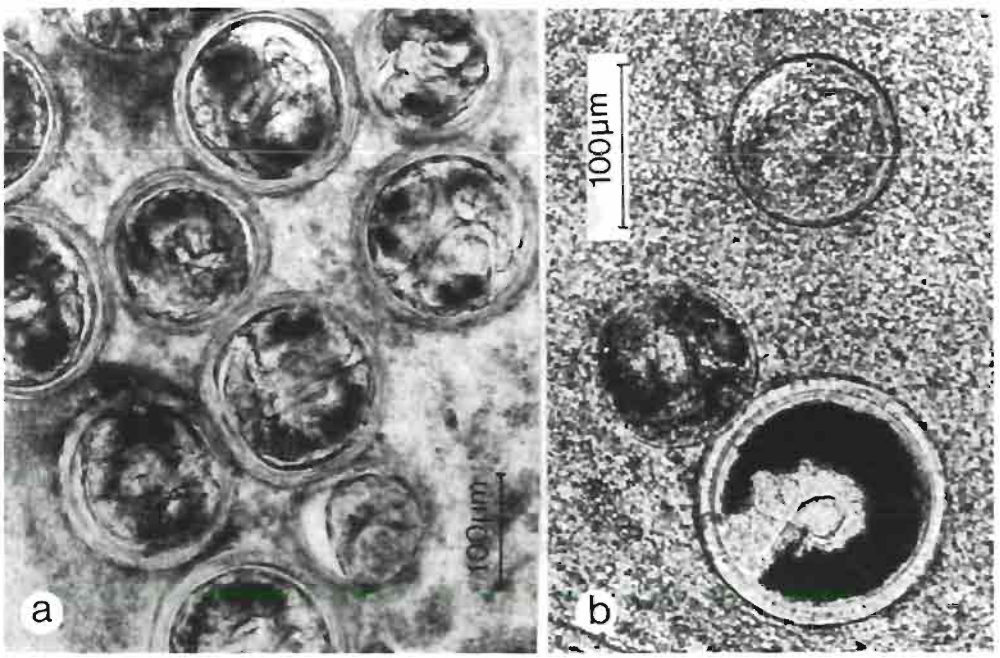


Fig. 13-109: *Renicola roscovita* metacercariae from *Cardium edule*. (a) Palp tissue 'paved' with fully grown cysts; (b) one intact metacercaria and 2 dead cysts in different stages of resorption. (Original.)

thousands of *C. parvicaudata* metacercariae to newly hatched *L. argentatus* and several other species of sea birds over extended periods of time. Adult renicolid worms were recovered from none of these.

The location of *Renicola roscovita* metacercariae in *Mytilus edulis*, *Cardium edule*, *C. lamarcki* and *Mya arenaria* is largely determined by the size of the host and the space available for encystment. Very young cockles ('autumn spat' in Table 13-20; mean shell length 6.86 mm) have tiny palps offering little space for the accomodation of greater numbers of cysts. Therefore, a larger proportion of larvae settle in the mantle margin and the visceral mass. As the hosts grow, the relative abundance changes in favour of the palps

Table 13-20
Cardium edule. Intensity of trematode infestation in 0-group individuals (Original)

	Summer spat	Autumn spat
Number of hosts inspected	23	24
Host-shell length (mm; mean ± standard deviation)	15.55 ± 2.05	6.86 ± 2.42
Total number of cysts recovered	8,592	316
Number of cyst hosts^{-1} (mean ± standard deviation)	373.6 ± 100.0	13.2 ± 9.9
Percentage of parasites involved		
Himasthla elongata	52.7	10.4
Himasthla interrupta	2.2	31.3
Renicola roscovita	45.1	57.3
Himasthla continua and *Psilostomum brevicolle*	0	1.0

('summer spat' in Table 13-20; mean shell length 15.55 mm). In larger mussels and cockles, the labial palps may become so densely 'paved' with *R. roscovita* metacercariae that their proper function as a transport and sorting device for food particles appears to be seriously impaired (Fig. 13-109). Metacercariae of *R. roscovita* and *Himasthla interrupta*, encysting in large numbers in the mantle margin of juvenile cockles (Table 13-17), interfere with the function of the mantle margin in disrupting or displacing pallial muscles. Heavily infested individuals are unable to maintain proper valve closure, thus facilitating entry of pathogens, small predators such as turbellarians, and detritus into the mantle cavity.

Sometimes — mainly in hosts collected in spring — dead and disintegrating *Renicola roscovita* metacercariae may be encountered in the tissues of the palps. Within these cysts, the metacercarial structures gradually fade and the cysts decrease in size (Fig. 13-109, b). Death of the larvae does not appear to result from the action of the host's internal defense mechanisms; it may rather be due to freezing (see also p. 705, *Himasthla elongata*).

Infestation with metacercariae of *Renicola roscovita* and *Himasthla elongata* has been shown to affect the resistance of bivalves to thermal stress. In an experiment, juvenile *Cardium edule* of 5 to 7 mm shell length were infested with cercariae of both species and subsequently exposed to a temperature of 38 °C for 45 min in sea water of 30 ‰ S. Fifty individuals were used in each experimental batch and 100 in the control. Sixty percent of the cockles harbouring *H. elongata*, but only 42 % of the individuals infested with *R. roscovita*, survived the exposure. Upon dissection, about 20 % of the cockles used as controls revealed light natural infestations with a few cysts of both species. Survival in this subgroup was 90 % as opposed by 96 % in the uninfested control. In order to trace after-effects ('delayed death') of the heat stress, the animals were kept unfed for a period of another 17 days at 22 °C, during which mortality was recorded (Fig. 13-107). By the end of this period, the respective survival figures in the 4 groups were as follows: Uninfested control – 30 %, control with light natural infestations – 15 %, *H. elongata* experimental infestations – 11 %, and *R. roscovita* experimental infestations – 4 %. The more deleterious effect of *R. roscovita* is believed to be due, at least in part, to a higher metabolic activity of the metacercariae of this species. Although *H. elongata* cysts are considerably larger (about 246 µm in mean diameter), those of *R. roscovita* grow within the second intermediate host (from about 113 µm initial size to approximately 170 µm final size) and, therefore, probably absorb more nutrients from the cockle's tissues than the non-growing *H. elongata* metacercariae. It is likewise possible that the physiological response of the cockle to physical changes in the environment is modified differentially by the presence of the two larval trematodes. Such a situation has been reported for other mollusc-trematode associations by Vernberg (1969).

The incidence as well as the intensity of infestation of *Cardium edule* and *Mytilus edulis* with *Renicola roscovita* metacercariae exhibit considerable spatial variation, which is strongly correlated with the infestation pattern in the first intermediate host, *Littorina littorea*. Obviously, *R. roscovita* prefers areas of higher exposure than *H. elongata*. Since the adults of both species usually occur in the same definite host individuals, mainly *Larus argentatus*, this situation is somewhat puzzling. It probably reflects differences in the life-cycle pattern of both parasites. In *R. roscovita* the egg has to be swallowed by the first intermediate host, and the miracidium hatches in the periwinkle's intestine, while in *H. elongata* the miracidium hatches in the open water and must locate and actively invade

a snail. Fjälling and co-authors (1980) found large numbers of *R. roscovita* metacercariae in 95 % of wild *M. edulis* from an exposed locality at Tjärnö Island (west coast of Sweden), whereas only 3 % of cultured mussels from a sheltered locality of that island were infested. Bowers (1969) observed a strong correlation between the increase in the percentage infestation by *H. elongata* metacercariae in *C. edule* and the population density of *L. littorea*.

Wherever *Littorina littorea* from the North Sea have been inspected for larval trematodes, more or less intense infestations with *Himasthla elongata* and *Renicola roscovita* have been recorded. One can state with certainty that the mussels and cockles from the same areas must necessarily be infested to a varying extent with the metacercariae of these species (Figs 13-102 and 13-103). In fact, their presence in these hosts has been documented by parasitologists, but has constantly been ignored by ecologists and malacologists. Growth, mortality, population dynamics and general ecology of *Cardium edule* and *Mytilus edulis* have been studied repeatedly, but the early workers (Orton, 1926; Stephen, 1931, 1932; Thamdrup, 1935; Bunting and co-authors, 1936; Wohlenberg, 1937; Linke, 1939; but also Cole, 1956b, and others) entirely disregarded the problem of trematode infestation. Orton (1933, 1934) mentioned the occurrence of parasites in *C. edule* undergoing heavy summer mortality on some British cockle beds, but he believed that parasitization was not important enough to account for the observed heavy losses. Kreger (1940), Kristensen (1957) and Theisen (1968) considered parasites and diseases as possible factors contributing to the mortality of cockles and mussels, but studies on this subject were not conducted. Smidt (1944) was clearly aware of the possible role of larval trematode infestation. He stated (p. 18/19):

> "The decrease of the older *Cardium* was so evident that it is a problem whether it depended on the food competition only. The possibility of this being due to an infection of trematodes must be considered but as no observations about this are available, this question cannot be answered. It must be mentioned, however, that in 1936 nearly the whole *Cardium* stock on Rønnerne at Frederikshavn (Kattegat) was infected with trematodes, and as the Skalling tidal area is a distinct wading bird locality, the possibility of an infection of trematodes is great here."

In fact, Lauckner (unpubl.) found up to 42.6 % of the *Littorina littorea* from tidal flats between the Skallingen peninsula and the Danish mainland to be infested with *H. elongata*. Incidences of that magnitude have hitherto not been reported in the literature. Consequently, *H. elongata* must be considered as an important factor affecting — and possibly controlling — population size and survival of *C. edule* in that area. Regrettably, in his later work, Smidt (1951) made no further mention of the trematode problem.

Conditions similar to those prevailing on Danish North Sea tidal flats are also characteristic of the Dutch Wadden Sea. In his studies on the ecology of *Cardium edule* from that area, Kristensen (1957, p. 49) admitted that "it is quite possible that parasites are an important factor of death but no sufficient data are available". He further stated (p. 50): "In the western Wadden Sea we admittedly never came across an infection by trematodes; but our observations on this point are only limited to a few." It must be concluded that Kristensen (1957), as well as Kreger (1940), who worked in the same area, have simply overlooked existing metacercarial infestations. Dietvorst (1972) reported

Himasthla elongata from 3.8 % of the *Littorina littorea,* and *Renicola roscovita* from 5.8 % of the *L. littorea* and 9.2 % of the *L. saxatilis* in the western Wadden Sea. Hence, the metacercariae must be expected to occur in appreciable numbers in bivalves from that area.

From the available information it must be concluded that, on North Sea tidal flats, *Himasthla elongata* and *Renicola roscovita* are the two most abundant and most important larval trematodes utilizing bivalves as second intermediate hosts. There is evidence that the cockle populations on the tidal flats of Sylt (German North Sea coast) are largely controlled by these parasites. The onset of emission of cercariae by infested *Littorina littorea* occurs in late May to early June and coincides with the settling of the young bottom stages of *Cardium edule* and other bivalve species on the tidal flats (Fig. 13-110). Cercarial invasion starts immediately after the spat has settled. Preliminary field studies, conducted by the reviewer, indicate that attacks by single cercariae of these species are lethal for bottom stages less than about 2 mm in shell length. From a length of about 5 to 7 mm on, cockles from the tidal flats at Sylt are almost 100 % infested, and intensities increase rapidly with age. In October, when cercarial production has ceased, individuals of 6.86 mm mean shell length ('autumn spat', i.e., members of a second spatfall settled in late summer or early autumn) were found to harbour an average of 13. 2 cysts, while 'summer spat' individuals, about 15.55 mm in length , contained, on the average, as many as 373.6 metacercariae. *H. elongata, H. interrupta* and *R. roscovita* make up over 97 % of the infestations (Tables 13-17 and 13-20). In addition to the total number, the relative frequencies of cysts of the 3 parasite species vary between 'summer' and 'autumn' hosts: While the relation of *H. elongata : H. interrupta : R. roscovita* is 24.3 : 1 : 20.9 in the former, it is 0.3 : 1 : 1.8 in the latter group. This reflects the differential maturation of the 3 larval trematodes in their first intermediate hosts in relation to environmental temperature (and, possibly, the physiological state of the snail host). Three- to 4-year-old cockles usually contain hundreds to thousands of metacercariae.

During the first summer in a cockle's life, growth is rapid but coincides precisely with the period of maximum cercarial attack (Fig. 13-111). Individuals settled in early summer may become so heavily infested that they are unable to survive and gradually disappear from the population until the end of the warm season. In the survivors, a significant negative correlation between shell length and the number of metacercariae is apparent.

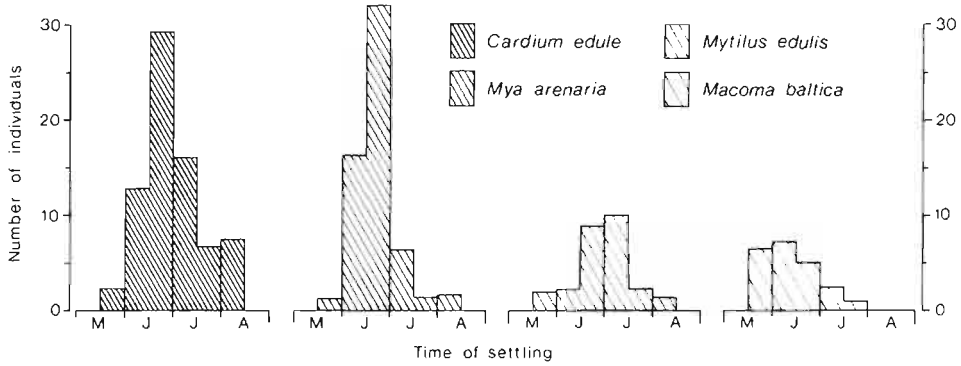


Fig. 13-110: Time of settling and average number of spat of several bivalve species on 20 dm^2 sediment area in the Dutch Wadden Sea in 1950. (After Baggerman, 1953.)

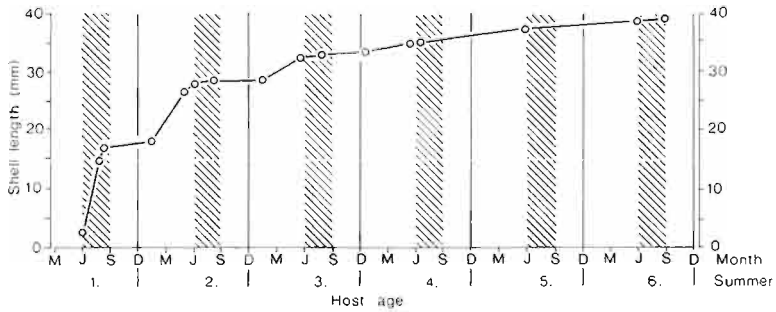


Fig. 13-111: *Cardium edule*. Relation between age, growth and major periods of cercarial attack (= crosshatched areas). (Based on Kristensen, 1957.)

This suggests that a heavy trematode burden may interfere with growth in juvenile bivalves. Most of the more heavily infested 0-group individuals die during their first winter; juveniles inspected in the following spring yield considerably lower numbers of metacercariae.

In some years, a second major spatfall of *Cardium edule* occurs in late summer or early autumn. By the end of the year, these animals ('autumn spat' in Tables 13-17 and 13-20) usually carry considerably lighter metacercarial burdens than individuals settled earlier in the summer and, consequently, have a better chance of survival. In the spring of the second year, maximum growth occurs prior to the onset of cercarial invasion (Fig. 13-111). This has some advantage for the host, since larger cockles can support larger numbers of metacercariae without being debilitated excessively. From the third year of life on, however, host growth cannot keep pace with the continuous 'bombardment' by larval trematodes. Three- to 4-year-old cockles accumulate hundreds to thousands of metacercariae and eventually become so debilitated that death ensues. Poor growth and reduced longevity of heavily infested cockles may well (although falsely) be attributed to 'unfavourable ecological conditions'. Kristensen (1957, p. 32) was aware "of the fact that mortality increases as the cockles grow more slowly", but was unable to offer an explanation for this observation. The major reason for the field biologists' total unawareness of the 'trematode problem' is probably due to the fact that these parasites typically cause a slow but constant attrition in numbers of adult hosts rather than spectacular epizootic mortalities. It is frustating to realize that even in the most recent publications on the ecology of *C. edule* (e.g., Hancock and Urquhart, 1965; Hancock, 1970; Franklin and Pickett, 1979) the effects of metacercarial invasion on growth and longevity of the host are either notoriously ignored or have remained undiscovered.

Digenea utilizing birds as final hosts: family Gymnophallidae

The Gymnophallidae represent another digenean family whose larval stages occur in bivalves. Gymnophallid metacercariae are ubiquitous in coastal marine environments, particularly in higher geographic latitudes, and affect a variety of commercially important molluscan species.

Although morphologically very similar — and virtually almost indistinguishable by the unexperienced observer — gymnophallid metacercariae belonging to different species

may occupy widely divergent microhabitats in their bivalve hosts. Sometimes these microhabitats may be so well circumscribed, and the host specificity so pronounced, that it is possible to identify the larval flatworms from the host species and the location within the host occupied by the metacercariae. In spite of their close morphological similarity, metacercariae of different species may elicit a wide range of host responses, ranging from virtually nil to severe pathology.

As already stated, many workers have failed to distinguish between cercariae and metacercariae (which, in the Gymnophallidae, are always unencysted), and regarded the latter as 'tailless cercariae' or 'cercariaea'. Unfortunately, the further life-cycle stages are known of only a few gymnophallid metacercariae. This gap in our knowledge precludes the development of effective measures of parasite-host contact avoidance in cultivated marine bivalves or measures of biological control in the field.

The ecology of larval gymnophallids is even less well known. Distribution and abundance of their metacercariae seem to be controlled by subtle differences in ecological factors. Therefore, gymnophallids may be serious metazoan parasites of bivalves in one area, but of no importance in others. An extreme case of interference of larval gymnophallids with commercial shellfisheries has been mentioned by Dubois (1901a). On some French mussel beds, *Mytilus edulis* was so heavily invaded by a pearl-producing gymnophallid that the mussels were rendered unsuitable for human consumption (see below).

Although there are numerous literature records of the occurrence of gymnophallid metacercariae in bivalves, there are remarkably few quantitative data on infestation intensities. From the available information, however, it becomes apparent that gymnophallid counts in these hosts may by far exceed those of all other species of metacercariae together, and may increase considerably — and sometimes dramatically — with host age and size. Obviously depending on the respective parasite-host system, as well as on physico-ecological factors, the increase in numbers of larvae may be more or less linear to host size (Fig. 13-120), or may be exponential (Fig. 13-112).

Most of the descriptions of gymnophallid metacercariae are so meagre with respect to the presentation of distinguishing morphological features and, in particular, of morphometric data, that reliable specific identification is difficult or even impossible. In many cases, merely the range of body dimensions has been specified. The uselessness of such data, especially in view of the constantly changing shape of living gymnophallid metacercariae, may be illustrated by the following example:

Let the specified range of length × width measurements of a hypothetical species of metacercaria be 100-150 × 50-75 μm. Does that mean that the smallest larva measures 100 × 50 μm and the largest 150 × 75 μm? Such a difference in body size, which corresponds to a 2.25-fold increase in projection area (Fig. 13-113, 1 and 2), appears very unlikely. Or are the measurements negatively correlated, i.e., is the shortest metacercaria also the widest one? Such a correlation (Fig. 13-113, 3 and 4), which is well in accordance with the changes in body shape observable in gymnophallids, appears more likely. However, unless this relation is not specified by a correlation coefficient obtained by statistical treatment of the raw data, exact morphometric comparisons cannot be made.

Another nuisance commonly encountered in trematodology is the presentation of measurements which have been obtained 'under slight cover-glass pressure'. What exactly is 'slight' and what is a 'little bit more than slight'?! The disadvantage of this method is illustrated in Fig. 13-114. Let 'a' and 'b' be 2 metacercarial cysts. In '1' the cover glass just touches the larger cyst ('b') but exerts no pressure upon it. The cyst dimensions are not distorted. In '2' the cover glass – now under slight pressure – touches cyst 'a' but without deforming it. Cyst 'b', on the other hand, is flattened under the pressure of the glass. Its 'apparent diameter' has increased by the increment 'x'. It is obvious that

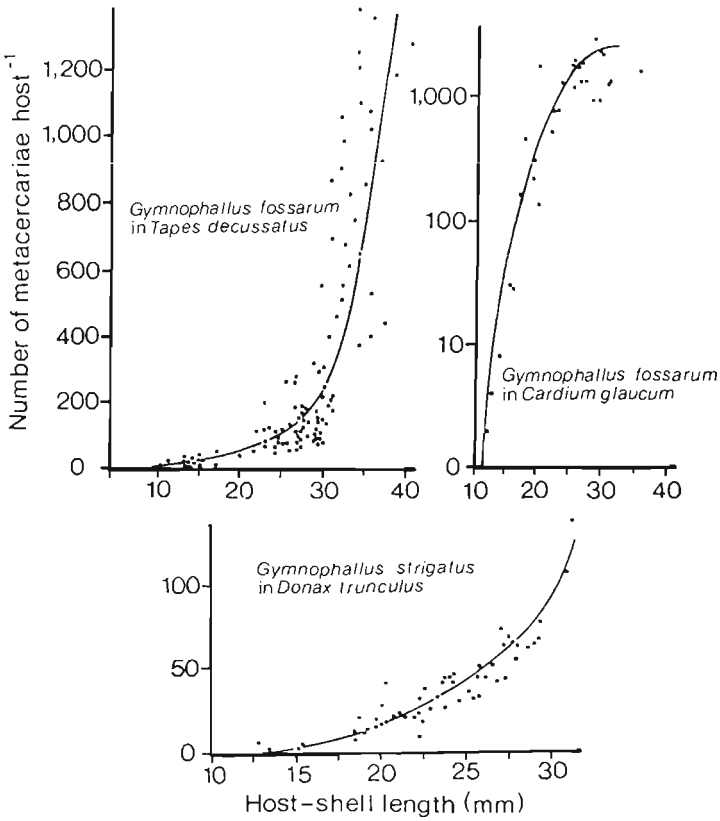


Fig. 13-112: Gymnophallid metacercariae in bivalves. Increase in infestation intensity with host size. (After Bartoli, 1974a.)

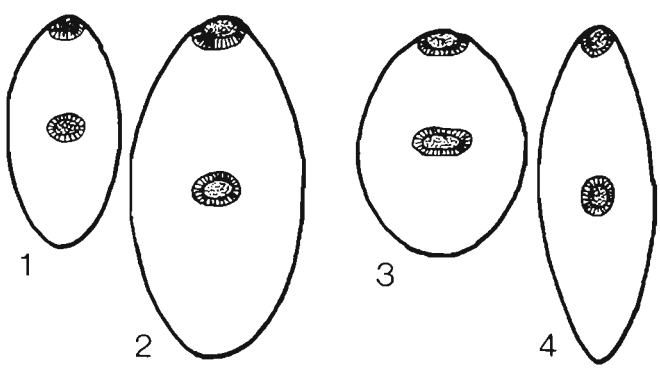


Fig. 13-113: Different interpretations of 'range of body dimensions' of metacercariae. For explanation see text. (Original.)

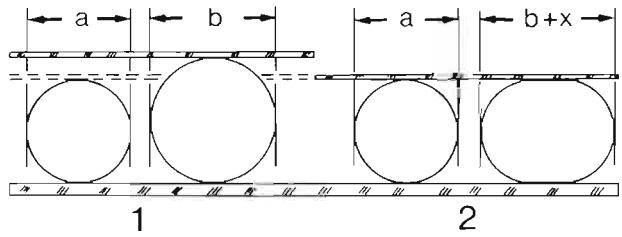


Fig. 13-114: Distortion of body dimensions by 'slight cover-glass pressure'. For explanation see text. (Original.)

measurements obtained under such conditions are entirely worthless. In order to avoid further increase in confusion, body dimensions of gymnophallid metacercariae given in the literature will not be specified below.

Donax vittatus, D. trunculus and *D. semistriatus* from the European Atlantic and Mediterranean coasts are frequently parasitized by the metacercariae of *Gymnophallus strigatus,* which occur either free in the extrapallial space, i.e., between mantle and shell, or become enveloped by proliferations of the mantle tissue. Both 'micro-ecotypes' are so different in aspect that they could be taken for larvae of two different trematode species (Fig. 13-115). Metacercariae from the extrapallial space are relatively transparent, while 'enveloped' ones have greatly distended excretory vesicles filled with large quantities of strongly refractive granules, which appear black in transmitted light. They are also smaller than the former (Bartoli, 1974a). The metacercariae parasitize a considerable number of bivalve species (Table 13-21). In hosts with thin, translucent valves, such as *Tellina tenuis* and *Angulus fabula,* aggregations of metacercariae are readily discernible from the outside due to the accumulation of yellowish conchiolinous material in the mantle portion surrounding the larvae (Giard, 1897b).

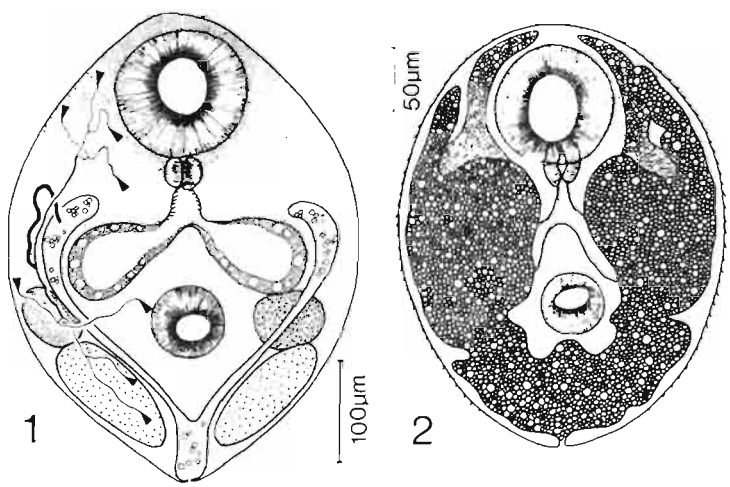


Fig. 13-115: *Gymnophallus strigatus* from *Donax trunculus.* 1: Metacercaria occurring free in extrapallial space; 2: metacercaria enveloped by mantle tissue. Ventral aspect. (After Bartoli, 1974a.)

Table 13-21
Gymnophallidae. Bivalve-invading species (Type-I life history pattern) and their natural secondary hosts in Camargue, France (After Bartoli, 1974a)

Gymnophallus rebecqui	*Cardium glaucum* *Abra ovata*
Parvatrema duboisi	*Mytilus galloprovincialis* *Brachidontes minimus* *Venus gallina*
Gymnophallus strigatus	*Donax trunculus* *Donax semistriatus* *Lentidium mediterraneum* *Solen marginatus* *Ensis ensis* *Pharus legumen* *Spisula subtruncata* *Loripes lacteus*
Gymnophallus fossarum	*Cardium glaucum* *Cardium tuberculatum* *Tapes decussatus* *Tapes aureus* *Tapes rhomboides* *Spisula subtruncata* *Solen marginatus* *Ensis ensis* *Pharus legumen*
Gymnophallus rostratus	*Loripes lacteus* *Venus gallina* *Tapes aureus* *Tapes decussatus* *Tapes rhomboides* *Divaricella divaricata* *Dosinia lupinus* *Solen marginatus* *Ensis ensis* *Donax trunculus* *Donax semistriatus* *Tellina tenuis* *Mactra corallina* *Abra ovata* *Scrobicularia plana* *Spisula subtruncata*

Incidence and intensity of *Gymnophallus strigatus* infestations can be high. Giard (1897b) and Dollfus (1912) found the metacercariae in large numbers in almost every individual of *Donax vittatus* from Boulogne-sur-Mer (France). Metacercariae in *Angulus* (*Tellina*) *fabula, T. tenuis* and *T. solidula* (= *Macoma baltica*), referred to *Brachycoelium* sp. (= *G. strigatus*) by Giard (1897b), supposedly include one or more separate species. In heavily parasitized clams, the inner surface of the valves may be covered with calcareous crests and ridges, deposited in response to hundreds of dead metacercariae. Such affected shells are frequently grossly deformed. In British waters, *G. strigatus* occurs commonly in

T. tenuis but rarely in *D. vittatus,* each of about 20 specimens of the former and only 1 out of a dozen specimens of the latter species, taken at Alnmouth, Northumberland, having been found infested (Lebour, 1908a, 1912). G. Rees (1939) recovered the metacercaria from 105 of 150 *T. tenuis* at Millport, Scotland. Her claim to have found "thin-walled, colourless, oval sporocysts of *G. strigatus* containing cercariae in various stages of development deep down in the digestive gland" of *Cardium edule,* is a false report. Miss Rees, who believed that gymnophallid cercariae are tailless, either saw sporocysts of *G. choledochus* containing metacercariae, or metacercariae of *Meiogymnophallus minutus* enclosed by host tissue. In the Mediterranean, *T. tenuis* has been identified as first intermediate host of *G. strigatus.* As shown experimentally, *T. tenuis* does not act as secondary host in this area. Similarly, *G. strigatus* cercariae do not penetrate Mediterranean *Cardium glaucum* (Bartoli, 1974a). Hence, Cole's (1956a) record of *Cercaria strigata* from British *C. edule* is probably also erroneous. In spite of numerous laboratoy trials to experimentally infest various sea birds with the metacercaria, the adult stage of *G. strigatus* is still unknown. Giard (1907) believed it to be identical with *G. somateriae* (Levinsen, 1881) Odhner, 1900b, but Lebour (1908a) pointed out that the adult of *G. somateriae* is smaller than the metacercaria of *G. strigatus.* Nevertheless, Dollfus (1912) named the metacercaria from *Donax vittatus G. somateriae* var. *strigatus.* But Ching (1973a) identified a metacercaria from *Macoma inconspicua* at Spanish Banks, Vancouver (Canada), as the larva of *G. somateriae.* It differed in several respects from those described by the above authors. In additon, Ching (1973b) found adult worms, recovered by Odhner (1900b, 1906) from the caecum of *Somateria mollissima* and ascribed to *G. somateriae,* to disagree with Levinsen's (1881) original description, and redescribed Odhner's specimens as *Paragymnophallus odhneri.* Levinsen (1881) tentatively identified *Hiatella* (*Saxicava*) *rugosa* from Greenland as metacercarial host for *G. somateriae.*

Gymnophallid metacercariae, situated between the mantle and shell of *Mytilus edulis,* are known for a long time as 'pearl trematodes'. As early as 1655, Olaus Worm in his 'Museum Wormiamum' witnessed the occurrence of pearls in mussels from Roskilde Fjord, Denmark. Garner (1873) recognized small distomes between mantle and shell as the causative agents of pearl formation in *M. edulis* in Britain. The discovery of pearls in mussels from Billiers on the French Atlantic coast by D'Hamonville (1894) initiated a series of studies on the phenomenon of 'margarosis' and its causes. Without giving a description of the larval worm, Dubois (1901a, b) namend it *Distomum margaritarum.* Jameson (1902) and Giard (1907) believed it to be the larval stage of *Lecithodendrium* (= *Gymnophallus*) *somateriae* but, according to Odhner (1906), it is more likely to represent *G. bursicola,* a species described by him (Odhner, 1900b) from the Bursa fabricii of Eider ducks *Somateria mollissima* in Sweden. On the basis of experimental studies, Stunkard and Uzmann (1958) tentatively identified metacercariae from *M. edulis,* taken on the North American Atlantic coast, as those of *G. bursicola.* Selikman (1962) found metacercariae, probably identical with those reported above, in White Sea mussels, but erroneously misidentified these as larval *G. choledochus.*

In his extensive studies on pearl formation in *Mytilus edulis,* Jameson (1902, 1903) introduced several gross errors, which have persisted in the literature for considerable time. He believed that 'tailless cercariae', developing in 'sporocysts' in *Cardium edule* and *Tapes decussatus,* attack the mussel and become the 'pearl trematode'. However, the 'cercariae' were actually metacercariae enclosed by host tissue, those in *C. edule* repre-

senting *Meiogymnophallus minutus* and those in *T. decussatus* probably *G. fossarum*. Both have nothing to do with the 'pearl trematode' in the mussel. Jameson further claimed to have produced experimental infestations in mussels with 'cercariae' from the cockle and the clam. No doubt his mussels had been infested previously. Furthermore, Jameson's relegation of the larva from *M. edulis* to an adult worm, obtained experimentally from *Melanitta nigra* and believed to represent *G. somateriae*, is erroneous since the adult is only about one-half the size of the metacercaria. Some of the errors introduced by Jameson (1902, 1903) were corrected in a subsequent paper (Jameson and Nicoll, 1913) but have been replaced by others. In spite of numerous subsequent studies, the first intermediate as well as the final host of the 'pearl trematode' from *M. edulis* are still unknown. Gymnophallid metacercariae occurring between the mantle and shell, and sometimes found to be associated with pearl formation, have been reported repeatedly from *M. edulis* on the European and American North Atlantic coasts (Nicoll, 1906a; Lebour, 1907c; Chubrik, 1966; Stunkard and Uzmann, 1958; Stunkard, 1959c; Loos-Frank, 1971b; Götting, 1979a; and others). Whether all of these represent a single species, i.e., *G. margaritarum sensu* Dubois (1901a), remains to be established. 'Pearls' or pearl-like calcareous concretions have been reported from a great number of bivalves (see section 'Abnormalities').

Parvatrema duboisi is responsible for pearl formation in *Mytilus galloprovincialis*. Dubois (1903a), who first recorded the metacercaria from Mediterranean mussels, stated that it is specifically different from the 'pearl trematode' described by him (Dubois, 1901a, b) from *M. edulis* as *Distomum margaritarum*. Unfortunately, he later (Dubois, 1907a) changed his opinion, but Dollfus (1923d) restudied the parasite, found it to be distinct from *D. margaritarum*, and named it *Metacercaria* (*Gymnophallus*) *duboisi*. Bartoli (1963, 1965b) regarded metacercariae recovered by him from mussels in the Gulf of Marseille as a new species and named it *P. timondavidi*, but later (Bartoli, 1974a) correctly changed its name into *P. duboisi*. *Mytilus* (*Brachidontes*) *minimus* and *Venus* (*Chione*) *gallina* were identified as additional second intermediate hosts. *'P. timondavidi'*, reported by Endo and Hoshina (1974) and Yasuraoka and co-authors (1974) from *Tapes philippinarum* in the Bay of Tokyo, is beyond doubt another species of *Parvatrema*.

The metacercariae of *Parvatrema duboisi* occur between the mantle and shell of *Mytilus galloprovincialis* or even more frequently at the bases of the gill lamellae. Some may be found to be surrounded by a mucous envelope, which forms the matrix for the deposition of calcareous material and eventually becomes the nucleus of a pearl. Up to 60 % of the mussels from Endoume, French Mediterranean coast, were found to be infested. The first intermediate host of *P. duboisi* is yet unknown (Bartoli, 1965b, 1974a).

Adolescaria perla and *Metacercaria* (*Gymnophallus*) *perligena*, described from Mediterranean and Black Sea *Mytilus galloprovincialis* (Sinitsin, 1911; Palombi, 1940; Cerruti, 1948), are most probably referable to *Parvatrema duboisi*, but *'G. margaritarum* Dubois, 1901', reported from the same host species by Palombi (1924), is beyond doubt a separate species, which has nothing to do with Dubois' (1901a, b) pearl trematode since it has an abbreviated life cycle, the metacercariae maturing inside the sporocysts in *M. galloprovincialis*.

Cardium edule, C. lamarcki and *Macoma baltica* from the German North and Baltic Sea coasts are metacercarial hosts for *Gymnophallus gibberosus* (Lauckner, 1971; Loos-Frank, 1971a, b). The larvae are almost invariably situated in the mantle portion

surrounding the insertions of the anterior adductor and siphon retractor muscles on the inner surface of the valves, but sometimes also invade the tissues of the adductor muscle. Non-migrating metacercariae may be surrounded, singly or in groups, by mucous envelopes — which Jameson (1902) mistook for sporocysts —, while migrating ones apparently feed on host tissue and cause the deposition of calcareous concretions (Figs 13-116 and 13-117; Lauckner, 1971). *G. gibberosus* metacercariae from *C. edule* and *C. lamarcki* are slightly larger than those from *M. baltica* and may be mistaken for another species. Although resembling the 'pearl trematode' from *Mytilus edulis* in several respects, *G. gibberosus* appears to be a separate species. Loos-Frank (1971a) found the metacercariae in *M. baltica* and *C. edule* from Jadebusen (German North Sea coast), but not in *M. edulis, Mya arenaria* and *Scrobicularia plana* from the same sites. With high probability, however, *G. gibberosus* occurs in *M. edulis* from Sylt (Lauckner, unpublished). Supposedly, some of the records of *G. strigatus* in various bivalves — particularly that by Giard (1897b), who has claimed to have found the latter in *Tellina solidula* (= *Macoma baltica*) — include larval *G. gibberosus*. In spite of the frequent occurrence and sometimes high abundance of the metacercaria, its first intermediate host has not yet been discovered.

Meiogymnophallus minutus is the trematode which, in its metacercarial form, has caused maximum confusion among European marine parasitologists. Jameson (1902), Nicoll (1906a) and Lebour (1907c) described "sporocysts containing tailless cercariae" and occurring "just under the umbo" in *Cardium edule* (Figs 13-118 and 13-119). The larvae were believed to be the 'young stage' of *Gymnophallus margaritarum*, the 'pearl trematode' of the mussel. As a consequence, Lebour (1912) redescribed the metacercaria as *'Cercaria margaritae'*. Jameson (1902), Nicoll (1906a) and Lebour (1912) observed and, in their figures, clearly depicted flame cells on both sides of the oral sucker of the metacercaria which they, however, interpreted as eye spots. Cole (1938) saw no eye spots, which he believed to be a feature distinguishing his *'Cercaria cambrensis'* from *'C. mar-*

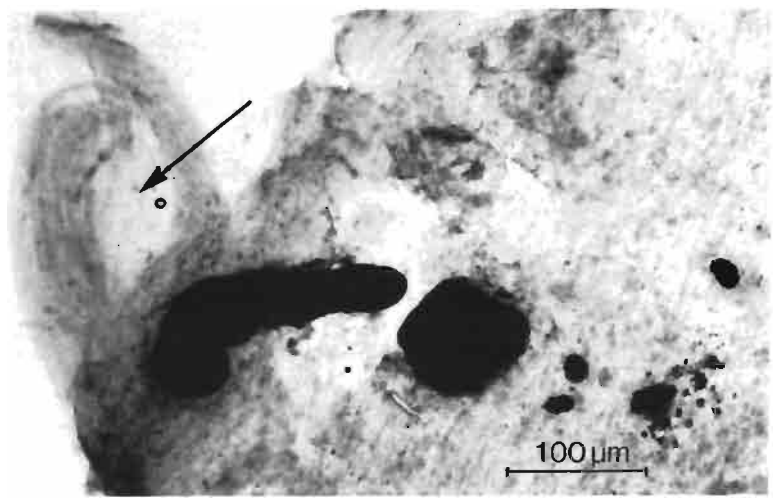


Fig. 13-116: *Cardium edule*. 'Comet tail' of calcareous concretions, produced by single migrating *Gymnophallus gibberosus* metacercaria in tissue of anterior adductor muscle. Squash preparation; chalky-white concretions appear black in transmitted light. Arrow: 'pit' in muscle tissue, produced and previously occupied by resting unencysted metacercaria. (Original.)

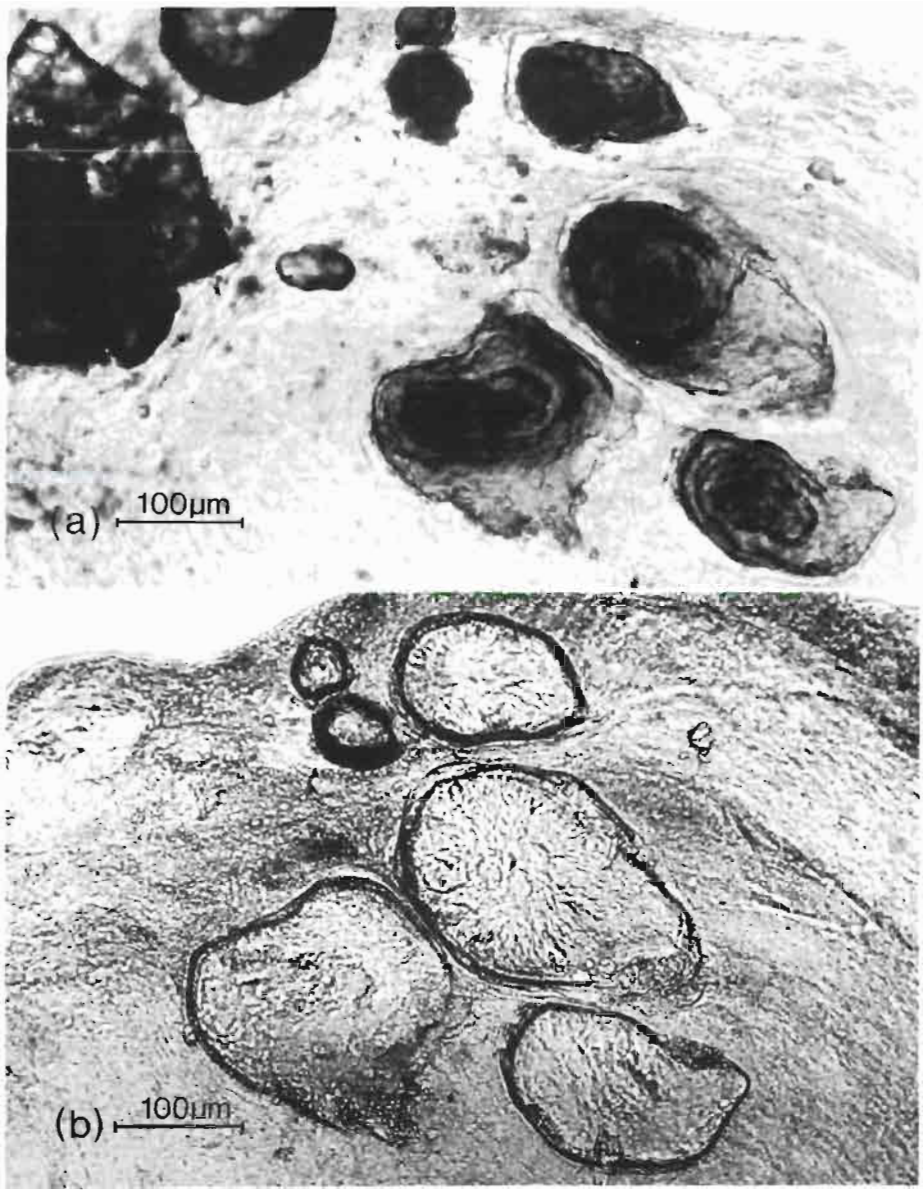


Fig. 13-117: *Cardium edule*. (a) Calcareous concretions in anterior adductor muscle, produced by *Gymnophallus gibberosus* metacercariae; (b) same, after decalcification, residues resembling metacercarial bodies of different size. (Original.)

garitae'. It remained to Bowers and James (1967) to show that all these forms are identical and represent the larval stage of a digenean, described superficially in the adult stage as *Distomum minutum* from the duodenum of oystercatchers *Haematopus ostralegus* by Cobbold (1859). Bowers and James (1967) furthermore demonstrated that the 'tailless cercariae' are, in fact, metacercariae enclosed by host tissue (Fig. 13-118). They recovered all stages — from recently established to fully formed metacercariae — from *C. edule* on

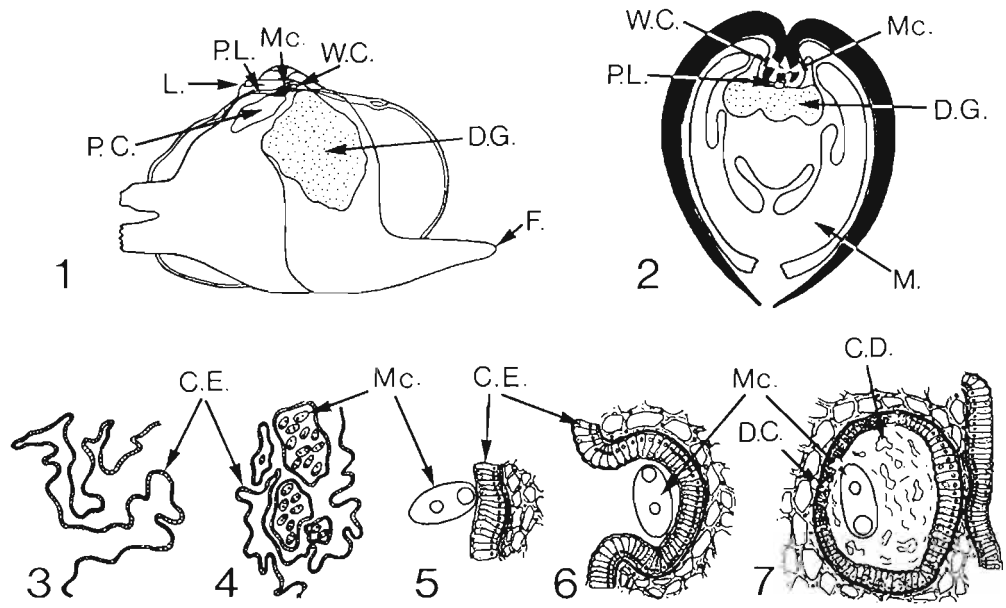


Fig. 13-118: *Cardium edule* harbouring *Meiogymnophallus minutus* metacercariae. 1: Schematic outline of host (lateral view, right valve and mantle removed) showing position of metacercariae; 2: transverse section of whole host; 3: transverse section of unparasitized pallial line; 4: same, heavily parasitized; 5, 6: recently attached metacercaria becoming enclosed by proliferation of columnar epithelium of pallial line; 7: host tissue completely enclosing older metacercaria and undergoing lysis. C.D. cellular debris, C.E. columnar epithelium of pallial line, D.C. destroyed cells, D.G. digestive gland, F. foot, L. ligament, M. mantle cavity, Mc. metacercaria, P.C. pericardial cavity, P.L. pallial line, W.C. wedge-shaped cavity below umbo. (After Bowers and James, 1967.)

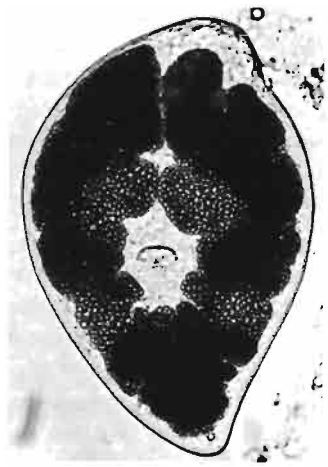


Fig. 13-119: *Meiogymnophallus minutus.* Metacercaria from *Cardium edule,* dorsal view. Note comspicuous refringent 'excretion granules' filling entire excretory vesicle. (Original.)

Llanrhidian Sands, Burry Inlet (South Wales). No earlier stages in development were seen in any of 11,410 cockles examined and, although all species of bivalves occurring in that area were inspected, no furcocercous gymnophallid cercariae, referable to *M. minutus*, were found. Its first intermediate host was later found to be *Scrobicularia plana* (p. 675; James and co-authors, 1977).

On Llanrhidian Sands, the metacercariae occurred in every cockle over 1 year old and in most spat over 12 mm long and 6 months of age. The number of larvae increased considerably with host size and age (Fig. 13-120). Up to 700 metacercariae have been dissected from older cockles. The increase in infestation intensity with host age suggests that most metacercariae survive in the cockle for a number of years (Bowers and James, 1967). Lebour (1912) found *Meiogymnophallus minutus* 'very frequently' in *Cardium edule* from Northumberland; Cole (1938) reported it from all of several thousand adult cockles from Conway (North Wales).

Loos-Frank (1971b) found about 50 % of the cockles from the East Frisian coast (Germany) to be infested with 5 to 50 metacercariae. At Sylt (North Frisian coast), both incidence and intensity of *M. minutus* infestation are extremely high. All of 3,600 adult *C. edule* examined were found to be infested; even spat of less than 6 mm shell length may yield one or several larvae (Lauckner, 1971). Similarly high infestation rates were observed in young cockles from Dutch commercial beds (Pistoor, 1969).

Meiogymnophallus minutus infestations are easily detectable. Cutting away the ligament of *Cardium edule* and pulling the valves apart exposes the chalky white mass of metacercariae lying immediately below the umbo. *M. minutus* displays pronounced host specificity, which is quite unusual for metacercariae. Although at Sylt every adult indi-

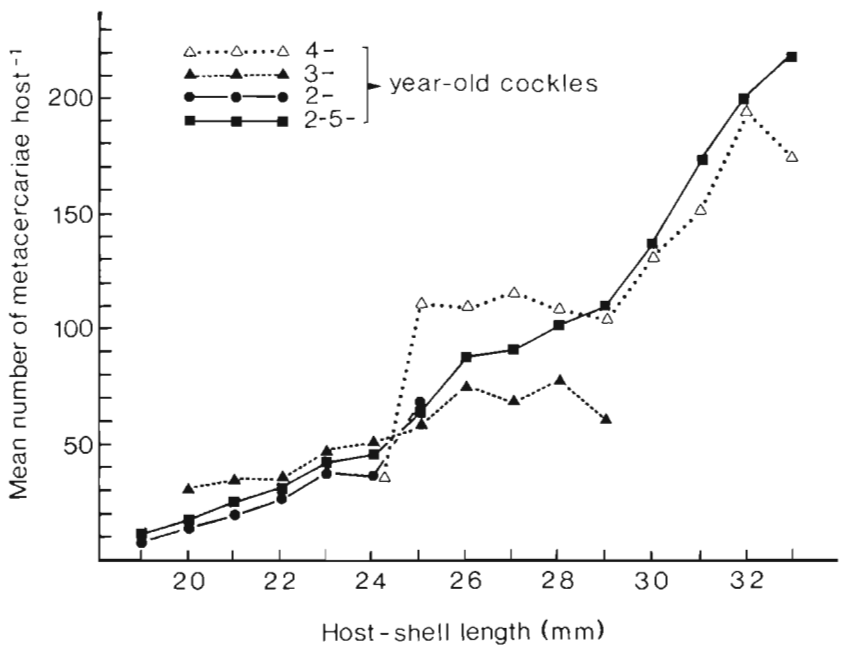


Fig. 13-120: *Cardium edule*. Increase in intensity of infestation with *Meiogymnophallus minutus* metacercariae in relation to host size and age. (After Bowers and James, 1967.)

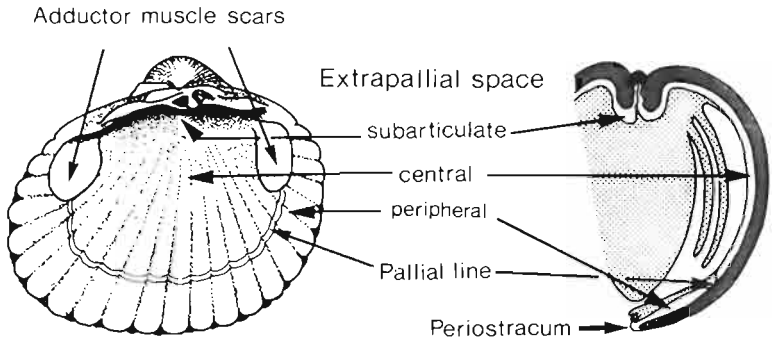


Fig. 13-121: Divisions of extrapallial space in Bivalvia. (After Bartoli, 1974a.)

vidual of *C. edule* is heavily parasitized by this species, *C. lamarcki* living side by side with *C. edule* is never infested. Both species of cockles have frequently been confounded and are sometimes difficult to separate, particularly in mixed populations. Where *M. minutus* occurs, specific identification of cockles is facilitated by checking for the presence or absence of the metacercariae under the umbo (Lauckner, 1971).

James and co-authors (1977) claim to have found *Meiogymnophallus minutus* also in *Cardium* (*Cerastoderma*) *glaucum* from different localities in England and Wales. In this host species, the metacercariae referred to that species do not occur under the umbo, but in the peripheral extrapallial space beneath the periostracum along the shell margin. The cockles studied by James and co-authors (1977) are probably *C. lamarcki*, a species differing morphologically from (Mediterranean!) *C. glaucum* (Lauckner, 1972). Both cockle species can also be separated by means of biochemical criteria (V. Brock, pers. comm.). James and co-authors apparently also believe that *M. minutus* and *Gymnophallus fossarum*, occurring in *C. glaucum* in the Mediterranean (p. 675), are specifically identical, which is not the case.

Cardium edule is metacercarial host for a second, specifically yet unidentified species of *Meiogymnophallus*, the larvae of which occur in the central extrapallial space, i.e., between the mantle and shell (Fig. 13–121). Aggregations of *Meiogymnophallus* sp. are easily discernible as reddish spots through the translucent mantle epithelium. Hundreds of metacercariae may occur in a single host individual. The larvae are slightly smaller than those of *M. minutus* and appear somewhat 'immature' due to the absence of the conspicuous masses of excretion granules characteristic of the former species. This led Loos-Frank (1971b) to believe that they represent recently established *M. minutus* metacercariae. However, the *Meiogymnophallus* sp. larvae are non-migratory. They inhabit 'pits' on the inner surface of the valves and are surrounded, singly or in groups, by a gelatinous matrix, which is suggestive of a 'stationary' mode of life.

In spite of the morphological and microhabitat differences between *Meiogymnophallus minutus* and *Meiogymnophallus* sp., the possibility exists that both are specifically identical, since metacercariae of the same species can differ morphologically and with respect to their microhabitat selection in relation to the host species invaded (Figs 13-115, 13-121 to 13-123). Unfortunately, the adult of the presumptive *Meiogymnophallus* sp. is not known. With respect to the adult stage it can be stated that, in addition to *M. minutus* occurring in *Haematopus ostralegus*, the only other known adult member of the genus

reported from the North Sea is *M. jamesoni*, described from the intestine of the common scoter, *Melanitta nigra*, by Bowers (1965b). Its comparatively large size suggests that it is probably not the adult stage of the mantle metacercaria from *C. edule*.

Macoma baltica from European North Atlantic waters is metacercarial host for *Gymnophallus gibberosus* (p. 721), *Lacunovermis macomae* and *Parvatrema affinis*. The metacercaria of *L. macomae* has first been described as *Cercaria macomae* by Lebour (1908a), who found it in low numbers between the mantle and shell of 3 of 20 *M. baltica* on Fenham Flats, Northumberland (England). Markowski (1936), unaware of Lebour's account, redescribed it as *Metacercaria mutabilis*. Of 300 *M. baltica* from Hel (Polish Baltic Sea coast), 103 harboured from 1 to 30 larval worms. Loos-Frank (1971b) reported *L. macomae* from about 60 % of *M. baltica* from various localities on the German North Sea coast. The metacercaria appears to be strictly host-specific; it has never been found in bivalves other than *M. baltica*. Loos-Frank (1970) experimentally demonstrated the adult of *L. macomae* to be identical with worms described as *Gymnophallus macroporus* from the intestine of the common scoter *Melanitta nigra* by Jameson and Nicoll (1913). Its first intermediate host is unknown, but in all probability *Cercaria baltica*, described by Markowski (1936) from the same host species (p. 678), is the first larval stage of *L. macomae* (Cable, 1953; Loos-Frank, 1970, 1971b).

Parvatrema affinis has an abbreviated life cycle (Table 13-14). Selikman (1953) and Reimer (1962) found young sporocysts containing cercariae, but the tailed stage appears to be of short duration, most sporocysts encountered in *Macoma baltica* harbouring only metacercariae. The conspicuous whitish, oval sporocysts, which measure up to 2,200 × 425 µm, infiltrate practically every organ of the clam except the gills, palps, siphons, intestine, or muscles. In addition to the metacercariae, they contain strongly refractive corpuscles of fairly uniform size, which probably represent 'excretion granules'

Table 13-22
Macoma baltica. Relation of shell length of 4 infested individuals to number of *Parvatrema affinis* sporocysts (After Swennen and Ching, 1974)

Shell length (mm)	Number of sporocysts
6.0	52
13.8	353
18.2	2,111
23.2	3,457

Table 13-23
Macoma baltica. Increase of *Parvatrema affinis* infestation with shell length (host age) (After Swennen and Ching, 1974)

Shell length (mm)	Age (years)	Infestation (%)
< 6	0	0
6–10	1–2	1
11–15	2–3	3
16–20	≥ 3	15
21–25	≥ 4	32

expelled from the metacercariae through the terminal pore of the excretory vesicle (Fig. 13-93). Average-sized (612 × 350 μm) sporocysts contain about 40 fully developed metacercariae, but large ones may yield numbers in excess of 200. The number of sporocysts per host increases considerably with host-shell length and age (Swennen and Ching, 1974; Tables 13-22 and 13-23).

Markowski (1936), who first described the larva as *Metacercaria morula*, found 2 of 300 *Macoma baltica* from Hel to be infested. In Kandalaksha Bay (White Sea), at Hiddensee (Baltic Sea coast of GDR), and on the German North Sea coast, infestation incidences ranged from 6.6 % to 7.5 % (Selikman, 1953, 1962; Reimer, 1962, 1971; Loos-Frank, 1971b). In the Dutch Wadden Sea, up to 44 % of the larger clams, predominantly those from higher shore levels, had *Parvatrema affinis* sporocysts and metacercariae (Hulscher, 1973; Swennen and Ching, 1974). Since heavy infestation causes complete parasitic castration, a considerable proportion of individuals is, therefore, excluded from the breeding population. Although *M. baltica* harbouring metacercariae survived for over a year in the laboratory, field observations suggest an increased mortality of infested clams under unfavourable ecological conditions (Hulscher, 1973). Oxygen deficiency of the host, due to the high metabolic activity of the larval trematodes, leads to an abnormal behaviour of such individuals (p. 749). The adult of *P. affinis* has originally been described from the intestine of *Melanitta nigra* as *Gymnophallus affinis* by Jameson and Nicoll (1913), and has subsequently been reported from a variety of other sea birds (Selikman, 1953; Reimer, 1962; James, 1964; Loos-Frank, 1970, 1971b; Swennen and Ching, 1974).

Macoma inconspicua from the North American Pacific coast is host for *Lacunovermis conspicuus* and *Meiogymnophallus multigemmulus*. Both cercariae and metacercariae of *L. conspicuus* develop in *M. inconspicua*, but in different individuals. At Spanish Banks (Vancouver, Canada), 72 % of the clams were infested with 1 to 7 (max. 26) metacercariae, although only 13 of 1,138 individuals harboured the sporocyst and cercarial stages. Experimental infestation of *M. inconspicua* with cercariae of *L. conspicuus*, as well as *in vitro* cultivation of 3-month-old metacercariae until the onset of egg production (which occurred after 48 h at 37 °C), were successful (Ching, 1965).

Meiogymnophallus multigemmulus has an abbreviated life cycle (Table 13-14). Three of 1,138 *Macoma inconspicua* from Spanish Banks were found to harbour sporocysts, which occupied almost all tissues of the clam except for the muscular foot and siphons, and which appeared like 'pink grains of sand' when dissected from the host. Twenty sporocysts, chosen at random, contained 37 to 108 metacercariae and a very small number of fork-tailed cercariae. The adults of both *M. multigemmulus* and *Lacunovermis conspicuus* parasitize in the intestine of several species of diving ducks (Ching, 1965).

Although oysters in various parts of the world have been inspected by parasitologists, apparently only a single gymnophallid metacercaria has been reported from members of the Ostreidae. This is *Gymnophalloides tokiensis*, infesting *Crassostrea gigas* in Japan. The parasites inhabit the extrapallial space where they usually occur in groups consisting of 3 to 90 individuals and resembling "la disposition d'une fleur de chrysanthème". Single patches may attain a diameter of about 3 mm, but larger assemblages derived from confluent patches are also seen. Each larva is firmly attached, by means of its powerful oral sucker, to the outer mantle epithelium. The metacercariae occur in significantly higher numbers in the extrapallial space of the left (lower) valve, the maximum (mean of a sample of 20

oysters) being 843 on the left and 324 on the right valve. In moderately high infestations, *G. tokiensis* shows a distinct preference for the anterior mantle portion, next to the mouth of the host, and only in extremely heavy infestations may the distribution be more uniform. The infestation intensity increases considerably with host age. One-year-old *C. gigas* from Yawata (Chiba Prefecture) harboured, on the average, 27 metacercariae in the extrapallial space of the right valve and 84 in that of the left valve. Respective figures for 2-year-old oysters were 235 and 1,026.

Gymnophalloides tokiensis occurs in largest numbers in oysters from muddy beds. At some sampling stations (for example, Tokyo Bay, Lake Hamana and Isé Bay), the incidence was 100 %, while it was zero at others (i.e., Hokkaido, Hiroshima Bay, Kaida Bay, Matsukawa Bay). Salinity appeared to be one of the factors interfering with the life cycle of the parasite, the metacercaria being sensitive to higher salinities. In young oysters taken from spat collectors, infestation incidences and intensities decreased significantly with the distance from the bottom. Similarly, spat from shaded parts of the collectors had higher metacercarial burdens than oysters from exposed collector surfaces. This indicates a negative phototactic behaviour of the cercaria.

Repeated raking of the oyster beds did not only improve growth and quality of *Crassostrea gigas* but obviously had a negative effect on the establishment of *Gymnophalloides tokiensis*. Of 20 oysters from an untreated bed, all were infested with a mean number of 402 metacercariae each, while of 20 oysters from a raked bed only 18 were infested with an average of 62 larvae (Fujita, 1925). In view of the severe pathology of *G. tokiensis* (Hoshina and Ogino, 1951; see below), the above ecological observations may be of practical importance for the selection of optimal conditions for the commercial raising of *C. gigas* in Japan. Unfortunately, neither the primary nor the definite hosts of *G. tokiensis* are known (Ching, 1972).

Further gymnophallid metacercariae and their respective bivalve hosts are listed in Table 13-24. Some of these have not yet been named or identified, and of others the cercarial and adult stages are unknown. Of particular interest are those species in which the metacercariae are located within sporocysts. For these forms, which have either an abbreviated or an alternative life cycle, the same parasitized bivalve individual acts as first and second intermediate host (see preceding subchapter and Table 13-14).

Multiple metacercarial infestations of individual bivalve hosts are the rule rather than exception. As far as several gymnophallids are involved, their metacercariae have often been confounded. Strict host specificity — like that observed in *Meiogymnophallus minutus* — is rare. Most gymnophallid metacercariae utilize more than 1 species of secondary host. Of the 5 gymnophallids from Camargue (French Mediterranean coast) with Type-I life cycles, the metacercariae of *Gymnophallus rebecqui* parasitize 2, those of *Parvatrema duboisi* 3, of *G. strigatus* 8, of *G. fossarum* 9, and those of *G. rostratus* even 16 species of bivalves in nature (Table 13-21). Experimental infestations have been achieved in additional hosts. On the other hand, some combinations not observed under natural conditions could also not be produced in the laboratory. Thus, it was impossible to experimentally infest *Cardium glaucum* with cercariae of *G. strigatus* and *G. rostratus*, or *Loripes lacteus, Venus gallina, Dosinia lupinus, Abra ovata* and *Mactra corallina* with cercariae of *G. fossarum* or *G. strigatus*. Cercarial penetration has been observed in some cases, but metacercariae did not develop (Bartoli, 1974a).

Bivalves infested with sporocysts and cercariae may be repellent to metacercariae of

Table 13–24
Some gymnophallid metacercariae with unknown life cycles from marine bivalves (Compiled from the sources indicated)

Species	Second intermediate host	Geographic area	References
Gymnophallus gibberosus	*Cardium edule, C. lamarcki, Macoma baltica*	North and Baltic Seas	Lauckner (1971), Loos-Frank (1971a, b)
'Distomum margaritarum' (= Gymnophallus bursicola?)	*Mytilus edulis*	North Sea	Dubois (1901a, b), Jameson (1902), Giard (1907)
'Metacercaria duboisi' (= Parvatrema timondavidi)	*Mytilus galloprovincialis, Brachidontes minimus, Venus gallina*	Mediterranean Sea	Dollfus (1923d), Bartoli (1963, 1974a)
Metacercaria (Gymnophallus) megalocoela	*Mytilus galloprovincialis*	Mediterranean Sea	Palombi (1934a)
Metacercaria (Gymnophallus) perligena	*Mytilus galloprovincialis*	Mediterranean Sea	Palombi (1940), Cerruti (1948)
Adolescaria perla	*Mytilus galloprovincialis*	Black Sea	Sinitsin (1911)
Gymnophallus sp. (spp.)	*Mytilus edulis, Modiolus modiolus, Macoma calcarea, Astarte elliptica, A. banksi, Musculus discors, M. laevigatus, Turtonia minuta, Yoldia hyperborea, Hiatella arctica, Chlamys islandicus, Nucula tenuis, Nuculana pernula*	Barents and White Seas	Chubrik (1966)
Gymnophallus somateriae (?)	*Hiatella (Saxicava) rugosa*	Greenland	Levinsen (1881)
Parvatrema sp.	*Tapes philippinarum*	Japan	Endo and Hoshina (1974), Yasuraoka and co-authors (1974)
(Meta-)Cercaria scrivenensis	*Tapes pullastra*	Scotland, Norway	Lebour (1912), Johannessen (1973)
Parvatrema sp.	*Gemma gemma*	U.S. Pacific coast	Obreski (1968)
Gymnophalloides tokiensis	*Crassostrea gigas*	Japan	Fujita (1925), Hoshina and Ogino (1951)

the same species. Such a case of immunity to autoinfestation has been demonstrated experimentally for *Loripes lacteus*, the only bivalve from Camargue serving as first and second intermediate host for the same species of gymnophallid with a Type-I life history, i.e., *Gymnophallus rostratus*. Cercariae never re-enter the host individual from which they have emerged, but attack and heavily parasitize other individuals of *L. lacteus* (Bartoli, 1974a). In contrast, cercariae of *Parvatrema isostoma* may re-enter and become established as metacercariae within the same individual of *Abra ovata* by which they had been shed (Dolgikh, 1968b).

Although hyperparasitization by sporozoans seems to occur more frequently in the

sporocyst and cercarial stages of digeneans, a few cases have also been documented for metacercariae. Giard (1897b) and Léger (1897a) described *Gymnophallus strigatus* ('*Brachycoelium* sp.') metacercariae in *Donax vittatus* from Boulogne-sur-Mer (France), which were packed with sporozoan spores of the group 'glugeidées'. Léger (1897a) assigned the hyperparasite to the genus *Pleistophora*, as he had observed variable numbers of spores assembled in spherical masses, 15 to 20 μm in diameter and enclosed by a membrane. He probably saw groups of spores within host-cell membranes which could be mistaken for pansporoblasts. Dollfus (1912, 1946a) found diffuse infiltrations of spores produced individually and not in pansporoblasts, identified the agent as a microsporan of the genus *Nosema*, and named it *N. legeri*.

Lightly hyperparasitized metacercariae were slightly larger (hypertrophied), more opaque and less active than healthy ones. Heavily affected individuals were almost spherical in shape and distinctly bloated. Their internal organs had entirely disappeared and they had become mere sacs of spores. These metacercariae contained yellowish crystalline material, believed to consist of calcium carbonate and to represent the initial stage of calcification, which eventually leads to the formation of pearls. Whenever an infestation by *Nosema legeri* occurred, almost all *Gymnophallus strigatus* metacercariae in a given *Donax vittatus* clam were involved, heavily attacked worms being killed. Since the host itself was not affected by the sporozoan, the hyperparasite was regarded as being beneficial for the clam in reducing its parasite burden (Dollfus, 1912).

In his investigation on the origin of pearls in *Mytilus edulis*, Jameson (1902) — apparently believing that he dealt with normal worms — figured metacercariae of *Gymnophallus margaritarum* with what appear to be initial and final stages of sporozoan infestation. Dollfus (1912) assumed that the hyperparasite in *M. edulis* might be identical with, or closely related to, *Nosema legeri*. Mediterranean *M. galloprovincialis* are sometimes heavily infested with metacercariae of *Parvatrema duboisi* which, in turn, are hyperparasitized by unidentified sporozoans (Dubois, 1901b). Masses of minute round bodies of unknown nature, presumably protozoan spores, caused the bodies of metacercariae of *P. donacis* in *Donax variabilis* from Texas to enlarge and become inactive and opaque (Hopkins, 1958a). Bowers and James (1967) identified *Nosema* sp. as a hyperparasite of *Meiogymnophallus minutus* in *Cardium edule* from British waters. The incidence varied irregularly from month to month, from 5 to 26 % of all cockles — mostly of those over 3 years of age — carrying infested metacercariae. The spores caused the body of the larval trematodes to enlarge, sometimes to almost twice the normal size, and to become opaque and greyish white. It appeared that *M. minutus* is killed by the microsporan.

Whether all these protozoan hyperparasites belong to the same species, remains to be established. Canning and Nicholas (1974) restudied the microsporan infesting *Meiogymnophallus minutus* in *Cardium edule* by means of light and electron microscopy and demonstrated that its sporonts are disporoblastic, but that nuclei in diplokaryon form, characteristic of *Nosema*, are not present at any stage. The agent shares morphological characteristics with members of the genus *Unikaryon*, as erected by Canning and co-authors (1974). Canning and Nicholas (1974) unfortunately misinterpreted Bowers and James' (1967) statement about the synonymy of *M. minutus* with larval gymnophallids described under various names. They assumed that *M. minutus*, the cockle trematode, is identical with Jameson's (1902) *Gymnophallus margaritarum*, the mussel trematode

(p. 720), as well as with *G. strigatus* from *Donax vittatus,* as reported by Giard (1897b), Léger (1897a), Dollfus (1912, 1946a), and Guyénot and co-authors (1925). As a consequence, they transferred *Nosema legeri* Dollfus, 1912, to the genus *Unikaryon* as *U. legeri.* But *M. minutus* is highly host-specific and never infests bivalves other than *C. edule* (p. 725). *G. strigatus* and *G. margaritarum* are likewise clearly distinct species. Taking into account the host specificity normally displayed by members of the Nosematidae, one may doubt that all the above-described microsporan hyperparasites represent a single species.

Measurements of 50 fresh spores of *Unikaryon legeri* from *Meiogymnophallus minutus* gave mean dimensions of 3.03 ± 0.30 μm × 1.76 ± 0.02 μm. In stained smears, the dimensions were 2.90 ± 0.13 μm × 1.66 ± 0.24 μm. Léger (1897a) and Dollfus (1912) reported the spores of *Nosema legeri* from *Gymnophallus strigatus* to measure 5 × 2.5 μm in sectioned material. However, Canning and Nicholas (1974), who re-inspected Dollfus' type slide, found that they measured only about 2.5 × 1.5 μm, which is approximately the size of the *U. legeri* spores. Although on Dollfus' slide "only spores were present and no details could be discerned other than the presence of the pale area of cytoplasm looking like a vacuole at the broader end of the spore" (Canning and Nicholas, 1974, p. 95), the authors regarded this as sufficient to synonymize *N. legeri* with *U. legeri.* (It should be pointed out, however, that Dollfus' relegation of his microsporan to the genus *Nosema* is, in any case, incorrect, as members of this genus are typically parasites of Lepidoptera. *Nosema* spp. reported from other invertebrates [Vol. III] have more recently been transferred to *Ameson* and other genera [Sprague, 1977]).

The mode of infestation of metacercariae by microsporans is unknown. Probably, the initial acquisition of the hyperparasite results from ingestion, by the bivalve host, of a spore, which then finds its way into a metacercaria. Experimental transmission of a *'Nosema'* hyperparasite of a larval strigeoid trematode of freshwater snails has, for example, been achieved by feeding its spores to the snail hosts (Cort and co-authors, 1960a, b). All asexual and sexual stages of *Unikaryon legeri* — uninucleate and binucleate schizonts and sporonts, uninucleate sporoblasts and spores — occur in *Meiogymnophallus minutus.* An intermediate host is not required. Spreading of the initial infestation to neighbouring metacercariae is indicated by the fact that, whenever an infestation occurs, most if not all larvae in a given host are hyperparasitized. It is also possible — although less probable — that the respective trematodes carry over their hyperparasites from the cercarial to the metacercarial stage, as reported for freshwater snails (Martin, 1936). Whether *U. legeri* can infest gymnophallid sporocysts and cercariae is not known.

Microsporan hyperparasitization of larval trematodes may be of practical (economic) importance. Firstly, beneficial effects can be expected to result from the fact that metacercariae killed by the protozoans can no longer harm the bivalve host. On the other hand, dead metacercariae enclosed by host tissue may undergo calcification and yield 'sandy' clams of reduced commercial value. Dubois (1907a, 1909) assumed that 'pearls' in molluscs may originate from direct irritation of host tissues by protozoan spores, regardless of the absence or presence of larval trematodes (p. 772).

Metacercarial — and, in particular, gymnophallid — infestations may exert profound effects on vital functions of their respective hosts. Such parasitic interference with host physiology is not normally considered in the malacological literature. In the 'Physiology of Mollusca' (Wilbur and Yonge, 1964, 1966), for example, only passing mention is made of parasites (in general). This is more than alarming because the physiologically most

thoroughly studied (and simultaneously commercially most important) species — members of the genera *Mytilus, Cardium, Mya, Mercenaria* and *Donax* — are among the favourite second intermediate hosts of bird trematodes. On North Sea tidal flats, *Cardium edule* — next to *Mytilus edulis* the most abundant intertidal bivalve in that area — is host for 11 species of metacercariae (including 4 gymnophallids). At certain sampling stations, at least 9 or even all of these larval trematodes may occur together in the same host individual. *C. lamarcki* and *M. edulis* from the same localities are parasitized by respectively 7 and 6 species of metacercariae and all of these may occur in a single host. In 3-year-old cockles, metacercarial burdens may be in excess of 3,000 larvae per host. General debilitation is the imperative consequence of such intense parasitic invasion.

Similar conditions have been reported from other intertidal areas of the European coasts. Bowers and James (1967), for example, found *Meiogymnophallus minutus* metacercariae in 100 % of *Cardium edule* older than 1 year in the Burry Inlet (Wales). Infestation intensities increased rapidly with host age (Fig. 13-120), but declined significantly in cockles between 4 and 5 years old. This decrease could neither be attributed to death of the metacercariae nor to interaction with the microsporan hyperparasite *Unikaryon legeri* (p. 732). The only remaining explanation is that the most heavily infested hosts die, thus reducing mean infestation intensity. This suggestion is supported by the absence of 6- to 7-year-old cockles in areas of high *M. minutus* prevalence. Concomitant absence of 0-group and 1-year-old individuals at such stations may also be due to excessive metacercarial attack (James and co-authors, 1977). Pistoor (1969) reported high *M. minutus* infestation intensities and heavy losses of young *C. edule* on Dutch cockle beds during the summer of settlement. In the light of these findings the statement by Hancock and Urquhart (1965, p. 10), that

> "there is no evidence that parasitization causes the early death of cockles, and even if it did it would not explain the different mortality rates observed between age groups",

can no longer be accepted as valid. Admittedly, adequate statistical treatment of sampling data is an irreducible prerequisite for the evaluation of parasite-induced differential mortality. Such exact data are, however, usually lacking in the ecological literature.

It is difficult to understand how *Meiogymnophallus minutus* kills cockles, because the metacercariae are restricted to a small, well circumscribed tissue area under the hinge (Fig. 13-117). James and co-authors (1977) suspect that a delayed hypersensitivity (allergic) reaction occurs in host cells surrounding the metacercariae, followed by systemic shock and death. Heavy infestations could also substantially lower stress resistance of the host. Accelerated death of heavily infested cockles kept under laboratory conditions supports this hypothesis.

Intertidal habitats are generally considered as 'stress habitats'. In a study on physiological responses of *Cardium edule* to environmental stress — i.e., changes in temperature, salinity, desiccation, freezing, etc. — it became apparent that the 'physiological capacity' of the test animals varied greatly between sampling stations. These differences were difficult to explain in terms of exposure and life history of the individuals under consideration. Closer examination of the causes of this variation revealed differences in the degree of parasitization — i.e., variations of the total metacercarial burdens in the range of 1 or 2 orders of magnitude — to be responsible for this (Lauckner, 1972; Lauckner, unpubl.). Some of the effects elicited by larval gymnophallids will be discussed below.

Gymnophallid metacercariae have often been reported to occur 'between the mantle and shell' of their respective bivalve hosts. This statement is, however, very unprecise, since the 'extrapallial space' can be divided into 2 or 3 well-defined areas (Fig. 13-121). Metacercariae of the same species may inhabit different microhabitats in different hosts (Table 13-25, Figs 13-122 and 13-123). The extrapallial space of certain bivalves has

Table 13-25
Gymnophallus spp. Microhabitats occupied by metacercariae in different host species (After Bartoli, 1974a)

Host species	Division of extrapallial space		
	peripheral	subarticulate	central
Cardium glaucum	*G. fossarum*	*G. fossarum*	*G. rebecqui*
Tapes decussatus *Tapes aureus*	*G. fossarum*	*G. fossarum*	*G. rostratus*
Abra ovata		*G. rebecqui*	*G. rebecqui* *G. rostratus*
Scrobicularia plana *Loripes lacteus* *Divaricella divaricata* *Venus gallina* *Dosinia lupinus* *Mactra corallina* *Tellina tenuis*			*G. rostratus*
Lentidium mediterraneum *Donax trunculus* *Donax semistriatus* *Spisula subtruncata*			*G. rostratus* *G. strigatus*
Cardium tuberculatum	*G. fossarum*	*G. fossarum*	
Solen marginatus *Ensis siliqua*	*G. fossarum*	*G. fossarum*	*G. fossarum* *G. rostratus* *G. strigatus*

access to the external medium, while it has no connection in others. Even with openings present, the extrapallial fluid may differ in composition from the external medium. Knowledge of the chemical composition of the extrapallial fluid is fragmentary (Wilbur, 1964).

The extrapallial fluid is the medium from which two solid phases form — the crystalline inorganic components and the organic matrix of the molluscan shell. Disturbances in the deposition of shell material are frequently associated with the presence of gymnophallid metacercariae within the extrapallial space. They may be due to (i) parasite-induced changes in the chemical composition of the extrapallial fluid or (ii) mechanical

and/or physiological disturbances of the mantle tissue, or both. The resultant pathology includes

(a) shell erosion,
(b) deposition of additional shell material in the form of calcareous concretions, blisters and crest- or ridge-like structures on the inner surface of the valves, or the formation of pearls,
(c) stunting or cessation of host-shell growth,
(d) destruction of the periostracum,
(e) hypertrophy of mantle tissues,
(f) paralysis of affected adductor muscles,
(g) depletion of host-body reserves,
(h) alterations of host behaviour, and
(i) general debilitation and morbidity.

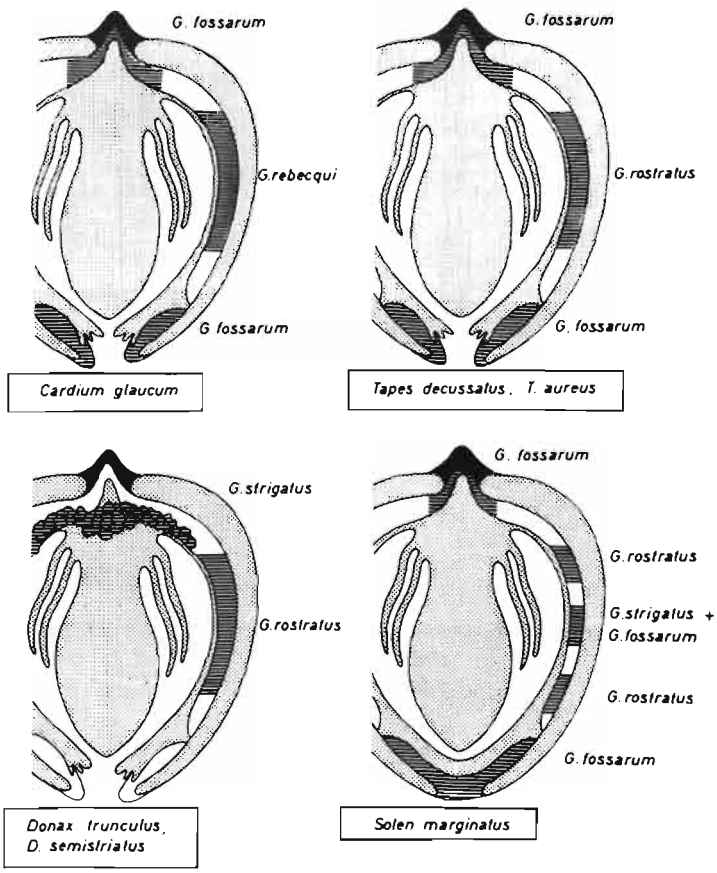


Fig. 13-122: Gymnophallid metacercariae in Mediterranean marine pelecypods. Microhabitat segregation exhibited in relation to host species involved. (After Bartoli, 1974a.)

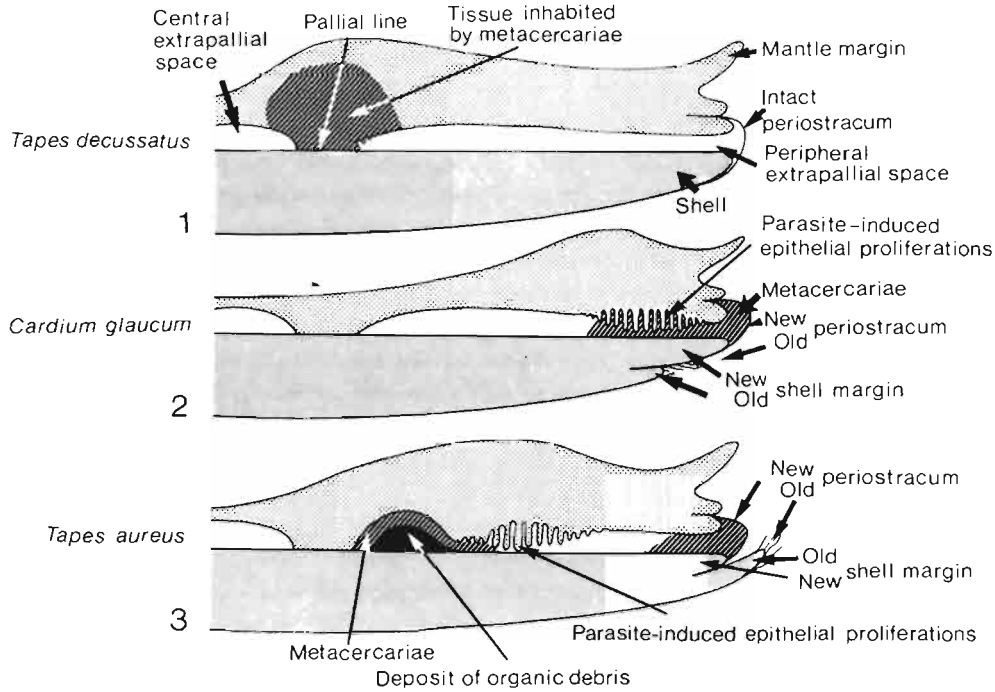


Fig. 13-123: *Gymnophallus fossarum*. Microbiotopes inhabited by metacercariae in different second intermediate host species. Hatched areas: space or tissue inhabited by metacercariae. (After Bartoli, 1974a.)

The severety of the response of the bivalve host varies with the parasite species involved, with the microhabitat occupied by the metacercariae, and with the intensity of infestation, from nil over local irritation and hypertrophy of the mantle tissues to profound pathology including disturbances in shell growth. *Gymnophallus rostratus* metacercariae, which parasitize at least 16 bivalve species from Camargue (Table 13-21), elicit virtually no histologically detectable host response. The larvae invariably occur free in the central extrapallial space (Fig. 13-122), but never in the peripheral or subarticulate region. They are relatively active and do not attach to the mantle epithelium. *G. rebecqui* inhabits mainly the dorsal region of the central extrapallial space of *Abra ovata*, where the metacercariae form dense aggregations attached to the mantle epithelium. Host cells in contact with the larvae are deeply staining and undergo considerable hypertrophy (Fig. 13-124). The reponse of *Donax trunculus* to metacercariae of *G. strigatus* varies in relation to the site of infestation. Single worms attaching to the middle portion of the mantle cause local hypertrophy. The parasites are not entirely engulfed by host tissue (Fig. 13-125, 1). Metacercariae on the mantle next to the base of the gills stimulate the epithelium to produce deeply indented proliferations (Fig. 13-125, 2). Host reaction against *G. strigatus* is most pronounced in the most dorsal portion of the mantle. Greatly hypertrophied tissue surrounds vesicles containing worms which, by this host response, have actually become internal parasites (Fig. 13-125, 3). In older, extremely heavily infested *D. trunculus*, the mass of tissue-embedded *G. strigatus* fills almost the total medio-dorsal body region of the host. The vesicles, which eventually become entirely closed and disconnected from the

extrapallial space, are similar to those produced by *Cardium edule* in response to *Meiogymnophallus minutus* (Fig. 13-118), and represent the earlier authors' 'sporocysts containing tailless cercariae'.

Another gymnophallid metacercaria eliciting strong host response is *Gymnophallus fossarum*. Although devoid of penetration glands (which are present in most other related larvae), the cercariae of this species are capable of easily penetrating *Cardium glaucum*. Entrance ports for part of the cercariae are the palps and gills. Upon passage across the epithelial barrier, they migrate to their microhabitat, the subarticulate extrapallial space. Apparently, this environment is not optimal for larval support, since most of the metacercariae die, causing the accumulation of blackish, stratified deposits, which largely consist of organic material. These formations are relatively soft in consistency and cause considerable thickening of the valves and modification of the aspect of the hinge. Other cercariae, which invade the peripheral extrapallial space directly via the mantle epithelium, settle between the epithelial folds of the mantle margin (Fig. 13-123, 2) where they cause extensive hypertrophy and formation of epithelial proliferations (Fig. 13-126). Concomitant lesions are observed in the valves of heavily parasitized *C. glaucum*. At first, growth of the periostracum is stimulated, excessively produced layers becoming folded upon each other along the shell margin. The production of shell material, on the other hand, is grossly impaired. Disturbance rings of considerable magnitude appear between, and parallel to, the normal annual growth rings. Successive disturbance rings mark consecutive periods of heavy cercarial attack, which takes place in the spring of each year. Sometimes the shell margin is so strongly deformed that the valves gape and the mantle margins lose contact (Fig. 13-127; Bartoli, 1973a, 1974a).

The shell lesions are probably due to parasite-induced alterations in the chemical composition of the extrapallial fluid, rather than to damage to the shell-secreting mantle margin. Such a mechanism is suggested by the fact that between 2 successive periods of cercarial attack shell growth is normal. Shell gaping and the resultant failure of the mantle margins to maintain close contact is of considerable disadvantage to sediment-dwelling bivalves. Entrance of sediment particles through gaps causes spoilage of the mantle cavity, the gills and the palps. In order to get rid of this foreign matter, the animal must produce excessive quantities of enveloping mucus which, in turn, may lead to emaciation. Ciliary activity of the gills — and thereby the filtration capacity and proper respiratory function of

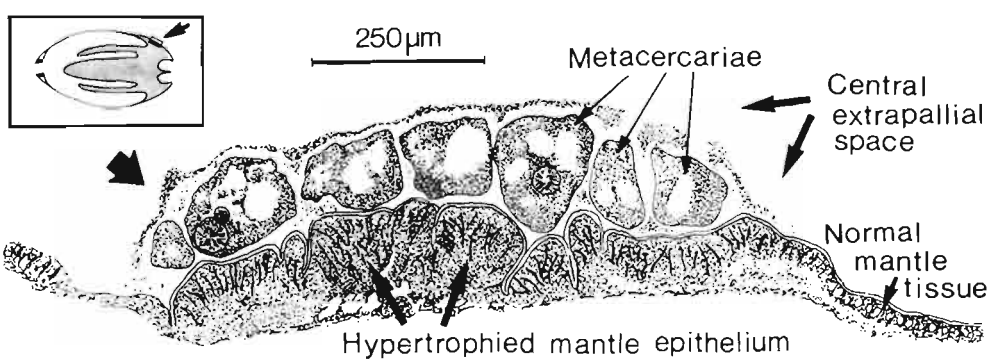


Fig. 13-124: *Abra ovata*. Mantle hypertrophy induced by *Gymnophallus rebecqui* metacercariae. (After Bartoli, 1974a.)

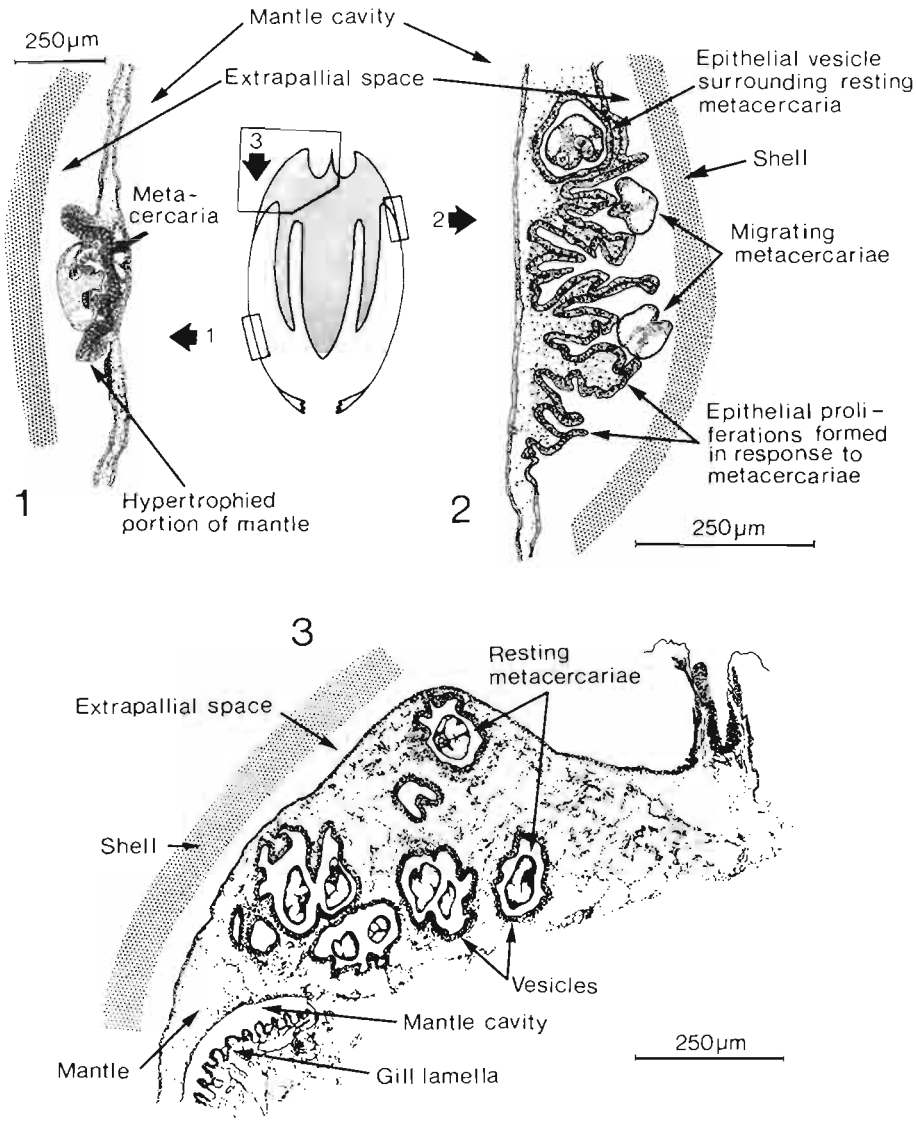


Fig. 13-125: *Donax trunculus.* Reaction of different mantle regions to metacercariae of *Gymnophallus strigatus.* (After Bartoli, 1974a.)

that organ — are also significantly impaired by layers of mucus and foreign particles covering the gill filaments. In extreme cases, contamination of the mantle cavity and oxygen deficiency may force the affected host to surface. This is, in turn, advantageous for the parasite because bivalves lying exposed on the sediment surface are easily accessible to the bird final host.

Individuals of different host species may respond in quite different ways to metacercariae of one and the same species. As in *Cardium glaucum*, the cercariae of *Gymnophallus fossarum* invade the peripheral extrapallial space in *Tapes decussatus.* But, instead of settling between the epithelial folds of the mantle margin as in *C. glaucum*, they

become installed in the mantle tissue adjacent to the pallial line, i.e., the border along which the mantle inserts on the shell (Fig. 13-123, 1). From this host-dependent microhabitat segregation, a superficial observer may gain the impression that he is dealing with two separate species of larval trematodes. Jameson (1902) and Lebour (1908a) mistook aggregations of *G. fossarum* metacercariae in *T. decussatus* from Billiers (France) and Northumberland (England) for 'tailless cercariae within sporocysts'.

Close contact of the *Gymnophallus fossarum* metacercariae with the clam's mantle tissue causes the epithelium to proliferate and to successsively engulf the parasites (Fig. 13-128, 2). Eventually, the connection with the extrapallial space is entirely disrupted, the metacercariae — now in *intra*pallial location — becoming enclosed in 'vesicles' formed by mantle tissue. Hyaline waste material accumulates in the vesicles, which steadily increase in volume. The surrounding mantle tissue becomes greatly hypertrophied, the radial mantle muscles disrupted (Figs 13-128, 3 to 13-130). As a consequence, the imprint of the pallial line on the inner shell surface becomes broadened and serrated.

The distribution of *Gymnophallus fossarum* in the peripheral extrapallial space of *Tapes decussatus* is not uniform. Fifteen days after the start of an infestation experiment with 200 larvae, the young metacercariae were rather evenly dispersed along the ventral margin of the pallial line, but after 60 days they had aggregated in the ventral sinusoidal

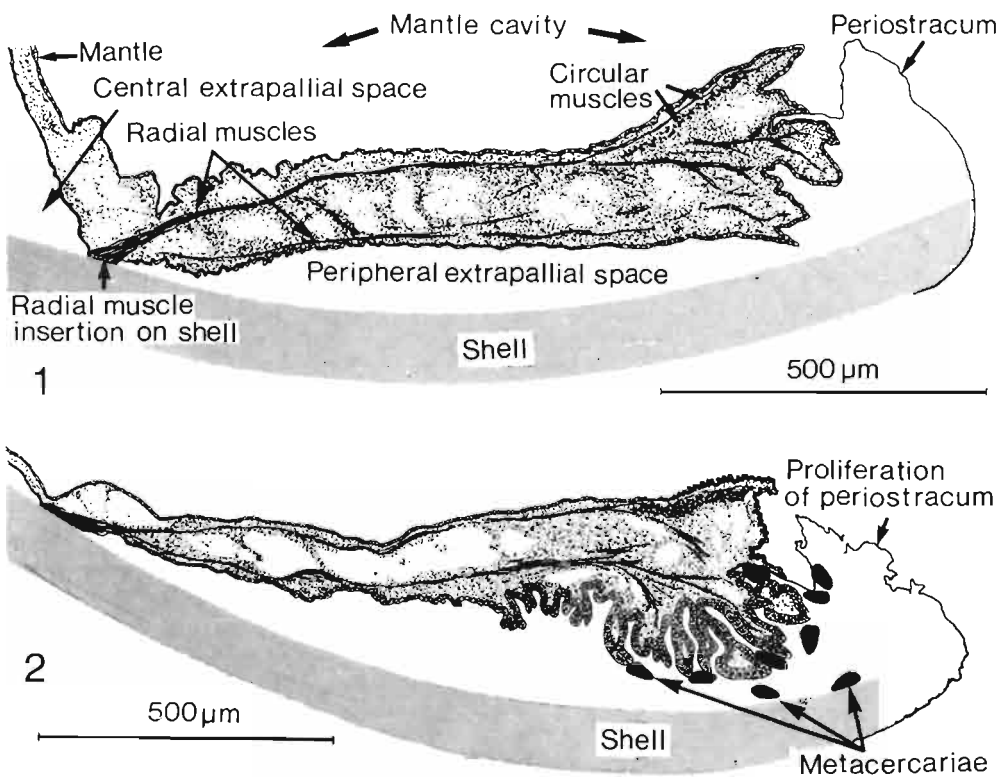


Fig. 13-126: *Cardium glaucum*. 1: Mantle and shell margin of healthy individual; 2: individual heavily parasitized by *Gymnophallus fossarum* (see Fig. 13-123, 2). Note extensive hypertrophy and epithelial proliferation of mantle margin caused by presence of metacercariae. (After Bartoli, 1974a.)

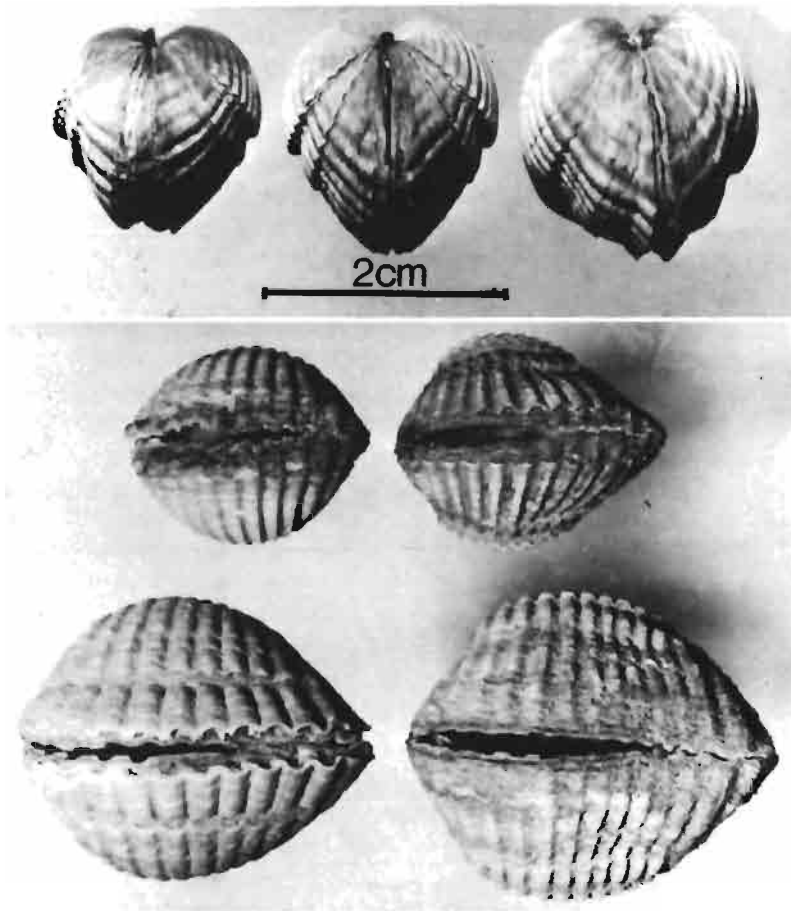


Fig. 13-127: *Cardium glaucum*. Shell pathology caused by *Gymnophallus fossarum* metacercariae. (After Bartoli, 1974a.)

portion of the peripheral extrapallial space next to the insertions of the siphon retractor muscles (Fig. 13-131). In heavily naturally infested *T. decussatus*, in which this preferred site is overpopulated, metacercariae also occur along the ventral margin of the pallial line. A 3.52-cm long clam from Lagune de Beauduc (Camargue), where heavy infestation intensities prevail, yielded a total of 3,137 *G. fossarum* metacercariae, a 4.73-cm host individual even 14,769 (Bartoli, 1973b; Fig. 13-132, Table 13-26 and 13-27).

In *Tapes aureus* (*Venerupis aurea*), *Gymnophallus fossarum* occupies 2 main microhabitats. One is the portion of the peripheral extrapallial space along the insertion of the pallial line on the shell; the other are the outer folds of the mantle edge, particularly the periostracal groove. Presence of metacercariae in the former location causes intense hypertrophy and villous proliferation of the mantle tissue, accompanied by the accumulation of large quantities of organic (conchiolinous?) material, which gradually forces the mantle away from the shell insertion on the pallial line. Larvae present in the periostracal groove and along the border between periostracum and shell cause destruction of the

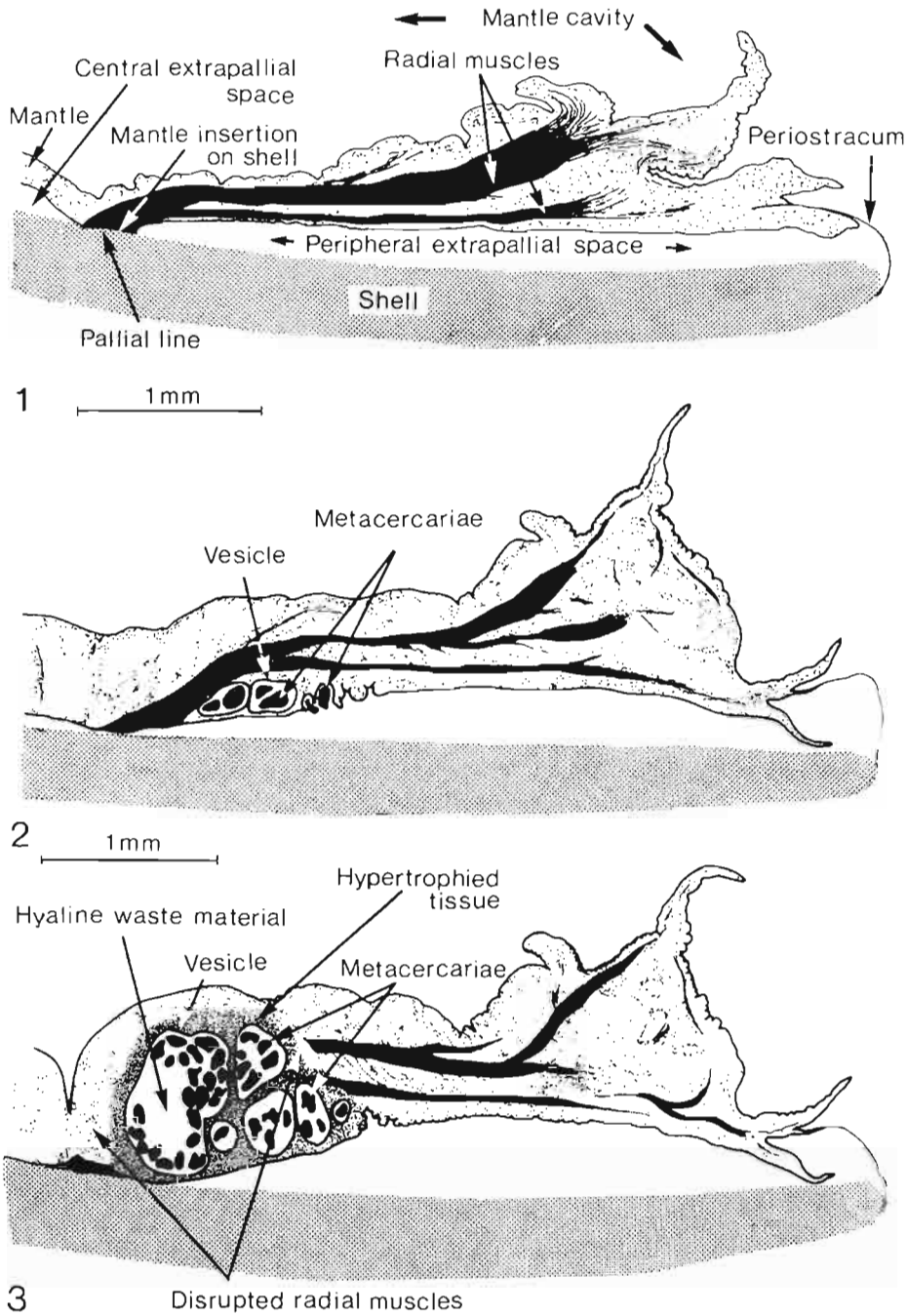


Fig. 13-128: *Tapes decussatus*. 1: Mantle margin of healthy individual; 2: individual lightly parasitized by *Gymnophallus fossarum;* 3: heavily infested individual. Note large vesicles harbouring metacercariae and being partially filled with hyaline waste material. Radial muscles disrupted near insertion on shell. (After Bartoli, 1974a.)

periostracum (Figs 13-133 to 13-135). As a consequence, shell growth ceases. The fluid of the peripheral extrapallial space is now in direct contact with the external medium. Upon subsequent reconstitution of the periostracum, shell growth continues, but due to the parasite-induced regression of the mantle, new shell substance is deposited on the inner surface of the valves instead along the margin (Fig. 13-133).

Recurrent periods of *Gymnophallus fossarum* attack and subsequent shell repair result in stunting and, eventually, entire ceasing of growth in *Tapes aureus*. Heavily infested clams are always smaller than healthy ones (Bartoli, 1976). Similar shell abnormalities — deformation of valve margins and production of 'extra lips' — are of frequent occurrence in various bivalve species, but remained largely unexplained or were attributed to external mechanical impacts. Hallam (1965) discussed various environmental factors responsible for stunting in living marine benthic invertebrates including bivalves and attempted to apply the results to cited instances among fossils. Trematode parasitism as a possible cause of such deviations from normal was not taken into consideration.

In *Tapes aureus,* heavily parasitized by *Gymnophallus fossarum* metacercariae, the large organic deposits situated in the sinusoidal region, and particularly along the ventral side of the posterior adductor muscle, interfere with the proper function of the siphons, forcing them into an approximately 45° ventrad position. This siphonal displacement does no longer enable the clam to maintain its normal burrowing position in the sediment; instead, it must tilt backward in order to maintain proper respiratory contact with the water (Fig. 13-136). Such individuals are normally not entirely burrowed, but expose large portions of their ventral mantle margin. Thereby, foreign material — particularly further *Gymnophallus fossarum* cercariae — may gain easy access to the mantle cavity and mantle

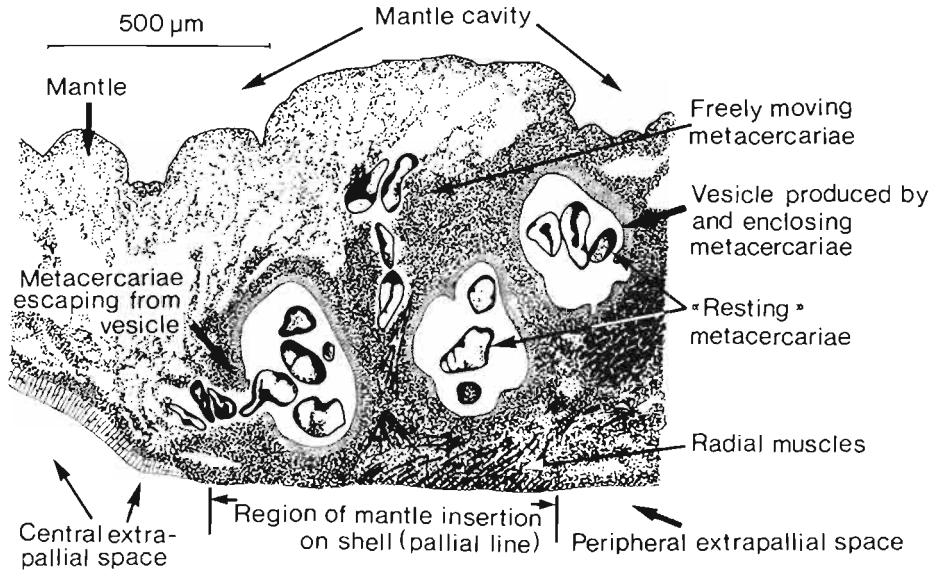


Fig. 13-129: *Tapes decussatus.* Mantle in region of shell insertion heavily parasitized by *Gymnophallus fossarum* (section corresponding to hatched area in Fig. 13-123, 1). Note 'resting' metacercariae enclosed by 'vesicle' formed by host tissue; some escaped from vesicles and moving freely within mantle tissue. (After Bartoli, 1974a.)

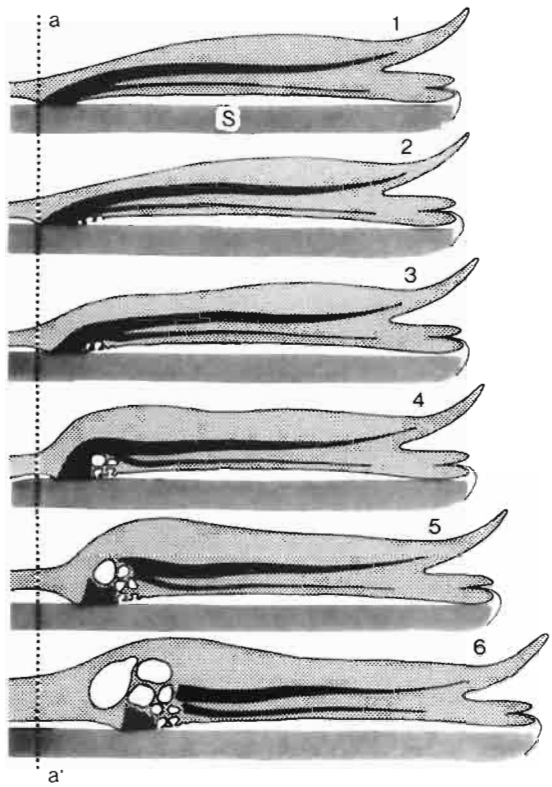


Fig. 13-130: *Tapes decussatus*. Time sequence of pathological alterations in mantle tissue caused by *Gymnophallus fossarum* metacercariae. 1: Healthy individual; 2, 3: few metacercariae present in peripheral extrapallial space, causing production of epithelial proliferations; 4: epithelial vesicles formed and enclosing older metacercariae, radial mantle muscles greatly displaced; 5, 6: metacercarial vesicles increasing in volume, radial muscles disrupted. Note massive hypertrophy of mantle tissue in region of pallial line. Insertion of mantle on shell (line a–a') displaced to the right as host grows. (After Bartoli, 1974a.)

tissues. Furthermore, *Tapes aureus* in this posture may fall an easy prey to oystercatchers, *Haematopus ostralegus*, the main final host of *G. fossarum* (Bartoli, 1976).

Shell lesions, caused by unidentified gymnophallid metacercariae, and similar to those seen in *Tapes aureus* from Camargue, have been observed in *T. pullastra* from Seljehølen, Norway. Infestation commenced when the clams were 1-to 2-year-old. Fifteen percent of the 3-year-old and 75 % of the 9-year-old individuals had lesions, and in those with heavier infestations the soft parts had dirty brown discolourations. Apparently, growth, longevity and burrowing activity were not impaired (Johannessen, 1973). The parasite was not identified; it is larger than *Gymnophallus fossarum*, as indicated by Bartoli (1974a), and may be identical with *'Cercaria scrivenensis'*, a meta(!)cercaria superficially described from *T. pullastra* in Loch Scriven, Scotland, by Lebour (1912).

A similar or identical metacercaria, tentatively identified as *Gymnophallus somateriae*, was found to cause serious shell damage to *Hiatella byssifera* from West Greenland. Infestation probably occurs in the spring. The larvae, which inhabit the extrapallial space, grow from about 0.1 to 0.4 mm in length during the second summer and die during the

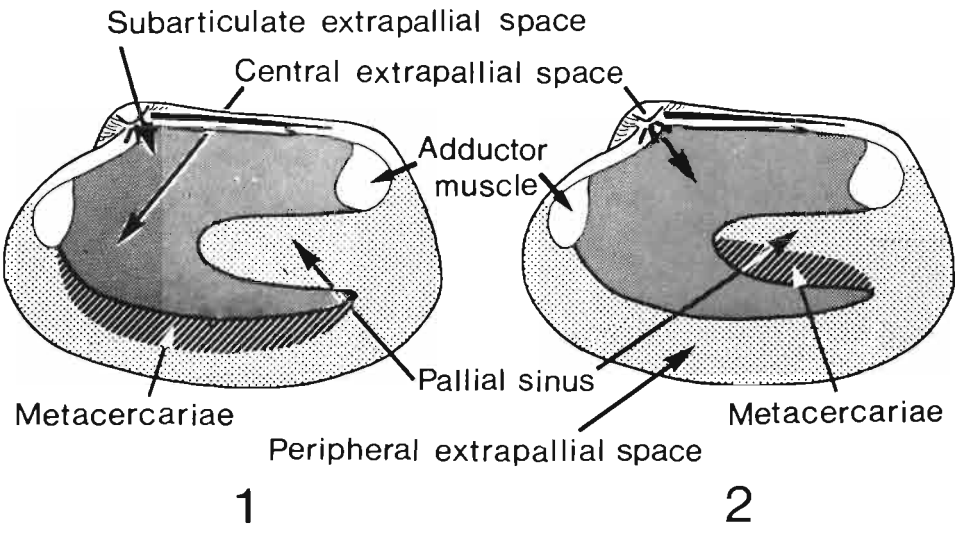


Fig. 13-131: *Tapes decussatus*. Distribution of *Gymnophallus fossarum* metacercariae in peripheral extrapallial space 15 days (1) and 60 days (2) after experimental infestation. (After Bartoli, 1974a.)

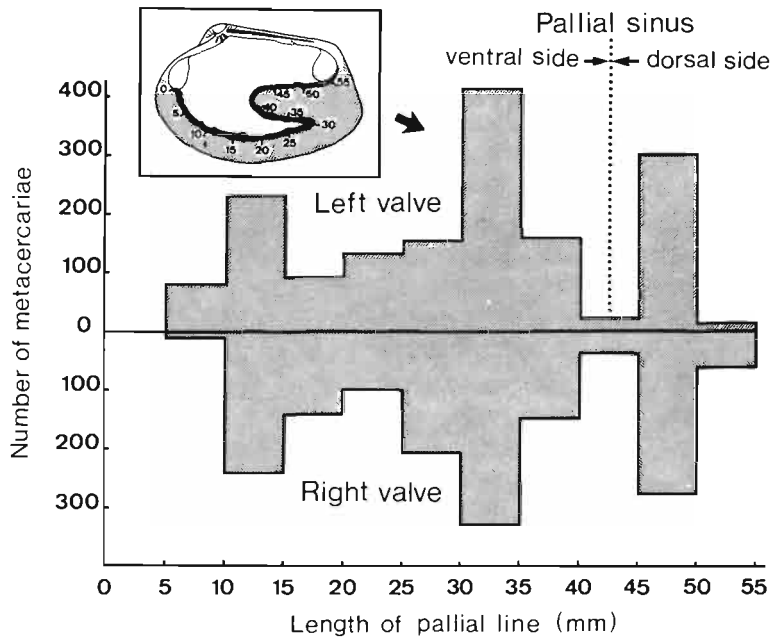


Fig. 13-132: *Tapes decussatus*. Distribution of *Gymnophallus fossarum* metacercariae in peripheral extrapallial space along pallial line in a 35.2-mm long host. (After Bartoli, 1974a.)

Table 13-26
Tapes decussatus. Number of *Gymnophallus fossarum* metacercariae recovered from marginal and sinusoidal portions of peripheral extrapallial space of a 3.52-cm long host (After Bartoli, 1973b)

Peripheral extrapallial space	Right valve	Left valve	Total
Sinusoidal region	862	899	1,761
Marginal region	678	698	1,376
Total	1,540	1,597	3,137

Table 13-27
Tapes decussatus. Number of *Gymnophallus fossarum* metacercariae recovered from peripheral and subarticulate regions of extrapallial space of a 4.73-cm long host (After Bartoli, 1973b)

	Region of extrapallial space		Total
	peripheral	subarticulate	
Right valve	6,919	428	
Left valve	7,422	0	
Total	14,341	428	14,769

third season, their remains becoming calcified. All clams, except the 0-group, were infested, sometimes heavily (Høpner Petersen, 1978).

Some gymnophallid metacercariae are known to cause shell erosion in their bivalve hosts. *Gymnophallus rebecqui* produces protuberances on the outer mantle epithelium of *Abra ovata*, composed of groups of metacercariae and hypertrophied host cells (Fig. 13-124). Each of these fits into a corresponding depression on the inner surface of the valves (Bartoli, 1974a). Similar 'pits' have been observed on the inside of the valves of *Donax vittatus* parasitized by *G. strigatus* metacercariae (Giard, 1897b; Dollfus, 1912), whereas no such structures are produced by the same parasite in *D. trunculus* and *D. semistriatus* (Bartoli, 1974a)*). Pit formation caused by gymnophallids, referred to *G. strigatus* (but supposedly including one or more other species), has also been reported from *Angulus (Tellina) fabula*, *T. tenuis* and *T. solidula* (= *Macoma baltica*) from Boulogne-sur-Mer (France) (Giard, 1897b). Dollfus (1912) believed the depressions to results from the fact that the deposition of new shell material is inhibited at the sites occupied by the metacercariae, whereas it proceeds at normal rates in the shell portions some distance away from these foci. It seems more likely, however, that pit formation is not a passive process but instead a parasite-induced active breakdown of host-shell substance.

What appears to be a 'decalcifying' mechanism has also been reported for other species of larval gymnophallids. Metacercariae of *Lacunovermis macomae*, occurring between the mantle and shell and most often near the umbo, are responsible for pit formation in *Macoma inconspicua* from Vancouver, Canada. The larvae are singly

*) The parasitized donax clam, identified as *D. trunculus* by Giard (1897b), actually belonged to *D. vittatus*, according to Dollfus (in Stunkard, 1959c, footnote p. 674).

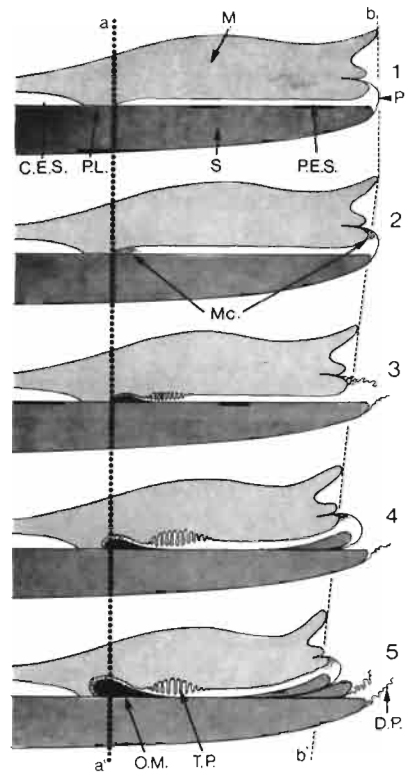


Fig. 13-133: *Tapes aureus*. Pathology caused by *Gymnophallus fossarum* metacercariae. 1: Mantle and shell of healthy host; 2: metacercariae present at 2 sites in peripheral extrapallial space; 3: accumulation of organic material, tissue hypertrophy, disruption of periostracum, shell growth ceasing; 4: periostracum restored, new shell margin ('extra lip') forming on inside of former margin; 5: situation after repeated shell repair, mantle margin distinctly regressed. – C.E.S. central extrapallial space, D.P. disrupted periostracum, M. mantle, Mc. metacercariae, O.M. organic material, P. periostracum, P.E.S. peripheral extrapallial space, P.L. pallial line, S. shell, T.P. tissue proliferation, line a–a' regression of pallial line, line b–b' regression of mantle margin. (After Bartoli, 1974a; modified.)

enclosed by a thin membraneous layer adhering to the shell. Older infestations may be covered by nacre secreted by the host (Ching, 1965).

Parvatrema obscurus lives in pits on the inside of the valves of North American Pacific coast clams *Transennella tantilla,* and *P. borealis* evokes similar effects in Atlantic clams *Gemma gemma.* An unidentified *Parvatrema* metacercaria parasitizes *G. gemma* from San Francisco Bay. Each larva is surrounded by a circular or slightly ovate gelatinous matrix fitting into a corresponding pit. In heavy infestations, the umbonal and more ventral shell area is densely pitted. Often several metacercariae are ensconced in large confluent depressions. Movement of the worms is suggested by the presence of grooved paths terminating in worm-inhabited pits (Obreski, 1968). Similarly, *Meiogymnophallus* sp. (*minutus*?) lives in pits forming on the inner surface of *Cardium edule* valves from the North Sea, and an unidentified larval gymnophallid causes shell erosion in Norwegian *Tapes pullastra* (Lauckner, 1971; Johannessen, 1973).

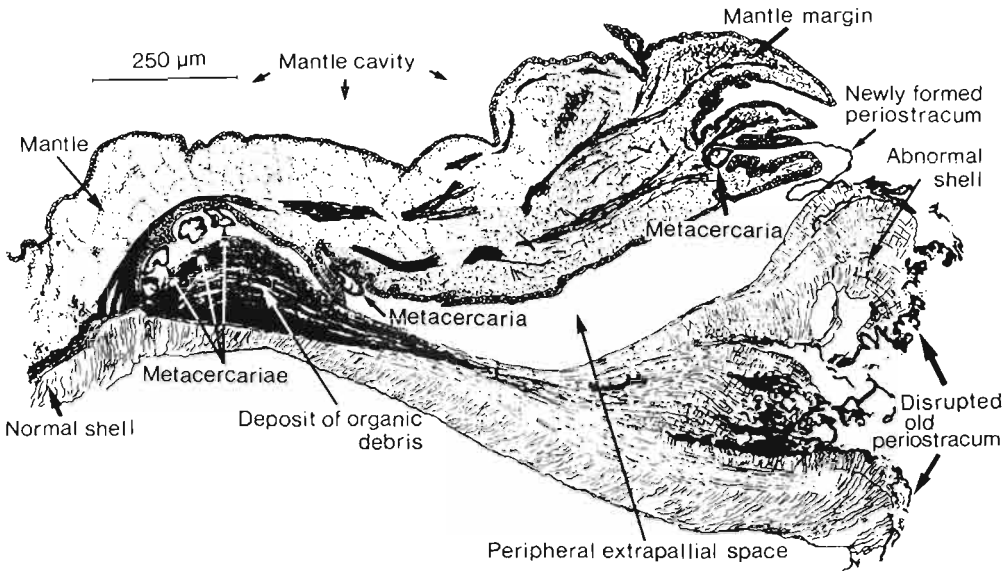


Fig. 13-134: *Tapes aureus.* Pathology of mantle and shell margin of individual heavily parasitized by *Gymnophallus fossarum* (see Fig. 13-123, 3). (After Bartoli, 1974a.)

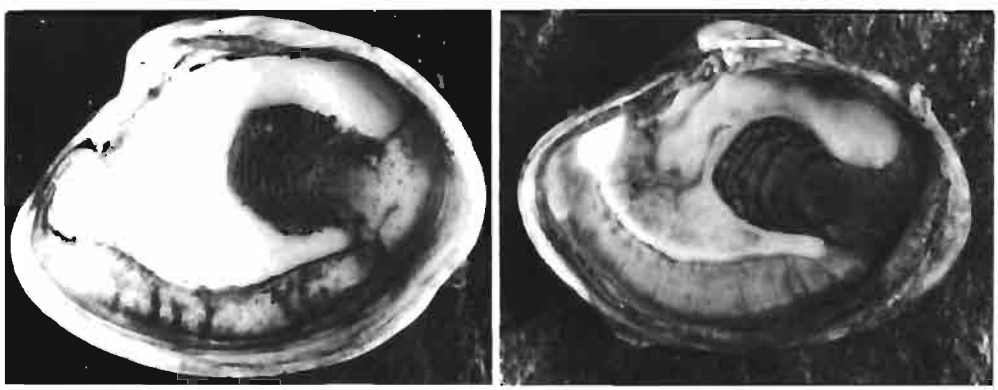


Fig. 13-135: *Tapes aureus.* Abnormal deposition of organic (conchiolinous?) material on inner surface of valves due to presence of *Gymnophallus fossarum* metacercariae in peripheral extrapallial space. (After Bartoli, 1974a.)

Metacercariae of *Meiogymnophallus minutus* occur, entirely enclosed by host tissue, under the umbo of *Cardium edule* (Fig. 13-118). Although not maintaining direct contact with the inner surface of the valves or the extrapallial space, they appear to be capable of causing shell erosion in cockles (Lauckner, 1972). In heavily infested individuals, the older parts of the valves, particularly the umbonal region, are thin and translucent. Some even have perforated umbos. The presence of well-defined radial ribs on the entire outer surface of the valves indicates that the erosion is not due to external factors but that the shell material has been removed *from the inside* of the valves. Stephen (1932) observed similar intense shell erosion in *C. edule* from the Clyde (Scotland) area, but the cockles

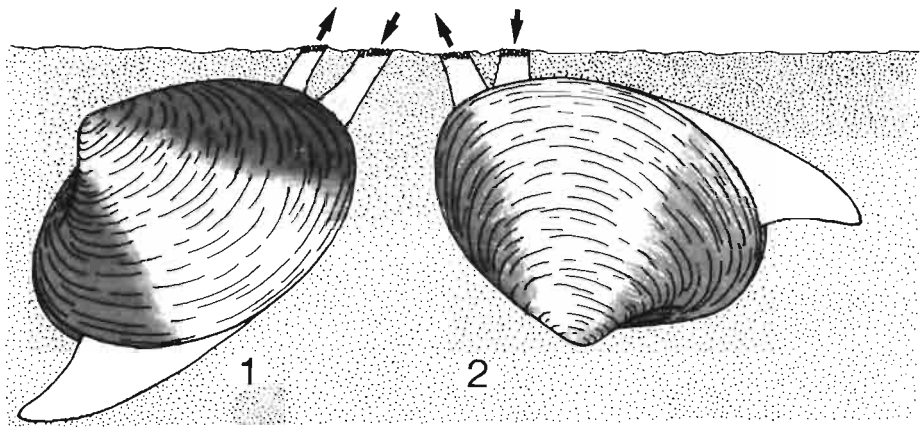


Fig. 13-136: *Tapes aureus*. Effect of *Gymnophallus fossarum* infestation on posture of burrowed host. 1: Normal posture of healthy individual; 2: abnormal posture of heavily infested individual. (After Bartoli, 1974a.)

were not checked for larval trematodes. *Gymnophalloides tokiensis* was found responsible for shell erosion in Japanese *Crassostrea gigas* (Hoshina and Ogino, 1951).

Since the metacercariae exhibit no anatomical capabilities for the physical abrasion of shell, they must either possess some chemical means for dissolving the bivalve's calcium carbonate or stimulate the host to do this. In fact, metacercariae — and in particular gymnophallids — accumulate large amounts of calcium carbonate and store them in so-called 'excretion granules' which appear as masses of dark spherules in transmitted light (white in reflected light), filling the entire lumen of the excretory vesicle (Figs 13-93, 13-115, 2 and 13-119). Although these 'excretion granules' are generally considered as metabolic waste products (Bowers and James, 1967), it has been demonstrated that their inorganic component consists mainly of calcium carbonate (Martin and Bils, 1964; Lauckner, 1972). With respect to their chemical composition and mode of formation, they are very similar to the so-called 'calcareous corpuscles' of cestodes (p. 772), and may serve to buffer acids during passage of the metacercaria through the final host's stomach. Dönges (1969) observed a direct correlation between the amount of calcium carbonate accumulated by developing metacercariae and their infestivity to the final host. The calcium making up the trematode concretions must come from the host's body fluids, and its active uptake against a concentration gradient would necessarily imply certain interferences with the host's metabolism. Whether the bivalve's shell-calcium carbonate is utilized in this process, is not known. Levels of tissue-Ca^{2+} in *Crassostrea gigas* showed great individual variation, but were slightly reduced in oysters infested with metacercariae of *Gymnophalloides tokiensis* (Hoshina and Ogino, 1951). Alteration (elevation) of host-tissue calcium, caused by larval trematodes, has also been observed in other species of molluscs (p. 709). On the other hand, etching or erosion of the inner shell surface has been reported as a normal consequence of anaerobic glycolysis during which shell material is utilized to buffer metabolic acids (Dugal, 1939; Wilbur, 1964; Crenshaw and Neff, 1969). Whether the presence of metacercariae enhances this process is not known.

Effects of metacercariae on other body components of the bivalve host have been

demonstrated for *Gymnophalloides tokiensis* infesting *Crassostrea gigas* in Japan. Heavily infested oysters had a higher water content and reduced levels of glycogen, protein and fat. Growth was also affected (Hoshina and Ogino, 1951). Parasite-induced depletion of body reserves and interference with vital functions may cause general host debilitation and reduced longevity. Thus, survival of tank-held *Tellina tenuis* harbouring metacercariae of *Gymnophallus strigatus* was 50 % after 5 months, while uninfested clams would normally have a survival of 60 to 80 % over a similar period (Trevallion, 1971).

Asymmetrical gaping of the valves — with the larger gap in the anterior region — is characteristic of *Cardium edule* and *C. lamarcki* infested with metacercariae of *G. gibberosus* (p. 721). Whether this gaping results from a specific effect of the parasite on the anterior adductor muscle — i.e., paralyzation caused by metabolic substances or toxins, disruption of muscle fibres by migrating larvae, irritation of the muscle by calcareous concretions dispersed throughout the muscular tissue (Fig. 13-116) — or from general debilitation, remains to be studied. In the literature, shell gaping in bivalves is usually considered to be associated mainly with post-spawning emaciation. Survival of cockles heavily infested with *G. gibberosus* is substantially reduced (Lauckner, unpubl.).

Infestation with sporocysts and metacercariae of *Parvatrema affinis* was found to alter the behaviour of *Macoma baltica*. Normally, these bivalves live deeply burrowed in the sediment. On Dutch tidal flats, certain individuals were seen to produce conspicuous crawling tracks on the sediment surface during low tide (Fig. 13-137). Subsequent examination revealed 100 % *P. affinis* infestations in these clams, as opposed by incidences of 13 % and 5 % in samples collected at random from below the sediment surface. The

Fig. 13-137: *Macoma baltica*. Crawling tracks on sediment surface produced by individuals infested with *Parvatrema affinis*. (After Swennen, 1969.)

tracks were found only at higher tide levels and occurred from December to the end of May, being most numerous in the spring. It was concluded that the presence of trematode infestations provided the stimulus for this abnormal behaviour (Swennen, 1969; Hulscher, 1973; Swennen and Ching, 1974).

Surfacing and track-digging has hitherto been interpreted as part of the normal behaviour of *Macoma baltica*, associated with foraging activities or displayed in response to oxygen deficiency (Brafield, 1963; Newell, 1979). Analysis of the looped tracks showed that most of the clams, exposed by the tide for about 1 h, were photopositive and moved toward the sun, but later, after 5 h exposure, became photonegative. This behaviour was believed to represent a means limiting the dispersal of *M. baltica* (Brafield and Newell, 1961). A parasitological survey of the clams was not undertaken.

Cardium edule and *Venus striatula* have been found to display a similar behaviour, interpreted as a directional response to light (Newell, 1979). But cockles from tidal flats at Sylt (German North Sea coast), crawling on the sediment surface, invariably showed significantly higher incidences of *Gymnophallus choledochus* infestation than normally burrowed individuals collected at random (Lauckner, unpubl.). Surfacing has also been observed in *Tapes decussatus* and *T. pullastra* from the French Atlantic coast, infested with sporocysts and cercariae of *Bacciger bacciger* (Jobert, 1894).

Although parasite-induced behavioural changes in intermediate hosts, most of which increase the chance of predation by the final hosts, are well documented (Holmes and Bethel, 1972), the underlying physiological mechanisms responsible for the abnormal behaviour of trematode-infested bivalves are as yet unknown. Interestingly, oystercatchers *Haematopus ostralegus* that feed to a great extent on *Macoma baltica*, sometimes reject individuals parasitized by *Parvatrema affinis* (Hulscher, 1973). It has been suggested that surfacing occurs in response to oxygen deficiency in infested hosts, due to the accelerated metabolic rate of the densely packed metacercariae within the tissues (Dineen in Swennen and Ching, 1974). In fact, oxygen uptake was higher in *Tellina tenuis* infested with metacercariae of *Gymnophallus strigatus* than in healthy individuals (Trevallion, 1971). On the other hand, surfacing may occur as an unspecific response to general debilitation or emaciation, since it is also observed in bivalves dying of causes other than trematode infestation.

The most conspicuous reaction of marine bivalves to invasion by larval gymnophallids is the formation of pearls. It is the result of the host's cellular response to tissue-invading foreign bodies in an attempt to wall off an intruder which, for whatever reason (size, resistance), cannot be phagocytized. Pearls may form around various foreign material including dead cestode larvae (see section 'Agents: Cestoda') or, possibly, gregarine spores (see section 'Agents: Apicomplexa'), but never around living trematode metacercariae, as assumed by Cheng and Rifkin (1970) and others. Pearl formation is essentially an abnormality and will be considered in detail under this heading (see section 'Abnormalities').

Bivalves as Final Hosts for Digenea

Typically, adult digenetic trematodes are parasites of vertebrates, but some species attain sexual maturity in invertebrates, mostly in molluscs. Digenean life cycles of this type are usually termed 'abbreviated' and regarded as representing an abnormal condition. As

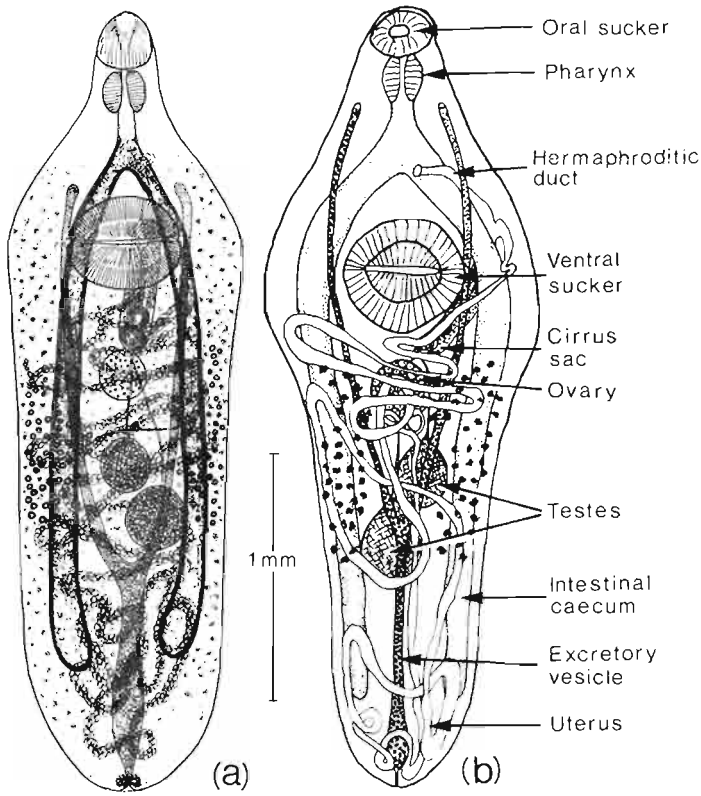


Fig. 13-138: *Proctoeces maculatus*. Adult worms from *Scrobicularia plana* (a, dorsal view) and *Mytilus edulis* (b, ventral aspect). (a after Loos-Frank, 1969c, b after Lang and Dennis, 1976.)

Table 13-28

Mytilus edulis. Incidence and intensity of infestation with adult *Proctoeces maculatus* in mussels from Shark River (New Jersey) in relation to host size (After Lang and Dennis, 1976)

Host-size class (mm)	'Dock' station No. of mussels	% infested	'Jetty' station No. of mussels	% infested	Average number of worms*
15.0–19.9	27	18.5	91	11.0	1.5
20.0–24.9	65	8.3	114	12.3	2.4
25.0–29.9	64	15.6	93	6.4	2.6
30.0–34.9	107	18.7	115	5.2	2.4
35.0–39.9	84	5.9	48	16.7	4.4
40.0–44.9	73	11.0	25	4.2	4.2
45.0–49.9	56	5.3	5	0	1.0
> 50.0	58	8.6	2	100.0	12.3

* Total number of worms recovered/number of infested hosts.

noted earlier, there can be little doubt, however, that originally the digeneans were parasites of molluscs, and that non-molluscan hosts have been adopted subsequently (Stunkard, 1957, 1959a, b, 1967, 1970b; Wright, 1960, 1966). In some of these 'primitive' life cycles the true picture may be obscured by the fact that fishes may act as additional or 'post-cycle' hosts. Fellodistomids of the genus *Proctoeces* are among the few species which employ gastropod (Vol. I, Chapter 12) and bivalve molluscs as definite hosts. Most of the reported *Proctoeces* spp. are probably specifically identical and referable to *P. maculatus* (Looss, 1901) Odhner, 1911.

During his studies on *Cercaria milfordensis*, infesting *Mytilus edulis* from Connecticut waters, Uzmann (1953; see p. 666) discovered several 'unencysted progenetic trematodes', which contained many eggs with well developed, motile miracidia. The worms, which were found free in the visceral mass of 3 mussels bearing *C. milfordensis* infestations, were interpreted as progenetic metacercariae. But 'progenetic metacercariae' *sensu stricto* are

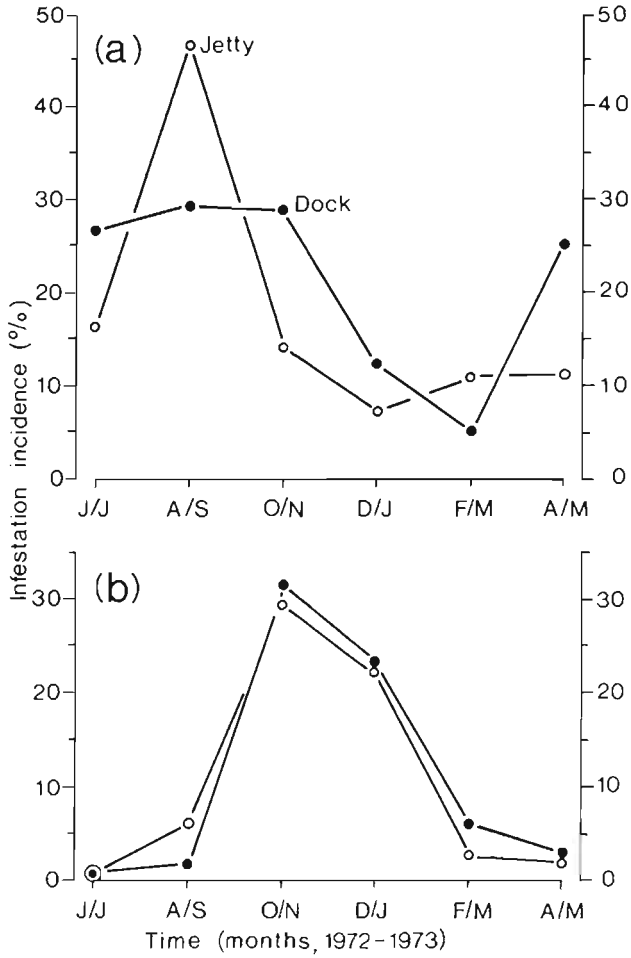


Fig. 13-139: *Mytilus edulis*. Seasonal incidence of infestation with larval (a) and adult (b) *Proctoeces maculatus* at 2 sites ('Dock', 'Jetty') in Shark River (Belmar, New Jersey). (After Lang and Dennis, 1976.)

normally encysted, exhibit subadult morphological characteristics and, in many if not most cases, can only attain the definite structure of the adult after transfer to a compatible vertebrate host. Subsequently, Stunkard and Uzmann (1959) demonstrated the existence of unencysted metacercariae and developmental stages from cercariae to gravid adults in the mussel. The fact that the eggs were misshapen in some individuals and the 'progenetic' worms were often not entirely normal was believed to reflect the abnormal condition of development in the molluscan host. On the basis of the morphology of the adult (Fig. 13-138), the worms from *M. edulis* were identified as *Proctoeces maculatus*. The species has originally been described from the intestine of labrid fishes *Labrus merula, Crenilabrus pavo* and *C. griseus* (= *C. cinereus*) from the Adriatic Sea by Looss (1901). Dennis and co-authors (1974) and Lang and Dennis (1976) found post-cercarial stages of *P. maculatus* in both *M. edulis* containing sporocysts and in mussels without sporocysts. Hence, the cercariae are, in spite of their lack of natatory ability, capable of invading new hosts. Unencysted metacercariae were found in the kidney and foot sinuses of the mussel, while the preferred sites of the adult stage were the heart and often the kidney.

Inspection of *Mytilus edulis* from 2 sampling stations ('Dock' and 'Jetty') in the Shark River (New Jersey, USA) yielded adult *Proctoeces maculatus* in 47 (10.5 %) of 493 'Jetty' mussels. Infestation intensities ranged from 1 to 6 worms per host (mean 0.2). At the 'Dock' station, 61 (11.4 %) of 534 mussels carried from 1 to 35 adult *P. maculatus* (mean 0.5). Of the 1,027 *M. edulis* examined, 18 contained live adults and sporocysts, 9 harboured dead adults and sporocysts, 81 had adults only, and 160 contained sporocysts only. Unlike sporocyst infestations (see p. 667), prevalences of adult worms did not increase with host size, whereas intensities did (Table 13-28). The prevalence of adult infestations showed a similar pattern at both sites and was distinctly seasonal, with peaks of about 30 % occurring in October-November, and lagging somewhat behind the maximum in the abundance of sporocyst infestations (Fig. 13-139). The incidence of adult *P. maculatus* dropped sharply between January and February, and by late May no adults were found. It seems likely that, once established, sporocysts of *P. maculatus* persist in *M. edulis* until host death, while adult infestations terminate with the death of the worms, presumably on a yearly cycle. As can be deduced from the succession of larval and adult generations (Fig. 13-139), the mature worms release eggs throughout the autumn and winter, and then die during the coldest months. Mature sporocysts that persist through the winter contribute some new adult infestations early in the spring, but substantial adult infestations do not reappear until October-November.

In addition to *Mytilus edulis, Proctoeces maculatus* employs a number of other bivalve and gastropod species as definite host. All of these worms have been described under different names. Freeman and Llewellyn (1958), Freeman (1962, 1963a, b) and White (1972) considered the features exhibited by adult *Proctoeces,* obtained from the kidney of *Scrobicularia plana* in British waters, to be consistent with the description of *P. subtenuis* (Linton, 1907) Hanson, 1950, a species previously reported from the hind gut of labrid and sparid fishes from tropical and subtropical waters (Linton, 1907; Manter, 1947; Hanson, 1950; Manter and Pritchard, 1962). As emphasized by Stunkard and Uzmann (1959) and Manter and Pritchard (1962), *P. subtenuis* is a junior synonym of *P. maculatus.* Dolgikh (1967) reported ovigerous 'progenetic metacercariae' of *P. maculatus* from the body cavity of *Rissoa splendida* in the Black Sea. '*P. major* (?)', occurring in the same host species, may represent a larger individual of *P. maculatus.* Bray and Gibson (1980) found

the parasite in *Thais* (*Nucella*) *lapillus* from Devon, England, and Dollfus (1965, 1966) described adult worms (which he, however, regarded as progenetic metacercariae) from gastropods *Gibbula umbilicalis* on the Atlantic coast of Morocco as *P. progeneticus* (Vol. I, p. 386). He also (1965) published a list of previous records of adult and 'progenetic' *Proctoeces* spp. occurring in marine fishes and invertebrates. Loos-Frank (1969c) found adult *Proctoeces* in *Scrobicularia plana* and prosobranchs *Buccinum undatum* (Vol. I, p. 385) from the German North Sea coast which she — after comparison with all other known adult forms — regarded as new species. These were named *P. scrobiculariae* and *P. buccini*, respectively.

On the basis of their extensive comparative studies, Bray and Gibson (1980) concluded that all of these trematodes are identical with *Proctoeces maculatus*, the type species of the genus. They furthermore assumed that non-ovigerous metacercariae obtained from the digestive gland of *Brachidontes senhausi* by Yamaguti (1938) and from between the epipodium and mantle of an abalone *Haliotis discus hannai* by Shimazu (1972), as well as ovigerous adult *Proctoeces* sp., dissected from the renal coelom of topshells *Batillus* (*Turbo*) *cornutus* by Ichihara (1965) in Japanese waters, are likewise referable to *P. maculatus*. Shimura and Egusa (1979a, b) and Shimura (1980), however, regarded *Proctoeces* sp. from *B. cornutus* as a separate species and named it *P. ichiharai*.

If Bray and Gibson's (1980) opinion is correct, *Proctoeces maculatus* must be a variable species occurring in widely dissimilar hosts. Perhaps its variability is the result of a wide host tolerance. Morphological comparison of worms from different hosts, relying upon the measurement of body dimensions, has, thus far, met with little success due to the above-mentioned inadequacy of morphometric procedures employed in helminthology. Even if one accepts Bray and Gibson's (1980) view, a number of questions remain. While in the Atlantic and adjacent waters *P. maculatus* (?) occurs in a wide variety of unrelated prosobranch and lamellibranch hosts — members of the orders Anisomyaria, Eulamellibranchiata, Archaegastropoda, Mesogastropoda and Neogastropoda — *P. ichiharai* in the Pacific infests archaegastropods only. On the English and German North Sea coasts, *P. maculatus* (*sensu stricto*) displays marked host preference. At Chalkwell (Essex, England), every one of about 1,000 *Scrobicularia plana* was found to be infested with an average of between 4 and 5 adult worms, but *Macoma baltica* and *Mya arenaria* occurring alongside *S. plana* on the Chalkwell mud flats were never infested (Freeman and Llewellyn, 1958). Similarly, Loos-Frank (1969c) found from 1 to 4 worms in 88 of 142 *S. plana* at Langwarden (German North Sea coast), but none of numerous individuals of *Mytilus edulis, Cardium edule* and *M. baltica* harboured a single *P. maculatus*. The factor(s) determining the host preference of adult *P. maculatus* in one case and the apparent lack of host preference in the other case remain to be studied experimentally.

The records of adult *Proctoeces maculatus* from the intestine of marine fishes also require further attention. It is difficult to believe that one and the same trematode should exclusively employ molluscs as definite hosts in higher-latitude waters and teleosts in subtropical and tropical waters. In the reviewer's opinion it appears more likely that fishes merely become 'accidentally' infested by ingesting molluscs harbouring adult worms and, hence, must be regarded as additional or 'post-cycle hosts'. This assumption is substantiated by the fact that adult *P. maculatus* survive for only a few days in fishes, but for more than 12 months in molluscs.

Although the specific identity of most of the above-mentioned *Proctoeces* spp.,

particularly of those from the Pacific, with *P. maculatus* requires conclusive confirmation, those from *Scrobicularia plana, Buccinum undatum* and *Thais lapillus* in Europe definitely belong to that species. One reason that led Loos-Frank (1969c) to regard '*P. buccini*' and '*P. scrobiculariae*' as distinct from *P. maculatus* was the fact that only a few (i.e., 5 of 93) *B. undatum,* collected on the German North Sea coast, were infested but that, if an infestation occurred, the number of worms (i.e., 30 to 180) per whelk was high. From this observation the author concluded that the entire life cycle of '*P. buccini*' is confined to *B. undatum.* With respect to '*P. scrobiculariae*' she found a more uniform distribution of the worms in the host population, 88 of 142 *S. plana* being infested with 1 to 4 worms per clam, and reasoned that an intermediate host must be involved in the life cycle of this trematode. Taking into account the feeding biology of *B. undatum* and *S. plana*, the differences in infestation pattern are easily explainable: The whelk is a carnivore feeding on invertebrates and dead fish. It acquires its worm burden by ingesting *Mytilus edulis* harbouring sporocysts, cercariae, metacercariae and adults of *P. maculatus.* Since each infested mussel usually contains hundreds of immature (and, in addition, a number of adult) worms, *B. undatum* may become heavily parasitized by devouring a single infested mussel. As the percentage of infested *M. edulis* is obviously low in the area under consideration, only a few whelks become parasitized. *S. plana*, on the other hand, lives deeply burrowed in the sediment and feeds on matter which it collects from the sediment surface by means of its long, protrusible siphons. *P. maculatus* cercariae (or metacercariae) dispersed in low numbers on the sea bottom, are 'pipetted' by the ingestion siphon along with the clam's food.

In the latter case, the distribution of *Proctoeces maculatus* in the *Scrobicularia plana* population should theoretically follow the negative binomial or one of the other empirical 'contagious' distributions (i.e., the Neyman Type A, B or C, or the Polya-Aeppli distribution), which are characteristic of the dispersal of parasites within their host

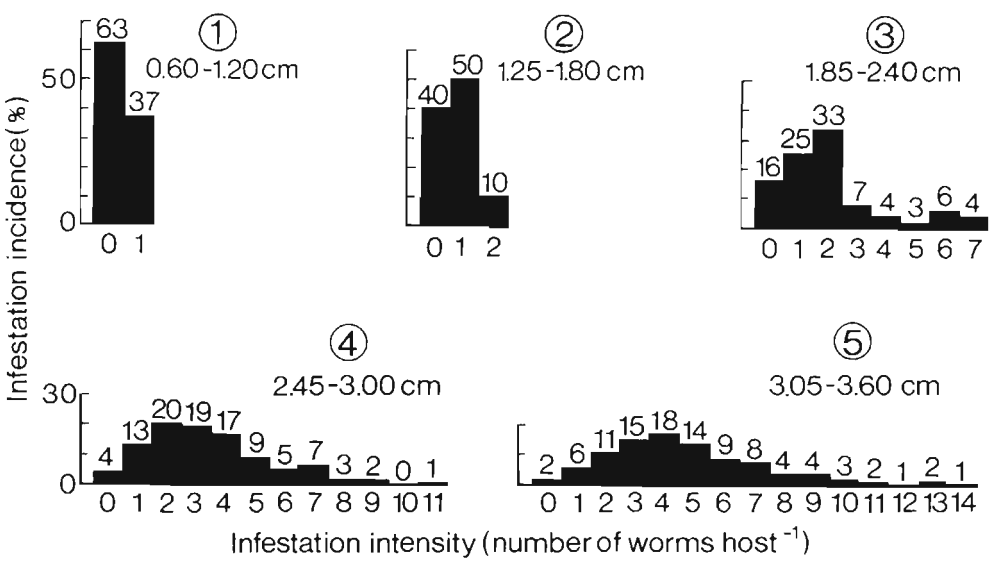


Fig. 13-140: *Scrobicularia plana.* Frequency distribution of adult *Proctoeces maculatus* in 5 arbitrary host-length groups (see Table 13-29). (After White, 1972; modified.)

Table 13-29
Scrobicularia plana. Frequency distribution of observed (O) and expected (E) occurrences of *Proctoeces maculatus* in 4 arbitrary host-length groups (Data recalculated from White, 1972; Fig. 13-140)

Host-length group	Number of parasites	O*)	E	$\bar{x}$	s^2	χ^2	p
①	0	100.8	100.9	0.37	0.23	0.0016 (0.00024)	0.94 (0.97)
	1	59.2	58.9				
②	0	64.0	65.0				
	1	80.0	77.2	0.70	0.41	0.58 (0.38)	0.45 (0.54)
	2	16.0	18.9				
④	0	6.4	7.5				
	1	20.8	20.2				
	2	32.0	29.4				
	3	30.4	30.8				
	4	27.2	25.9	3.47	4.50	6.79 (4.21)	0.56 (0.84)
	5	14.4	18.6				
	6	8.0	11.9				
	7	11.2	6.9				
	8	4.8	3.7				
	9	3.2	1.8				
⑤	0	3.2	2.3				
	1	9.6	9.1				
	2	17.6	18.6				
	3	24.0	27.7				
	4	28.8	27.1	4.32	4.67	4.97 (3.06)	0.76 (0.93)
	5	22.4	23.3				
	6	14.4	17.0				
	7	12.8	10.8				
	8	6.4	6.1				
	9	6.4	3.1				

*) = numbers above columns in Fig. 13-140 multiplied by factor 1.6. For explanation see text.

population. Such a consistency has as yet not been demonstrated for *P. maculatus* but may, as illustrated below, be deduced from the information available in the literature. White (1972) has indicated the frequency distribution of *P. maculatus* in 5 size groups of *S. plana* (Fig. 13-140). The range as well as the mean number of parasites per host increase with shell length, the maximum number recorded in a single host being 14 and the mean number increasing from 0.37 in Group 1 to over 4 in Group 5 (Table 13-29). Regrettably, White (1972) did not submit his data to statistical analysis. In a re-evaluation of his data, conducted by the reviewer, a few simplifying assumptions had to be made: In White's graph (Fig. 13-140) columns represent percentages rather than absolute numbers (the numbers above the columns, indicating the respective percentages, have been added for convenience). The total number of *S. plana* in White's sample was n = 806, divided into 5 arbitrary shell-length groups. Assuming equality of the 5 groups, each will contain 806/5 = 161 clams. For calculation purposes, the percentages indicated in Fig. 13-140 have, therefore, been multiplied by 1.6 to yield quasi-absolute values. These (column 'O' in

Table 13-29) have been compared to the expected values (column 'E') of the negative binomial distribution. (Frequencies in classes > 9 have been excluded from the computations in order to avoid errors possibly associated with the low O's at the right-hand ends of the strongly tailed distributions No. 4 and 5). Group 3, which is obviously heterogenous with respect to the distribution of *P. maculatus*, and probably represents a mixture of 2 age classes of *S. plana*, has been omitted from the calculations. In the remaining 4 samples, the agreement between observed and theoretically expected occurrences is immediately apparent and verified by the χ^2 test. Substitution of the calculated 'O' values in Table 13-29 by the numbers (percentages) given in Fig. 13-140 yields even lower χ^2's and larger p's, indicating a still closer fit of the data to the negative binomial distribution. The respective figures are added in parantheses (columns 'χ^2' and 'p' in Table 13-29). This re-evaluation of White's (1972) field data exemplifies the eminent suitability of the *S. plana-P. maculatus* association for basic studies of host-parasite systems. Beyond doubt, the examination of White's original data (i.e., the absolute numbers of parasites in individual clams) would have permitted an even deeper insight into the dynamics and structure of the *P. maculatus* population under consideration, particularly with respect to his heterogenous shell-length group number 3.

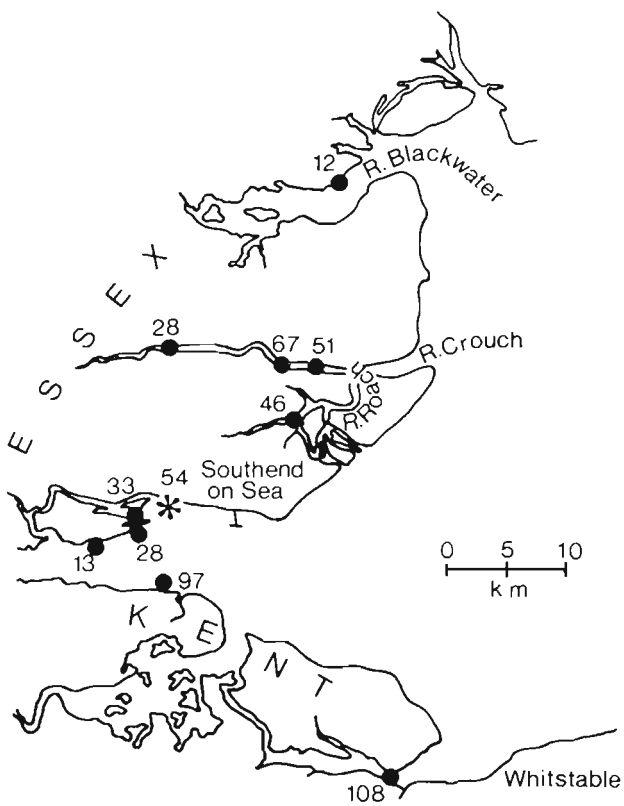


Fig. 13-141: *Proctoeces maculatus*. Distribution in the Thames estuary and surrounding area. ● *Scrobicularia plana*, uninfested; ✻ *S. plana* with adult *P. maculatus* infestation. Numbers indicate host-sample size. (After White, 1972.)

In their studies on *Proctoeces maculatus* (misidentified as *P. subtenuis*) in Britain, Freeman and Llewellyn (1958) observed an extremely localized occurrence of the parasite. They found an average of 4 to 5 worms in everyone of approximately 1,000 *Scrobicularia plana* from Chalkwell on the north coast of the Thames estuary, and single worms in 3 of about 150 *S. plana* from Whitstable on the opposite bank of the Thames (Fig. 13-141). In the severe winter of 1962-63, *S. plana* was eliminated from the area where it had previously been found infested with *P. maculatus*; but on its return a build-up of infestation was observed (Freeman, 1963b; Bray and Gibson, 1980). Examination of numerous *S. plana* from various localities in Essex, Devon, Wales and Suffolk, performed by Freeman and Llewellyn (1958), yielded constantly negative results. Subsequently, however, sexually mature *P. maculatus* have been found by White (1972) in *S. plana* collected from Dawlish Warren in the estuary of the River Exe, Devon; but specimens from that locality, examined more recently by Bray and Gibson (1980), were again uninfested. Worms, if present, are easily detected due to their conspicuous pink to distinctly red colouration and, hence, can hardly be overlooked.

Freeman and Llewellyn (1958) hypothesized about the strict localization of *Proctoeces maculatus* in the Thames estuary, and suggested that it might be a fairly recent

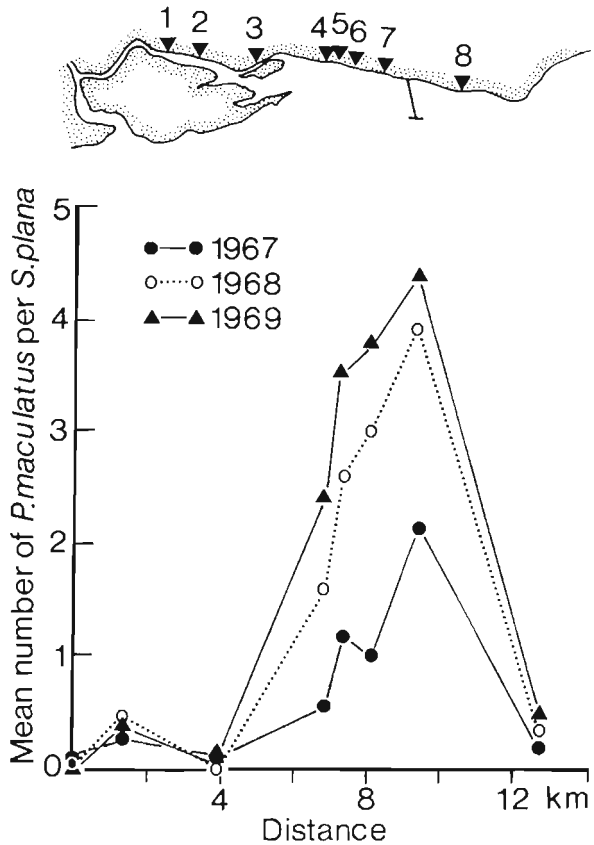


Fig. 13-142: *Proctoeces maculatus*. Distribution of adult worms in *Scrobicularia plana* along north coast of Thames estuary. (After White, 1972.)

Table 13-30
Scrobicularia plana. Incidence and intensity of *Proctoeces maculatus* infestation at sites indicated in Fig. 13-142. (After White, 1972)

Site No.	1967			1968			1969		
	No. of *S. plana* examined	% infested by *P. maculatus*	Mean no. of *P. maculatus* per *S. plana*	No. of *S. plana* examined	% infested by *P. maculatus*	Mean no. of *P. maculatus* per *S. plana*	No. of *S. plana* examined	% infested by *P. maculatus*	Mean no. of *P. maculatus* per *S. plana*
1	20	5.0	0.05 ± 0.05	38	2.6	0.026 ± 0.026	42	Nil	–
2	20	20.0	0.25 ± 0.12	30	33.3	0.46 ± 0.14	47	23.4	0.32 ± 0.092
3	28	7.1	0.07 ± 0.05	39	Nil	–	63	1.58	0.016 ± 0.016
4	40	42.5	0.57 ± 0.12	38	68.4	1.60 ± 0.22	47	91.5	2.40 ± 0.26
5	10	70.0	1.20 ± 0.42	43	97.6	2.59 ± 0.21	54	94.4	3.52 ± 0.31
6	32	65.9	1.00 ± 0.20	42	95.2	3.00 ± 0.31	50	92.0	3.78 ± 0.31
7	26	92.3	2.15 ± 0.28	55	100	3.90 ± 0.29	47	98.0	4.40 ± 0.42
8	12	16.6	0.17 ± 0.11	37	24.4	0.30 ± 0.094	54	40.7	0.50 ± 0.10

addition to the British fauna, possibly accidentally introduced via an appropriate fish final host or an infested first intermediate host. As emphasized by Freeman (1963a), however, *P. maculatus* has never been recorded from a fish in British waters. Unfortunately, Freeman and Llewellyn (1958), obviously unaware of Uzmann's (1953) description of *Cercaria milfordensis* (the larval stage of *P. maculatus*), did not inspect *Mytilus edulis*, the first and second intermediate host, in their study area.

White (1972), who extended Freeman and Llewellyn's (1958) investigation into a 3-year study of the ecology of *Proctoeces maculatus*, confirmed the previous authors' findings of an extreme localization of the parasite. Infested *Scrobicularia plana* were only found around Chalkwell (Essex), but not at any of 10 neighbouring stations on the Essex and Kent coasts (Fig. 13-141). At Chalkwell, *P. maculatus* was first recorded in 1954 when Freeman and Llewellyn (1958) started their field work on that parasite. When White (1972) terminated his collections in 1970, it became apparent that *P. maculatus* had maintained a stable population, for at least 16 years, around Chalkwell, but had not spread to adjacent areas. Along the coast to the east or west of Chalkwell, the parasite was found to display a distinct spatial pattern which was repeated, with only minor variation, in 3 consecutive years (Fig. 13-142; Table 13-30). The pronounced variation in incidence and intensity of infestation over short distances — sometimes less than a mile — remained unexplained. It was suggested, however, that this could have been due to the lack of 'dispersal power' on behalf of the larval stages and to a more restricted distribution of the intermediate host. Unfortunately again, White (1972), although making reference to the paper of Stunkard and Uzmann (1959), did not examine *Mytilus edulis* in order to discover the pre-adult stages of the parasite. Simultaneous monitoring of the prevalence of sporocysts and cercariae in mussels from the north coast of the Thames estuary would have contributed to a far better understanding of the dynamics of the *P. maculatus* population in the Chalkwell area.

During White's (1972) 3-year study, *Proctoeces maculatus* proved to be a highly successful parasite. At most of the 8 collecting sites the incidence as well as the intensity of infestation increased considerably with time, the intensity also with host size (Figs 13-142

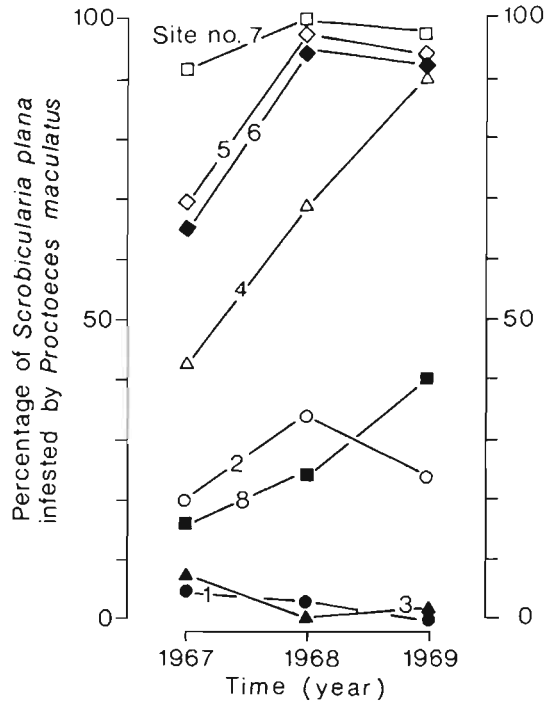


Fig. 13-143: *Scrobicularia plana*. Changes in incidence of infestation with adult *Proctoeces maculatus* in clams from north coast of Thames estuary (Sites as indicated in Fig. 13-142). (Original; based on data presented in Table 13-30.)

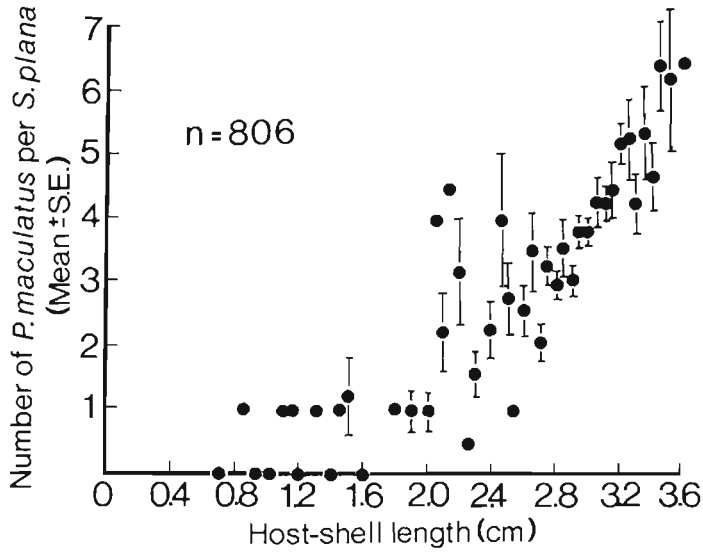


Fig. 13-144: *Scrobicularia plana*. Relation between host-shell length and number of adult *Proctoeces maculatus* in clams from Site 7 in Fig. 13-142. Standard errors of mean plotted for sample sizes ≧ 4. (After White, 1972.)

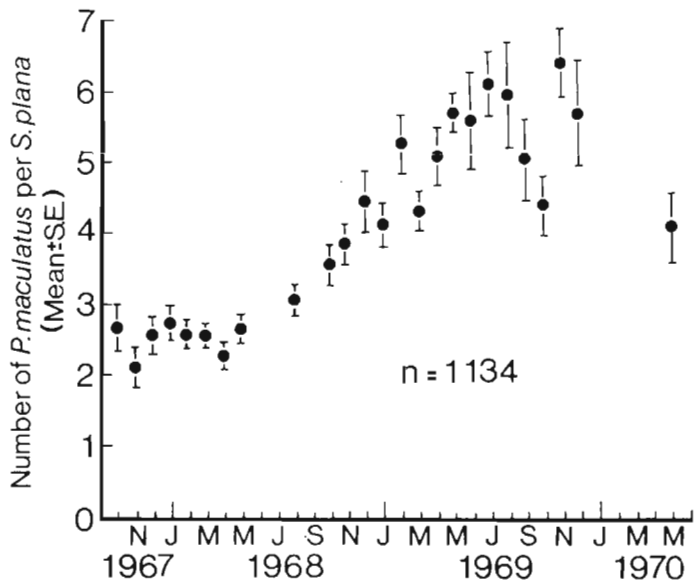


Fig. 13-145: *Scrobicularia plana*. Changes in intensity of infestation with adult *Proctoeces maculatus* in clams from Site 7 in Fig. 13-142. (After White, 1972.)

to 13-145; Table 13-30). There was no marked seasonal variation comparable to that reported by Lang and Dennis (1976) for the abundance of *P. maculatus* in *Mytilus edulis*. Loos-Frank (1969c), however, noted reduced reproductive activity — i.e., atrophied testes and the presence of unembryonated eggs during the second half of the winter — in worms from *Scrobicularia plana* on the German North Sea coast. An interesting fact is the frequent occurrence of dead but preserved *P. maculatus* within the kidney of *S. plana*. At Site 7, the incidence of clams with dead parasites increased from less than 10 % in 1968 to over 30 % in 1969, with a maximum number of 4 worms recorded from a single host (Fig.

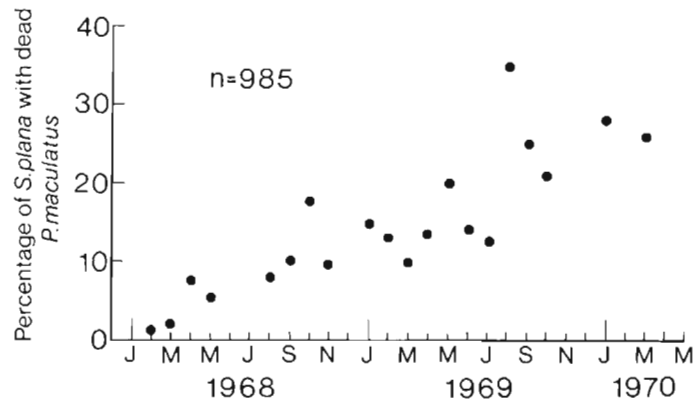


Fig. 13-146: *Scrobicularia plana*. Occurrence of dead preserved adult *Proctoeces maculatus* in clams from Site 7 in Fig. 13-142. (After White, 1972.)

13-146). The observed increase closely paralleled that of the overall infestation level. The dead worms always occurred side by side with living ones in the same host individual. Similar conditions have also been reported by Freeman and Llewellyn (1958) and Loos-Frank (1969c). Dead specimens varied in colour between light brown and almost black, and also varied in texture, the darker ones being harder and literally 'petrified'. The fact that they did not disintegrate *post mortem* suggests that the bivalve kidney is a sterile environment. As the body fluids of bivalves are saturated with calcium (Potts and Parry, 1964), it is not surprising that dead worms become calcified.

Nothing has been reported on the pathology of adult living or dead *Proctoeces maculatus* in *Mytilus edulis, Scrobicularia plana* or any of the other definite hosts. Dennis and co-authors (1974) merely state that the epithelial cells of the *S. plana* kidney bordering parasites present in the lumen exhibit a slight increase in acid phosphatase activity. The fact that, in the Chalkwell area, *P. macultus* has for many years maintained a high abundance, apparently without affecting the population structure and density of *S. plana*, suggests a low overall pathogenicity of the adult parasite. The ability to complete its entire life cycle in a single invertebrate host, *M. edulis*, renders *P. maculatus* ideally suitable for qualitative and quantitative studies of host-parasite interrelationships. Its apparent lack of 'dispersal power', together with the presumed low pathogenicity, also suggests its usability for experimental field studies. Considering the obviously narrow range of ecological conditions under which *P. maculatus* is capable of dissemination, the artificial introduction of the parasite into secluded marine ecosystems should be expected to represent a minimum hazard for commercial mussel culture.

Agents: Cestoda

The 'tapeworms', well-known and frequently harmful parasites of man and animals, literally stand for the term 'disease'. However, none of the larval or postlarval tapeworms occurring in marine bivalves are known to infest humans. Metacestode infestation of pearl oysters may even be beneficial from the economic point of view, because the worms are believed to be involved in pearl formation. On the other hand, large worm burdens may significantly deteriorate the condition of oysters and detract from their market value.

Larval tapeworms have been reported from a great variety of marine invertebrates (Vol. I, Chapters 6, 7, 9, 10, 11 and 12). Although molluscs as a group have been considered as uncommon hosts for these parasites (Cheng, 1967), recent findings suggest that the opposite holds true. Representatives of the class Cestoda utilizing marine bivalves as intermediate hosts are members of the orders Trypanorhyncha (or Tetrarhynchidea), Lecanicephalidea, Tetraphyllidea and Diphyllidea. The adults of all of these are parasitic in the spiral valve or intestine of sharks, skates or rays. Therefore, larval cestode infestations in bivalves are most common in tropical and subtropical waters where elasmobranchs constitute an important proportion of the vertebrate fauna.

In spite of the large number of larval and adult cestodes of elasmobranchs described thus far, no complete life cycle has yet been worked out experimentally (Euzet, 1979). Suggested identities of larval and adult stages, as reported in the literature, are merely based on morphological similarities. There may also be problems regarding the recognition of the exact developmental stage of pre-adult cestodes encountered in invertebrates. The terminology of tapeworm development is highly confused. The term 'metacestode', used

by many authors to describe stages found in invertebrate (intermediate) hosts, applies to any larval form between the egg and adult or to larvae in the general or collective sense. Freeman's (1973) system of metacestode classification (see legends to Figs 13-147 and 13-150) is much more descriptive and matches the diversity of cestode morphological and developmental features better than the older designations (like 'plerocercoid I', 'plerocercoid II', etc.).

The picture is further obscured by the obvious ability of pre-adult cestodes to survive passage from one trophic level of the marine food web to the next (i.e., ingestion, by predator, of parenteral stages present in prey organisms, and re-establishment in predator organism): The carrier may be an obligate host, necessary for the completion of the parasite's life cycle, or a non-essential, 'accidentally' infested paratenic host. Usually, little morphological development occurs in hosts of the latter group. The functional morphology of cestode larvae has been reviewed in detail by Šlais (1973).

In the majority of cases discussed below, marine bivalves appear to act as primary hosts for pre-adult tapeworms. The filter- and/or deposit-feeding bivalves (as well as gastropods relying upon the same methods of food collection) become infested by either ingesting reproductive products (eggs or gravid proglottids) released with the faeces of the definite (selachian) host, or by attracting free-swimming coracidia with the ciliary currents of their gills. Carnivorous (molluscivorous) gastropods, on the other hand, appear to serve mainly as additional or paratenic (transport) hosts for larval cestodes, acquiring these parasites by feeding on infested bivalves or gastropods. The first mode of infestation is based on circumstantial evidence (Cheng, 1966a; see below); the second has been demonstrated experimentally. Thus, banded tulips *Fasciolaria lilium hunteria*, infrequent hosts for *Rhinebothrium* sp. (Cake, 1975), exhibited statistically significant increases in total numbers of larvae when fed ponderous arks *Noetia ponderosa* infested with plerocercoids of this species (Cake, 1977a, b). Cake (1976, p. 163) concluded:

> "For the parasitologist who is interested in surveying the cestode fauna of any coastal marine habitat, molluscivorous gastropods would serve as ideal 'indicator' organisms (or cestode collectors)."

Cake (1976) has assembled a key to larval cestodes of shallow-water, benthic molluscs of the northern Gulf of Mexico, the only one of its kind. In that study, 2,470 molluscs representing 36 gastropod, 55 bivalve and 1 cephalopod species, were examined. Eleven distinct species of cestode larvae, representing 9 or 10 recognized genera in 7 families and 4 orders — the Trypanorhyncha, Lecanicephalidea, Tetraphyllidea and Diphyllidea — were conditionally identified. Because the plerocercoids lack taxonomically important characteristics, most could be identified only to generic level. During the study it became apparent that most of the larval tapeworms occurring in Gulf of Mexico molluscs lack host specificity.

Information pertaining to the adult stages of the larval forms covered here is more complete. Consult Dollfus (1942, 1946b) and Campbell and Carvajal (1975) for Trypanorhyncha; Subhapradha (1951) for *Polypocephalus*; Southwell (1925), Young (1954a), Euzet (1959), Baer and Euzet (1962), Alexander (1963) and R. A. Campbell (1970) for Tetraphyllidea; Young (1956) and H. H. Williams (1966) for *Echeneibothrium*; Alexander (1953), Goldstein (1967) and H. H. Williams (1969) for *Acanthobothrium*; R. A. Campbell (1970) for *Rhinebothrium*; H. H. Williams (1968) for *Phyllobothrium* and Phyllobothriidae; G. Rees (1961) and Alexander (1963) for *Echinobothrium*

and Diphyllidea; Wardle and McLeod (1952) for general information. Reference to these keys, species descriptions and host records may facilitate future larval identification.

Most records of larval cestodes from marine molluscs are merely descriptive. There are only a few studies on the pathology of tapeworm infestation in bivalves. From this limited information, however, it becomes apparent that molluscs react considerably more severely to larval cestodes than to larval trematodes. From extended studies on cestodes occurring in Gulf of Mexico molluscs, Cake (1977a) concluded that heavy infestations certainly cause physiological stress which may affect growth, reproduction and, in commercially exploited species, edibility.

Larval lecanicephalidean cestodes of the genus *Tylocephalum* occur, sometimes in great abundance, in a variety of marine bivalves, particularly in oysters of the genus *Crassostrea* and in pearl oysters of the genus *Pinctada* from tropical and subtropical waters (for rectifications in the highly confused taxonomy of pearl oysters consult Ranson, 1961). Seurat (in Giard, 1903) depicted encapsulated, acaudate glando-procercoids (metacestodes), dissected from South Pacific *Margaritifera margaritifera cumingi* (= *Pinctada magaritifera*), which he initially mistook for encysted amphistome trematodes but subsequently identified as larval cestodes of the genus *Tylocephalum*. Mature tapeworms, believed to represent the adult stage of the pearl-oyster worm, were found in the spiral valve of rays *Aetobatus* (= *Myliobatis*) *narinari* and named *T. margaritiferae* (Seurat, 1904, 1906). Herdman (1904, 1906), Shipley and Hornell (1904, 1906) and Herdman and Hornell (1906) reported *Tylocephalum* metacestodes from Ceylonese *M. vulgaris* (= *P. radiata*) and window-pane oysters *Placuna placenta*, but failed to recognize their generic identity. They simultaneously found tetrarhynchidean postplerocercoids, named *Tetrarhynchus unionifactor*, in the pearl oysters, and believed that those represent a later developmental stage of the *Tylocephalum* metacestodes. Although no intermediate stages were ever seen, Herdman and Hornell (1906) constructed a hypothetical developmental sequence leading from the (lecanicephalidean!) procercoid to the (trypanorhynchan!) postplerocercoid in the pearl oyster. Their error was recognized by Southwell (1910a, 1912), Jameson (1912) and Dollfus (1924).

Subsequently, *Tylocephalum* sp. metacestodes have been found mainly in oysters — in *Crassostrea virginica* from Hawaii (Sparks, 1963; Cheng, 1966a; Rifkin and Cheng, 1968, 1969; Rifkin and co-authors, 1969a, b), from the Gulf of Mexico (Sparks, 1963; Sindermann, 1964; Cake, 1976, 1977a), and from Georgia (Sindermann and Rosenfield, 1967); in *C. gigas* from Japan, Hong Kong and the People's Republic of China (Sakaguchi, 1973; Cheng, 1975c); in *C. madrasensis* from Karnataka, India (Stephen, 1978b); in *C. commercialis* (= *Saccostrea cucullata*) from New South Wales and southern Queensland, Australia (Wolf, 1976b); and in *C. echinata* from Northern Territory, Australia (Wolf, 1976b). *Tylocephalum* sp. metacestodes have also been detected in pearl oysters *Meleagrina occa* (= *Pinctada chemnitzi*) and *M. irradians* (= *P. albina*) from Nossi-Bé, Madagascar (Dollfus, 1923b), in *Tapes semidecussatus* from Hawaii (Cheng and Rifkin, 1968), and in *Argopecten irradians concentricus, Macrocallista nimbosa* and *Spisula solidissima raveneli* from St. Teresa Beach, Florida (Cake, 1973; Fig. 13-147, 1). Sakaguchi (1973) reported larval *Tylocephalum* from 10 species of gastropod and bivalve molluscs in Japan, and Cake (1976) identified 16 gastropod and 33 bivalve species in the northern Gulf of Mexico as hosts for these metacestodes. Predaceous (molluscivorous) gastropods function as paratenic hosts and acquire their *Tylocephalum* infestations by

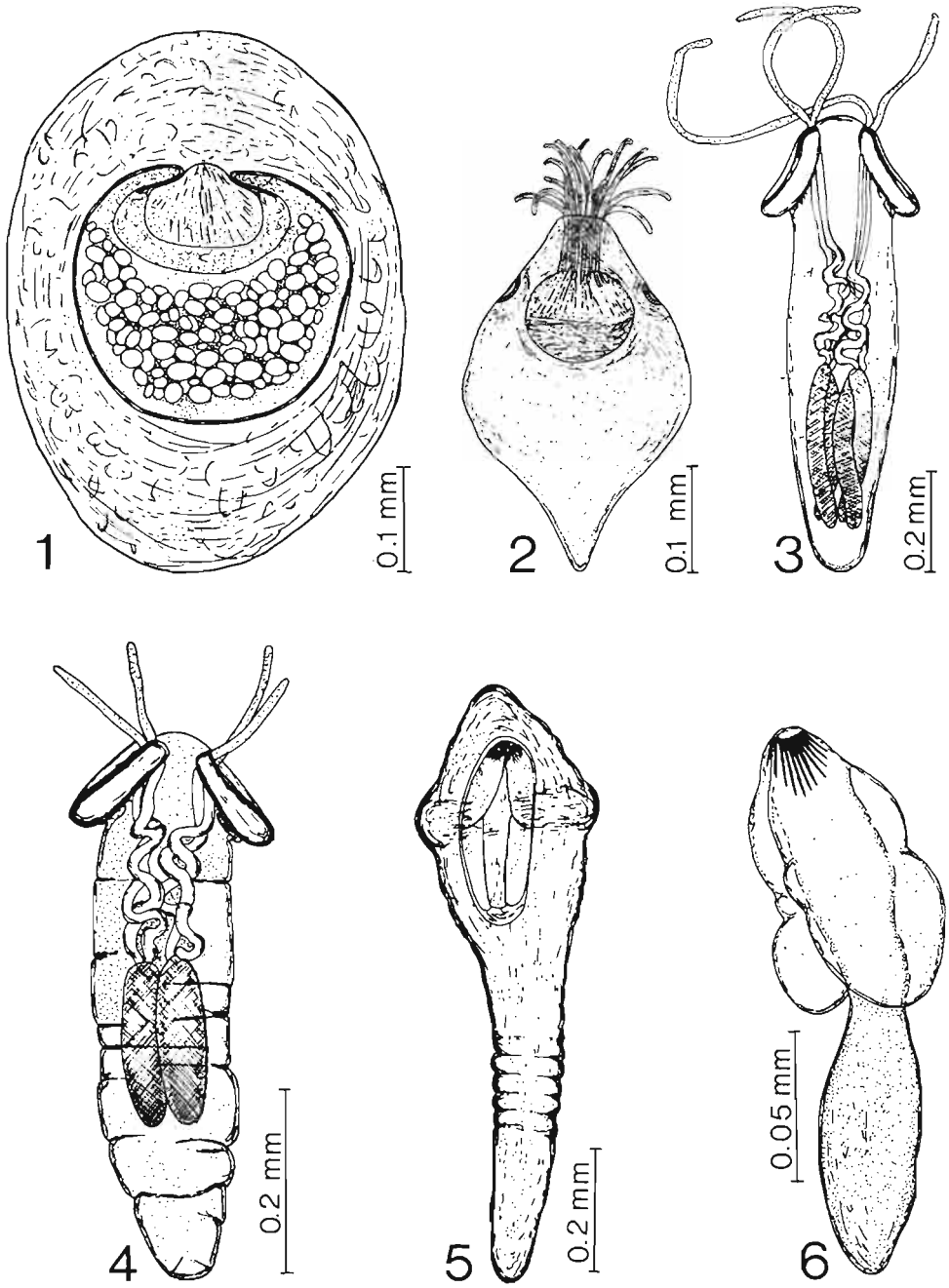


Fig. 13-147: Larval cestodes from marine bivalves and gastropods. 1: Encapsulated, acaudate glando-procercoid of *Tylocephalum* sp. from *Argopecten irradians concentricus;* 2: tentaculo-plerocercoid of *Polypocephalus* sp. from *A. irradians concentricus;* 3: tentaculo-neoplerocercoid of *Eutetrarhynchus* sp. from gastropod *Pleuroploca gigantea;* 4: tentaculo-neoplerocercoid of *Parachristianella* sp. (*dimegacantha?*) from *Macrocallista nimbosa;* 5: longicaudate, invaginated acanthoro-stellobothridio-cysticercoid of *Echinobothrium* sp. from gastropod *Nassarius vibex;* 6: scolex of *Echinobothrium* sp. from *N. vibex.* (After Cake, 1976.)

devouring metacestode-carrying bivalves (Cake and Menzel, 1979, 1980). The larvae from the various Gulf of Mexico molluscs were morphologically indistinguishable and believed to represent a single species. If this is correct, larval members of the genus *Tylocephalum* display little if any host specificity. It seems that their host range and distribution is wider than previously accepted (Table 13-31).

Apparently, however, these helminths require tropical or at least subtropical conditions for successful transmission. In spite of extensive histological investigations by Roughley (1926) and Wolf (1976b), which amount to approximately 2,000 sectioned specimens, no larval *Tylocephalum* have ever been found in *Saccostrea cucullata* from central and southern parts of the New South Wales coastline, i.e., at southern latitudes higher than 30°. In Japan, various gastropod and bivalve molluscs in Tanabe Bay (ca. 33.5° N) and Sukumo Bay (ca. 33° N) were found to be abundantly infested with larval *Tylocephalum,* whereas those from the colder waters of Ago Bay and Hiroshima Bay (ca.

Table 13-31
Tylocephalum sp. (or spp.). Prevalence of metacestodes in bivalves from different geographical areas (Compiled from the sources indicated)

Host species	Number examined	Number (%) infested	Number of metacestodes recovered	Locality	Source
Crassostrea virginica	153	52 (34.0)	n.i.*)	Pearl Harbor, Oahu, Hawaii	Rifkin and Cheng (1968)
Tapes semidecussatus	100	over 50	1 to 23 in 20 clams	Kaneohe Bay, Oahu, Hawaii	Cheng and Rifkin (1968)
Crassostrea virginica	138	60 (43.5)	950	Eastern Gulf of Mexico	Cake (1977a)
Argopecten irradians concentricus	78	68 (87.2)	5,639		
Macrocallista nimbosa	69	39 (56.5)	462		
Mercenaria campechiensis	26	6 (23.1)	101		
M. mercenaria	4	3 (75.0)	477		
Spisula solidissima similis	35	8 (22.9)	42		
Crassostrea gigas	35	35 (100)	+*)	Tanabe Bay, Wakayama Prefecture, Japan	Sakaguchi (1973)
Pinctada martensi	50	50 (100)	++		
Mytilus edulis	456	52 (11.4)	+		
Pinna attenuata	10	10 (100)	++		
Chlamys nobilis	25	5 (20.0)	+		
Spondylus barbatus	22	22 (100)	++		
Crassostrea commercialis (= *Saccostrea cucullata*)	117	23 (19.7)	n.i.	Northern New South Wales, Australia	Wolf (1976b)
Saccostrea cucullata	132	70 (53.0)	n.i.	Southern Queensland, Australia	
Crassostrea echinata	11	2 (18.2)	n.i.	Northern Territory, Australia	

*) n.i. not indicated; + light infestation; ++ heavy infestation

34.5° N) were uninfested (Sakaguchi, 1973). Burton (in Sparks, 1963) found *Tylocephalum* metacestodes in *C. virginica* from Chincoteague Bay, Maryland (ca. 38° N). These oysters, however, had been transplanted from Apalachicola Bay, Florida, to Maryland waters, and had probably acquired their parasites prior to transplantation. Similarly, *Tylocephalum*-infested oysters reported from North Carolina (ca. 35° N) by Sindermann and Rosenfield (1967), had been transplanted from more southerly waters. Hence, also this geographic record may be artificial (Wolf in Cheng, 1975c).

Whether all the *Tylocephalum* sp. metacestodes reported from bivalves represent a single species is doubtful and remains to be studied. The globular larvae commonly encountered measure approximately 0.7 to 1.5 mm × 0.5 to 1.0 mm. Much smaller (0.1 to 0.2 mm) metacestodes, found in Ceylonese *Pinctada margaritifera,* were believed to belong to a separate species (Jameson, 1912; Southwell, 1924), but may as well represent younger stages of the same parasite. Reimer (1975) recognized 2 distinct types of *Tylocephalum* metacestodes. Those found in bivalves *Meretrix casta, Mactra mera* and *Arca inaequivalvis* from the Madras coast, India, were particularly large (872 to 5,420 × 139 to 990 μm) and differed from the typical members of the genus in lacking the characteristic powerful myzorhynchus. They may not belong to *Tylocephalum* but to *Tetragonocephalum, Lecanicephalum* or *Disculiceps.*

Most of the reported *Tylocephalum* metacestodes have been described so superficially and inadequately that reliable comparisons and specific identifications cannot be made (Yamaguti, 1959). The genus *Tylocephalum* has been erected by Linton (1890) to contain *T. pingue*, an adult lecanicephalid recovered from the spiral valve of a ray, *Rhinoptera quadriloba*, caught off Woods Hole, Massachusetts. Adult *Tylocephalum* spp. have been reported from elasmobranchs in tropical and subtropical waters, particularly in areas where oysters or pearl oysters are infested with metacestodes. At least 5 species occur in Ceylonese sharks and rays (Shipley and Hornell, 1906). Experimental attempts to link the metacestode(s) in oysters and pearl oysters with any of the known adult worms have failed thus far (Southwell, 1910a, 1911b; Jameson, 1912; Cheng, 1966a). Sakaguchi (1973) succeeded in obtaining partial development of *Tylocephalum* metacestodes from *Pinctada fucata* (= *P. martensi*) and other molluscs in *Heterodontus japonicus.* Metacestodes emerged from their capsules in the shark's stomach within 24 h and migrated to the spiral valve within 7 days, but formation of segments and genital organs did not take place. On the other hand, larvae administered to other species of sharks and teleosts were digested and discharged from the anus within 2 days.

The remainder of the life cycle of *Tylocephalum* sp. is obscure. Ciliated 'coracidia' (Fig. 13-148), about 264 μm long and 160 μm wide, were observed closely associated with the gill surfaces of *Crassostrea virginica* from Hawaii. One of these larvae was in the process of shedding its external ciliated epithelium. Other 'coracidia' were seen in the oysters' stomach, and one of these had already shed its ciliated epithelium. Obviously these larvae were preparing to penetrate the oyster. A prominent invagination at the anterior end of the 'coracidium' was interpreted as a developing myzorhynchus typical of *Tylocephalum* larvae (Cheng, 1966a). Wolf (1976b) observed the presence of similar ciliated 'coracidia' in the gills and palps, as well as infestations of varying intensities and stages of encapsulation, in the digestive diverticula of 95 of 249 *Saccostrea cucullata* from northern New South Wales and southern Queensland (Australia).

Cake and Menzel (1980) doubt that the organisms observed by Cheng (1966a) and

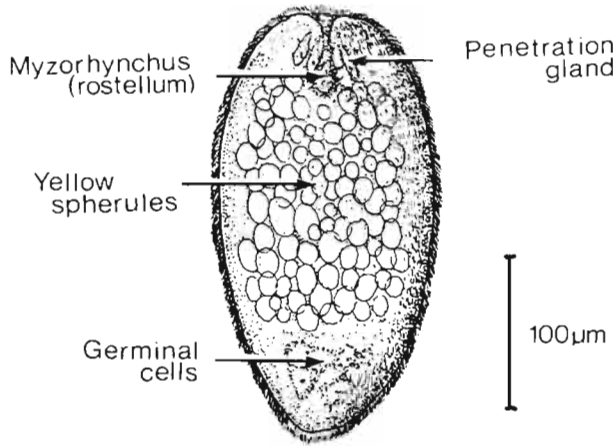


Fig. 13-148: *Tylocephalum* sp.(?). 'Coracidium' from gill surface of *Crassostrea virginica*. (After Cheng, 1966a.)

Wolf (1976b) are actually larval cestodes. They point out that no coracidial stage has yet been demonstrated in the ontogeny of any known lecanicephalidean cestode. Moreover, Cheng's 'coracidia' are 5 to 7 times larger than those known from marine pseudophyllideans and tetrarhynchideans, and no penetration glands (as in Fig. 13-148) have been observed in marine coracidia (Rybicka, 1966). Cheng's 'coracidia' also lack the larval hooks typical of other similar cestode larvae, and the ciliated epithelium of his form appears to be part of an epidermis rather than a ciliated embryophore that surrounds the oncosphere of a true coracidium. Cake and Menzel (1980) conclude that the organisms seen by Cheng (1966a) and Wolf (1976b) are probably not the precursors of *Tylocephalum* metacestodes but perhaps advanced metazoan larvae of another oyster symbiote. If no coracidium exists in the lecanicephalidean life cycle, filter-feeding molluscs may become infested by ingesting planktonic or demersal cestode eggs containing oncospheres. However, the possibility exists that bivalves may ingest the remains of small, infested crustacean first intermediate hosts, such as copepods.

Tylocephalum metacestodes are said to be capable of asexual reproduction. Willey (1907) reported as many as 20 buds developing within a large metacestode from *Placuna placenta*, and Southwell (1910a, b) observed endogenous budding of *Tylocephalum*, resulting in the production of numerous larvae of different sizes, in Ceylonese pearl oysters *Margaritifera vulgaris* (= *Pinctada radiata*). Cake (1975), however, did not specifically confirm the above authors' observations during his studies on *Tylocephalum* infestations in Gulf of Mexico pelecypods.

Incidence and intensity of larval *Tylocephalum* infestation may locally reach epizootic proportions, particularly in oysters and pearl oysters (Table 13-31). A medium-sized Ceylonese *Pinctada margaritifera* may contain over 200 metacestodes, and up to 36 capsules could be seen in a single section of an Australian *Saccostrea cucullata* specimen (Southwell, 1912; Wolf, 1976b, 1979). In individuals of *Placuna placenta*, *Tylocephalum* metacestodes were observed in dense aggregations in the superficial layer of the mantle (Herdman and Hornell, 1906). Both incidence and intensity of *Tylocephalum* infestation were higher in Japanese pearl oysters from natural populations than in those from

cultivated beds (Sakaguchi, 1973). Heavily infested *S. cucullata* had a poor, transparent and watery consistence, and the condition indices of similarly affected *C. virginica* were consistently much lower than normal for the species for the season of the year (Sparks, 1963; Wolf, 1976b). Although, apparently, oyster mortalities directly related to *Tylocephalum* have not been documented, the impact of heavy metacestode infestation on local oyster fisheries should not be underestimated. Wolf (1979) pointed out that badly infested oysters are hardly marketable.

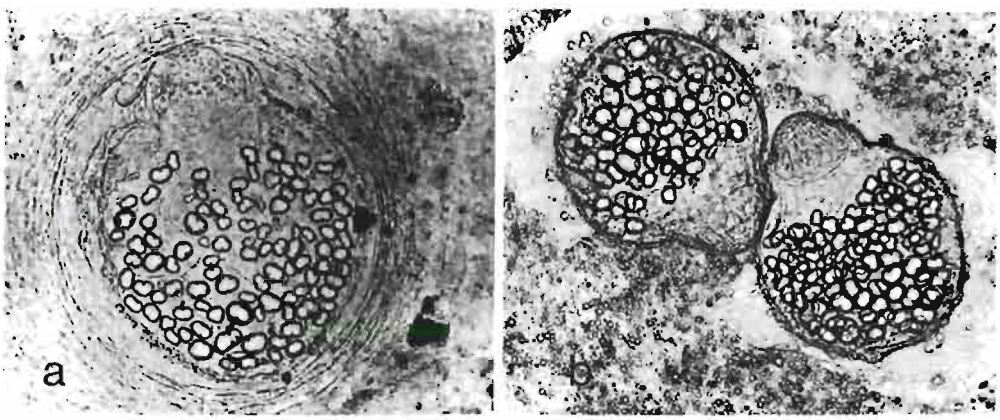


Fig. 13-149: *Tylocephalum* sp. (a) Metacestode encapsulated in digestive-gland tissue of *Crassostrea virginica;* (b) metacestodes excised from capsules, × 630. (After Sparks, 1963.)

The pathology of larval *Tylocephalum* infestation varies with host species. Response elicited by *Crassostrea virginica* against metacestode invasion is severe and comprises the formation of thick-walled fibrous capsules of host origin around the intruders (Fig. 13-149). The typical location of these 'cysts' is the loose connective tissue between the digestive diverticula and beneath the gut, but larvae may also occur in the gills and other tissues of the oyster. By far the most common invasion route is through the alimentary wall, primarily the stomach wall. While passing through the lining epithelium, the body movements of the larva may produce lesions in the surrounding oyster tissues. There is no appreciable host response while the parasite is passing through the gut wall, but shortly after it comes in contact with the stratum of connective tissue at the base of the lining epithelium, a conspicuous layer of connective tissue fibres begins to form around it.

Capsule formation commences when the invading parasites compress the surrounding Leydig cells during migration; it leads to a progressive thickening of intercellular fibrous material between the Leydig cells and an infiltration of haemocytes. Specifically, the capsules consist of 3 types of cells and 2 types of extracellular fibres embedded in a homogenous matrix of medium electron density. The cellular elements are fibroblast-like cells which are primarily concentrated in the innermost region of the capsule, haemocytes which are scattered throughout the cyst, and 'brown cells' which are situated along the periphery. The major type of extracellular fibres is of medium electron density and non-periodic (i.e., non-collagenous). The second type is also non-periodic and similar to the matrix in electron density. The spatial relationship between the extracellular fibres and the cellular constituents of the capsule suggests that extracellular fibrillogenesis is influenced

by these cells (Sparks, 1963; Cheng, 1966a; Rifkin and Cheng, 1968, 1969; Rifkin and co-authors, 1969a, b; Cheng and Rifkin, 1970).

The capsules around *Tylocephalum* metacestodes situated in the zone surrounding the alimentary tract of *Crassostrea virginica* are consistently thicker than those around larvae located in the interdiverticular spaces of the digestive gland. This is probably due to the fact that there is less Leydig tissue, and hence less intercellular material, in the digestive gland than in the area surrounding the alimentary tract. Healthy appearing metacestodes in the oyster are usually not or only slightly encapsulated, while heavy capsules consisting of connective-tissue fibres infiltrated with numerous haemocytes contain larvae in the process of being resorbed (Cheng, 1966a; Rifkin and Cheng, 1968).

Host response to *Tylocephalum* sp. is even more pronounced in *Tapes semidecussatus*. In this clam, metacestodes occur mainly encapsulated in the intertubular spaces between the digestive diverticula, which suggests invasion, by the coracidia (?), from the mantle cavity rather than from the alimentary tract. The capsules formed around the parasites are similar in structure and arrangement of the eosinophilic fibres to those produced by *Crassostrea virginica*. The only striking difference is that there appear to be more haemocytes intermingled with the encapsulating fibres in *T. semidecussatus*. Twenty *T. semidecussatus* from Kaneohe Bay, Oahu, Hawaii, contained from 1 to 23 larvae, 56 % of which were found to be in the process of being resorbed. Initially, a conspicuous massing of haemocytes occurs until each metacestode becomes enveloped by a thick tunic consisting of densely packed blood cells and numerous 'brown cells' which, according to Mackin (1951), Stein and Mackin (1955) and Cheng and Burton (1965b), are probably part of the molluscan internal defense system. The formation of this cellular coat initiates the gradual disintegration of the enclosed parasite, which is followed by the breakdown of the fibrous capsule, haemocytic infiltration, and final phagocytosis of the cestode's cellular debris. The large percentage of such disintegrating parasites suggests that *T. semidecussatus* is not a totally compatible host for larval *Tylocephalum* sp. (Cheng and Rifkin, 1968).

Comparable stages of resorption of *Tylocephalum* sp. metacestodes were also seen in Sydney rock oysters *Saccostrea cucullata* (Wolf, 1976b), but capsules produced by *Crassostrea gigas* in response to what appears to be the same parasite species were less dense and none of the enclosed larvae were in the process of being resorbed (Cheng, 1975c). Judging from the observations by Shipley and Hornell (1904), Herdman and Hornell (1906), Seurat (1906), Jameson (1912) and Southwell (1912, 1924), resorption of metacestodes apparently does also not occur in pearl oysters *Pinctada* spp., which are considered to be normal intermediate hosts for *Tylocephalum* sp.

In spite of heavy parasite burden (up to 125 metacestodes per host) and prominent capsule formation around the intruders, none of the 60 infested *Crassostrea virginica* individuals inspected by Cake and Menzel (1980) were weak or moribund, or exhibited any significant loss of body volume and weight. In no case was the digestive tract blocked by massive infestations, as observed by Cake (1975, 1976) in the case of encapsulated *Parachristianella* sp. postplerocercoids (see below).

Tylocephalum *sp. metacestodes — originally misinterpreted as a stage in the life cycle of a trypanorhynch,* Tetrarhynchus unionifactor *— have been incriminated in the formation of pearls in* Pinctada *spp. (Giard, 1903; Herdman, 1903–1906; Seurat, 1904, 1906; Southwell, 1912, 1924; Hornell, 1922). Shipley and Hornell (1904) and Dollfus (1923a, b) described and figured* Tylocephalum *obtained from the centre of decalcified pearls.*

Rubbel (1911a, b), however, concluded that parasites are not the cause of pearl formation, at least not in fresh-water pearl mussels *Margaritana margaritifera*. Similarly, Jameson (1912) failed to confirm the occurrence of larval worms within pearls and, on the basis of an elaborate study, discarded the cestode theory of pearl formation. His view has been accepted - without experimental reconfirmation, however — by most modern workers.

One reason for the rejection of the above hypothesis is the fact that *Tylocephalum* metacestodes

> "are found in the soft tissues on the interior of bivalves and not on the mantle, as are gymnophallid trematode metacercariae which do stimulate pearl formation, and hence could not stimulate the nacre-secreting mantle to form pearls"

(Cheng, 1967; p. 256). The assumption that the presence of mantle epithelium cells is an imperative prerequisite for pearl formation, is clearly disproven by the occurrence of so-called 'muscle pearls' in *Pinctada* spp. and other bivalves at sites where no trace of mantle cells can be found. Muscle pearls in *Pinctada* spp., when present, are usually abundant. Thus, a single Ceylonese *P. radiata* was found to contain 23 small pearls visible to the naked eye, while, under the dissecting microscope, 170 additional tiny spherules could be seen. Muscle pearls are commonly situated in the muscular tissue adjacent to the insertions of the adductor and pallial muscles. Their mode of formation is unknown, but they are believed to originate from minute 'calcospherules' developing in centres of tissue irritation (Herdman and Hornell, 1906). Muscle pearls lack the lustre of 'true' or 'cyst' pearls and have no commercial value. Recent findings (Götting, pers. comm.) indicate that mantle cells are definitely not required to initiate pearl formation. Protein-calcium complexes are synthesized at sites other than those at which pearl formation occurs, and are transported to the latter via a special type of blood cell yet to be described in detail.

A critical evaluation of the pros and cons of the cestode theory of pearl formation suggests that it cannot be rejected straight away. Pearls may be formed in response to a variety of quite unrelated stimuli, abiotic as well as biotic, natural as well as artificial (Tsujii, 1960). The capability of trematode larvae to induce the production of pearls has been demonstrated repeatedly. There is no reason to assume that cestode larvae should not be able to stimulate the pearl oyster in a similar manner. Since Shipley and Hornell (1904) and Dollfus (1923a, b) have obtained 'mummified' metacestodes from decalcified pearls, there is no doubt that larval cestodes *can* cause the formation of pearls in molluscs. It might be interjected that bivalves respond to metacestode invasion by extensive fibrosis, and that nacrezation — i.e., the deposition of nacre around parasites of molluscs which irritate or invade the mantle region (Cheng, 1967) — and fibrotic response are mutually exclusive (Cheng and Rifkin, 1970). But *Tylocephalum*-induced fibrosis is generally less pronounced in *Pinctada* spp. than in other molluscs, and the fibrotic response to larvae invading the mantle tissues has, apparently, not yet been studied. It may even be lacking. Since the presumed invasive stage of *Tylocephalum* is capable of penetrating the alimentary, as well as the gill epithelium of *Crassostrea virginica* (Cheng, 1966a), it may also be able to invade and penetrate the mantle epithelium. In fact, Herdman and Hornell (1906) observed 'cysts', which were the smallest they ever saw, in the mantle tissues of *Margaritifera vulgaris* (= *Pinctada radiata*). The capsules contained an 'embryo' of simple morphological organization, presumably a developing *Tylocephalum* metacestode. Similar early stages were seen in the gills. The further fate of these larvae is unknown, but it is possible that some of them eventually penetrate the mantle and settle in the extrapallial

space where they stimulate the epithelium to secrete a pearl. Such a mechanism would not necessarily imply that pearls are formed around living parasites (which is neither the case in trematode-induced pearl formation). It appears more likely that the pearl nucleus incorporates dead larvae or their debris (Hornell, 1922; Southwell, 1924).

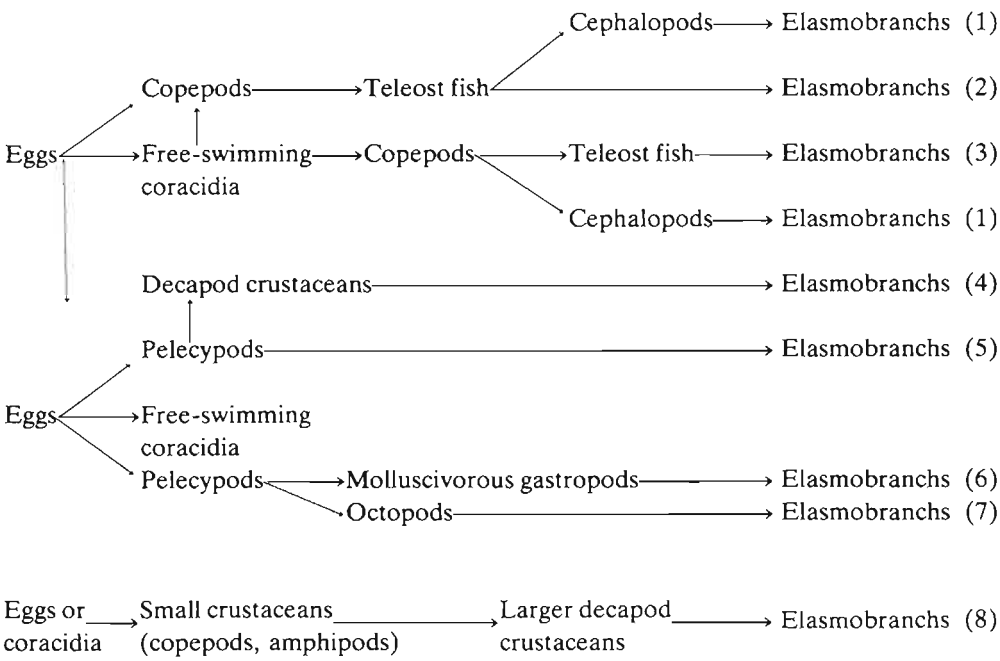
It might be tempting, in this connection, to speculate upon the possible involvement of cestode calcareous corpuscles in the formation of pearls. The parenchyme of numerous cestodes, particularly that of larval forms, is known to contain masses of strongly refringent corpuscles. Such bodies occur in large quantities in *Tylocephalum* metacestodes (Figs 13-147, 1 and 13-149), and 'yellow spherules' of similar structure have been observed in organisms believed to be *Tylocephalum* coracidia (Fig. 13-148). Chemically, the corpuscles consist of an organic base bound to inorganic matter, mainly calcium, magnesium, phosphorus and carbon dioxide. They may constitute as much as 41 % of the dry weight of the organism. Although numerous studies have been devoted to the structure, morphogenesis, histochemistry, chemical composition and function of these bodies (Diamare, 1930; Schopfer, 1932; Starcoff, 1939; Chowdhury and co-authors, 1955a, b, 1962; von Brand and co-authors, 1960, 1965a, b, c, 1967, 1969; Scott and co-authors, 1962; Desser, 1963; Kegley and co-authors, 1969, 1970; Nieland and von Brand, 1969; von Brand and Nylen, 1970; Chowdhury and DeRycke, 1974a, b, 1976; and others), no definite answer has been obtained as to their true significance. Several functions have been attributed to them, a major one being the neutralization of the acidic products of anaerobic metabolism. The latter role, however, would be meaningful only for the endoparasitically living adult and pre-adult worms but not for the free-swimming, aerobically living 'coracidium'. The large carbonate content of the calcareous corpuscles in metacestodes (and possibly of the 'yellow spherules' in *Tylocephalum* 'coracidia') rather suggests that they may serve to protect the metacestodes as they pass through the stomach of the definite host (and the 'coracidia' as they invade the bivalve intermediate host via the stomach). The same function has been attributed to the 'excretion granules' of trematode metacercariae (p. 748). It appears possible that the 'calcospherules' in the tissues of pearl oysters, which give rise to the formation of muscle pearls, are in fact condensation products forming around cestode calcareous corpuscles derived from disintegrated, resorbed metacestodes or 'coracidia'.

Still another hypothesis on the origin of pearls in molluscs exists. Dubois (1907a) claimed to have found sporozoan spores in the centre of decalcified pearls from the mantle of *Margaritifera vulgaris* (= *Pinctada radiata*) from the Tunisian coast. The ovoid spores were very similar to those described by Giard (1897b) and Léger (1897a) from gymnophallid metacercariae (*Gymnophallus strigatus*) parasitizing various bivalve species off Boulogne-sur-Mer, France (p. 731). It could be possible that the spores in the mantle tissue of the pearl oyster originate from either mollusc-invading sporozoans (microsporans?) or from sporozoans hyperparasitic in helminths in the pearl oyster. Consequently, Dubois (1907a; p. 311) raised the question: "On peut se demander si ce ne serait pas le *parasite du parasite* du mollusque qui produirait la perle."

Regardless of the definite mechanism of pearl formation in *Pinctada* spp., the latter is a 'rare event'. Thus, a normal Ceylon pearl oyster may contain as many as 200 encapsulated *Tylocephalum* metacestodes but not a single pearl (Southwell, 1912). Herdman and Hornell (1906) obtained 'true' or 'cyst' pearls from only 69 of 1,400 and 130 of 1,491 Ceylonese *P. radiata*.

Another larval lecanicephalidean parasitizing marine bivalves is *Polypocephalus* sp. Its pyriform tentaculo-plerocercoids (Fig. 13-147, 2), 260 to 410 µm long, occur singly or in clumps of up to 8 individuals in thin, transparent capsules in the connective tissue of the digestive gland of *Argopecten irradians concentricus*. In contrast to *Tylocephalum* sp., which was found in all but 3 of 43 cestode-infested mollusc species from the northeastern Gulf of Mexico, *Polypocephalus* metacestodes only occurred in bay scallops, 42 of 55 individuals, collected from 4 sampling stations, being infested (Cake, 1973, 1975, 1976, 1977a). Nothing is known about its pathology and further life cycle. *A. irradians concentricus* appears to be a regular intermediate host of this lecanicephalidean (Cake, 1979), although larval *Polypocephalus* sp. have more frequently been recovered from penaeid shrimp in Gulf of Mexico waters (Vol. III). The adults are intestinal parasites of elasmobranchs. Nine species are known worldwide (Subhapradha, 1951).

Postlarvae (tentaculo-neoplerocercoids) of members of the Trypanorhyncha (Tetrarhynchidea) have mostly been found in cephalopod molluscs and decapod crustaceans (Vol. III), as well as in teleost fishes (Vol. IV), but several species occur in bivalves and gastropods. On the basis of his own, extended studies, Cake (1975) has summarized our present knowledge on suggested life-cycle pathways of trypanorhynch cestodes as follows (in brackets: source of information):



(1) Yamaguti (1959): *Nybelinia* spp.; (2) Young (1954b), Yamaguti (1959), Mudry and Dailey (1971): *Lacistorhynchus* spp.; (3) Ruszkowski (1932, 1934), Riser (1956): *Grillotia erinaceus* and *L. tenuis*; (4) Kruse (1959), Aldrich (1965), Mudry and Dailey (1971): *Parachristianella monomegacantha* and *Prochristianella penaei*; (5) Cake (1975): *Parachristianella* sp.; (6) Cake (1975): *Eutetrarhynchus* sp.; (7) Cake (1975): circumstantial evidence from *Octopus joubini*; (8) Aldrich (1965): *Prochristianella penaei*.

Pelseneer (1906, footnote) briefly mentioned a postlarval trypanorhynch, "ressemblant fort à *Tetrarhynchus ruficollis* Eysenhardt", parasitic in the visceral mass of *Ostrea edulis* from a Belgian oyster bed. Neoplerocercoids of this species, now known as *Eutetrarhynchus ruficolle*, are normally found in decapods, and the adults in rays and sharks (Dollfus, 1936, 1942). Another postlarval *Eutetrarhynchus* sp. (Fig. 13-147, 3) has been recovered from *Argopecten irradians concentricus, Atrina rigida, A. seminuda* and *Dosinia discus*, as well as from 5 species of molluscivorous and 1 species of filter-feeding gastropods in the eastern Gulf of Mexico. Infestation incidences in the bivalves were lowest in *A. irradians concentricus* (2 of 78 = 2.6 %) and highest in *D. discus* (7 of 16 = 43.8 %) (Cake, 1975, 1976). Only one species of adult *Eutetrarhynchus, E. lineatus* (Linton, 1908), has been recorded from that area to date. *Donax variabilis* and *Atrina seminuda* from Galveston Beach, Texas, are hosts for the neoplerocercoid of *Nybelinia* sp., reported as '*Scolex* sp. VIII' by Wardle (1974).

Several larval members of the genus *Tetrarhynchus* have been reported from *Pinctada* (*Margaritifera*) spp. and *Pinna* sp. from various localities in the Indopacific. Of these, *T. unionifactor* has found ample scientific attention because it was (erroneously) held responsible for pearl formation. Its adult is believed to parasitize in the spiral valve of rays *Rhinoptera javanica,* sharks *Ginglymostoma concolor,* and probably other elasmobranch species, but experimental proof of this is lacking (Southwell, 1910a, 1911a, b). The specific identity of the other larval Indopacific tetrarhynchids remains obscure. The genus *Tetrarhynchus* is abundantly represented in tropical waters. At least 14 species of adult worms occur in rays and sharks from Ceylonese waters (Shipley and Hornell, 1906). The circuitous literature dealing with *T. unionifactor* and related larval forms has been reviewed critically by Dollfus (1923a, 1942).

Argopecten irradians concentricus, Anadara transversa, Atrina spp., *Donax variabilis, Chione cancellata, Spisula solidissima similis, Macrocallista* spp., *Noetia ponderosa* and *Raeta plicatella*, as well as 5 species of gastropods from the northern Gulf of Mexico, are hosts for postlarvae (tentaculo–neoplerocercoids) of trypanorhynchs *Parachristianella* sp., probably *P. dimegacantha* (Fig. 13-147, 4). The encapsulated larvae occur, mostly singly, along the walls of the intestine of all hosts and in the foot musculature of *Macrocallista nimbosa* and *Spisula solidissima similis*. Infestation incidences and intensities were highest in southern scallops, sunray venus clams and surf clams. Forty-two of 78 *A. irradians concentricus* yielded a total of 222 neoplerocercoids, 60 of 69 *M. nimbosa* had 1,674, and 30 of 35 *S. solidissima similis* had 520 worms. Heavy infestation severely restricts the passage of food material through the host's intestine. In one heavily parasitized sunray venus clam the intestine was completely blocked (Cake, 1975, 1976, 1977a). There was no unusual host-tissue reaction against these postlarval trypanorhynchs, and they appeared normal and quite viable. Degenerate *Parachristianella* sp. postlarvae were found in only 1 of 194 bivalve hosts, and these may have been 'over-aged' individuals (Cake, 1975).

The occurrence of large numbers of viable *Parachristianella* sp. postlarvae in bivalves suggests that pelecypods are normal, compatible intermediate hosts for trypanorhynch cestodes. *P. dimegacantha* neoplerocercoids occur, although in much lesser abundance, in penaeid shrimp from the Gulf of Mexico (Vol. III). Previously, bivalves had been regarded as abnormal, accidentally infested invertebrate hosts (Wardle and McLeod, 1952; Dollfus, in Cake, 1975).

Cysticercoids of *Echinobothrium* sp. (the only genus in the order Diphyllidea) occur

in the tissues, mainly in the foot, of *Solen vagina* (*S. marginatus*) from Arcachon, France (Kunstler, 1888). The larvae were not identified to species, but Monticelli (1890) believed them to be identical with *E. levicolle*, found by Lespès (1857a) in the digestive gland of mud snails *Nassarius reticulatus*, also from Arcachon. Pintner (1889) assigned, with some reservation, Lespès' larva to *E. musteli*, a species described by him from the spiral valve of *Mustelus canis*. Vaullegeard (1901), on the other hand, referred to larval *Echinobothrium* found in *Cardium edule* and *Solen* sp. as *E. typus* van Beneden, 1849. *Echinobothrium* sp. cysticercoids, recovered from the digestive gland of *N. vibex* and *Cantharus cancellarius* in the northeastern Gulf of Mexico, have also been attributed to *E. musteli*, the only adult species reported from that area (Cake, 1976, 1977a; Fig. 13-147, 6). These carnivorous snails have probably acquired their larval worms by devouring infested bivalves or crustaceans, another group of hosts for these cysticercoids.

The overwhelming majority of cestodes parasitizing marine bivalves are members of the Tetraphyllidea. Lack of distinguishing morphological features is particularly characteristic of this group of larval tapeworms and renders specific or even generic identification tenuous. Length-width range measurements usually given for plerocercoids have little if any diagnostic value. The only paper presenting exact data (mean ± standard deviation, number of observations, range) suitable for statistical comparison is that by Hamilton and Byram (1974).

In some cases, tetraphyllidean plerocercoids have been reported as 'enigmatic organisms' and have not even been recognized as representatives of the phylum Platyhelminthes (Vol. I, p. 181). Modern methods of *in vitro* cultivation, developed for the artificial maintenance of non-marine larval and adult cestodes (Rothman, 1959; W. C. Campbell and Richardson, 1960; W. C. Campbell, 1963; McCaig and Hopkins, 1965; Voge, 1967; Voge and Seidel, 1968; Evans, 1970; Goodchild and Davis, 1972; and others; reviews by Voge, 1973, and Lackie, 1975), provide a promising tool for the study of developmental stages and life cycles of marine forms. Cultivation of larval stages of selachian cestodes requires a special glucose-enriched elasmobranch saline medium (Read and co-authors, 1960; Hamilton and Byram, 1974). Application of such methods has facilitated generic identification of several tetraphyllidean and lecanicephalidean larvae obtained from marine molluscs (Hamilton and Byram, 1974; Cake, 1976, 1977a). Further improvement of culture techniques may permit identification, to species level, of many of these larvae in the future.

Numerous tetraphyllidean plerocercoids have been reported from a wide variety of marine invertebrates — including siphonophores, ctenophores, turbellarians, gastropods (Vol. I, Chapters 6, 7, 10 and 12), bivalves, cephalopods, copepods, decapods and chaetognaths —, as well as from teleost fishes. To this group of larvae the collective names '*Scolex pleuronectis* Müller, 1788' or '*S. polymorphus* Rudolphi, 1819', have been applied (van Beneden, 1850; Monticelli, 1888; Curtis, 1911; Dollfus, 1923a, 1936, 1953, 1964, 1967, 1976; Linton, 1924; Southwell, 1925; Riser, 1956; Euzet, 1959; Regan, 1963; Friedl and Simon, 1970; Reimer, 1975; and others). Monticelli (1888) assembled a list of junior synonyms applied to larvae of this type collected from fish hosts. Many, if not most, of these scolices are probably members of the genus *Acanthobothrium* in the family Onchobothriidae (Dollfus, 1936, 1953; Reichenbach-Klinke, 1956a, 1957; Cake, 1976, 1977a). About 70 species of adult *Acanthobothrium* spp. are known, but a large number of species remain to be discovered and described. The adult worms display a very high

degree of host specificity, but the larval stages apparently do not. The taxonomy of the genus is highly confused. Some of the species labelled *Acanthobothrium* are not even tetraphyllideans (Goldstein, 1967; H. H. Williams, 1969).

Bivalve hosts for unidentified '*Scolex pleuronectis*' or '*S. polymorphus*' include *Solen vagina* (*S. marginatus*) from Arcachon, France (Kunstler, 1888), *Meretrix casta* and *Donax scortum* from Madras, India (Anantaraman, 1963; Reimer, 1975), and *Argopecten irradians concentricus, Chione cancellata, Anomalocardia auberiana, Cyrtopleura costata, Dosinia discus, D. elegans, Ensis minor, Modiolus modiolus squamosus* and *Noetia ponderosa* from the northern Gulf of Mexico (Cake, 1975, 1976).

A small thin-walled, whitish cyst, attached to the outer surface of the intestine just below the stomach of a channelled duck clam *Raeta* (= *Anatina*) *plicatella* from the Gulf of Mexico, at first mistaken for a piece of ovary, was found to contain several dozen cestode larvae, each coiled to form a sphere. Released from the capsule into sea water, the worms squirmed with peristaltic movement and everted the terminal sucker. The colourless body, about 54 μm long, was filled with small hyaline granules. No internal anatomical details could be made out, but affinities with the Phyllobothriidae or Onchobothriidae were suggested (Harry, 1969).

Cake (1975, 1976) refound the *Scolex pleuronectis* of Harry (1969) in *Argopecten irradians concentricus, Ensis minor, Macoma constricta, Pseudomiltha floridana, Raeta plicatella, Tagelus divisus, T. plebeius*, as well as in moon snails *Polinices duplicatus* from the northern Gulf of Mexico. The bothridio-plerocercoids of this type, which range from 0.2 to 0.9 mm in length, occur encapsulated singly or in groups of up to 150 individuals per capsule in the host's gut wall pouches or distended digestive diverticula immediately adjacent to the intestine. By means of *in vitro* cultivation, the larvae could be identified as belonging in the onchobothriid genus *Acanthobothrium*. On the basis of scolex morphology they were tentatively referred to *A. brevissime*, described by Linton (1908) and Goldstein (1964, 1967) from sting rays *Dasyatis sayi* and *D. sabina* in the Gulf of Mexico. The unincubated *Acanthobothrium* sp. plerocercoids (Fig. 13-150, 5), recovered from bivalve hosts, have quadriloculate bothridia but transform into a triloculate and accessory sucker configuration in the adult worm (Cake, 1975). As pointed out by Southwell (1925), the anterior bothridial loculus of quadriloculate tetraphyllidean larvae may be a transient structure, disappearing during development or becoming an accessory sucker. Changes in scolex morphology during *in vitro* development have also been described for a larval *Acanthobothrium* by Hamilton and Byram (1974).

Another '*Scolex pleuronectis*', originally reported by Regan (1963) from crowned conchs *Melongena corona* at Live Oak Point, Florida (USA), was similarly identified, by means of *in vitro* cultivation, as a member of the genus *Acanthobothrium*, possibly *A. paulum* Linton, 1890 (Cake, 1976). It was also found free in the gut of bivalves *Noetia ponderosa* and of 10 species of gastropods from the Gulf of Mexico (Friedl and Simon, 1970; Hamilton and Byram, 1974; Wardle, 1974; Cake, 1976, 1977a). Regan (1963) figured and described the bothridio-plerocercoid from *M. corona* as triloculate, whereas the other authors reported it to be quadriloculate. Friedl and Simon (1970) considered only the last 3 loculi to be permanent structures, with the most anterior muscular concavity being a possible modification of a bothridial muscular pad or a primordial accessory sucker. Hamilton and Byram (1974) substantiated the latter authors' speculations by *in vitro* development of the plerocercoid.

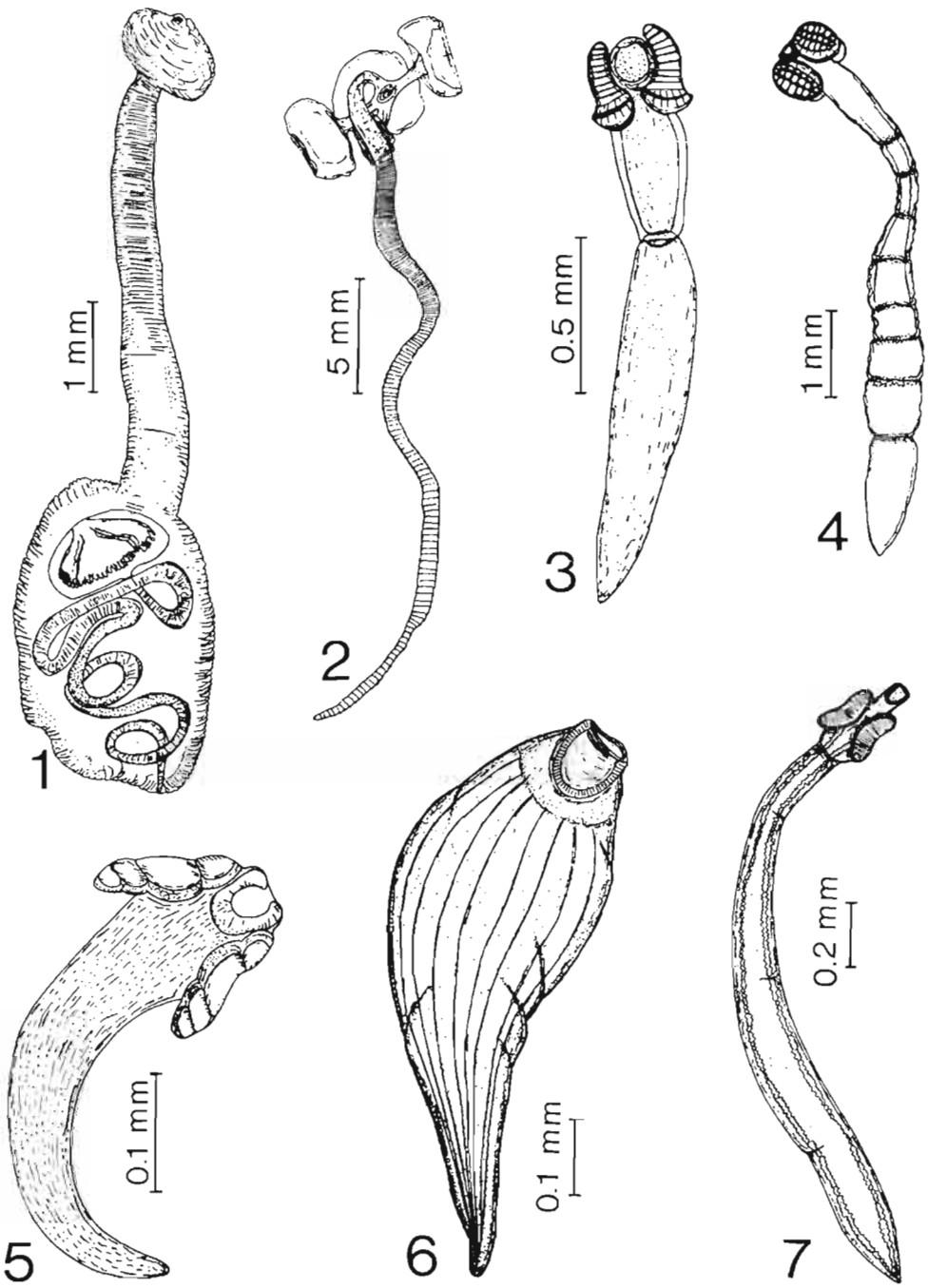


Fig. 13-150: Larval cestodes from marine bivalves and gastropods. 1: Claviform capsule containing bothridio-postplerocercoid of *Rhodobothrium* sp. from *Anadara transversa* (cut-away view showing coiled postplerocercoid); 2: postplerocercoid excised from capsule; 3: bothridio-plerocercoid of *Rhinebothrium* sp. from *Argopecten irradians concentricus;* 4: bothridio-plerocercoid of *Dioecotaenia cancellata* from *Chione cancellata;* 5: bothridio-plerocercoid of *Acanthobothrium* sp. (*brevissime?*) from *Ensis minor;* 6: uniacetabulo-plerocercoid of *Rhinebothrium* sp. from *Chione cancellata;* 7: bothridio-plerocercoid of *Rhinebothrium* sp. from gastropod *Busycon spiratum pyruloides.* (After Cake, 1976.)

Gaper clams *Schizothaerus nuttallii* (= *Tresus nuttalli*) from Elkhorn Slough, California, were found to be heavily infested with encapsulated cestode larvae. Up to 140 'cysts', each containing 1 to 5 worms, were dissected from the foot musculature of a single clam. Occasionally, individuals of *Macoma nasuta* and *Paphia* (= *Protothaca*) *staminea* from Elkhorn Slough, as well as *Pecten circularis aequisulcatus* from Newport Bay, also yielded this parasite whereas Washington clams *Saxidomus nuttalli*, which often occur in the same bed with *T. nuttalli*, were never found to be infested with encysted plerocercoids. The larvae were initially assigned to the genus *Phyllobothrium* and subsequently to *Anthobothrium*. The adult was believed to occur in bat sting rays *Aetobatus* (= *Myliobatis*) *californicus* (MacGinitie, 1935; MacGinitie an MacGinitie, 1949). The description given by these authors lacks sufficient detail to be diagnostic, but their figure appears to characterize the plerocercoid from *T. nuttalli* as a member of the genus *Echeneibothrium* or, more likely, *Rhinebothrium*, in the family Phyllobothriidae. Assuming that the drawing in MacGinitie and MacGinitie (Fig. 13-151) is sufficiently accurate and to scale, the plerocercoid is approximately 1.3 mm in length and has 4 bothridia, about 0.45 mm long and equipped with some 25 loculi separated by transverse septa and subdivided by a longitudinal septum.

Katkansky and co-authors (1969b) found what might be the same parasite in clams from Drakes Estero, California. Encapsulated plerocercoids occurred in the foot of nearly every *Tresus nuttalli* individual examined, and in one case a 'ciliated embryonic form' (probably a coracidium) was seen in sectioned material from the intestine of a gaper clam. In addition, free plerocercoids, as well as larvae considered to be precursors of the plerocercoid stage, were found in the gut lumen of both *T. nuttalli* and *Saxidomus nuttalli*, but encapsulated cestodes were not seen in the latter host, which was considered refractory to the tissue-invading stage. Whether the larval cestodes from the gaper clam and the Washington clam are specifically identical has not been established. The plerocercoids

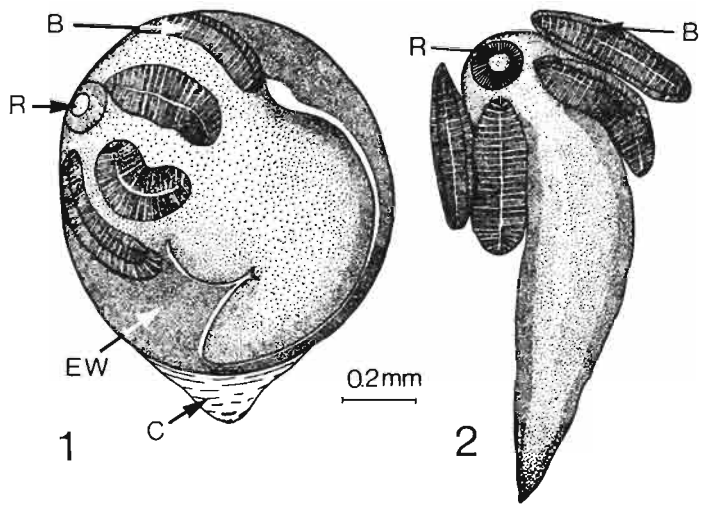


Fig. 13-151: Plerocercoid of *Echeneibothrium* sp. (or *Rhinebothrium* sp.?) from foot of *Tresus nuttalli*. 1: Larva enclosed by capsule of host origin; 2: plerocercoid excised from capsule. B bothridium, C capsule, EW metabolic wastes filling capsule lumen, R apical sucker. (After MacGinitie and MacGinitie, 1949.)

excysted from the gaper clams could not be identified to species level since the number of loculi on their bothridial surfaces exceeds that of any species described. The live plerocercoids from the intestine of the Washington clams, on the other hand, were believed to match the description of *Echeneibothrium maculatum*, as given by Wardle and McLeod (1952). As stated earlier, scolex morphology must not necessarily be identical in successive larval and adult stages. Thus, Hamilton and Byram (1974) and Cake (1976) observed transformation of the initially quadriloculate bothridia of an *Acanthobothrium* sp. plerocercoid from snails *Fasciolaria tulipa* into triloculate bothridia with an anterior accessory sucker and muscular pad, accompanied by loss of the muscular apical sucker, during *in vitro* development (see above).

Littleneck clams *Protothaca (Venerupis, Paphia) staminea*, found exposed on gravel beds on Bird Island in Humboldt Bay, California, proved to contain numerous small yellowish cysts in the mantle and foot, each of which harboured a tetraphyllidean plerocercoid identified as *Echeneibothrium* sp. Often more than 35 cysts per cross section were seen closely packed throughout all the body tissues (Fig. 13-152). The enclosed larvae (Fig. 13-153) exhibited a domelike rostellum (myzorhynchus) with an internal spherical muscle mass and a terminal opening in the centre. Each of the 4 bothridial surfaces was subdivided into 10 loculi. According to the key of Wardle and McLeod (1952), the plerocercoids appeared to represent *E. myzorhynchum* with respect to the number and arrangement of the bothridial loculi, but were more close to *E. fallax* with respect to the characteristics of the myzorhynchus (Sparks and Chew, 1966).

Double infestations involving 2 larval tetraphyllideans have been found in *Protothaca staminea* from Humboldt Bay by Warner and Katkansky (1969a). Cysts occurred in two distinct size groups. The larger ones, averaging 1.2 to 1.6 mm in diameter, were situated predominantly in the visceral area, while the smaller ones, approximately 0.6 to 0.9 mm in diameter, were mainly found in the peripheral and mantle region. Live plerocercoids excised from the large cysts were 2.0 to 2.4 mm in length, and each of the 4 broad, trumpet-like bothridia (0.6 to 0.8 mm long) was divided into 11 loculi subdivided by a longitudinal septum. The domelike, muscular rostellum described by Sparks and Chew (1966) was also seen. Larvae from the smaller cysts were 0.8 to 1.4 mm in length and each of the 4 narrow bothridia (0.4 to 0.5 mm long) was divided by transverse septa into a single row of 22 loculi. The rostellum was on an extensible pillar having an internal muscular mass with a terminal opening. In agreement with Sparks and Chew (1966), and according to Wardle and McLeod (1952), the larger plerocercoids more closely resembled *Echeneibothrium myzorhynchum* although disparities exist. The smaller larvae appeared to be referable to *E. maculatum* although positive identification was not possible.

Another larval *Echeneibothrium*, believed to be specifically different from the one described by Sparks and Chew (1966) in *Protothaca staminea*, parasitizes rough-sided littleneck clams *P. laciniata* in Morro Bay, California. This clam species is of rare occurrence in that area, and all of the 4 individuals found were heavily infested with plerocercoids, each enclosed by a yellowish capsule. Several cross sections of the clams' foot were examined and no fewer than 270 of these 'cysts' were counted. The living plerocercoids are approximately 1.2 to 1.8 mm in length and each of the 4 bothridia is divided by transverse septa into 24 loculi. The myzorhynchus is domelike, with an internal, spherical muscular mass and a terminal opening. The larvae resemble both *E. maculatum* and *E. fallax* with respect to certain morphological features although not close enough for

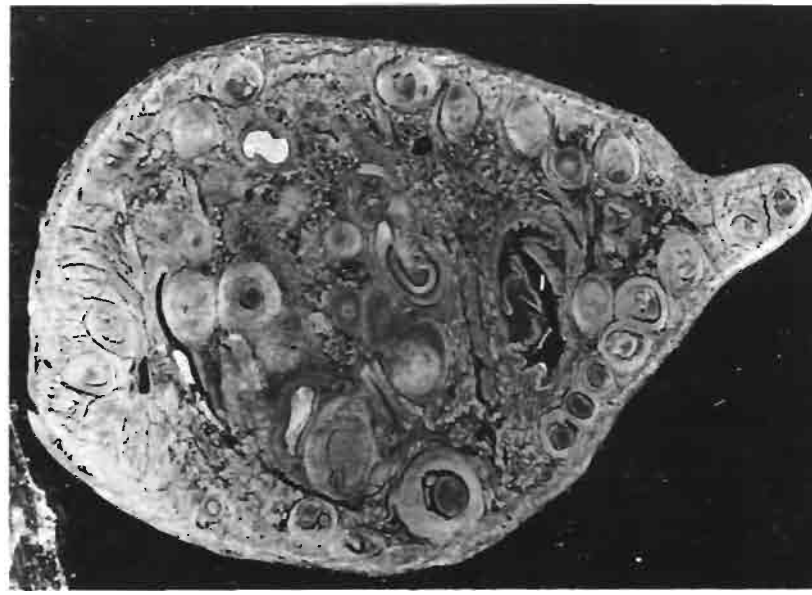


Fig. 13-152: *Protothaca staminea.* Cross section of entire specimen showing numerous encapsulated *Echeneibothrium* sp. plerocercoids throughout tissues, × 7.88. (After Sparks and Chew, 1966.)

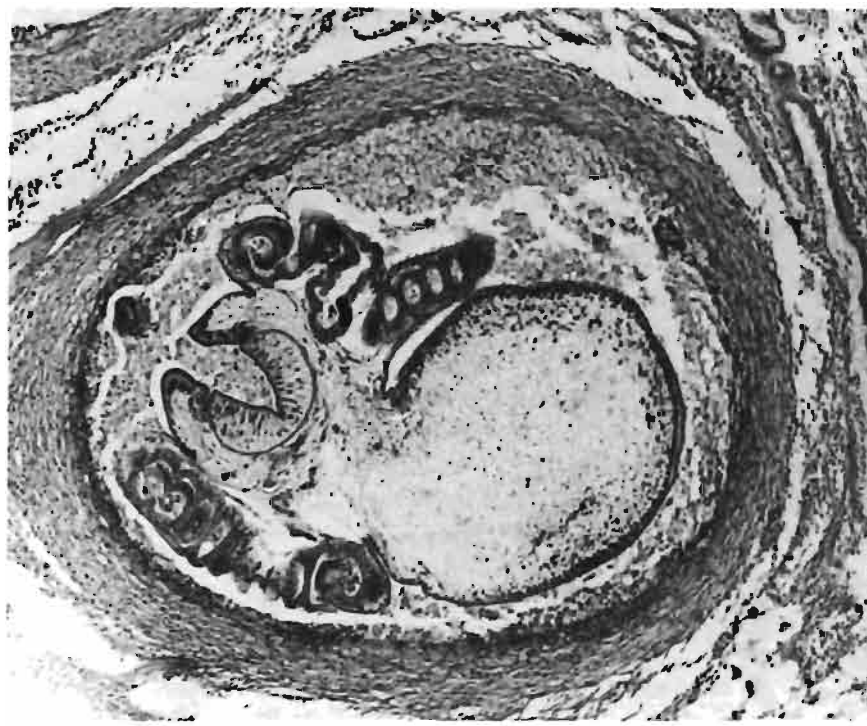


Fig. 13-153: *Echeneibothrium* sp. Plerocercoid encapsulated in tissue of *Protothaca staminea.* Note thick capsule of host origin surrounding larva, × 75.6. (After Sparks and Chew, 1966.)

positive identification (Katkansky and Warner, 1969). Cake (1975), however, believes that the above-described plerocercoids appear to be unidentified species of *Rhinebothrium* rather than *Echeneibothrium.*

Pismo clams *Tivela stultorum,* collected from the ocean beach near Watsonville, California, were found to be parasitized by plerocercoids tentatively identified as *Echeneibothrium myzorhynchum.* Its unusually large, yellowish-white cysts, measuring 3.2 to 3.8 mm in diameter, were located in the connective and gonadal tissue adjacent to the intestine. Very few cysts were found in each infested clam and they were extremely inconspicuous, being nearly identical in colour to the surrounding host tissue. Excised from their capsules, the larvae were 3.4 to 4.2 mm long (with rostellum everted) and possessed 4 oval bothridia, 1.6 to 1.8 mm in length and 0.8 to 1.0 mm in width, and with the bothridial surfaces divided into 10 loculi by 9 transverse septa and subdivided by 1 longitudinal septum. A domelike rostellum was present and contained an internal muscular mass, 0.50 to 0.65 mm in length when everted and 0.40 to 0.45 mm in width (Warner and Katkansky, 1969b). Wardle and McLeod (1952) state that the rostellum of *E. myzorhynchum* is cylindrical, while that of the larvae in *T. stultorum* more closely resembles the rostellum of *E. fallax* in being domelike. Moreover, the worms in the Pismo clam had bothridia of up to twice the maximum length reported by Hart (1936) in his original description of *E. myzorhynchum.* But Wardle and McLeod's (1952) and Hart's (1936) descriptions are of adults, and the discrepancies noted in the plerocercoids may be due to developmental changes presumed to occur in the definite host (Warner and Katkansky, 1969b). Riser (1955) points out that Hart's (1936) description of *E. myzorhynchum* was actually of a complex of species parasitizing big skates *Raja binoculata.*

Meretrix casta from the Madras coast (India) is host for another larval *Echeneibothrium* sp. Only 2 plerocercoids were found in the musculature of a clam. They measured 2,140 and 2,520 μm in length and 314 and 294 μm in width. The bothridia, with about 22 loculi, were 190 to 204 × 103 to 110 μm, and the myzorhynchus 60 to 72 × 82 to 99 μm in dimension (Reimer, 1975). All known species of *Echeneibothrium* parasitize elasmobranchs of the family Rajidae (Euzet, 1979).

Other phyllobothriid plerocercoids reported from marine bivalves include members of the genera *Anthobothrium* (= *Rhodobothrium*) and *Rhinebothrium.* Postlarvae of the former genus occur in *Argopecten irradians concentricus, Anadara transversa, Donax variabilis, Spisula solidissima similis, Tellina versicolor* and *Macrocallista nimbosa* from the Gulf of Mexico. The bothridio-postplerocercoids (Fig. 13-150, 2) are encapsulated singly in muscular, thin-walled, claviform sac-like blastocysts (Fig. 13-150, 1), the smaller end of which is embedded in the visceral mass of the host while the larger, bulbous end containing the coiled postplerocercoid hangs free in the mantle cavity. No precursor stages (procercoids, plerocercoids) of this cestode were encountered in any of the infested bivalves. Prevalence of the postlarvae was low, only 10 of a total of 247 bivalves harbouring 1 parasite each. Incidences were highest in *T. versicolor* (1 of 9 = 11.1 %) and *A. transversa* (6 of 56 = 10.7 %), and lowest in *A. irradians concentricus* (1 of 78 = 1.3 %) and *M. nimbosa* (1 of 69 = 1.4 %) (Cake, 1975, 1976, 1977a). Initially identified as *Rhodobothrium* sp. (Cake, 1973), the postlarvae were subsequently assigned to *Anthobothrium* (Cake, 1975). Campbell and Carvajal (1979) retransferred them to *Rhodobothrium* and speculated on their possible identity with *R. pulvinatum* parasitizing *Dasyatis americana* in the Gulf of Mexico (Linton, 1890).

Larvae, morphologically very similar to those reported by Cake and depicted in Fig. 13-150 (1, 2), have been described by Gallien (1950) from 2 of 900 *Mactra solida* at Saint-Malo (French coast of English Channel) as *Proboscidosaccus enigmaticus*. Anthouard (in Dollfus, 1964) obtained 3 further individuals from *M. solida* samples collected in the Loire estuary, and Dollfus (1974) found a single *P. enigmaticus* in an individual of *Ostrea edulis* from Quiberon (French Atlantic coast). The claviform sacs in *M. solida* measure from 14 to 20 mm in total length, the bulbous end being 4 to 5 mm long and 2.5 to 3 mm wide, and the peduncle measuring 10 to 15 mm in length and about 0.5 mm in diameter. The lumen of the bulbous extension is almost entirely filled by the coiled postplerocercoid. The individual found in *O. edulis* had a total length of 36 mm, the bulbous end measuring 6 × 4 mm and the peduncle 30 mm in length and 0.7 to 0.9 mm in diameter. *P. enigmaticus* was considered to be the larval stage of *Sphaerobothrium lubeti* Euzet, 1959, a phyllobothriid parasite of eagle rays *Myliobatis aquila* (Dollfus, 1974; Euzet, 1979).

Clams *Mesodesma donacium* from Chile are hosts for another similar larval cestode, named *Proboscidosaccus mesodesmatis* by Bahamonde and López (1962). Campbell and Carvajal (1979) discussed the synonymies existing in the phyllobothriid genera *Rhodobothrium* Linton, 1889, *Inermiphyllidium* Riser, 1955, and *Sphaerobothrium* Euzet, 1959, and assigned *Proboscidosaccus enigmaticus* and *P. mesodesmatis* to the genus *Rhodobothrium*. By means of *in vitro* cultivation, Carvajal and co-authors (1982) eventually confirmed the identity of the larvae from *M. donacium* with the postplerocercoid of *Rhodobothrium mesodesmatum*. The adult matures in the spiral valve of bat stingrays *Myliobatis chilensis*.

Plerocercoids of *Rhinebothrium* sp. (or spp.?) parasitize in the digestive diverticula of *Argopecten irradians concentricus, Anadara transversa, Amygdalum papyrium, Atrina rigida, A. seminuda, Donax variabilis, Dosinia discus, Ensis minor, Mactra fragilis, Noetia ponderosa, Periploma inaequale, Raeta plicatella, Spisula solidissima similis, Tagelus divisus, T. plebeius* and *Trachycardium egmontianum*, as well as free in the digestive tract of 14 species of molluscivorous and filter-feeding gastropods from the Gulf of Mexico. The bothridio-plerocercoids occurring in the bivalves are small, 0.3 to 4.6 mm long, and have spoon-shaped bothridia, divided into a narrow anterior region with 8 transverse loculi and a cuplike posterior region with 8 radially arranged loculi (Fig. 13-150, 3). Those found in the alimentary tract of molluscivorous gastropods are larger, 0.4 to 15.1 mm long, more advanced, and possess leaflike bothridia with up to 33 transversely arranged loculi (Fig. 13-150, 7). The generic identity of both forms has been confirmed by experimental cultivation and infestation studies. In addition to the above bothridio-plerocercoids, small uniacetabulo-plerocercoids (Fig. 13-150, 6) frequently occur in the digestive gland of bivalves concurrently infested with advanced bothridio-plerocercoids. They probably represent early larvae of *Rhinebothrium*. In some instances, a complete developmental sequence of plerocercoids is present in a single host. Overall incidence of infestation with *Rhinebothrium* sp. in the above pelecypods was 44.8 % (212 of 474) and ranged from 4.2 % in *T. egmontianum* to 96.2 % in *N. ponderosa*. The infested bivalves yielded a total of 2,918 plerocercoids, with an average of 13.8 larvae per infested host and a maximum of 200 in *A. irradians concentricus* (Cake, 1975, 1976, 1977a, b).

Only 1 of at least 11 distinct species of larval cestodes parasitizing Gulf of Mexico molluscs could be identified to species level. This is *Dioecotaenia cancellata*, whose large (ca. 8 mm) bothridio-plerocercoids (Fig. 13-150, 4) occur in low abundance in the

digestive diverticula of *Chione cancellata* and *Anadara ovalis*, as well as in the stomach of crown conchs *Melongena corona*. Its bothridia are oval, slightly cupped, and divided into 21 loculi (Cake, 1976). The adult has first been described as *Rhinebothrium cancellatum* from the spiral valve of a cow-nosed ray *Rhinoptera bonasus* taken off Woods Hole, Massachusetts, by Linton (1890). Schmidt (1969) redescribed the species as *Dioecotaenia cancellata*.

Despite the frequent occurrence of tetraphyllidean cestodes in marine bivalves, next to nothing is known about their pathology. Sparks and Chew (1966) described the heavy capsule of host origin formed around the plerocercoids of *Echeneibothrium* sp. in littleneck clams *Protothaca staminea*. It was believed to consist of a compact network of fine collagenous fibres and numerous haemocytes. Within the capsules were quantities of amorphous material, probably excretory products of the enclosed worm, and occasional clumps of host haemocytes. The remaining host tissues were normal in appearance except for the 'crowding effect' of the numerous cysts. Cheng (1967), Rifkin and Cheng (1968) and Cheng and Rifkin (1970) doubt that the capsule fibres formed around the plerocercoids of *Echeneibothrium* sp. are collagenous. They point out that Sparks and Chew (1966) only examined sections stained with Harris' hematoxylin and eosin and hence their conclusion concerning the chemical nature of the fibres cannot be sustained. Katkansky and Warner (1969) and Warner and Katkansky (1969a), being aware of the objections by the above authors, subjected *Echeneibothrium* sp. capsules in *Protothaca staminea* and *P. laciniata* to the collagen staining procedure of Pauley (1967b), and collagen was noted to be present.

Although accelerated mortality of bivalves infested with larval tetraphyllideans has not been documented in the literature, there can be little doubt that infestations of such a magnitude as those reported by MacGinitie (1935), MacGinitie and MacGinitie (1949), Sparks and Chew (1966) and Katkansky and Warner (1969) severely affect the health of infested clams. 'Surfacing' of normally burrowed bivalves, also commonly observed in trematode-infested individuals (pp. 749 and 750), may be interpreted as an abnormal, pathological response to larval helminth invasion. Sparks and Chew (1966) noted large numbers of littleneck clams *Protothaca staminea* to occur exposed on gravel beds at Bird Island, Humboldt Bay, California. Since this clam typically spends its entire post-larval life burrowed, a diseased condition was immediately suspected. Such 'kickouts' are known to occur sporadically in *Protothaca* populations on Washington beds. Inspection of exposed clams found at Bird Island revealed heavy infestation with *Echeneibothrium* sp. plerocercoids (p. 779). Although the authors were not quite sure that this surfacing response was elicited by the parasites, they (1966, p. 415) felt that there are strong arguments in favour of such speculation:

> "If the littleneck clam or other burrowing molluscs serve as the only hosts of the plerocercoid stages of *Echeneibothrium*, then some means of making the clams accessible to rays and skates would facilitate completion of the life cycle."

In contrast, Warner and Katkansky (1969a) collected *Echeneibothrium*-infested *Protothaca staminea* from a sediment depth of at least 10 cm. Since the infestation intensity observed by them was much lower than the previously reported one — maximal 8 capsules per clam, as opposed by up to 35 cysts per cross section found by Sparks and Chew (1966) — they concurred with the latter authors' hypothesis. On the other hand, even heavy infestation of *P. laciniata* with another species of larval *Echeneibothrium*

apparently did not cause these clams to leave their normal habitat (Katkansky and Warner, 1969). However, only 4 rough-sided littleneck clams have been found during that study.

Questions have been raised as to whether larval cestode infestations in edible or potentially edible marine bivalves may present a hazard for human health. Among the commercially exploited species, particularly American oysters *Crassostrea virginica*, Atlantic bay scallops *Argopecten irradians concentricus* and sunray venus clams *Macrocallista nimbosa* have been found to be heavily infested. A single species, *Tylocephalum* sp., has been reported from *C. virginica*, but infestation incidences and intensities may locally be very high. Larval helminth infestations in oysters require particular attention because large quantities of these molluscs are consumed living (Cheng, 1967). Other bivalves are occasionally eaten raw by epicurean beachcombers. In the Gulf of Mexico, *A. irradians concentricus* was infested by the largest number of species, i.e., seven. Again, *Tylocephalum* sp. was the most abundant parasite, followed (in descending order) by *Rhinebothrium* sp., *Polypocephalus* sp., *Parachristianella* sp. (*dimegacantha*?), *Eutetrarhynchus* sp. and *Acanthobothrium* sp. *Macrocallista nimbosa* was most heavily parasitized by *Parachristianella* sp. (Cake, 1977a). None of these selachian tapeworms is known to infest humans.

Agents: Nematoda

Marine bivalves, as a group, are rather uncommon hosts for nematodes, but ascaridoidean and spiruroidean roundworms have been reported on several occasions from representatives of this molluscan class, mainly from commercially important species. All nematodes encoutered in marine pelecypods are larvae. Pathology associated with their presence, in molluscan tissues, is moderate. Some species are potentially hazardous to human health.

Ceylonese pearl oysters *Pinctada margaritifera* have been found to harbour, mainly in the gonad, larval ascaridoidean nematodes, described as *Ascaris meleagrinae* by von Linstow (in Shipley and Hornell, 1904). The worms were believed to mature in the intestine of teleost fishes. Baylis (1936) tentatively ascribed these larvae to the genus *Paranisakis*, whose members parasitize as adults in the intestine of elasmobranchs. N. A. Cobb (1930) described *Paranisakis pectinis* on the basis of a single larva, recovered from "the visceral mass of a scallop (*Pecten*)" probably *P.* (*Aequipecten*) *maximus*, from Beaufort, North Carolina (USA). Hutton (1964), who refound what appears to be the same nematode in *Argopecten gibbus* from the east coast of Florida, disagreed with Cobb's designation and transferred the species to the genus *Porrocaecum* as *P. pectinis*, under which name it has subsequently been listed by Cheng (1967, 1973b), Cheng and Rifkin (1970), Sindermann (1970a) and Broom (1976). The genus *Porrocaecum*, however, contains only members parasitic as adults in birds, not in marine vertebrates (Hartwich, 1957, 1959, 1975). Perkins and co-authors (1975, 1977), Lichtenfels and co-authors (1976) and Perkins (1979) provisionally assigned third- and fourth-stage larvae recovered from *Spisula solidissima* to the genus *Paranisakiopsis*, whose members are parasitic as adults in macrourid fishes (Yamaguti, 1941). Attempts to identify the larvae by means of feeding experiments and *in vitro* cultivation were hampered by the occurrence of a disease-causing haplosporidian hyperparasite in nearly 100 % of the nematodes. Neither of the above allocations is correct.

In his revision of the nematode superfamily Ascaridoidea, Hartwich (1957) erected the genus *Sulcascaris* to contain *Ascaris sulcata*, described by Rudolphi (1819) from marine turtles *Chelonia mydas* and *Caretta caretta*. Allison and co-authors (1973) disagreed with Hartwich's (1957) concept and misclassified adult worms taken from stomachs of these hosts as *Porrocaecum sulcatum*. It remained to Sprent (1977) to demonstrate experimentally the specific identity of the larval nematodes — recovered, in this case, from scallops *Amusium balloti* and *Chlamys* sp. in Queensland (Australia) coastal waters — with adult *Sulcascaris sulcata* from the stomach of *Chelonia mydas* and *Caretta caretta*. Berry and Cannon (1981) traced the entire life cycle of the worm using moon snails *Polinices sordidus*, as well as bivalves *Pinctada* spp. and *Melina ephippium* as experimental intermediate hosts and laboratory-reared loggerhead turtles *C. caretta* as definite hosts. Eggs laid by adult females are negatively buoyant, sink to the bottom, and tend to adhere to the substratum. There are 2 moults in the egg, second-stage larvae appearing 4 days, and third-stage ones 5 days after the eggs had been laid. Third-stage larvae, about 0.4 mm in length, hatch spontaneously in sea water of 25 °C from the seventh day on. These hatched larvae infest the molluscan intermediate host, in which they grow to a length of about 5.3 mm in 140 to 210 days. From about day 115 on, the larvae commence to moult into fourths, which grow from an initial size of 8.09 ± 2.61 mm (n = 6) to 17.82 ± 4.57 mm (n = 10) by day 177. Fourth-stage larvae in molluscs ingested by *C. caretta* attach at the oesophago-gastric junction and moult into adults in 7 to 21 days. Subsequent growth to mature adults takes at least 5 months.

The stage of *Sulcascaris sulcata* normally encountered in naturally infested molluscs is the fourth-stage larva, although third-stage larvae may be found occasionally. Worms from *Amusium balloti* were 31.76 ± 4.86 mm in length and 0.57 ± 0.06 mm in width (n = 16). Body dimensions of larvae dissected from different host species show considerable variation. Whether all these larvae belong to a single species, *S. sulcata*, remains to be established. Most authors have given only the *range* of the respective measurements, but these cannot be submitted to exact statistical comparison. The only papers providing exhaustive data (mean ± standard deviation, number of observations) are those of Cannon (1978) and Berry and Cannon (1981). Unfortunately, studies on growth allometry, which would be helpful in identifying and comparing larvae and adults of different size and from different hosts, have not yet been conducted. At present, *S. sulcata* is the only recognized species in the genus, but Sprent (1977) has recovered adult *Sulcascaris* from *Caretta caretta*, which differ from 'normal' *sulcata*. Detailed descriptions, drawings, light and electron micrographs and morphometric data of larval and adult *S. sulcata*, provided by Allison and co-authors (1973), Sprent (1977), Cannon (1978), Lichtenfels and co-authors (1978) and Berry and Cannon (1981), may be consulted for comparison.

Sulcascaris sulcata is widespread in warm seas and has a considerable host range. Thus far, its fourth-stage larvae have been found in 9 bivalve and 5 gastropod species. Additional mollusc species have been infested experimentally. Prevalences may vary considerably with locality and host species (Table 13-32). The worms normally occur singly, but up to 7 larvae have been recovered from individual surf clams on the U.S. Atlantic coast (Payne and co-authors, 1980). One spiny oyster *Spondylus ducalis* from Bundaberg, Queensland, even contained 15 fourth-stage *S. sulcata* (Cannon, 1978). Small bivalves usually harbour fewer nematodes than larger ones. Mean numbers present in 916

Table 13-32
Sulcascaris sulcata. Host range and prevalence of 4th-stage larvae from marine molluscs (Compiled from the sources indicated)

Host species (in alphabetical order)	Prevalence (n.i. not indicated)	Geographical area	Source*)
Amusium balloti	n.i.	Queensland, Australia	*15, 17*
" "	25 of 467; 1.08 worms host^{-1}	Bundaberg, Queensland	*1*
" "	up to 64 %	Shark Bay, Western Australia	*6*
Argopecten gibbus	n.i.	East Florida coast	*4, 16*
" "	n.i.	U.S. Atlantic coast	*8*
" "	up to 40 %	Cape Canaveral, Florida	*9*
Argopecten irradians	2.3 % of 400	North Carolina	*2*
" "	n.i.	U.S. Atlantic coast	*8*
Busycon canaliculatum	n.i.	New Jersey to North Carolina	*7, 8*
Chlamys sp.	n.i.	Queensland	*15*
Cypraea tigris	1 worm in 1 host	Great Barrier Reef, Australia	*1*
Fasciolaria lilium hunteria	n.i.	Cape Canaveral	*9*
Lunatia heros	n.i.	New Jersey to North Carolina	*7, 8*
Pecten sp. (*maximus*?)	1 worm in 1 host	Beaufort, North Carolina	*3*
Pinctada margaritifera	5.6 %	Gulf of Mannar, Ceylon	*14*
Pinna menkei	1 worm in 1 host	Moreton Bay, Queensland	*1*
Pleuroploca gigantea	n.i.	Dry Tortugas, Florida	*10*
Spisula solidissima	16 % of 894	North Carolina, Virginia	*13*
" "	up to 50 %	New Jersey to North Carolina	*7, 8*
" "	173 of 1,293 (overall), locally from 2 to 78 %	New Jersey, Maryland, Virginia	*5*
" "	752 worms in 1,894 hosts (max. 0.37 worms host^{-1})	U.S. Midatlantic States	*11*
" "	771 of 3,358; 0.51 worms host^{-1}	New Jersey, Delaware, Maryland, Virginia	*12*
Spondylus ducalis	1 individual infested with 15 worms	Bundaberg, Queensland	*1*
Polinices sordidus	13 larvae in 1 snail	Experimental infestations	*17*
Pinctada spp.	4 to 10 larvae per host		
Melina ephippium	1 to 36 larvae per host		

*) Sources: *1* Cannon (1978), *2* Cheng (1973b), *3* Cobb (1930), *4* Hutton (1964), *5* Kern (1977), *6* Lester and co-authors (1980), *7* Lichtenfels and co-authors (1976), *8* Lichtenfels and co-authors (1978), *9* Lichtenfels and co-authors (1980), *10* Overstreet (in Lichtenfels and co-authors, 1980), *11* Payne and co-authors (1977), *12* Payne and co-authors (1980), *13* Perkins and co-authors (1975), *14* Shipley and Hornell (1904), *15* Sprent (1977), *16* Lichtenfels and co-authors (1977), *17* Berry and Cannon (1981).

Amusium balloti, divided into six 10-mm size classes ranging from 56 to 115 mm shell height, were 0, 0.1, 0.9, 0.44, 0.83 and 2.16, respectively. The coefficient of dispersion indicates that the distribution of the worms among the scallops was clumped (mean 0.75, variance 1.71) (Lester and co-authors, 1980). There is no apparent seasonal fluctuation of incidence, indicating that the parasites persist and accumulate in the hosts. Samples of *Spisula solidissima* from more northerly regions of the U.S. Atlantic coast have fewer worms than samples from lower latitudes. Overall prevalences were found to increase from 2 % in clams from New Jersey and Maryland to 32 % in clams from Virginia, with local

variations between 0 and 8 % in the New Jersey and Maryland samples and between 4 and 78 % (!) in the Virginia samples (Kern, 1977). This corresponds to the geographical distribution of the reptilian definite hosts (Payne and co-authors, 1980).

In *Spisula solidissima*, *Sulcascaris sulcata* inhabits all tissues, and its numbers in the edible and non-edible portions are similar. In particular, 60 % of the 173 worms recovered from 1,293 clams were found in the visceral mass, 27 % in the foot, 12 % in the adductor muscles and 1 % in the mantle. Parasitized clams tended to be slightly larger than unparasitized ones (Kern, 1977).

Normally, there is no gross tissue reaction in response to the invaders, but occasionally a thickening of clam tissue surrounding the larvae may be observed. Some worms occur embedded in individual cysts of host origin (Payne and co-authors, 1980). In scallops, *S. sulcata* inhabits mainly, although not always, the adductor muscle. The worms are white and inconspicuous when small, and yellow to pale orange or brown when larger. They usually lie loosely coiled within the superficial (1 to 5 mm deep) regions of the muscle, causing it to assume a brownish discolouration and to become caseous. There is considerable cellular infiltration about the larvae, which may contribute to the staining of the tissues. The presence of more than one larva causes sufficient damage to the muscle that much of its tonicity is lost. Removal of affected muscle portions during processing may cause an increase of costs (Cheng, 1973b; Cannon, 1978). In *Argopecten gibbus* screened by Lichtenfels and co-authors (1980), all *S. sulcata* occurred in the gonads, and were found more frequently in larger scallops. Gonad colour is commonly used to determine ovarian development in *A. gibbus*. Misleading ovarian colour changes may be produced by *S. sulcata* infestations (Miller and co-authors, 1980).

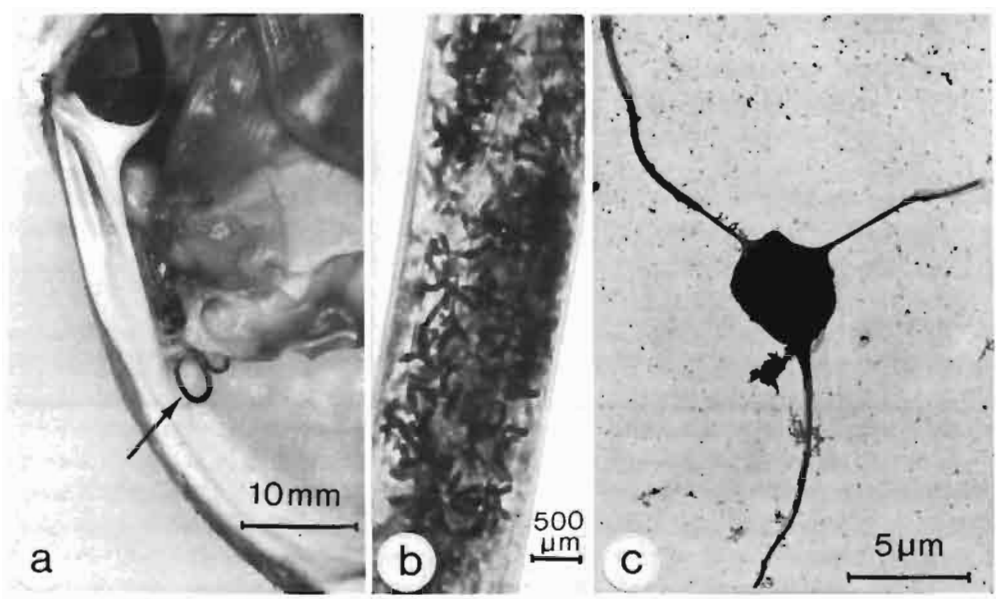


Fig. 13-154: *Urosporidium spisuli*. (a) Individual of *Spisula solidissima* showing *Sulcascaris sulcata* (arrow), hyperparasitized by *U. spisuli, in situ* between adductor and foot retractor muscles. (b) Hyperparasitized nematode containing numerous vermiform *U. spisuli* sporocysts. (c) *U. spisuli* spore showing 3 appendages. (After Perkins and co-authors, 1975.)

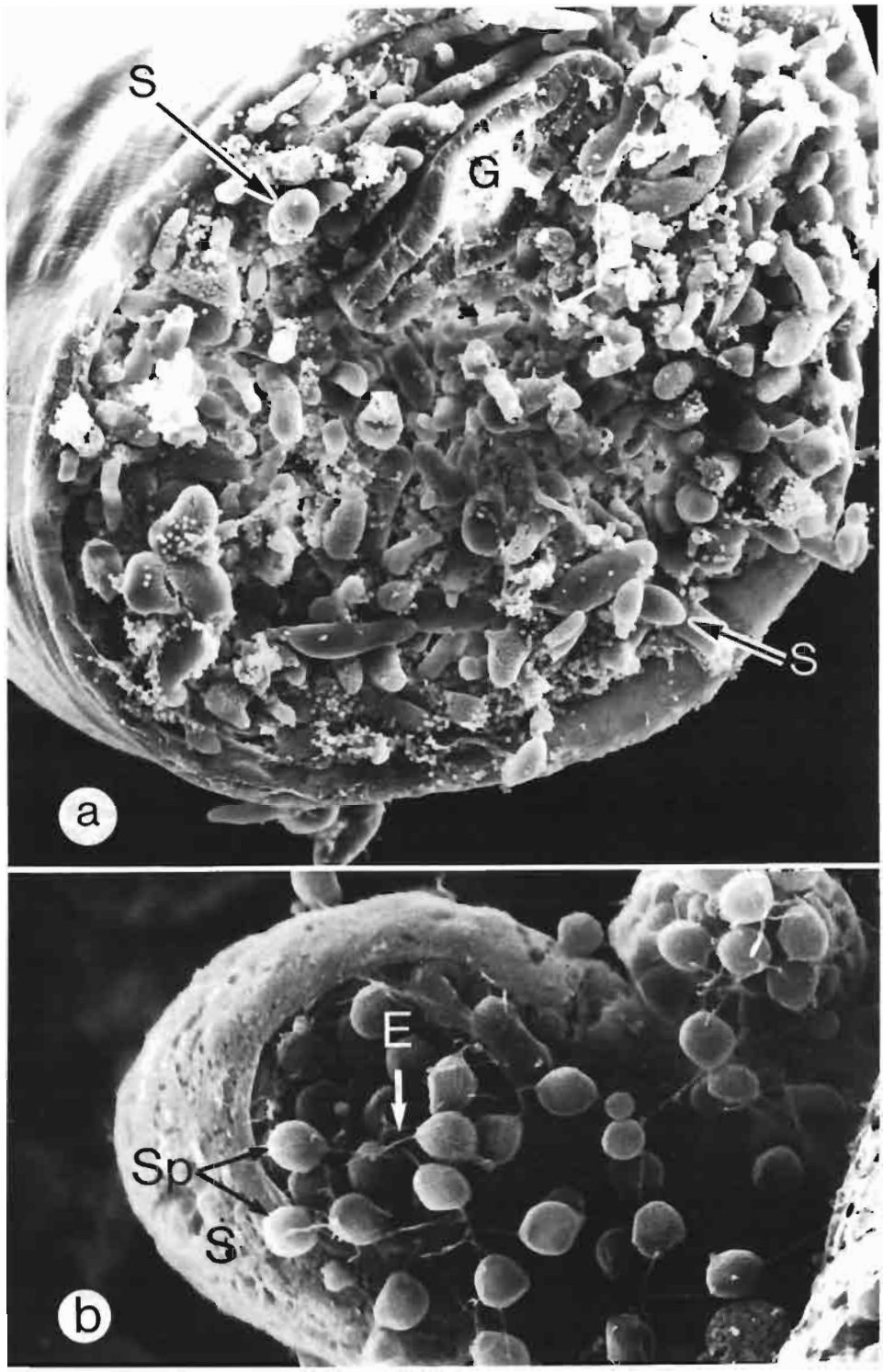


Fig. 13-155: *Urosporidium spisuli.* (a) Densely packed sporocysts in pseudocoel of *Sulcascaris sulcata,* × 140. (b) Ruptured sporocyst with liberated spores, × 2,000. E extension of epispore cytoplasm, G nematode gut, S sporocyst, Sp spores. (After Perkins and co-authors, 1977.)

Spisula solidissima has become an important species in the United States seafood industry. In 1973, for instance, the value of processed surf clam products reached a record level of about 38 million $. It was, therefore, of considerable economic concern when clammers and processors complained of 'long dark worms' in the clam meat (Fig. 13-154, a). It was this observation that actually led to the discovery of the presence of *Sulcascaris sulcata* in *S. solidissima*. The worms were not previously noticed or reported, probably because they are normally rather inconspicuous and similar in colour and density to the tissues of this clam. It was then determined that the nematodes become dark brown to brownish-black when a haplosporidian hyperparasite, occurring in the pseudocoel, sporulates and forms dense masses of golden brown spores enclosed by vermiform sporocysts (Fig. 13-154, b). Sightings of the brown worms resulted in clams being withheld from the commercial trade in Maryland (Perkins and co-authors, 1975).

The hyperparasite was determined to be a new species of *Urosporidium*, termed *U. spisuli*, and being the first recognized representative of the genus parasitizing a nematode. Its vermiform sporocysts (Fig. 13-155, a) are branched or unbranched and up to 0.5 mm long. Living spores are ovoidal and measure $4.9 \pm 0.05 \times 4.4 \pm 0.04$ µm ($\bar{x} \pm$ S.E., $n = 100$). The epispore cytoplasm extends centrifugally into 2 or 3 conspicuous, slender extensions, about 7 to 14 µm long (Figs 13-154, c and 13-155, b) (Perkins, 1975, 1979; Perkins and co-authors, 1977).

In the mid-seventies, an epizootic spread of *Urosporidium spisuli* in *Sulcascaris sulcata* infesting surf clams appears to have occurred along the United States Atlantic coast. According to Perkins and co-authors (1975), Lichtenfels and co-authors (1976, 1978), Kern (1977) and Payne and co-authors (1980), the proportion of hyperparasitized nematodes was 100 % or close to that level in surf clams collected from New Jersey to North Carolina waters in 1974-75. By the end of 1975 or during the first half of 1976, the epizootic appears to have subsided abruptly. Of 3,358 *Spisula solidissima*, collected during July 1976 to September 1977 from New Jersey, Delaware, Maryland and Virginia commercial beds, 771 (23 %) had *S. sulcata* infestations. Of the 1,696 worms obtained from these clams, only 15 (0.88 %) were hyperparasitized (Payne and co-authors, 1980). A similar decline was recorded by Kern (1977).

Published data are too scanty to determine with confidence whether there was a concomitant overall decrease in *Sulcascaris sulcata* infestations in surf clams, but such a decline appears likely. As indicated by the inspection of hyperparasitized worms, as well as by fruitless feeding and cultivation tests, the haplosporidian debilitates the nematodes. Diseased worms were 'generally larger' (hypertrophied?) than healthy ones (Lichtenfels and co-authors, 1976, 1978). Ingestion, by the definite host, of such debilitated larvae would result in a disruption of the life cycle of the parasite and, hence, prevent subsequent reinfestation of the molluscan intermediate hosts. Therefore, the conclusion of Payne and co-authors (1980, p. 152),

> "Because unparasitized nematodes are pale and difficult to see in the clam flesh, a false impression may have been created among casual observers that surf clams in 1976-1977 contained fewer worms than in the preceding years",

appears untenable.

Different conditions appear to prevail in *Argopecten gibbus*. Samples of this scallop species, collected near Cape Canaveral, Florida, seven years apart, showed a consistent level of *Sulcascaris sulcata* infestation (1970: 40 %, 1976: 38 %, 1977: 35 worms in 35

scallops, percentage infestation not indicated) (Lichtenfels and co-authors, 1980). It appears worth mentioning, in this context, that nematodes in the scallops are never hyperparasitized. The occurrence of normal fourth-stage larvae in molluscan 'reservoir' hosts other than *Spisula solidissima*, which are capable of maintaining the life cycle of the parasite, merits special attention.

From the evidence presented, the *Spisula solidissima – Sulcascaris sulcata – Urosporidium spisuli* complex appears to be an ideal model for the study of biological interaction and control. As far as has been determined, only worms in the surf clam become hyperparasitized (Sawyer and co-authors, 1980). Lichtenfels and co-authors (1980), who examined *S. sulcata* from several lots of *Argopecten gibbus*, found no trace of the haplosporidian. *U. spisuli* has not been reported from anyone of the remaining 12 molluscan hosts of *S. sulcata*. This may lead one to speculate that *S. solidissima* is an essential, obligate host for one or several stages in the life cycle of *U. spisuli*. Unfortunately, the entire life cycle of the haplosporidian, as well as the mode of infestation of the nematodes, are not known. It is unlikely that *U. spisuli* is transmitted directly from worm to worm. With the possible exception of *Haplosporidium* ('*Minchinia*') *pickfordae* (Barrow, 1965), no haplosporidian spores have as yet been shown to produce infestations in individuals of the same species from which they were obtained (Pixell-Goodrich, 1915; Taylor, 1966; Perkins and co-authors, 1975).

Depending on whether a clam or scallop is consumed cooked or uncooked, *Sulcascaris sulcata* may be regarded as filth to be removed for aesthetic reasons, or as a threat to public health. At present, neither the nematode nor its haplosporidian hyperparasite are regarded as hazardous to humans. Cannon (1978) failed to experimentally infest teleost and elasmobranch fish, and worms did not become established in laboratory-held chickens and cats (Berry and Cannon, 1981). *S. sulcata* does not survive at 37 °C or above (Bier in Lichtenfels and co-authors, 1980). However, it has been demonstrated that other larval nematodes that do not normally survive in warm-blooded hosts will survive when adapted to higher water temperatures (Ko, 1976, 1977; Norris and Overstreet, 1976; see below).

Larval spiruroidean gnathostomatid nematodes of the genus *Echinocephalus* have occasionally been reported from bivalves, particularly from oysters and pearl oysters, in tropical and subtropical marine waters. The genus *Echinocephalus* has been erected by Molin (1858) to contain *E. uncinatus*, an adult nematode parasitic in the spiral valve of sting rays *Trygon brucco* in the Adriatic Sea. As pointed out by Baylis and Lane (1920), his description was based on material containing individuals of 2 different species. The latter authors claimed to have found a single larva of *E. uncinatus* encysted in a *Pinna* sp. from Ceylon. They furthermore assumed that this larva is specifically identical with those reported from the adductor muscle of Ceylonese pearl oysters *Pinctada margaritifera* as *Cheiracanthus uncinatus* by von Linstow (in Shipley and Hornell, 1904) and as *Echinocephalus gracilis* by Stossich (in Shipley and Hornell, 1906). Johnston and Mawson (1945) and Anantaraman (1964) assigned to *E. uncinatus* larvae recovered from Australian and Indian marine gastropods. Probably neither of these larval forms is identical with *E. uncinatus*.

Ko and co-authors (1974) briefly reported on the occurrence of larval *Echinocephalus* sp. in *Crassostrea gigas*, cultivated in a brackish-water area in Hong Kong. Subsequently, Ko (1975) described adult worms recovered from the intestine of eagle rays *Aetobatus flagellum*, caught in the vicinity of oyster beds from which infested *C. gigas* were obtained,

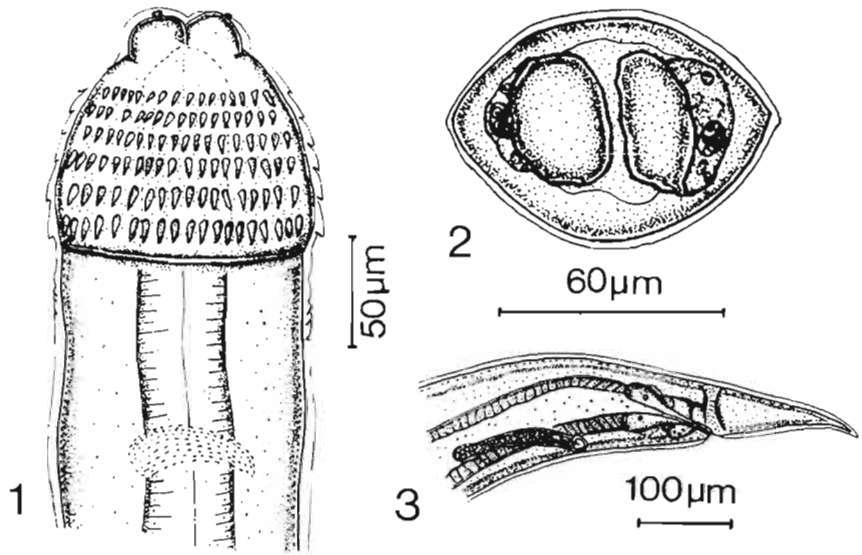


Fig. 13-156: *Echinocephalus sinensis*. Second-stage larva from *Crassostrea gigas*. 1: Anterior region, lateral aspect; 2: *en face* view; 3: posterior region of female, lateral aspect. (After Ko, 1975.)

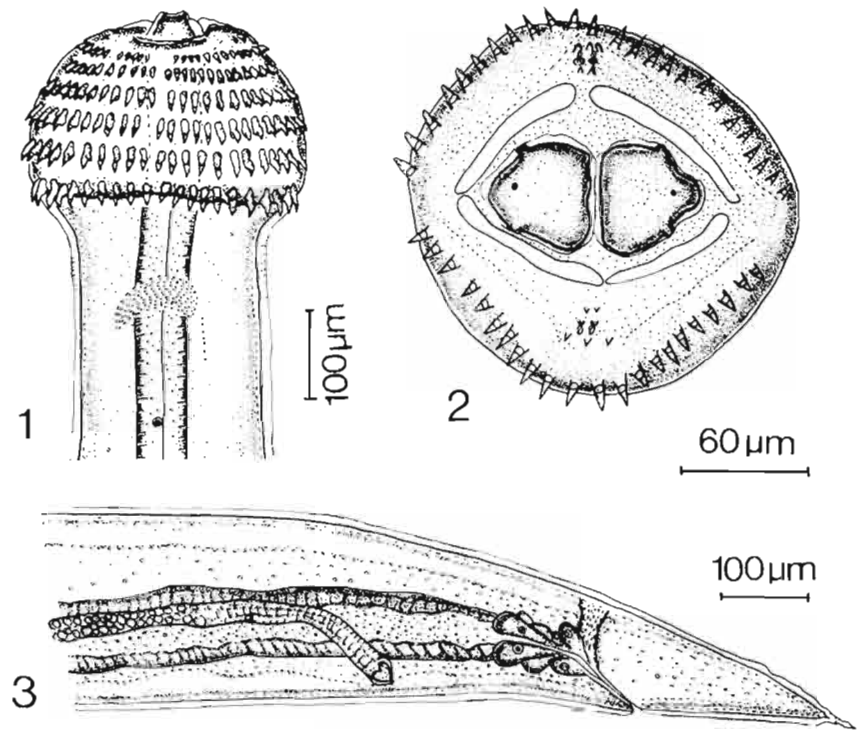


Fig. 13-157: *Echinocephalus sinensis*. Third-stage larva from *Crassostrea gigas*. 1: Anterior region, lateral aspect; 2: *en face* view; 3: posterior region of female, lateral aspect. (After Ko, 1975.)

as *E. sinensis*. Second- and third-stage nematodes, found in *C. gigas*, were believed to represent larval *E. sinensis*, but infestation experiments ascertaining this assumption were not performed. In a parallel study, Cheng (1975a) described the larval worms from *C. gigas* as *E. crassostreai*. Since Ko's species description was published before that of Cheng, the correct name for the oyster nematode is, according to the law of priority, *E. sinensis*, provided that the larva from the oyster and the adult from the eagle ray are specifically identical.

Male second-stage larvae of *Echinocephalus sinensis*, taken from *Crassostrea gigas*, are 6.4 ± 0.8 mm (n = 5) in length; females measure 7.1 ± 1.2 mm (n = 4). Corresponding dimensions of male and female third-stage larvae are 11.6 ± 1.1 mm (n = 20) and 11.2 ± 0.8 mm (n = 20), respectively. Second-stage larvae have a conical head with 6 rows of cephalic spines, 19 to 22 μm in length, while third-stage individuals, which represent the most commonly encountered type, have a bulbous head with 7 rows of cephalic spines, 7 to 26 μm long, the first row being inconspicuous, with only 6 small spines (Figs 13-156 and 13-157). Sexual dimorphism is noticeable in both types of larvae. First-stage larvae were not recovered. As suggested by Millemann (1963), the life cycle of echinocephalids probably involves only one (molluscan) intermediate host. Eggs probably embryonate in the external environment. How the early larvae invade the oyster remains unknown (Ko, 1975).

Echinocephalus sinensis larvae inhabit primarily the lumen of the oyster's gonoduct. There are no histopathological changes in the cells proper lining the gonoduct in the vicinity of the parasites, but there is a formation of a tunic of reaction elements surrounding the gonoduct (Fig. 13-158, a). This tunic, which averages 0.15 mm in thickness, consists of connective-tissue fibres, haemocytes (primarily granulocytes), myofibres, tightly packed Leydig cells and brown cells. A comparable structure does not exist in unparasitized oysters (Cheng, 1975a). Ko and co-authors (1975), however, found the lumina of infested gonoducts to be greatly enlarged. Desquamation, erosion and disruption of the ciliated duct epithelium also occurred, possibly due to the action of the spines of *E. sinensis* as it moved. Most of the normal pseudostratified columnar ciliated epithelium had been transformed into a cuboidal or squamous type. Disruption of the duct epithelium, if it occurred, was associated with invasion, of the lumen, by massive numbers of hypertrophied haemocytes, some of which were seen adhering to the cuticle of the nematodes (Fig. 13-158b, d). Occasionally, the duct lumen became occluded by haemocytes. It is not known whether *E. sinensis* in heavy infestations can affect the reproductive capacity of *Crassostrea gigas* by obstructing the genital tracts.

The presence of *Echinocephalus sinensis* in the gonoduct of *Crassostrea gigas* has also been found to cause hyperactivity of gland cells embedded in the epithelial lining of the duct. The rare occurrence of worms in the oyster's gonad appears to result in displacement, shrinkage, rupture and compression of ova in regions adjacent to the nematodes. Because of the spatial separation of the intragonoductal parasites from the periductal reaction complex, the hypothesis was advanced that the reaction is stimulated by molecules secreted and/or excreted by the worms and being able to permeate the lining epithelium and subtending basal lamina (Cheng, 1975b, 1978).

The prevalence of *Echinocephalus sinensis* in *Crassostrea gigas* from Hong Kong is fairly high. Cheng (1975a) found 27 of 200 oysters, collected in July and August 1974 in Deep Bay, New Territories, to be infested with the larvae. Ko and co-authors (1974, 1975)

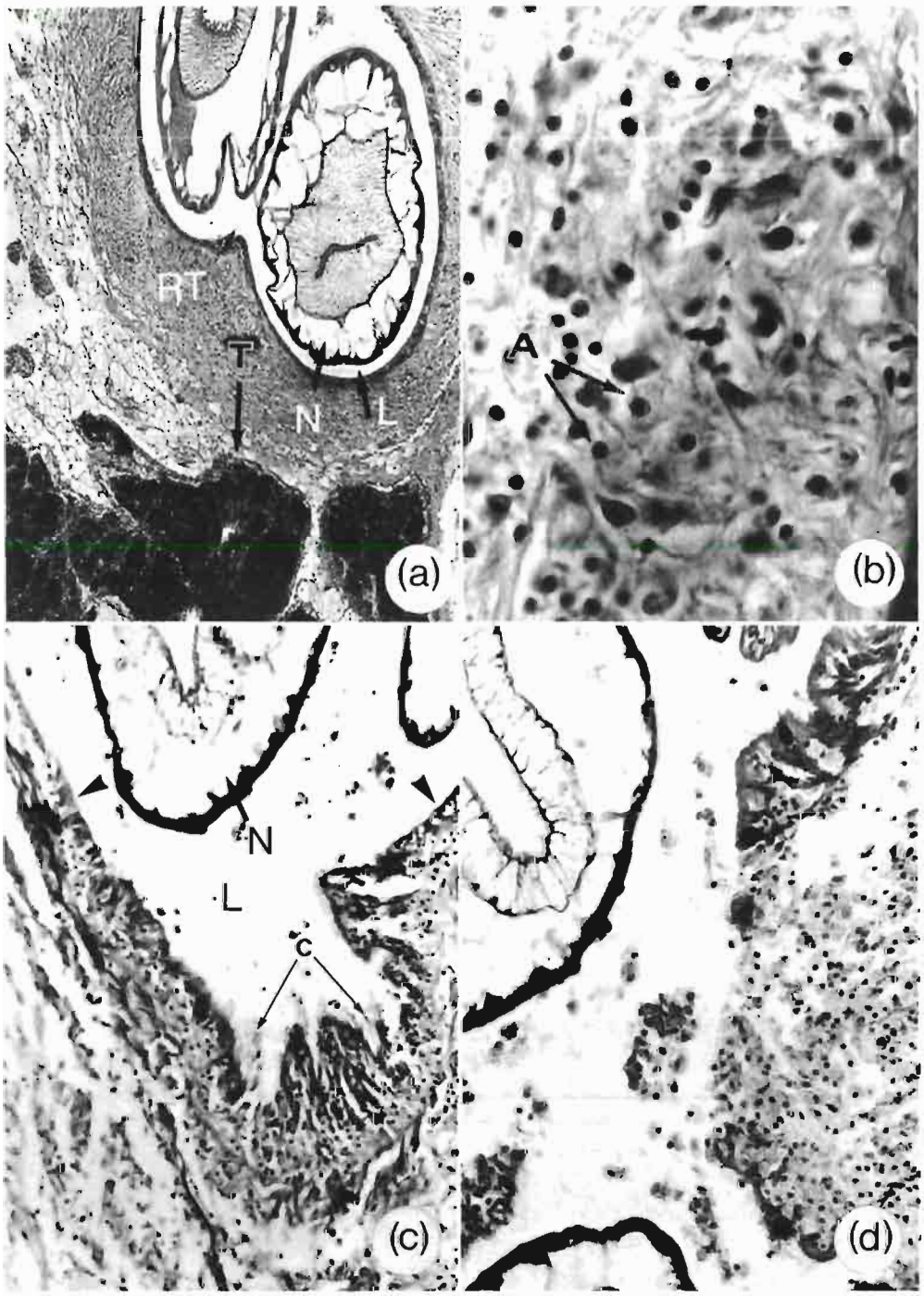


Fig. 13-158: *Crassostrea gigas*. Histopathology associated with *Echinocephalus sinensis* infestation. (a) Formation of reaction tunic (RT) in perigonoductal region, × 113. (b) Heavy infiltration of haemocytes (A) in duct wall, × 1,130. (c) Erosion (arrows) of ciliated duct epithelium, × 235. (d) Disruption of epithelium leading to invasion of duct lumen by hypertrophied haemocytes, × 470. A haemocytes, C cilia, L gonoduct lumen, N nematode, RT reaction tunic, T oyster testis. (After Ko and co-authors, 1975.)

observed *E. sinensis* in 31 % of 240 oysters collected from the same localities during 1973. Infestation intensities increased with host age — from an average of 1.0 per infested 1-year-old oyster to 1.9 worms in 4-year-old individuals. The maximum number of larvae recovered from a single oyster was 7. Incidence and intensity of infestation appear to have increased in 1974 when Ko (1976) recorded a 55 % prevalence of *E. sinensis* in 1,159 oysters collected at monthly intervals (except March). The average number of worms per host was 4.23. There was a marked seasonal variation in both incidence and intensity of infestation, reaching a distinct peak (93 %, 18 worms per host) in August (Fig. 13-159). This peak may be the result of mass infestation due to the increased abundance, in the summer months, of the ray host, *Aetobatus flagellum*, in inshore waters. The subsequent decrease is not fully understood. Perhaps the marked drop in water temperatures late in the year might result in high mortality among heavily parasitized oysters, which might also be more susceptible to predation by the rays.

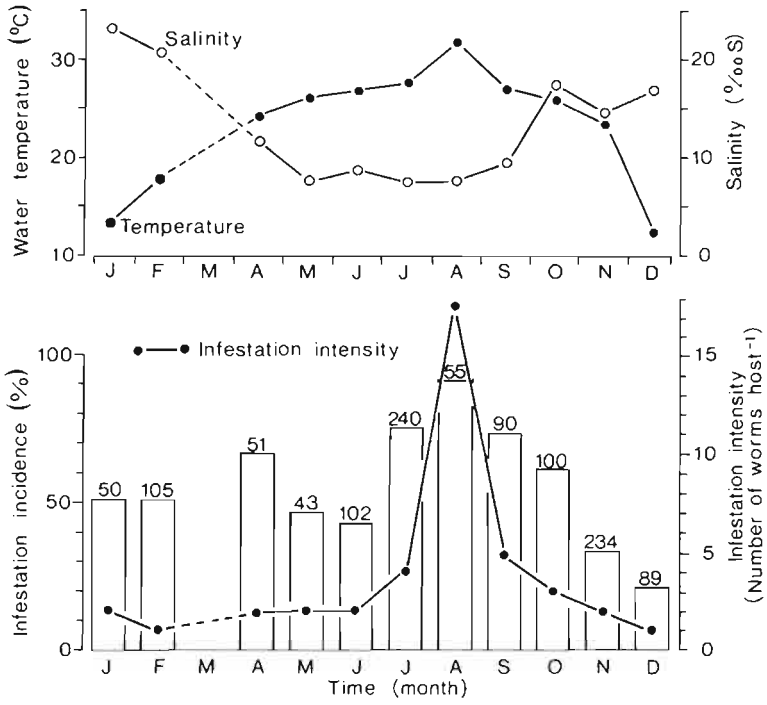


Fig. 13-159: *Echinocephalus sinensis*. Seasonal prevalence in *Crassostrea gigas* in relation to temperature and salinity. Numbers above bars indicate sample size. No sample taken in March. (After Ko, 1976; modified.)

Echinocephalus sinensis infestations in oysters are of possible importance for public health. Experiments, conducted by Ko and co-authors (1975) and Ko (1976, 1977), indicate that third-stage larvae from oysters, administered to kittens and a rhesus monkey, are capable of penetrating the walls of the stomach and the small and large intestine, causing massive cellular infiltrations at the infested sites. The infestivity of the larvae is directly related to the ambient temperature of the molluscan host. Human consumption of infested raw or poorly cooked oysters may, therefore, represent a potential health hazard.

It was speculated that human cases of *E. sinensis* infestation in Hong Kong may have been overlooked or misdiagnosed (Ko, 1976). The potential importance of the occurrence of larval nematodes is stressed by the discovery of Burton (1963), who observed unidentified nematodes, tightly coiled in the region of the digestive gland, in histological sections of *Crassostrea virginica* from Maryland waters. Lester and co-authors (1980) found 2 specifically unidentified larval *Echinocephalus*, 11 and 18 mm long, in a sample of 10 *Amusium balloti* from Shark Bay, Western Australia. *Cheiracanthus* (= *Echinocephalus*) *uncinatus*, a larval nematode of uncertain affinities (see above), has been shown to be involved in pearl formation in Ceylonese *Pinctada margaritifera* (Herdman and Hornell, 1906).

Crassostrea virginica and *Mercenaria mercenaria* have been shown to be capable of acting as aberrant intermediate hosts for the rat lungworm *Angiostrongylus cantonensis*, the causative agent of human eosinophilic meningo-encephalitis in parts of Asia and the Pacific Basin. Normally, the life cycle of this metastrongylid nematode involves terrestrial or amphibious molluscs as intermediate hosts, and non-marine molluscivorous crustaceans can act as paratenic hosts. Humans may become infested by accidentally ingesting unprocessed intermediate or paratenic hosts (Mackerras and Sandars, 1955; Alicata and Brown, 1962; Alicata, 1965). Within the bivalve's body, the larvae can be distributed via blood vessels. During the intravascular phase, a characteristic histopathological syndrome, termed 'perivascular leukocytosis', is evident. The condition is characterized by the aggregation of large numbers of host haemocytes around blood-vessel walls, suggesting the attraction of blood cells to some substance elaborated by the parasite and probably capable of permeating the blood-vessel wall. This unidentified substance may be in the form of the nematode's moulting fluid. Within the oyster's tissues, the *A. cantonensis* larvae are not encapsulated; rather, they are motile and, as a result, cause lesions, particularly in the Leydig tissue. Haemocytic response to motile larvae is apparent although destruction of the intruders does not appear to occur. Experimentally, American oysters and quahaugs were challenged with approximately 3,000 first-stage larvae of *A. cantonensis*. Viable second-stage larvae in the process of moulting, as well as third-stage larvae, were recovered from the bivalves 6 days later. The latter were used successfully to infest young white rats (Cheng and Burton, 1965b; Cheng, 1966b).

These findings could have special importance on some of the Pacific islands where the rat lungworm exists and clams and oysters may be eaten raw or imperfectly cooked. Knapp and Alicata (1967), in an attempt to duplicate Cheng's above-mentioned experiments, failed to achieve *Angiostrongylus cantonensis* infestations in *Venerupis* (= *Tapes*) *philippinarum* and *Crassostrea virginica*. As pointed out by Cheng (1967, 1973b), this failure was clearly due to the totally inadequate experimental procedure employed by these authors.

Second-stage larvae of the 'codworm' *Phocanema decipiens* occur in the intestinal tracts of various invertebrates including *Mytilus edulis* and *Mya arenaria*. No development of these nematodes takes place in the invertebrates which merely act as accidental 'hosts' for the transport of the larvae into the true (teleost) intermediate hosts. Adult *P. decipiens* are parasitic in the intestine of seals (Myers, 1960). Numerous nematodes may be found free in the mantle cavity of oysters. Closer inspection reveals these as individuals of normally free-living species, which have accidentally found their way into the oysters (Schuurmans Stekhoven, 1942).

Agents: Nemertea

Nemerteans of the genus *Malacobdella* occur as inquilines in the mantle cavity of several marine bivalves. Coe (1945) recognized 4 valid species, of which 3 have each been reported from a single host species. *M. grossa*, which has also been described under several other names, utilizes at least 23 different hosts, extending from Europe to North America (Gibson, 1967, 1968; Ropes and Merrill, 1967; Jones and co-authors, 1979). The host spectrum of the worm includes commercially important pelecypods, such as *Crassostrea virginica, Mercenaria mercenaria, M. campechiensis* and *Mya arenaria* (Guberlet, 1925; Coe, 1943; Ropes, 1963; Porter, 1964). On the average, about 60 % of the molluscs screened for *Malacobdella grossa* have been found infested, but figures may run as high as 100 % (Gibson, 1967, 1972; Høpner Petersen, 1978). In more than 90 % of the cases, the worms occur singly, but occasionally double or multiple infestations (with max. 5 individuals per host) have been recorded. In *Cardium edule* from Shetland, double occurrences involved up to 78.6 % of the cases (Jones and co-authors, 1979).

Detailed analyses of the diet and feeding mechanism of *Malacobdella grossa* clearly characterize this nemertean as an unselective microphageous omnivore, not as a parasite (Jennings and Gibson, 1969; Gibson and Jennings, 1969). McMillin (1924; in Gibson, 1968) reported *Malacobdella*-caused tissue damage in razor clams *Siliqua patula*. Guberlet (1925), however, emphasized that tissue destruction sometimes observed in bivalves harbouring *M. grossa* cannot be attributed to the action of these worms. Host-epithelial cells, occasionally observed in the gut of the nemerteans, were believed to be an incidental food component resulting from the normal exfoliation of host tissues. Gibson (1968) was unable to trace any measurable effect or tissue damage caused by *M. grossa* in oval piddocks *Zirfaea crispata* from the Yorkshire, England, coast, but apparently, histological examinations have not been performed during this investigation. At least some degree of local irritation is likely to result from the attachment of the worms by means of their large posterior sucker. More subtle impairment of vital host functions, such as filtration activity, have not been studied. In *Mya truncata* from Disko Bay, West Greenland, the proportion of the wet weight of *M. grossa* to that of the soft parts of the host was found to rise from 1 % for the small to about 12 % for the large individuals (Høpner Petersen, 1978). Impairment of the clams' filtration capacity must be expected to result from the presence of such large 'foreign bodies' in the mantle cavity.

Curiously, Stout (1970) found *Malacobdella grossa* not within the mantle cavity but instead among the sand grains and detrital material accumulating between the lamellae of the siphonal plates of *Tresus nuttalli* from South Humboldt Bay, California.

Malacobdella grossa, in turn, is host for protozoan parasites (Vol. I, Chapter 11). Of these, *Haplosporidium malacobdellae* has also been recorded from free-living, intertidal monostiliferous hoplonemerteans *Amphiporus lactifloreus* (Varndell, 1980), which may act as an infestation source for *M. grossa*.

Gering (1911) reported an average of 57.5 % *Malacobdella grossa* infestation in *Cyprina* (*Arctica*) *islandica* from Kiel Bay, Western Baltic Sea. Incidences were 32.0 % in host-size class 20 to 35 mm, 69.1 % in size class 36 to 55 mm and 71.5 % in clams over 55 mm in shell length. Arntz (1972), who restudied the bivalve-nemertean association in the same area in 1968/69, found only 1.4 %, 29.4 % and 50.4 % of the respective host-size classes to be infested (average: 27.1 %). Whether this drastic decline may be

attributable to hyperinfestation of *M. grossa* by *Haplosporidium malacobdellae,* has not been studied.

Agents: Gastropoda

The gastropod family Pyramidellidae comprises a very large group of small to very small (usually less than 5 mm) opisthobranch snails, which are well known as ectoparasites of invertebrates, mainly of molluscs and polychaetes (Robertson and Orr, 1961). The structure and mode of life of the pyramidellids, particularly the buccal apparatus and its function, have been described in detail by W. E. Ankel (1949a, b), Fretter and Graham (1949) and Maas (1965). All species studied thus far feed on the body fluids of their hosts by means of a long suctorial proboscis. Penetration of the host tegument is facilitated by a protrusible stylet (Vol. I, Chapter 9, Fig. 9-12).

Pyramidellids occur in all seas but are most abundant in warmer waters, with the greatest concentration in the Pacific Ocean (Laseron, 1951, 1959). At present, the systematics in the Pyramidellidae are highly confused. Most descriptions are based on shell morphology only, disregarding structural differences in the soft parts. Species of *Odostomia* have been identified as ectoparasites of oysters, mussels and other commercially important marine bivalves in the North Atlantic Ocean. Winckworth (1932) included 41 pyramidellid species in his list of British marine molluscs, and W. E. Ankel (1936) mentioned 27 in the fauna of the North and Baltic Seas.

Mainly on the basis of the occurrence of spermatophores (which are lacking in the European *Odostomia* spp.), Robertson (1978) separated the eastern North American odostomioid pyramidellids parasitizing marine molluscs from the respective European counterparts and included them in the new genus *Boonea.* Robertson and Mau-Lastovicka (1979) assembled a list of literature records of *Boonea* spp. and their 'hosts'. It must be emphasized that actual feeding of these pyramidellids on their carriers has not been observed in all of these cases. In the laboratory, *B. seminuda* fed on 22 out of the 36 gastropod and bivalve species offered; *B. bisuturalis* fed on 37 out of 45, and *B. impressa* fed on 36 out of 37 'hosts'. Thus, *Boonea* spp. are not host-specific. It should be pointed out, however, that under artificial laboratory conditions pyramidellids will occasionally feed on 'hosts' they would never encounter in the field. In other pyramidellids, feeding is observed with great difficulty, even when the presumed natural host is offered.

The degree to which pyramidellids are host-specific remains, therefore, unknown. Fretter and Graham (1949) emphasized that European odostomes are host-specific. Berry (1955) even went so far as to suggest that host specificity accounts for the large number of pyramidellid species. There is little evidence to support this hypothesis (Allen, 1958; F. Ankel and Møller Christensen, 1963; Bullock and Boss, 1971). Although not being strictly host-specific, odostomioids may display various degrees of host preference. In choice experiments involving various gastropod and bivalve species, *Boonea bisuturalis,* for example, will become attached to all of these molluscs except slipper limpets *Crepidula plana,* but shows a distinct behavioural preference for *Argopecten irradians* and *Crassostrea virginica* (Boss and Merrill, 1965). Although accepting *C. virginica,* as well as various gastropod species as hosts (Allen, 1958; Wells, 1959b), *B. impressa* was not attracted to *Mercenaria mercenaria* and *Modiolus modiolus* (Hopkins, 1956b). *Odostomia rissoides* (= *O. scalaris*) is attracted to *Mytilus edulis* but not to species of *Tapes, Tellina*

or *Mactra* (Pelseneer, 1913, 1914). Host-shell texture may be a factor determining attractiveness (Clark, 1971).

Subtle ecological conditions may influence host choice in odostomioid pyramidellids. *Boonea seminuda*, originally described from *Argopecten irradians*, was very common on the upper valves of this species, as well as on the 'ears' of *A. gibbus* at Beaufort, North Carolina (Hackney, 1944; Wells and Wells, 1961), but did not occur on *A. irradians* from Woods Hole, Massachusetts, although nearly every *Crepidula fornicata* from the same localities was found with attached *B. seminuda* (Robertson, 1957). Preference of *B. seminuda* for *C. fornicata* was also demonstrated in choice experiments although a graded affinity to other bivalve and gastropod species was noted (Boss and Merrill, 1965).

Host age (or size) is another factor apparently determining the attractiveness of bivalves for pyramidellids. Thus, *Boonea bisuturalis* attacks primarily small *Crassostrea virginica* (Loosanoff, 1956), whereas *B. impressa* shows a preference for large oysters. A hundred or more individuals may be found on a single host. Snails placed in an aquarium with oysters of graded sizes, 26 to 76 mm long, assemble in the largest numbers on the largest hosts and in proportionally smaller numbers on the smaller oysters (Hopkins, 1956b). European *Odostomia* (*Brachystomia*) *ambigua* show a similar preference for larger individuals of *Chlamys opercularis* (W. E. Ankel, 1938).

Commercially important bivalves on which true feeding by *Boonea* spp. has actually been observed include *Argopecten gibbus* (by *B. seminuda*: Wells and Wells, 1961; Wells

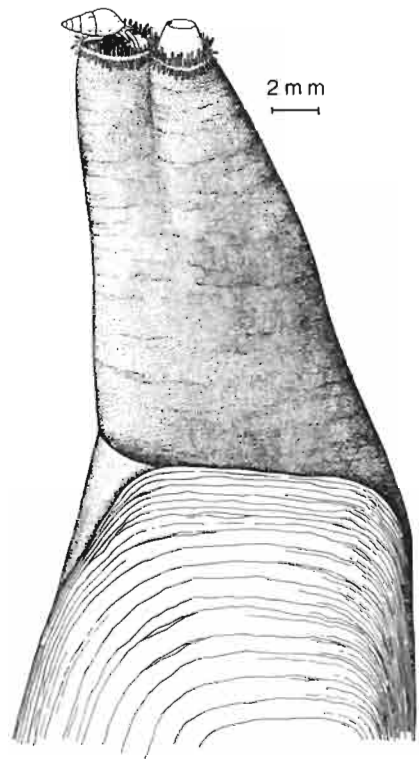


Fig. 13-160: *Odostomia ambigua* sucking on siphon of *Saxicava rugosa*. (After Ankel, 1959.)

and co-authors, 1964), *Crassostrea virginica* and *Mytilus edulis* (by *B. bisuturalis*: Loosanoff, 1956; Merrill and Boss, 1964; Boss and Merrill, 1965; Bullock and Boss, 1971) and *C. virginica* (by *B. impressa*: Allen, 1958; Wells, 1959b). *Odostomia trifida* (= *B. bisuturalis*) was reported from the siphon tips of *Mya arenaria* from the Gulf of St. Lawrence but feeding was not observed (Medcof, 1948). In the laboratory, Robertson and Mau-Lastovicka (1979) observed *B. seminuda* to feed on 7 (44 %) of 16, *B. bisuturalis* on 16 (80 %) of 20, and *B. impressa* on 17 (94 %) of 18 bivalve species offered. It appears therefrom that *B. impressa* is the least specialized of the 3 odostomioids. In European waters, species of *Odostomia* have been seen to feed on *Mytilus edulis* (*O. 'eulimoides'* [= *O. ambigua*] and *O. scalaris*: Pelseneer, 1913, 1914; Fretter and Graham, 1949; Cole and Hancock, 1955; Rasmussen, 1973), on *Cardium edule* and *C. lamarcki* (*O. scalaris*: Rasmussen, 1973) and on *Saxicava rugosa* (*O. ambigua*: W. E. Ankel, 1959; Fig. 13-160).

The pyramidellids normally occur externally on the hosts' shell near the mantle edge. Attachment to the shell surface may be secured by a byssus-like attachment thread (Hoffman, 1979). *Odostomia tellinae* has been observed to live *within* the mantle cavity of *Tellina* sp. from Chinese waters (Pelseneer, 1912). At first view, little host damage might be expected to result from attack by these tiny ectoparasites. Their body weight usually amounts to only 0.03 to 0.17 % of that of their hosts (Robertson and Mau-Lastovicka, 1979). However, constant irritation of the bivalve's mantle margin by the penetration of the pyramidellid's proboscis during feeding may result in withdrawal of the affected portions of the mantle.

A sample of 100 2-year-old *Ostrea edulis* from the River Roach, Essex (England) was found to contain no less than 46 individuals, which showed evidence of attack by *Odostomia eulimoides* (= *O. ambigua*). The snails were lodged in characteristic small pockets just inside the ventral shell margin, and obviously formed in response to the parasites. The odostomes, which appeared to be fairly sedentary within their pockets, usually occurred in pairs. In many pockets, spawn had been deposited (Cole, 1951c). Similar pockets are produced by *Boonea bisuturalis* and *B. impressa* in *Crassostrea virginica*. Up to 17 snails have been recovered from a single oyster (Allen, 1958).

The cumulative effect of parasitization by odostomioids may be most pronounced in older oysters. Individuals of *Ostrea edulis* from Essex, England, waters, 5 to 6 years of age and about 76 mm in average diameter, showed serious shell deformities due to *Odostomia ambigua* attack. Most of the 40 oysters studied had confluent pockets covering almost the entire shell margin, which had become thickened, in the worst cases being about 1 cm wide with double or triple lips. Great ridges of shell substance had been laid down following the path of the withdrawn mantle margin. Some of the pockets had penetrated farther down toward the adductor muscle. When the process continued, the muscle became almost completely covered by a thorn-like ridge of brownish shell material. In one severe instance, only a narrow band of 1/10 of the adductor muscle remained fully functional. Such affection causes the oyster to gape, with the consequence that sand and silt will be driven into the mantle cavity and, eventually, suffocation and death ensue. Of the 40 oysters examined, 20 were so severely affected that the adductor muscle was damaged, and of these, 11 were dying or already dead from the effects of *O. ambigua*. Examination of 3-year-old brood oysters from Essex waters revealed varying incidences of odostome infestation, with a maximum of about 30 % (Cole and Hancock, 1955; Hancock, 1969).

In North American waters, from the Gulf of St. Lawrence to Florida, *Boonea*

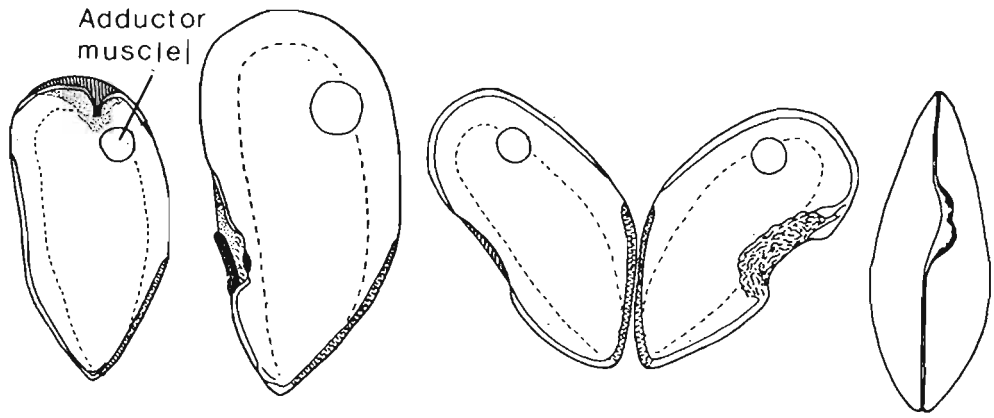


Fig. 13-161: *Mytilus edulis*. Shell lesions produced by *Odostomia scalaris*. (After Cole and Hancock, 1955.)

bisuturalis affects mainly young individuals of *Crassostrea virginica*. Although it may not actually kill oysters more than 10 mm in length, it interferes with their normal development and growth. Heavily invaded oysters have characteristically deformed shells with deeply cupped valves and thickened edges (Loosanoff, 1956).

Odostomia scalaris has been found in great numbers on *Mytilus edulis* from Menai Bridge, Wales. Pockets similar to those in oysters were seen in several mussels. They usually occurred on one valve only, while the opposite valve had overgrown slightly in an effort to close the gap (Fig. 13-161). Snails attached to the outer surface of the valves were seen to protrude their long proboscis into the siphonal aperture of the mussels. They remained in this feeding position for several days (Cole and Hancock, 1955).

Two specifically unidentified pyramidellids of the genus *Turbonilla* from Buzzards Bay (Massachusetts) were considered an enigma. One of these was the eighth most common animal species in that area, but suitable hosts appeared to be lacking. It was tentatively suggested that, in this environment, *Turbonilla* is a deposit-feeder, using its buccal pump to draw in the flocculent superficial sediment (Sanders, 1958, 1960). This conclusion is somewhat surprising in view of the fact that all known pyramidellids are ectoparasites. *T. interrupta*, for instance, which is abundantly distributed along the North American Atlantic coast, is known to attack oysters, scallops and venus clams (Morton, 1967).

There is a single report on hyperparasitism in a pyramidellid. Pelseneer (1913, 1914) identified *Odostomia rissoides* (= *O. scalaris*), attacking *Mytilus edulis* at Wimereux (France), as host for larval monstrillid copepods. The crustaceans, which did not cause parasitic castration in the odostomes, were identified as *Monstrilla helgolandica*.

Muricid and thaidid gastropods of the genera *Urosalpinx, Eupleura, Ocenebra* and *Thais*, commonly known as oyster drills, attack a wide variety of gastropod and bivalve molluscs. Although the large drills are predators rather than parasites, they will be mentioned here in passing because they constitute an important threat to the oyster industry, particularly on the North American Atlantic coast.

General accounts of the role of muricid and thaidid snails as oyster pests have been given in almost all pertinent textbooks and reviews on oyster biology (e.g. Needler, 1941;

Korringa, 1952a; Cole 1956a; Cole and Hancock, 1956; Galtsoff, 1964). The most widely distributed of these predaceous gastropods is the common oyster drill, *Urosalpinx cinerea*. The voluminous literature on its predatory behaviour has been reviewed by Carriker and van Zandt (1972), and its mechanism and physiology of boring have been studied mainly by Carriker and his associates (Carriker, 1969, 1972, 1978; Carriker and co-authors, 1963, 1967, 1978; Person and co-authors, 1967; Nylen and co-authors, 1969; Smarsh and co-authors, 1969; Carriker and Chauncey, 1974).

Economic losses on oyster and mussel beds caused by *Urosalpinx cinerea* undoubtedly amount to thousands of dollars yearly (Field, 1923). The species may even attack highly motile bivalves, such as scallops. In a laboratory study of attack success, conducted by Ordzie and Garofalo (1980), an average of 72.3 % of *U. cinerea* attacks on *Argopecten irradians* led to death of the scallops. Being a temperate-water species, *U. cinerea* seems to be bordering on extinction in Canadian waters where it, therefore, does not constitute a threat to the east-coast oyster industry (Medcof and Thomas, 1969). The lower temperature limit for feeding by *U. cinerea* is about 7.5 °C, the optimum about 25.0 °C (Hanks, 1957). Stauber (1950) discussed the question of the existence of physiological races of *U. cinerea* adapted to different environmental temperature ranges.

Native to the east coast of North America, *Urosalpinx cinerea* has accidentally been introduced into Europe during the 1920's where it now has become an important pest (Korringa, 1952a; Hancock, 1969). Cole (1951b) estimated that about 75 % of the *Ostrea edulis* spat may be killed by drills on Essex, England, oyster beds during their first year of life. A second muricid drill, *Eupleura caudata*, has about the same distribution as *U. cinerea* but is usually less abundant.

In laboratory trials, adult *U. cinerea* and *E. caudata* were not affected by hyperosmotic salt solutions, but their embryos were readily eradicated by a 5-min exposure to saturated 'rock salt' solutions. Caution should be exercised, however, in exposing seed oysters with damaged bills to such procedures, since they are likewise killed by high salt concentrations (Shearer and MacKenzie, 1961). The literature on control of *U. cinerea* has been reviewed by Carriker (1955).

Dog-whelks of the genus *Thais* (syn. *Purpura, Nucella*) represent another genus of oyster drills no less destructive than *Urosalpinx*. In the spring and summer of 1967, for instance, 80 to 95 % of *Crassostrea virginica* spat, as well as oysters less than 5 cm long, were killed by *Thais haemastoma* in Alabama waters (May, 1968). High losses of spat and larger oysters, due to predation by southern oyster drills, have also been recorded in Mississippi Sound (Chapman, 1959) and at Port Aransas, Texas (Menzel, 1955). Being an estuarine species of southerly distribution, *T. haemastoma* feeds most voraciously at optimum salinities of about 20 ‰ S. Feeding ceases at salinities below 7.5 ‰ S and temperatures below 10 to 12.5 °C (Garton and Stickle, 1980).

Japanese drills *Ocenebra japonica* are of considerable concern in oyster cultures on the North American Pacific coast (Galtsoff, 1964; Quayle, 1969). Among the 5 species of oyster drills common to Japanese waters, *T. tumulosa clavigera* is the most serious pest (Fujiya, 1970). *T. carinifera* was observed to devour spat and to bore holes into the valves of adult *C. gryphoides* from Bombay, India (Durve, 1964). For a review of the Thaididae from the western Atlantic consult Clench (1947).

The mechanism of shell boring by thaidids has been studied in *T. lapillus*, a drill from more northerly American and European Atlantic waters, by Chétail and Binot (1967),

Chétail and co-authors (1968), Rosenberg and co-authors (1968), Chétail and Fournié (1969, 1980) and Webb and Saleuddin (1977). Experiments conducted by McGraw and Gunter (1972) indicate that *T. haemastoma* utilizes a paralytic mucous secretion, produced by the hypobranchial gland, in attacking oysters. Thirty-four percent of the oysters eaten by the snails showed no evidence of drilling on the shell. The authors concluded that drilling appears to be a secondary process in many cases.

An interesting study by Cooley (1958, 1962) considered the possible use of *Parorchis acanthus* in the biological control of oyster drills. This trematode utilizes *Thais lapillus*, *T. haemastoma* (*floridana*) and *Urosalpinx cinerea* as first intermediate hosts. Infested drills undergo parasitic castration (Vol. I, Chapter 12) and are, therefore, lost for the breeding population. Lebour and Elmhirst (1922) inferred a life cycle involving bivalves as second intermediate hosts, but Stunkard and Cable (1932) showed that the cercariae of *P. acanthus* (misnamed *P. avitus* by these authors) encyst free on solid surfaces (Vol. I, Fig. 12-28). Natural infestations of *P. acanthus* in *T. haemastoma* from Gulf of Mexico waters were low, 2.5 % in 7,604 drills, and, although exposure to miracidia resulted in a nearly 60 % infestation in experimental hosts, Cooley (1962) was too pessimistic as to the utilizability of this trematode in drill control under field conditions. It should be noted that Feare (1970) has found up to 69 % of *T. lapillus* from Robin Hood's Bay, Yorkshire (England), to be naturally infested with *P. acanthus*. It seems, therefore, that ecological conditions favouring the dissemination of this trematode in drill populations merit further study.

Another, potentially even more effective biological drill control could be the phycomycete fungus *Haliphthoros milfordensis*. It infects selectively the ova and early developmental stages of *Urosalpinx cinerea*. Veligers and protoconchs are not affected. Ganaros (1957) discussed the utilizability of this fungus in the biological control of oyster drills. How extensive damage done by similar fungi can be, has been shown by Johnson (1958). *Lagenidium chthamalophilum*, attacking ova of *Chthamalus fragilis*, was held responsible for the obvious reduction in population density of this barnacle in the Beaufort Inlet region, North Carolina. Under natural conditions, *L. chthamalophilum* appears to be host-specific to *C. fragilis* (Johnson, 1960; Johnson and Sparrow, 1961). On the other hand, the apparent wide host spectrum of *H. milfordensis* (Vol. I, Chapter 12) might preclude its use in oyster-drill control.

Agents: Bivalvia

Burrowing clams are known to excavate a wide variety of substrates including corals (Vol. I, Chapter 6) and molluscan shells. The basic structure of the Lamellibranchia ideally fits them for burrowing (Yonge, 1963). On the other hand, the normally thin shells of bivalves, as well as their burrowed mode of life, makes them a rather unsuitable substrate for large burrowing bivalves. Oysters, with their thick-walled valves, make an exception.

Crassostrea virginica may at times be attacked by the pholadid burrowing clam *Diplothyra smithi*. This species is frequently referred to as *Martesia* sp., a genus containing only wood-boring clams (Turner, 1955). *D. smithi*, which may reach about 1.25 cm in length, is usually found inside the oyster's shell material in a cavity which increases in size as the clam grows. In southern waters of North America, the burrowing clam is very

common, particularly on some reefs on the Texas coast. On one occasion, over 200 *D. smithi* of various sizes were found in a single oyster. As the cavity bored by the clam increases and approaches the inner valve surface, the oyster protects itself by depositing layers of conchiolin over the nearly perforated areas. On the outer surface of the oyster valve, the presence of clams is indicated by small holes only. Weakening of the shell structure is the main effect of *D. smithi* on *C. virginica* (Galtsoff, 1964). Perforation of the shell in the region of the adductor muscle may lead to the formation of yellow pustules or abscesses in the muscle tissue. Bacteria (as secondary invaders) may or may not be found in smears. Some abscesses appear to be sterile. A small percentage of such affected oysters die. Most abscesses are, however, isolated by fibrous capsules and masses of haemocytes and sloughed off subsequently. Freed pustules may occasionally be found in the shell cavity and may be covered with thin layers of shell material (Mackin, 1962).

Pholadids *Penitella conradi* have been found to burrow into the valves of *Mytilus californianus* and *Pododesmus* sp., as well as into the shells of gastropods *Haliotis* spp. and *Astraea* sp. from the North American Pacific coast (Turner, 1955). Mytilids *Lithophaga* spp. and *Botulina coralliophaga*, as well as gastrochaenids *Rocellaria* spp., attack the valves of silver-lip pearl oysters *Pinctada maxima* from the Arafura Sea, Japan, and may, hence, detract considerably from the commercial value of the shells (Takemura and Okutani, 1956). In addition to *'Martesia'* (= *Diplothyra*) *smithi* (see above), *Lithophaga bisulcata* and *Rocellaria hians* have been reported from burrows within the shell of *Crassostrea virginica* from Beaufort, North Carolina (Wells, 1961).

The clams appear to excavate their burrows by purely mechanical means, using the external surfaces of their strongly modified valves as an abrasive tool. No chemicals, such as acids, appear to be involved in the process (Elliott and Lindsay, 1912; Turner, 1954; however, see Vol. I, pp. 209/210).

Wiborg (1946) reported on the unusual 'parasitic' occurrence of a blue mussel *Mytilus edulis* within the mantle cavity of a horse mussel *Modiolus modiolus*. Bivalves may settle as larvae on the left (upper) valve of *Placopecten magellanicus*. When the growing organisms become lodged between the mantle and shell of the sea scallop, it attempts to wall off the intruders by secreting a thick layer of conchiolin and depositing additional calcium carbonate. Curious deformities of the shell margin may result from this process. A total of 30 *Mytilus edulis*, 28 *Saxicava* (*Hiatella*) *arctica* and 23 *Anomia aculeata* were recovered from a single large scallop from Cape Cod Bay, Massachusetts (Merrill, 1960). *S. arctica* may also nestle in empty holes previoulsy excavated by burrowing sponges *Cliona vastifica* into the shells of *Placopecten magellanicus* (Evans, 1969). Erycinacean bivalves associate with members of numerous invertebrate phyla. Some of these associations have been labelled parasitic. Erycinaceans found with other bivalves are nestlers. There is no indication of parasitism (Boss, 1965).

Agents: Bryozoa and Phoronidea

While a considerable number of encrusting bryozoans are members of the fouling community living epizoically on molluscan shells (Osburn and Soule, 1953; Wells and co-authors, 1964), ectoprocts of the orders Cheilostomata and Ctenostomata burrow into calcareous and non-calcareous substrata including molluscan shells. As they penetrate the

valves only superficially, these organisms have little negative effect on their bivalve 'hosts'. Consequently, they will here be considered only in brief. For exhaustive information, the reader is referred to the publications cited below, which contain numerous references.

The ectoproct bryozoans as burrowing organisms have not received the recognition in study that some of the larger, more readily recognized groups have enjoyed. Initially known by their bore holes only, the burrowing Penetrantiidae, Immergentiidae and Terebriporidae have received systematic treatment from Fischer (1866), Marcus (1938), Silén (1946, 1956), Osburn and Soule (1953), Bobin and Prenant (1954), Soule and Soule (1969b), and others. Soule and Soule (1969a) summarized the information pertaining to the systematics and biogeography of the burrowing bryozoans, and Silén (1947) gave a detailed account of the anatomy and biology of the Penetrantiidae and Immergentiidae.

From the available information it becomes apparent that many of the approximately 4,000 living species of ectoprocts are circumboreal, circumtropical or even cosmopolitan in distribution. Numerous species have been reported from bivalves, including members of the Ostreidae, Mytilidae, Cardiidae, Pectinidae and Limidae (Soule and Soule, 1969a). While most of these occur in both dead and living gastropod and bivalve shells, *Penetrantia concharum* has hitherto only been reported from dead shells (Silén, 1946, 1947; Soule, 1950).

The mechanism by which the delicate ectoprocts penetrate into hard substrata, is not readily understood. Apparently, it has only been studied in any detail by Silén (1947). The author assumed that the process is chemical, involving phosphoric acid (or a secretion from which phosphate ions separate), because relatively high concentrations of phosphate ions were found to be associated with the growing tips of the stolons. However, efforts to identify specific secretory cells in the stolonal tips were not successful. Although the ectoprocts apparently prefer shell areas for initial attachment where the periostracum has worn away, the zoids are nevertheless capable of penetrating shells with intact periostracum. Silén concluded that another chemical agent, possibly an enzyme, may be responsible for the dissolution of the conchiolinous periostracum. As pointed out by Soule and Soule (1969a), the small size of the zoids (less than 1 mm) and of the stolonal tips (which are only 10 to 15 μm in diameter), plus their complete immersion in the molluscan shell, tends to discourage histochemical studies of enzymatic activity.

Of the class Phoronidea, which contains only some 20 known purely marine species, a few display a mode of life similar to that of the burrowing ectoprocts. Marcus (1949) and Silén (1952, 1954) have given descriptions of the anatomy, systematics and behaviour of the shell-burrowing phoronids. *Phoronis ovalis,* a species of cosmopolitan distribution, occurs almost exclusively in dead shells. Marcus (1949) found it in densities of up to 150 burrows per cm^2 in shell fragments of the oyster drill *Thais floridana*. The circular tube openings are 0.2 to 0.3 mm in diameter. Shells of *Mytilus* and balanids were also found to be infested. Silén (1956) reported a well developed pseudocolony of *P. ovalis* from the shell of a living individual of the green-lipped mussel *Mytilus* (= *Perna*) *canaliculus* from Little Papanui, Otago Peninsula (New Zealand).

Although burrowing bryozoans and phoronids inflict little direct injury upon bivalve shells, the tiny burrows and etchings produced by them may well provide suitable entrance ports for more powerful and destructive burrowing or fouling organisms (Fischer, 1866).

Agents: Annelida

The cosmopolitan spionid polychaete genera *Polydora* and *Boccardia* contain a large number of free-living (tube-building) and burrowing (boring) species of 'mudworms'. Several of these burrow into dead and living molluscan shells. The taxonomic status of both genera is in considerable disorder and misidentifications are the rule rather than exception. In particular, burrowing forms have frequently been confounded with actually non-burrowing species. In their informative review of *Polydora* and related genera, J. A. Blake and Evans (1973, p. 247) conclude that "considerable work remains to straighten out the taxonomic status of several of the 'better known' species of *Polydora*".

The 'mud-' or 'blisterworms', as they are called, are of considerable economic concern as they have been accused of causing substantial mortalities, particularly among oysters and mussels. J. A. Blake (1971) has revised the genus *Polydora* occurring on the Atlantic coast of North America. Of the several species of mudworms found in the intertidal zone of the North American Atlantic and Pacific coasts, only *P. websteri* and *P. ligni* (and possibly *Boccardia hamata*), are important to oyster ecology. *P. websteri* damages the oyster directly by burrowing into the shell, while the non-burrowing *P. ligni* indirectly affects the oyster, in that excessive numbers of worms occurring epizoically on the shell surface cause smothering of their living substrate. *P. websteri* also invades scallops. In European waters, *P. ciliata* and *P. hoplura* are responsible for mortalities among oysters and mussels. *P. ciliata*, a form of dubious cosmopolitan occurrence, has been reported from various parts of the world ocean, particularly from Australian waters (Haswell, 1886; Whitelegge, 1890; Roughley, 1925). Whether the specific identifications, in these records, are correct remains to be established. They very probably include other, yet undescribed species. Not even the taxonomic status of one of the most common representatives, *P. ciliata*, is quite clear. Two well-marked ecotypes of '*P. ciliata*' exist, which may prove to be separate species (Korringa, 1951a; Cole and Hancock, 1956). Rasmussen (1973) found transitional stages bordering on 'typical' *P. ciliata* and *P. ligni* among samples from Isefjord, Denmark, and included the latter in *P. ciliata*. Michaelis (1978b) found no such overlap in individuals of both species from the German North Sea coast.

Species identification in the genus *Polydora* is based, to a large extent, on the morphology of the setal armature of the 5th setiger. In addition to true interspecific

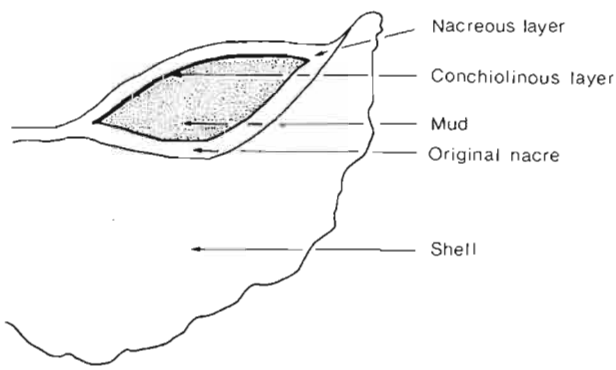


Fig. 13-162: *Crassostrea virginica*. Diagrammatic section of shell and mud blister produced by *Polydora websteri*. (After Lunz, 1941.)

differences, individual spines may exhibit varying degrees of wear, which, as has already been pointed out by Mesnil (1896), may lead (and has, in fact, led) to misidentifications, also between *P. ciliata* and *P. ligni* (Michaelis, 1978b). Rearing of mudworms from egg to adult under controlled laboratory conditions, as partially achieved by D. P. Wilson (1928) and J. A. Blake (1969b), may help to clarify taxonomic problems in the genus *Polydora*.

In molluscan shells, *Polydora* produces 2 types of unsightly 'mud blisters' (Figs 13-162, 13-164 and 13-165). Larvae settling on the outer surface of the valves excavate U-shaped tunnels, which are subsequently 'paved' with compacted mud. Blisters of another type, common in heavily infested oysters in late summer, result when juvenile or adult worms crawl between the mantle and inner shell surface. Attempting to wall off the intruders, the oyster secretes a thin, transparent sheet composed of conchiolin followed by calcite layers. Concurrently, the worms fill the newly formed space between blister wall and shell with loose mud, and then compact it, leaving U-shaped channels equipped with two 'chimneys' at the ends, which protrude from the substrate and communicate with the exterior. Burrowing continues after the blisters have formed (Hempel, 1957a; Haigler, 1969; J. A. Blake and Evans, 1973).

Very little structural adaptation distinguishes the mollusc-associating polychaetes from free-living forms, and the question whether or not physiological adaptations have evolved remains unknown for the most part (Clark, 1956). Several hypotheses have been put forward as to the exact mechanism by which *Polydora* spp. penetrate shell substrate. Lankester (1868a, b), who was the first to describe the burrows and burrowing of *Leucodore* (= *Polydora*) *ciliata*, concluded that penetration of calcareous substrata is accomplished by worm-produced respiratory carbonic acid and/or some other acid secretion, aided by mechanical action of the setae. McIntosh (1868), however, strongly disagreed with the 'chemical (or acid) theory' and favoured the 'purely mechanical penetration hypothesis'. Söderström (1923) maintained that burrowing in *P. ciliata* is chemical, while Hannerz (1956) hypothesized that burrowing involves both a chemical and a mechanical component in larval worms, but that it is purely mechanical in the adult.

A historical account of the various opinions, most of which are based on circumstantial observations, has been presented by Hempel (1957a, 1960), who concluded that *P. ciliata* bores by purely mechanical means, using the heavily developed, modified spines of its 5th setiger in the excavation of the shell substance. The proper process of burrowing has not been followed directly, but mechanical action has been inferred from the detection of scars, presumably produced by the worm's spines (giant setae), on the walls of incomplete channels, as well as from traces of wear on the spines of the 5th setiger, which were absent from the spines of presumably non-burrowing individuals. Similar wear may be seen on the spines of shell-boring *P. websteri* but seems to be less pronounced in sediment-dwelling, tube-building *P. ligni*.

Contrary to the opinion of various authors, it is now generally accepted that the spines are not used to excavate the substrate. Using natural shell, as well as 'artificial blisters' composed of transparent plastic films sealed to Iceland spar substrates, Haigler (1969) studied the burrowing mechanism in metamorphosing larvae and adults of *Polydora websteri*. Worms penetrated all layers of oyster shell, including prismatic, calcite-ostracum, hypostracum, periostracum and internal conchiolin layers. Individuals induced to settle directly on test substrates at room temperature excavated chalky deposits within 24 h, calcite-ostracum and hypostracum within 1 week and conchiolin layers within 1

month. As worms from which the giant setae of the 5th setiger had been amputated, produced burrows identical to those made by intact individuals, it was concluded that the setae are not essential for burrowing. No particulate abrasion products were found; apparently, all the calcite was removed by dissolution, which also was the case when the giant setae were retained. Observation of adult *P. websteri* in artificial blisters indicated that the setae seldom contacted the substrate. For extended periods of time, however, the worms lay motionless with their venters appressed to the substrate, and sometimes moved slowly forward and backward, still maintaining close contact with the substrate. Individuals kept in sea-water agar produced acid while displaying such behaviour, which suggests that *P. websteri* excavates shell by localized chemical (acid) dissolution of calcium carbonate. Hempel (1957a), however, drew attention to the fact that acid is also produced by non-burrowing polychaete species. Dorsett (1961b) hypothesized that a chelating agent linked to the biochemistry of mucus produced by the worms' subtegumental glands is the responsible chemical agent.

Haigler's (1969) observations and experiments on the mechanism of shell penetration by *P. websteri* have been confirmed and extended by Zottoli and Carriker (1974), particularly on the basis of ultrastructural studies. Individuals of *P. websteri,* when placed on preparations of polished *Mytilus edulis* shell under transparent plastic film, secrete a viscous fluid, which dissolves the interprismatic and interlamellar organic matrices, and then dissolves the exposed calcite crystals. Etched prisms and lamellae revealed a complex pattern of internal dissolution. The authors concluded that enlargement of the burrow by the worm is accomplished by chemical dissolution of shell and probably also by flushing of loosened, partially dissolved prisms and lamellae. Concomitantly, a detrital tube is formed within the burrow from material collected and pressed in place by the worm. It is during this process that the modified setae of the 5th setiger are used to consolidate the burrow walls and to keep the diameter of the inner tube constant throughout its length. However, the setae are not used in the excavation process proper.

The chemical nature of the 'viscous fluid' observed by Zottoli and Carriker (1974) in *Polydora websteri* penetrating *Mytilus edulis* shell material has not been determined and, in essence, no definite explanation regarding the true nature of the burrowing process has been obtained. The possible participation of enzymes, such as acid phosphatase, carbonic anhydrase or proteases (see section 'Agents: Porifera') — particularly in the process of penetration of periostracum and internal conchiolin layers — has not been investigated.

Mud worms in various parts of the world have been identified as *Polydora ciliata.* At least the North American east-coast records from *Crassostrea virginica* (Lunz, 1940, 1941, 1943; Needler, 1941; Medcof, 1946), *Argopecten irradians* (Turner and Hanks, 1959) and *Mercenaria mercenaria* (Landers, 1967), are misidentifications which, in fact, represent *P. websteri* (O. Hartman, in Loosanoff and Engle, 1943; Hopkins, 1958b; Galtsoff, 1964; J. A. Blake, 1969a, b, 1971; Blake and Evans, 1973). The same may hold true for '*P. ciliata*' in *C. gigas* from the Pacific coast (Quayle, 1969). Further North American Pacific (California) coast polydorid records include *P. elegantissima* (in *Tivela stultorum*) and *P. convexa* and *Boccardia berkeleyorum* (in *Pododesmus macroschisma*) (J. A. Blake and Woodwick, 1971a, b).

Hopkins (1947) has summarized the literature on *Polydora* infestations. He points out that a number of reports blame *Polydora* as a dangerous oyster pest being definitely incriminated in outbreaks of oyster mortalities in Australia, Belgium and the United

States. Equivocal opinions as to the destructiveness of these annelids may well reflect species differences among the spionids under consideration (Cheng, 1967). Thus, Mortensen and Galtsoff (1944) considered the infestation of *Crassostrea virginica* by *'P. ligni'* to be a purely accidental phenomenon. But their material, in all probability, contained individuals of both *P. websteri* and *P. ligni*. As has been emphasized by Rasmussen (1973), considerable morphological variation may occur among individuals of *Polydora* presumed to be representatives of one and the same species. For rapid sorting, *P. websteri* may be dinstinguished from the ever-present *P. ligni* by the fine longitudinal lines of black pigment on the palps, as opposed to the diffuse, brown lines on the palps of *P. ligni* (Haigler, 1969).

The magnitude of the damage done to molluscan shells may also reflect host-species differences. Thus, imported individuals of *Crassostrea gigas*, planted in Louisiana waters, became much more heavily invaded by *Polydora websteri* than native oysters *C. virginica*. Since *P. websteri* always settles along the shell margin, the differences were tentatively explained in terms of variations in shell closure in the two species (Kavanagh, 1941).

There are reports on *Polydora websteri* infestation throughout the entire distributional range of *Crassostrea virginica* in North America. Incidence and intensity of infestation may vary considerably with local ecological conditions. Salinity, water temperature and composition of the substrate (mud or firm bottom) appear to be main factors determining worm abundance. Prevalences of up to 100 % are not uncommon, and large numbers of mud blisters may be observed in individual oysters, particularly on the south and southeast coasts (Loosanoff and Engle, 1943; Owen, 1957) but diminish in northerly direction. In Canadian Atlantic waters, prevalences of *P. websteri* in *C. virginica* varied greatly between localities, ranging from 0 to 75 % (Medcof, 1946).

Aside from the unsightly appearence of the mud blisters on the shell, worm penetration in the region of the adductor muscle may result in the formation of yellowish pustules or abscesses, probably due to the introduction of mud into the muscle tissues. About 56 % of the abscesses in *Crassostrea virginica* were found to be associated with penetration by either *Polydora* (*websteri*), burrowing bivalves '*Martesia*' (probably *Diplothyra*; see section 'Agents: Bivalvia') or burrowing sponges of the genus *Cliona*. Of these, *Polydora* was the most destructive. Smears from worm-produced pustules revealed enormous numbers of moribund haemocytes, as well as basophilic granules, which stain purplish-blue with Giemsa and appear to be degeneration products. Bacteria may or may not be found in such abscesses; some seem to be sterile. The pustules were most common during the high-temperature period of late summer and gradually disappeared in late autumn. Isolation by fibrous capsules and masses of haemocytes occurred, followed by sloughing of the entire pustule. Such cast-off pustules may occasionally be found in the mantle cavity, and may be covered with thin layers of shell substance. 'Muscle pearls', nacreous excrescences in the muscle scar, represent healed abscesses in the muscle tissue. 'Yellow pustule disease' was held responsible for the death of a small percentage of affected oysters (Mackin, 1962).

From careful evaluation of the published information it appears that *Polydora websteri* is not as destructive as the other bivalve-invading representatives of the genus. Although, in certain areas, a positive correlation may occasionally be found between oyster mortalities and the degree of *P. websteri* infestation, such a correlation was not substantiated by results obtained from controlled laboratory experiments and might, therefore, be caused by other factors (Loosanoff and Engle, 1943; Owen, 1957).

On the other hand, *Polydora websteri* infestation was believed to cause general

weakening and poor condition of affected oysters and to retard growth (Lunz, 1940, 1941; Owen, 1957). Loosanoff and Engle (1943) and Medcof (1946) found no reduction in meat yield in *Polydora*-infested *Crassostrea virginica*. At any rate, although infested oysters are not generally unfit as sea food, mud blisters may detract substantially from the commercial value of oysters since higher-grade, select specimens for the half-shell trade are more difficult to obtain from beds with high *P. websteri* prevalence (Lunz, 1940, 1941; Needler, 1941; Medcof, 1946). Also, *Polydora* blisters may interfere with shucking (Haigler, 1969).

Some North American oyster mortalities attributed to *Polydora websteri* ('*P. ciliata*') may, in fact, be due to *P. ligni*. Worms of this species do not burrow into molluscan shells (J. A. Blake and Evans, 1973), but frequently build their tubes on the surface of oyster shells and secrete strands of thick mucus to form a network which retains sediment, oyster faeces and rejected material. Decomposition of this mass covering the valves produces much hydrogen sulphide. As a consequence of this poisoning, the oysters die, the mortality sweeping over acres of beds and reaching a peak in late February to March. During 1940, *P. ligni* was believed to have destroyed several hundred thousand dollars worth of oysters in Delaware Bay (Nelson and Stauber, 1940).

Similar effects of oyster smothering and attack may be expected to result from invasion, of inshore waters and oyster beds, by *Boccardia hamata*, a hitherto largely neglected faunal element of the North American coasts (Blake, 1966, 1969a; Dean and Blake, 1966). This spionid had previously been included in the genus *Polydora*. Larsen (1978) reported on the discovery of a large population of *B. hamata* on James River (Virginia) oyster beds. Its abundance was greater than that of *P. websteri*. Worms were recovered from internal galleries in *Crassostrea virginica* valves, as well as from tubes on oyster shells.

In addition to oysters, *Polydora websteri* is known to affect scallops, which appear to suffer more severely from attack by this spionid. While, in the majority of cases, oysters are able to cope with the worms by secreting sufficient shell substance to wall off the burrows, scallops may be challenged by these annelids. *P. websteri* infestation was considered a factor contributing to unusually high mortalities of bay scallops *Argopecten irradians* in Fairhaven, Massachusetts (USA). In contrast to oysters, scallops have a delicate shell with little variation in thickness from the hinge to the ventral margin. Only the mantle margins seem to be capable of elaborating nacre in quantity, with the remaining area secreting mainly conchiolin when irritated. Thus, the scallop may protect itself from *Polydora* attacks near the periphery by walling off the worms with calcifications but appears to be unable to cope with burrows made closer to the hinge where they may interfere with the attachment of the adductor muscle, causing it to pull loose under violent contraction. Also, affected adductor muscles (the only portion marketed) appear to be of poor quality (Turner and Hanks, 1959).

By means of radiography, Evans (1969) studied the burrows produced by *Polydora websteri* and *P. concharum* in the shells of sea scallops *Placopecten magellanicus* from Newfoundland (Canada) waters. In contrast to burrowing sponges *Cliona vastifica*, which settle almost exclusively on the lower valve, the polychaetes occupy mainly the upper valve.

In the St. Lawrence estuary, Canada, *Polydora websteri* was found to display a pattern of selective attack. While Arctic wedge clams *Mesodesma deauratum* were frequently infested with this spionid, southern Arctic wedge clams *M. arctatum* were not, even though

P. websteri does occur within their distributional range. Although there is little distributional overlap, the habitats and burrowing habits of the two bivalve species are essentially alike. It was considered possible that differing ecological conditions, especially water temperature, may influence not only the abundance of burrowing polychaetes but also the intensity and selectivity of their invasiveness (J. D. Davis, 1967).

Although not normally invading bivalves that burrow into the sediment, *Polydora* (*websteri*) was present in the valves of 5 to 10 % of individuals of *Mercenaria mercenaria* living under stress (pollution) conditions in Narragansett Bay tributaries. This may indicate that the clams had emerged from the sediment in response to irritant environmental conditions (Jeffries, 1972). J. D. Davis (1969) attributed similar *Polydora* infestation in *M. mercenaria* from Nantucket Harbor, Massachusetts, to partial exposure following storm-induced sediment dislocation and failure of the clams to re-enter the substratum. Landers (1967) described accidental and experimental *P. websteri* (misidentified as *P. ciliata*) infestations in laboratory-reared *M. mercenaria* grown in trays without substrate. Perforation of the valves of 5 to 8 mm long clams occurred within 18 days but took twice as long in clams 30 to 35 mm in length. Calcified mud blisters appeared on the inner shell surfaces within 30 days after perforation. In spite of considerable damage to the shells, particularly in small individuals, mortality did not appear to be increased significantly.

The 2 European bivalve-invading *Polydora* species, *P. hoplura* and *P. ciliata*, differ with respect to their distribution, their ecology and, particularly, their mode of shell penetration. In *Ostrea edulis*, *P. hoplura* produces mud blisters along the shell margin, which are similar to those made by *P. websteri* in *Crassostrea virginica*. *P. ciliata*, on the other hand, excavates U-shaped burrows all over the shell surface. The burrows are often extended by 'chimneys' composed of mud, which protrude from the oysters's valve surface, giving heavily infested individuals a 'hairy' appearance. Korringa (1951a, 1952a) considers *P. hoplura* to be more destructive to oysters than *P. ciliata*. Losses among cultivated Mediterranean oysters have been attributed to the former species (Carazzi, 1895).

Polydora hoplura, a more southerly species, is particularly abundant along the French Atlantic coast where it attacked almost every individual of *Ostrea edulis* (Giard, 1881). This species has also posed problems in Devon and Cornwall (England) oyster beds into which it had been introduced with oysters relaid from Bretagne, France (Cole, 1956a). *P. hoplura* invasion has been blamed to account for growth retardation in *O. edulis*. Three hundred worms per host are not uncommon in certain oyster-growing areas on the British west coast. Such numbers of worms were believed to be capable of checking shell growth very severely and to play a major part in the production of stunted oysters (Cole, 1956a; Cole and Waugh, 1958). Houlbert and Galaine (1916) found the meat yield of oysters infested with *Polydora* ('*Sclerocheilus*') to drop to 75 % or less of that of healthy individuals. Elimination of worms by dipping infested oysters in dilute phenol, cresol or copper sulphate solutions resulted in marked improvement in shell-growth rate. However, frequently rapid re-infestation ensued in relaid oysters, masking possible beneficial effects of the treatment (Korringa, 1951a, c, 1952a; Cole, 1956a; Cole and Hancock, 1956; Cole and Waugh, 1958).

In the colder waters of the North and Baltic Seas, *Polydora ciliata* replaces *P. hoplura* and commonly invades oysters and mussels (Lebour, 1907c; Atkins, 1931a; Korringa, 1951a, c, 1952a; Cole, 1956a; Hempel, 1957a, b; Dorsett, 1961b; Daro and Polk, 1973; Michaelis, 1978b; and others.) The longer planktonic life of *P. ciliata* larvae may also

account for its wider distribution (D. P. Wilson, 1928). *P. ciliata* larvae occur in large numbers in the meroplankton for several months of the year (Dorsett, 1961a; Daro and Polk, 1973). Although 'maladie du ver' resulting from *P. ciliata* attack has reportedly caused shell deformities and mortalities among *Ostrea edulis* on some beds (Leloup, 1937), Korringa (1952a) maintained that it is less destructive to oysters than *P. hoplura* and *P. websteri*. However, heavily *P. ciliata*-infested oysters have brittle shells that break easily during shipment.

Mytilus edulis, with its thinner shells, may suffer even more severely from *Polydora ciliata* invasion than oysters. Infested mussels exhibited a significant ($p < 0.001$) reduction in shell strength, as determined by the compression load at shell fracture (Fig. 13-163). There was a distinct negative correlation between the number of worms per valve and shell strength, yielding a correlation coefficient of $r = -0.47$ ($n = 47$) for mussels 71 to 75 mm in length, and $r = -0.40$ ($n = 85$) for individuals 61 to 65 mm long (Kent, 1977, 1981). Such a reduction in shell strength may considerably increase the mussel's vulnerability to predation and give the impression of 'size/age-selective natural mortality'. Predation is one of the main causes of mussel mortality and limits its vertical distribution (Seed, 1969a).

The absence of *Mytilus edulis* from some sublittoral regions has been attributed to the predation pressure of crabs *Cancer pagurus* (Kitching and co-authors, 1959). In intertidal regions, mussels with weakened shells may succumb more quickly to the predatory activities of shore crabs *Carcinus maenas* (Elner, 1978; Elner and Hughes, 1978) and birds, such as oystercatchers *Haematopus ostralegus* (Norton-Griffiths, 1967). At Sylt, German North Sea coast, gulls *Larus argentatus* were seen to drop mussels from consider-

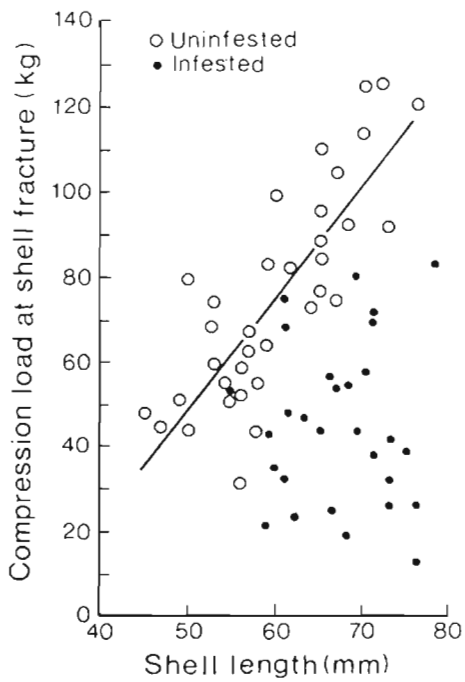


Fig. 13-163: *Mytilus edulis*. Effect of *Polydora ciliata* infestation on shell strength. (After Kent, 1981.)

able heights onto a concrete platform in order to gain easy access to the otherwise difficultly attainable flesh. The mussels so opened were invariably large (> 60 mm), and their shells were heavily infested with *P. ciliata* (Lauckner, pers. obs.). That *C. pagurus* in fact feeds selectively on mussels with weakened shells, has been demonstrated experimentally by Kent (1977, 1981). Caged crabs, offered mussels 63 to 69 mm in length, crushed only 28 % of mussels having 0 to 9 *P. ciliata* tubes but 75 to 77 % of individuals having 30 to 59 worm burrows in their shells.

Similar to shell strength, valve closure of *Mytilus edulis* was found to be affected by *Polydora ciliata,* presumably because of a reduction in the relative size of the posterior adductor muscle. The force necessary to pull the valves apart has been determined under constant (2.55 kg) and linearly increasing (0.5 cm sec^{-1}) load. In both cases, infested mussels could be opened significantly ($p < 0.001$) more easily (Kent, 1977). The load applied by Kent is well in the range of the traction power (2 to 5 kg) exerted by *Asterias rubens,* a significant mussel predator, and other asteroids (Feder, 1955; Lavoie, 1956; Hancock, 1965, 1974). Hancock (1965) suggested that Danish mussels, which have large posterior adductor muscles, are capable of resisting sea star attack better than British mussels, which have smaller adductors. Apart from increased vulnerability to sea star predation, weakening of the adductor muscle and resultant shell gaping may significantly reduce the mussel's tolerance to adverse physicoecological conditions (Kent, 1977).

In mussel shells heavily infested with *Polydora ciliata,* large confluent blisters and pearly excrescences on the inner valve surface (Figs 13-164 and 13-165) do not only produce a disgusting appearance but may also interfere with muscular attachment. Shell penetration in the region of the adductor or byssal retractor muscles results in atrophy and destruction of muscle tissue (Lebour, 1907c; Field, 1923). Significant reduction, by the large blisters, of available shell-cavity volume interferes with the general physiology of the mussel. Stress from *P. ciliata* alters the condition index*) and water content**). The differences in mildly and heavily infested individuals of *M. edulis* from Fowey Estuary, Cornwall (England), were statistically significant in most of the monthly samples (Figs 13-166 and 13-167; Kent, 1977, 1979). Field (1923, p. 220) has stated that *P. ciliata* "often interferes with the production of the genital products wherever the calcareous ridges press against the mantle" of the mussel. Similarly, Pillai (1965) suggested that *Polydora*-produced blisters may deform the unterlying mantle tissue and thereby affect gamete production. Kent's (1979) findings seem to indicate that the reduction of the condition index of the mantle (which contains the gonadal tissue) might be associated with reduced fecundity of mussels. Although no tests were done on the possible effect of heavy *P. ciliata* infestation on individual *M. edulis* gametes, it is known that physiologically stressed mussels may produce larvae which exhibit reduced vigour and slower growth than larvae from unstressed individuals (Bayne and co-authors, 1975).

Considering the high percentage of *Mytilus edulis* badly affected by *Polydora ciliata* in

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*) *Dry-weight condition index* $= \dfrac{\text{Total tissue-dry weight (g)}}{\text{Shell-cavity volume (ml)}} \times 1{,}000$

**) Water content (%) $= \dfrac{\text{Tissue-wet weight} - \text{Tissue-dry weight}}{\text{Tissue-wet weight}} \times 100$

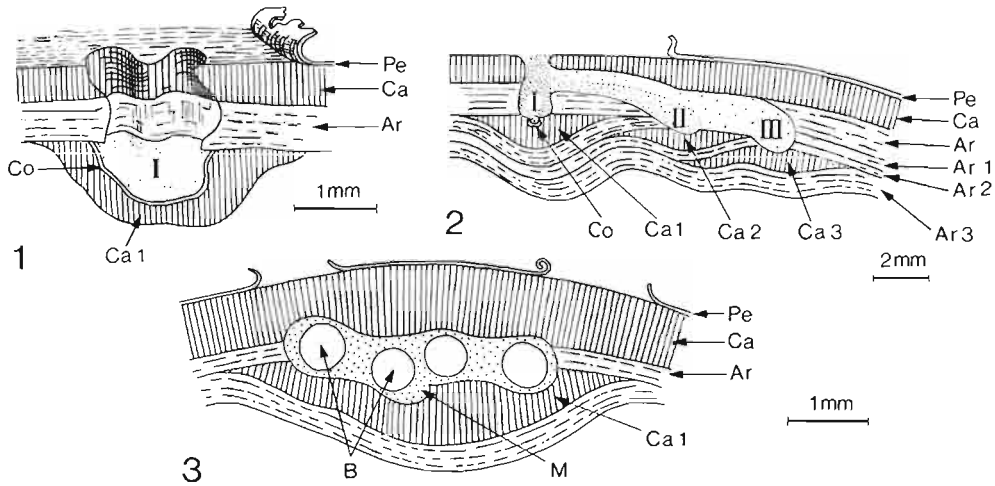

Fig. 13-164: *Mytilus edulis*. Diagrammatic section of shell exposing *Polydora ciliata* burrows. 1: Perpendicular primary burrow (I) excavated by juvenile worm in shell portion devoid of periostracum (Pe). Burrow penetrating inner aragonite shell layer sealed off by thin conchiolin sheet (Co) followed by calcite layer (Ca 1) protruding from inner shell surface. 2: As the worm grows, a horizontal burrow (II) is excavated and subsequently enlarged (III). Mussel responds by deposition of alternating layers of calcite (Ca 1–Ca 3) and aragonite (Ar 1–Ar 3). 3: Section of thickened shell portion showing accumulation of mud (M) and cross sections of U-shaped burrows (B) of 2 adjacent worms occupying a common blister. (After Michaelis, 1978b; modified.)

certain areas, possible effects of these spionids on mussel fecundity merit further study. In view of the wide range of ecological conditions under which *M. edulis* occurs throughout its geographical range, and concomitant differences in growth rate and overall condition, material for comparative purposes must be taken from the same locality. Crowley (1972), for example, was unable to detect any adverse effects of *P. ciliata* on Irish mussels, but he compared uninfested and infested individuals from different localities. Much remains also to be learned about the ecology of *P. ciliata*. Michaelis (1978b) reported on the mass occurrence of these worms in mussels from Norderney, German North Sea coast, in the winter of 1973/74, the causes of which remained unexplained. There can be little doubt that, locally, *P. ciliata* mass infestation can cause appreciable mortalities among mussels. Lebour (1907c) stated that, on Northumberland (England) mussel beds, particularly at Budle, where *P. ciliata* is "a real evil", it has been responsible for repeated devastations. Hertweck (1971) found *P. ciliata* infestations in *Cardium edule* which, when healthy and living under normal ecological conditions, lives burrowed in the sediment. It should be noted that, in addition to molluscan shells, *P. ciliata* can live in a variety of substrates including limestone (Giard, 1881; Hempel, 1957a, b). According to Joyeux-Laffuie (1891), the species is responsible for large-scale destruction of limestone beach along the French coast of the English Channel. He estimated that about 1.575 l of material per m^2 rock surface may be broken down by *P. ciliata*.

There are several records of '*Polydora ciliata*' from outside Europe, mainly from Australia. Haswell (1886) emphasized that worms affecting rock oysters *Saccostrea cucullata* on Hunter River beds were morphologically indistinguishable from the European *P. ciliata*. He (p. 274) stated that he has not been able "to find any record of such extensive

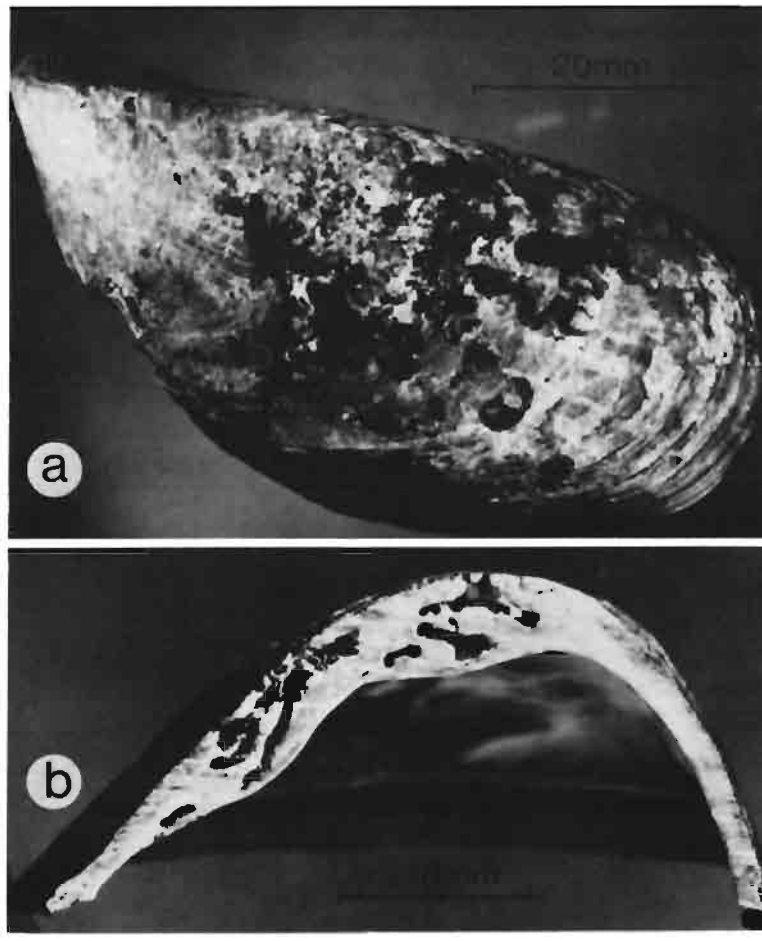

Fig. 13-165: *Mytilus edulis*. (a) Surface view of right valve showing numerous *Polydora ciliata* burrows; (b) crosscut through same specimen exposing worm burrows in thickened shell portion, ventral valve margin to the right. (Original.)

destruction of oysters effected by this little annelid on the European coast as seems to be taking place on the Hunter River". Similarly, Whitelegge (1890) and Roughley (1925) reported that mudworm infestations on New South Wales oysters beds may become so heavy that very few oysters survive to develop to a marketable size. The extensive mortalities eventually brought about the development of intertidal and off-bottom culture methods in New South Wales. In addition to *S. cucullata*, Whitelegge (1890) found *Pinna menkei* and other bivalves from New South Wales (Australia) waters to be commonly affected by what was believed to be *P. ciliata*. Stephen (1978a) found these worms in Indian backwater oysters *Crassostrea gryphoides* and *C. madrasensis*, respectively. Effects on these oyster species appeared to be mild, and the prevalence of '*P. ciliata*' in the Mulki estuary near Mangalore (India) appeared to be checked by low salinities during the monsoon period.

From the descriptions of Whitelegge (1890) and Stephen (1978a) of the burrowing

sites occupied by these Indopacific spionids — the worms settled and caused blister formation on the shell margins rather than boring into the shell from the outside of the valves — it appears that they are not at all *Polydora ciliata* but rather *P. websteri* or another, yet unidentified species. In fact, J. A. Blake (in Blake and Evans, 1973) has re-examined some of Haswell's (1886) material from Australian *Saccostrea cucullata* and has found that the specimens agree more closely with *P. websteri* than with *P. ciliata*. Durve (1964) identified spionids from *Crassostrea gryphoides* farmed near Bombay, India, as *P. coeca* rather than as *P. ciliata*.

In addition to the presumed *'Polydora ciliata'*, several other spionids have been reported from the Indopacific. Worms taken from pearl oysters *Pinctada margaritifera* have been named *P. hornelli* and *P. pacifica*, respectively (Herdman, 1906; Takahashi, 1937). Although not being believed to cause serious harm, the worms' irritating action has been shown to cause pearly excrescences on the inner valve surface of pearl oysters from the Gulf of Mannar (Ceylon). Mohammad (1972) described spionids from the same pearl-oyster species in Kuwait, Arabian Gulf, as *P. vulgaris*. Pillai (1965) identified spionids 'found among oysters' at Binakayan, Cavite, Manila Bay, as a new species, *Polydora cavitensis*. At Batticaloa (Ceylon), *Crassostrea virginica* was found to be affected by 2 spionids. One of these, believed to be *P. armata*, excavated horizontal burrows in oyster shells, each of which often harboured more than one worm. The presence of the other species, *Polydora* sp., was frequently associated with blister formation on the inner shell surface, which did not occur in the *P. armata* infestations. Larger blisters were found to have caused degeneration, discolouration and loss of normal consistency of adjacent mantle tissue. Where a blister had developed in the region of the adductor muscle, the fibres had degenerated and the muscle was weakened (Perera and Arudpragasam, 1966).

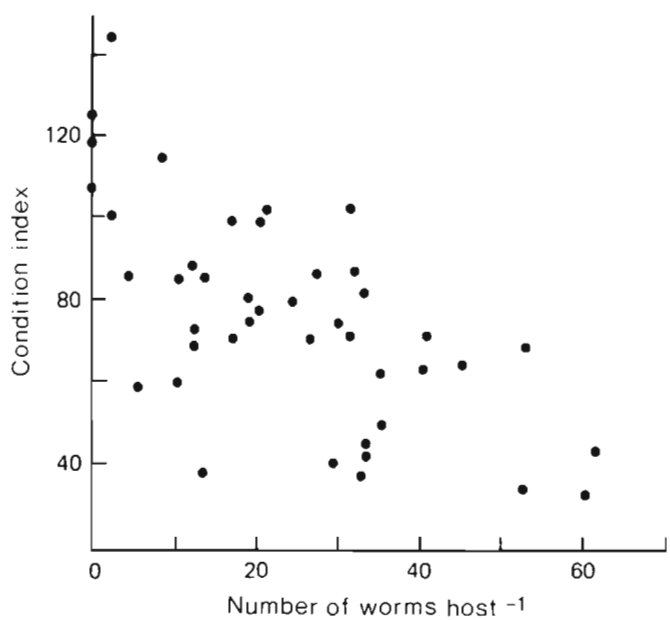

Fig. 13-166: *Mytilus edulis*. Decline of condition index in relation to number of *Polydora ciliata* per mussel. (After Kent, 1979.)

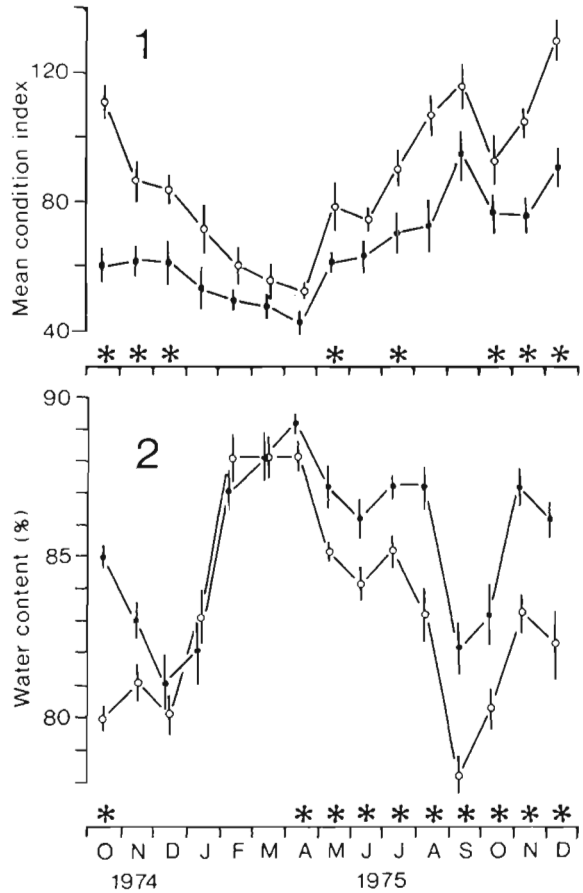

Fig. 13-167: *Mytilus edulis*. Effect of *Polydora ciliata* infestation on monthly changes in mean condition index (1) and water content (2) of mildly (○) and heavily (●) infested mussels. Vertical bars: standard errors; ★: means significantly different at 5 % level. (After Kent, 1979.)

Menzies' (1957) annotated bibliography on marine boring organisms, as well as a number of symposium papers edited by Carriker and co-authors (1969), make further reference to *Polydora*.

A number of polychaetes other than spionids are known to associate with marine bivalves. Cirratulids *Dodecaceria concharum* may settle in empty *Polydora* spp. burrows in shells of *Placopecten magellanicus*, enlarge these as they grow, and eventually form a distinctive burrow of their own (Evans, 1969). *D. concharum* has also been observed to inhabit *Polydora ciliata* burrows in shells of *Mytilus edulis, Mercenaria mercenaria* and other molluscs (Atkins, 1931a; J. A. Blake, 1969a). Nereids *Nereis limbata* occupy channels in oysters shells excavated by burrowing sponges *Cliona* sp. *Hydroides hexagonus* builds calcareous tubes attached to the outer surfaces of shells of dead and living oysters. When abundant, the tubes may kill young oysters (Nelson and Stauber, 1940). Sabellids *Pseudopotamilla reniformis* have been found in the shells of *Placopecten magellanicus* from Maine (USA). Some affected shells had large ridges on the inside, presumably

formed in response to worm penetration. The large size of the worms (up to 100 mm) and the reaction of the bivalve to its burrowing may constitute a potential menace when infestations are massive. Up to 30 large tubes have been removed from a single scallop shell (J. A. Blake, 1969a). *Potamilla* (*Pseudopotamilla*) *reniformis* has also been reported from shells of *Ostrea edulis, Pecten* spp. and *Anomia* spp. in France. *P. torelli* excavates burrows in *O. edulis* shells (Lamy and André, 1937).

Nereids *Neanthes arenaceodentata* (syn. *N. caudata*) have been identified as producers of unsightly shell blisters in *Mercenaria campechiensis* from Boca Ciega Bay, Florida. Incidences of blisters in 16 monthly samples consisting of 100 clams each ranged from 30 to 51 %. In 91 % of the affected clams, worm burrows were present in one valve only, at the posterior end. In heavily affected specimens, up to one-half of the inner shell surface was raised by the blisters, and in some cases the posterior adductor and retractor muscles were partially destroyed. Detrital material, accumulated by the worms in the blister cavity, gave the affected southern quahaugs an unattractive appearance, rendering them unacceptable for commerce in half-shell and steamer clams (Taylor and Saloman, 1972). Individuals of *Nereis* sp., up to 25 mm in length, burrow into the shell of *Crassostrea virginica* from Batticaloa (Ceylon). The excavations made by these worms could easily be distinguished from those made by *Polydora* spp. They were long, rather wide, and lined by a horny material. Their external openings appeared as minute pores on the shell surface. Forty-one of 50 oysters examined harboured these polychaetes. The largest number collected from any one oyster was 19 (Perera and Arudpragasam, 1966).

Ceratonereis tridentata may occur not only among the epifauna but also between the mantle and shell of *Argopecten gibbus*, causing blister formation. Although the worm blisters normally open to the exterior, some may also communicate with the mantle cavity through a perforation in the mantle tissue. Such penetration often occurs in conjunction with a malformation of the gills or gonads (Wells and Wells, 1962; Wells and co-authors, 1964; Wells, 1965). Sindermann (1971) stated that fouling by tube worms and other encrusting organisms can reduce survival of calico scallops. Eunicids *Eunice harassi* may inhabit the mantle cavity of *Ostrea edulis* (Gravier, 1900).

Although such associations merely represent cases of commensalism, a certain degree of host irritation inflicted by these inhabitants cannot be ruled out entirely. Paris (1955) and Clark (1956) briefly reviewed literature records of incidences of parasitism and commensalism in the polychaete annelids, which include annelid-bivalve associations.

Agents: Copepoda

Marine bivalves have many copepod parasites, more than any other invertebrate class. Some — like the well known *Mytilicola* — have greatly modified bodies, and live as endoparasites in the intestine. Others — like *Modiolicola, Ostrincola, Pseudomyicola, Conchyliurus, Myocheres, Paranthessius* and *Myicola* — are relatively untransformed and occupy the mantle cavity where they cling to the surface of the gills. Most of the copepods associated with molluscs are Cyclopoida; only a few members of the Caligoida, Harpacticoida, Lernaeoida and Monstrilloida are mollusc parasites, and Calanoida and Notodelphyoida have not been reported from this host class. Moreover, freshwater molluscs have no known copepod parasites.

Among most of the copepods reported from molluscs, host specificity does not seem to be very strict, but further studies are required to ascertain this. Damage to the molluscan hosts from ectoparasitic Copepoda appears to be slight, but mortalities in mussels have been attributed to the presence of endoparasitic Mytilicolidae.

The body of literature on the European mussel copepod *Mytilicola intestinalis* (Fig. 13-168) that has accumulated since its discovery in 1902 is vast. It appears that, in the course of the decades, every renowned shellfish biologist from each of the marine biological laboratories along the North European coasts has commented on this parasite whose importance relative to mussel mortalities is, nevertheless, as yet imperfectly understood. Only limited reference can be made here to the more relevant papers.

The adult stage of *Mytilicola intestinalis*, a cyclopoid copepod, was first described from the intestine of Mediterranean *Mytilus galloprovincialis* in the Gulf of Trieste, Adriatic Sea (Steuer, 1902, 1905). Pesta (1907) added descriptions of the free-swimming nauplius, metanauplius and first copepodid stages, but failed to infest mussels. Caspers (1939) accomplished experimental infestation of *M. edulis* with first-stage copepodids cultivated from the egg in the laboratory, and described the second and third copepodid stages (Figs 13-169 and 13-170). Further aspects of the life cycle and biology of *M. intestinalis* have been studied by Ahrens (1937, 1939a, b), Grainger (1951), Costanzo

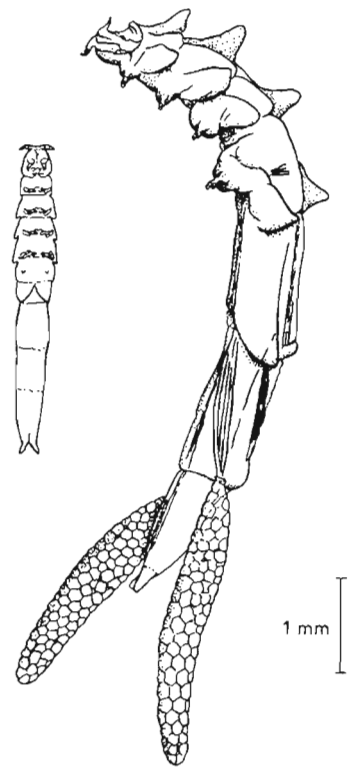

Fig. 13-168: *Mytilicola intestinalis* from intestine of *Mytilus edulis*. Adult male (left, ventral aspect) and female with egg-sacs (right, dorso-lateral view). Note dorso-lateral processes by which female appresses its ventral surface with appendages against opposite wall of host's intestine. (After Hockley, 1951.)

(1959), C. S. Williams (1969a, b, c, d), Davey and Gee (1975) and Davey and co-authors (1978).

Mytilicola intestinalis has previously been assigned to the family Clausiidae (= Clausidiidae) (Monod and Dollfus, 1932). Although M. S. Wilson and Illg (1955) have emphasized that this highly modified copepod does not appear to be referable to the latter family, and although Bocquet and Stock (1957b) have erected the family Mytilicolidae to include *M. intestinalis* as type species, several more recent authors (Cheng, 1967; S. A. Campbell, 1970) erroneously continue to assign *M. intestinalis* to the Clausidiidae.

Since its first recording in North European waters, *Mytilicola intestinalis* has repeatedly been accused of causing large-scale mortalities among mussels. As a consequence, a tremendous body of literature dealing with this mussel parasite has since accumulated. Prevalence and epizootiology of *M. intestinalis* have been monitored in England by Cole (1951a, 1961), Cole and Savage (1951), Hockley (1951), Bolster (1954), Waugh (1954), Hepper (1955), C. S. Williams (1969a, b), S. A. Campbell (1970), Davey and Gee (1976) and Davey and co-authors (1978), in Scotland by H. J. Thomas (1953) and Mason (1966), in Ireland by Grainger (1951) and Crowley (1972), in Portugal by Vilela and Monteiro (1958) and Monteiro and Figueiredo (1961), in Spain by Bassedas (1950), Andréu (1960a, b, 1963, 1965, 1976) and Figueras and Figueras (1980), in France by Heldt (1950, 1951), Dollfus (1951), Fleury and co-authors (1951), Korringa and Lambert (1951), Lambert (1951b, c), Brienne (1964) and His (1979), in Belgium by Leloup (1951, 1960), in the Netherlands by Korringa (1950b, 1951b, 1952b, 1953, 1956, 1957, 1959, 1968), in

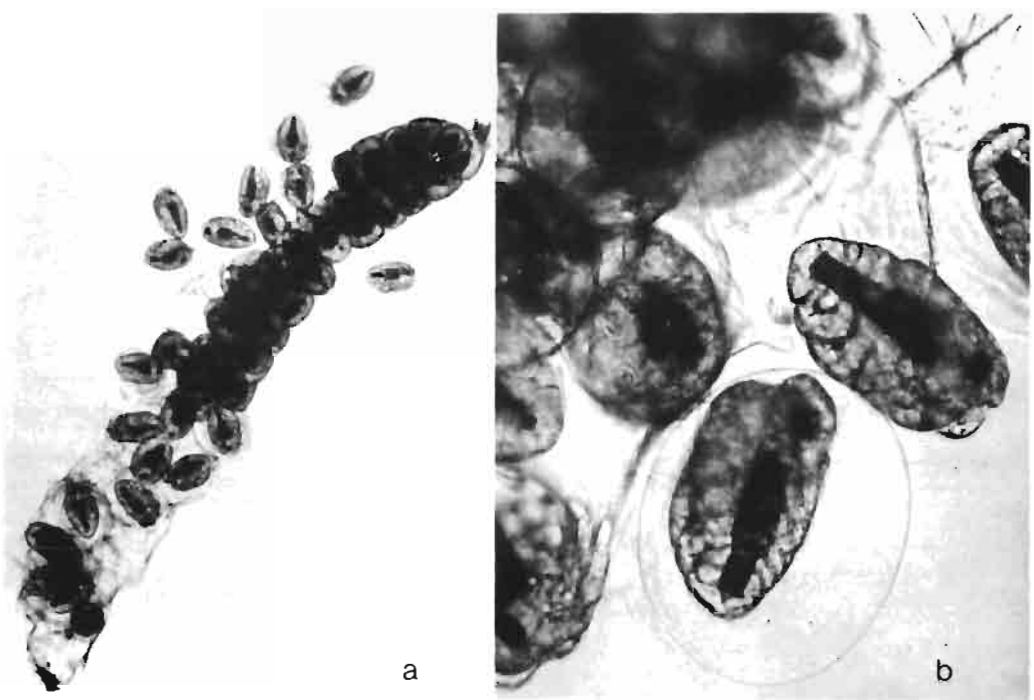

Fig. 13-169: *Mytilicola intestinalis* from *Mytilus edulis*. (a) Egg-sac with hatching nauplii; (b) same at higher magnification. (After Caspers, 1939.)

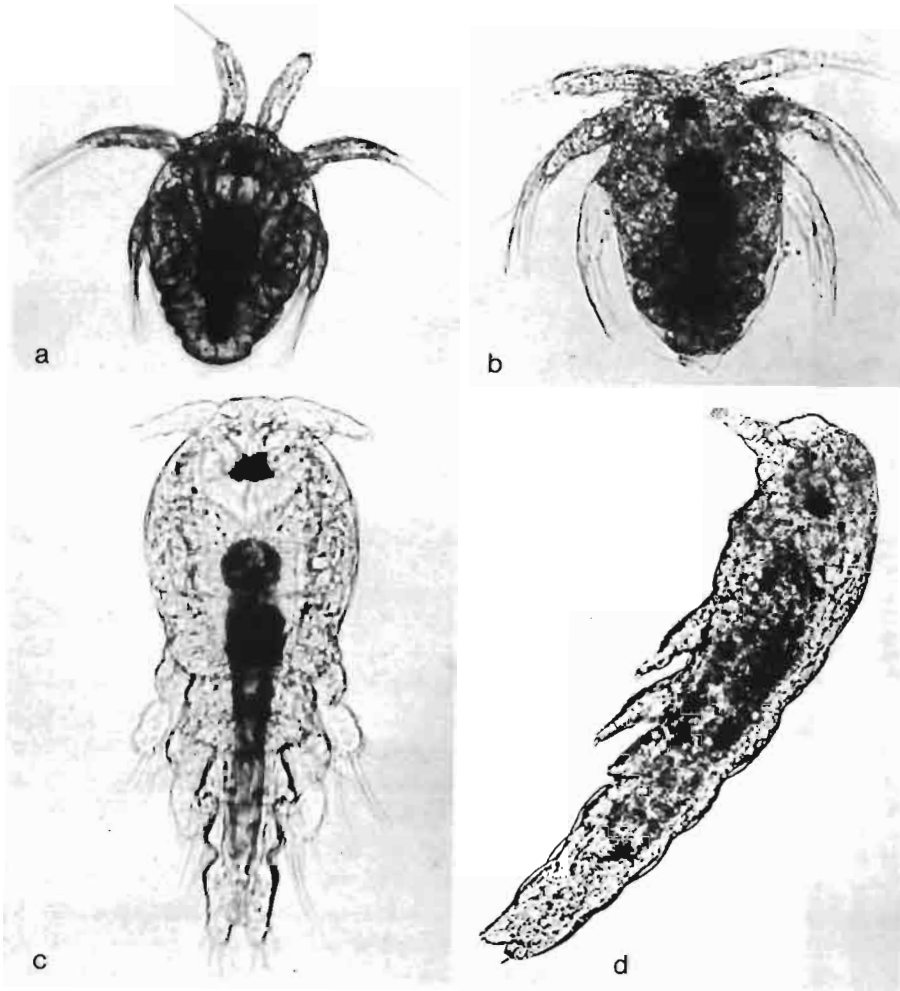

Fig. 13-170: *Mytilicola intestinalis* from *Mytilus edulis*. (a) Free-swimming nauplius; (B) metanauplius; (c) Copepodid I (1st parasitic stage from intestine of mussel), dorsal view; (d) Copepodid II (2nd parasitic stage), lateral view. (After Caspers, 1939.)

Germany by Meyer-Waarden (1951, 1956a, b, 1960, 1963), Meyer-Waarden and Mann (1950, 1951, 1952, 1954a, b, 1956) and Dethlefsen (1970, 1972, 1974b), and in Denmark by Theisen (1964, 1966). Parallel studies were conducted in the Mediterranean by Meyer-Waarden and Mann (1954c), Genovese (1959), Hrs-Brenko (1964, 1967) and Cormaci (1973).

From the various reports, the impression arises that *Mytilicola intestinalis* first occurred in the Mediterranean and has subsequently spread to the North Sea (Havinga, 1951). The initial records came from the Adriatic (Steuer, 1902, 1905; Pesta, 1907) and from the western Mediterranean (Cerruti, 1931; Monod and Dollfus, 1932; Bassedas, 1950; Heldt, 1950, 1951; Korringa and Lambert, 1951), where *M. intestinalis* mainly invades *Mytilus galloprovincialis* but has also been found in *M. edulis*. The first North Sea

record of *M. intestinalis* is that by Caspers (1939), who found 100 % infestation in mussels from tidal flats off Neuharlingersiel and Cuxhaven (German North Sea coast), with a maximum of 24 copepods per mussel. The author suspected that *M. intestinalis* might have occurred enzootically, and hence undetected, in *M. edulis* from the southern North Sea, as he had found an infested mussel in the collections of the Biologische Anstalt Helgoland from November, 1936. Ellenby (1947) reported the parasite as a new faunal element from the River Blyth, Northumberland, although there are at least two unconfirmed previous reports of its occurrence in England, one dating back to 1937 and the other to 1944 (Cole, 1951a).

North-eastward migration of the parasite, if it occurs, is apparently slow. *Mytilicola intestinalis* is rarely found north of the Elbe estuary along the coast of Schleswig-Holstein, Germany (Fig. 13-171). On the other hand, mussels from various localities in the western Limfjord, Jutland (Denmark) exhibited incidences of up to 97 % (Theisen, 1964). Zero infestation in the eastern part of the Limfjord, as well as in the Mariager Fjord and Randers Fjord, Danish Kattegat coast, suggests immigration of the parasite from the North Sea. No copepods have, however, been detected in mussels from the Danish North Sea tidal flats adjacent to the Limfjord entrance.

Despite the apparent slow geographic spreading, local infestations may develop rapidly and assume epizootic proportions. Thus, *Mytilicola intestinalis* was found in the Limfjord for the first time in January, 1964. In nearly all highly infested mussels there was a break in the contours of the shells, indicating the size at which they had become affected

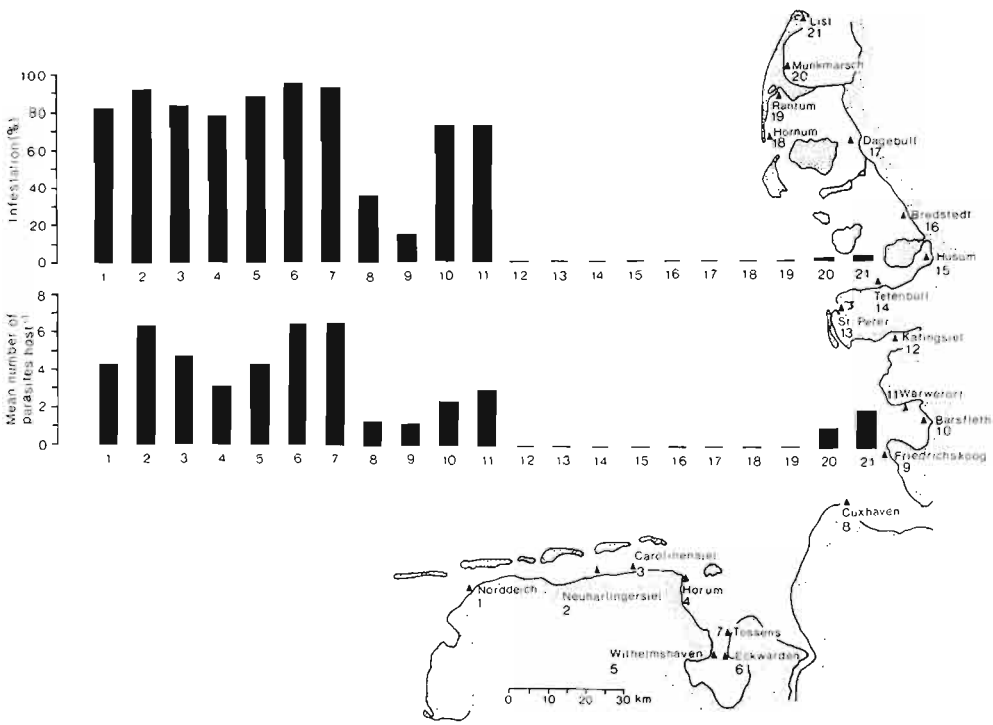

Fig. 13-171: *Mytilus edulis*. Incidence and intensity of *Mytilicola intestinalis* infestation at intertidal sampling stations. (After Dethlefsen, 1972.)

to such an extent that the growth rate had decreased drastically. The breaks were invariably found near the margin of the valves, suggesting that the massive *M. intestinalis* invasion had occurred in 1963, and that none of the animals had grown more than a few millimeters after having become infested. None of 83 Limfjord mussels, inspected for parasites in 1962, revealed any *Mytilicola* infestation (Theisen, 1964). Whether the observed growth-rate decrease was, in fact, due to *Mytilicola* or to other (ecological and/or disease) factors was not ascertained.

Mussels from the island of Föhr (German North Sea coast) and the Flensburger Förde (Baltic Sea), which were devoid of *Mytilicola intestinalis*, acquired copepods when transplanted to areas in the Jadebusen where high infestation levels prevailed. Within 4 months, up to 70 % of the transplanted individuals were found to harbour 2 to 4 parasites, and after 6 months 90 to 94 % of the introduced mussels contained 6 to 8 parasites (Meyer-Waarden and Mann, 1954a). Uninfested *Mytilus galloprovincialis* from Padstow (England) became infested to a level similar to or heavier than that of *M. edulis* when transplanted into an area where the copepod was present (Hepper, 1957). In contrast, experiments with *M. galloprovincialis* conducted in Lago di Ganzirri near Messina, Sicily (Italy), yielded no infestation in individuals placed in the vicinity of native, infested mussels (Meyer-Waarden and Mann, 1954c). It was concluded that *M. intestinalis* may be enzootic in low proportions of mussels in various areas and become epizootic only under adverse environmental conditions.

Prevalences of *Mytilicola intestinalis* may be very high locally but vary in relation to a multitude of abiotic and biotic factors. Infestation incidences of 100 % are not uncommon, and more than 30 copepods may be dissected from a single mussel. Hosts from lower shore

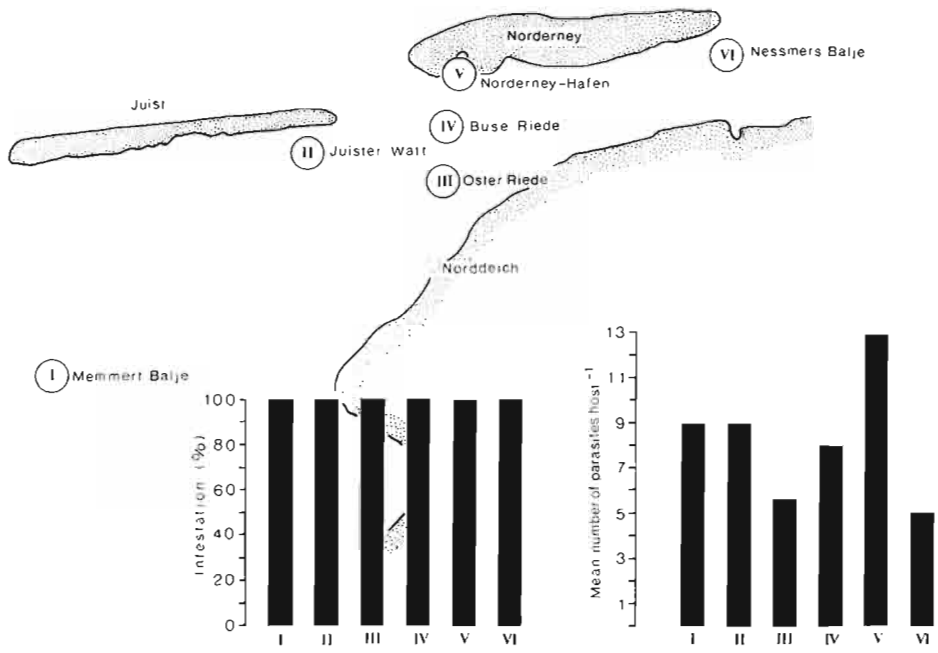

Fig. 13-172: *Mytilus edulis*. Incidence and intensity of *Mytilicola intestinalis* infestation at subtidal sampling stations. (After Dethlefsen, 1972.)

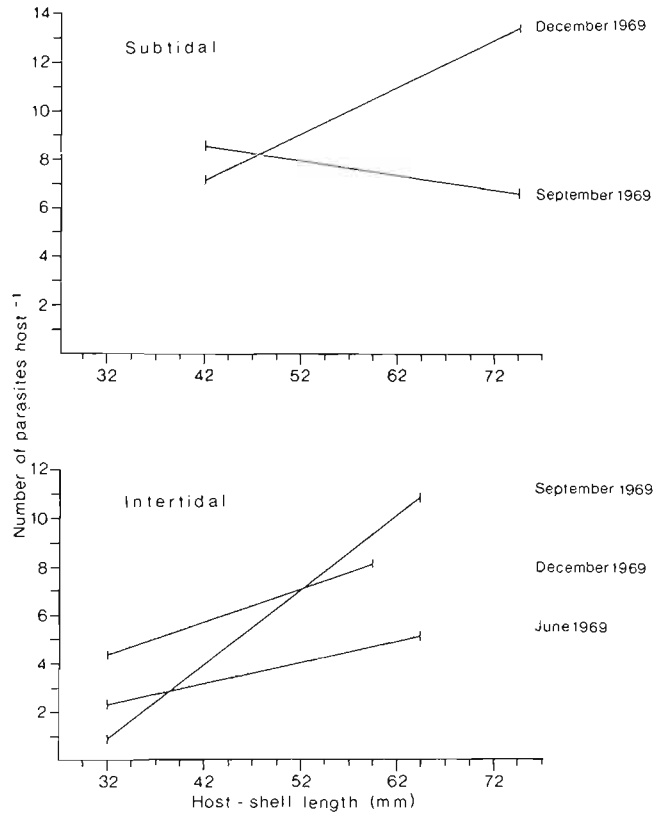

Fig. 13-173: *Mytilicola intestinalis.* Relation between number of copepods and shell length of host, *Mytilus edulis,* from subtidal and intertidal sampling stations during different seasons. (After Dethlefsen, 1972.)

levels and from sheltered areas are invariably more heavily invaded than individuals from high-shore levels and exposed localities (Figs 13-171 and 13-172). Usually, there is a positive correlation between infestation intensity and host size (Fig. 13-173), and larger mussels harbour larger worms (Fig. 13-174). Juveniles less than 10 mm in length are rarely infested (Hockley, 1951; Bolster, 1954; Hepper, 1955; Andréu, 1960a; Brienne, 1964; C. S. Williams, 1967; S. A. Campbell, 1970; Dethlefsen, 1972; Davey and Gee, 1976; His, 1979).

Possibly, biotic interferences of *Mytilicola intestinalis* with other parasites exist. Mussels showing a decline in condition due to heavy *Polydora* invasion can support fewer *M. intestinalis*, perhaps because there is less food available for the copepod (C. S. Williams, 1968). Usually, mussels infested by *M. intestinalis* have not been screened for other disease agents, such as microbial or protozoal pathogens. Therefore, disease symptoms tentatively attributed to the presence of the copepods, may have, in fact, been caused by etiologically different 'hidden infections'.

Quantitative recovery of *Mytilicola intestinalis* from mussels by dissection of guts is a tedious job. Dare (1977) has developed a technique for enzyme extraction of copepods from whole fresh or deep-frozen mussels using papain. By means of this method, large

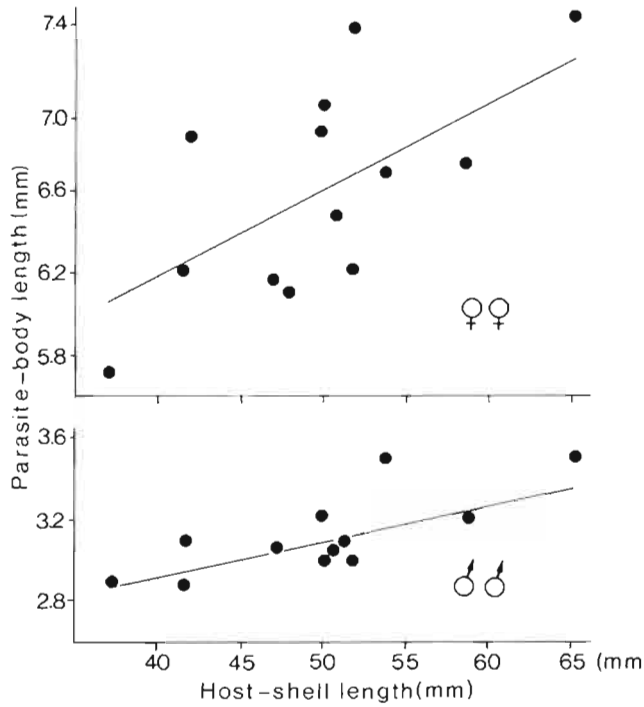

Fig. 13-174: *Mytilicola intestinalis*. Relation between body length of female and male copepods and shell length of host, *Mytilus edulis* (n = 240 measurements per regression line). (After Dethlefsen, 1972.)

numbers of mussels can be processed quickly, thoroughly and cheaply, thus enabling rapid assessment of *Mytilicola* distribution to be made over wide areas, particularly at low levels of infestation.

The presence of *Mytilicola intestinalis* causes metaplastic changes in the gut epithelium of *Mytilus edulis*, involving the replacement of normal ciliated columnar cells by non-ciliated cuboidal cells. However, as the metaplastic areas are localized, and in view of the known mobility of the copepod (Hockley, 1951), it was concluded that repair of the damaged areas is rapid, and that *M. intestinalis* has no significant effect on the basic cellular functions in *M. edulis* (Moore and co-authors, 1978). Occasionally, the copepods may invade the digestive gland of the mussel, which then assumes a reddish discolouration. Durfort (1980) and Durfort and co-authors (1982) reported pathological alterations of the oocytes of mussels harbouring 3 to 8 copepods in their digestive gland. The same changes, however, were also observed in mussels from an oil spill site, as well as in individuals parasitized by the trematode *Proctoeces maculatus* (*'Cercaria tenuans'*).

In spite of its apparent low degree of pathogenicity, *Mytilicola intestinalis* has been blamed for mass mortalities of *Mytilus edulis*, which commenced in 1949 in the Netherlands and spread to German waters in 1950, bringing the related industry to a standstill. Relative to the mode of action of *M. intestinalis*, the numerous investigations conducted by various authors are equivocal. Most of the early workers expressed — without definite experimental proof and with meagre observational data — the opinion that this copepod

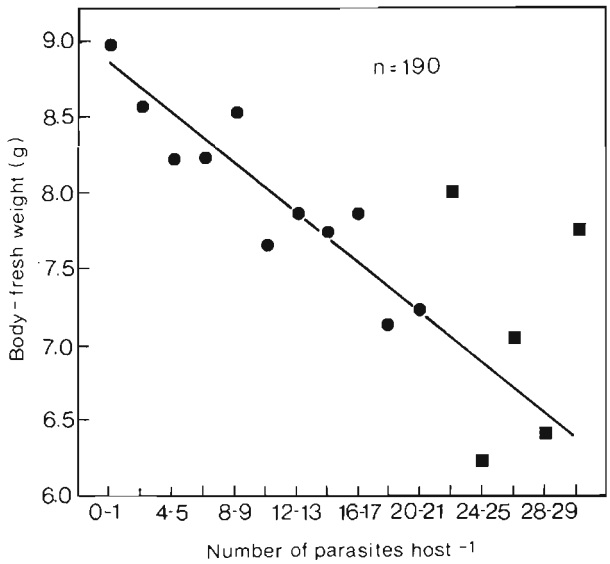

Fig. 13-175: *Mytilus edulis*. Effect of *Mytilicola intestinalis* infestation on soft-tissue fresh weight of mussels 70 to 75 mm in length (squares represent groups of 1 to 4 mussels only). (After Andréu, 1960a.)

parasite is highly detrimental to *M. edulis*. Thus, Cole and Savage (1951) found an inverse relationship between the condition of parasitized mussels of the size group 5.5 to 5.9 cm and both the mean number of copepods per host and the mean number of *M. intestinalis* over 1.5 mm in length. Moreover, they reported a mean flesh weight of 3.206 g in parasitized mussels of the size group 5.5 to 5.9 cm, as compared to 5.975 g in unparasitized specimens. Since parasitized and unparasitized mussels were obtained from different stations (!), these figures can in no way be compared to each other, and no conclusions can be drawn from these findings. Andréu (1960a) established a negative correlation between the soft-tissue wet weight of mussels from the Spanish Atlantic coast and the number of copepods per host. The line in Fig. 13-175 corresponds to the regression equation

$$\text{Host-body fresh weight} = 8.9849 - 0.0943 \times \text{number of parasites.}$$

Table 13-33
Mytilicola intestinalis. Effect on physiology and biochemistry of *Mytilus edulis* (Based on data presented by Meyer-Waarden and Mann, 1950)

	Non-parasitized	Parasitized
O_2 consumption (mg O_2 h^{-1} kg^{-1} at 21 °C)	76	132
Filtration activity (India ink clearance by test individuals within 90 min; relative values)	1.0	0.47
Meat content (% wet weight)	27.1	22.6
Water content (%)	45.8	36.5
Residues (% ash content)	8.9	7.6
Lipids (%)	6.7	6.4
Proteins (%)	58.2	55.6
Weight of digestive gland (%)	15.3	10.7

Again, the possible interference of *M. intestinalis* with other — undetected — disease agents has not been excluded.

Profound effects of *Mytilicola intestinalis* on the filtration activity, oxygen consumption and various body constituents of *Mytilus edulis* have been demonstrated by Meyer-Waarden and Mann (1950). On the basis of their results, which are summarized in Table 13-33, the authors arrived at the conclusion that the pathogenic effects of *M. intestinalis* are severe and could lead to the death of affected mussels. Detrimental effects of *M. intestinalis* on *M. edulis* have also been inferred by Mann (1951, 1956), Hockley (1951), Couteaux-Bargeton (1953), Bolster (1954), Meyer-Waarden and Mann (1954b), Korringa (1950b, 1955), Cole (1956a), and others. Despite the obvious lack of sound experimental procedures and the equivocalness of the various opinions, Korringa (1968) continued to blame 'red-worm disease' for mass mortalities among *M. edulis*. The findings of most of these authors could not be duplicated by modern workers.

It must be emphasized that in the above-cited papers virtually no attempts have been made to submit the observational data to even the most primitive statistical analysis. Consequently, the 'results' obtained merely reflect opinions and wishful thinking of the respective authors, who, being confronted with mass mortalities of *Mytilus edulis*, looked for an agent that could be made responsible for the epizootics. They found *Mytilicola intestinalis* to occur in high abundance in mussels and did not hesitate to accuse it — without definite supporting experimental evidence — of being the causative agent.

No search for the possible presence of microbial or other pathogens in diseased mussels has been made by these authors, although the coincidence between mussel mortalities and the apparent slow migration of *Mytilicola intestinalis* from the Mediterranean northward bears much resemblance to the spread of a contagious disease. Never in these papers did the idea arise of the presence of a contagious malady of possible viral, fungal or protistan etiology although, at that time, such diseases were known to occur in *Crassostrea virginica* in North America (Needler, 1941; Needler and Logie, 1947; Mackin and co-authors, 1950; H. C. Davis and co-authors, 1954). It appears worth mentioning, in this context, that increased mortalities observed in *Mytilicola orientalis*-infested *Crassostrea gigas* from Humboldt Bay, California, were believed to be attributable to heavy infections by an unidentified micro-organism in the tissues of moribund oysters (Chew and co-authors, 1965).

Being aware of these discrepancies, Dollfus (1951, p. 82/83) stated:

> "Mon opinion est que *Mytilicola* n'est pas pathogène et que la cause de la maladie est à chercher dans une autre direction . . . On ne doit pas considérer un parasite comme pathogène avant d'avoir expérimentalement reproduit la maladie à partir de celui-ci."

Unfortunately, this sound warning has been ignored by his contemporaries.

The probable involvement, in the mussel mass mortalities, of a contagious microbial or protistan agent is indicated by the facts that (i) bivalves other than *Mytilus edulis*, which harbour no *Mytilicola*, underwent unusual mortalities during the course of the mussel epizootics (Dollfus, 1951), (ii) even highly *Mytilicola*-infested *M. edulis* could be seen, which were in excellent condition (Hepper, 1955; Meyer-Waarden, 1956b, 1960, 1963; Monteiro and Figueiredo, 1961; Andréu, 1963; Brienne, 1964; Theisen, 1966; S. A. Campbell, 1970), and (iii) several investigators were virtually unable to detect adverse effects of *M. intestinalis* infestation on *M. edulis* and *M. galloprovincialis*, respectively

(Caspers, 1939; Genovese, 1959; Monteiro and Figueiredo, 1961; Hrs-Brenko, 1964; C. S. Williams, 1969c; Dethlefsen, 1974a, 1975; Davey and co-authors, 1978). The major objection against the '*Mytilicola* hypothesis', however, is the fact that mass mortalities of European mussels have virtually subsided in spite of persistent high *Mytilicola* prevalences in some areas (Dethlefsen, 1972).

The findings of modern workers relative to the pathogenicity of *Mytilicola intestinalis*, obtained by means of sound experimental procedures and statistically backed long-term observations may be briefly summarized as follows:

There is no traceable adverse effect of *Mytilicola intestinalis* on the condition index and the biochemical constituents of *Mytilus edulis*, with the possible exception of highly infested sublittoral mussels during the winter months. In all other cases studied, the magnitude of seasonal variations in mussel biochemistry and condition index due to variations in host-body size, stage of gonad development, seasonal cycles and environmental factors are greater than those due to parasitism (C. S. Williams, 1969c; Dethlefsen, 1975; Gee and co-authors, 1977; Fig. 13-176).

Various levels of *Mytilicola intestinalis* infestation had no effect on the rates of oxygen consumption by *Mytilus edulis*. Filtration rates were significantly affected by the infestation intensity, high levels (>10 copepods per mussel) causing a depression. But only at high temperatures (22 or 23 °C) and low density of food organisms did this depression of feeding lead to a decline in the scope for growth, which would result, in the long run, in a decline in host condition (Bayne and co-authors, 1978a). Thus, none of the above authors were able to corroborate the results obtained by Meyer-Waarden and Mann (1950).

Mytilicola intestinalis is not specific to mussels. In addition to *Mytilus edulis* and *M. galloprovincialis, Ostrea edulis, Cardium edule* and *Tapes decussatus* have been found to be naturally infested, although incidences and intensities were low. The above hosts have also been infested experimentally, but attempts to obtain infestations in *Scrobicularia plana, Pecten* (*Chlamys*) *varius, P. maximus* and *Macoma baltica* were unsuccessful (Baird and co-authors, 1951; Hepper, 1953, 1955, 1956). Cheng (1967) listed several trochid snails as hosts for *M. intestinalis*, following a number of authors in assuming that worms described from these snails as *Trochicola enterica* by Dollfus (1914) are identical with *M. intestinalis*. Cheng (1967) seems to have been unaware of the papers by Bocquet and Stock (1957b) and Bocquet and co-authors (1963) who, in accordance with Monod and Dollfus (1932), proved *T. entericus* to be a separate species invading gastropod hosts only.

Unikaryon mytilicolae, a microsporan hyperparasite, occurred in about 20 % of the *Mytilicola intestinalis* recovered from mussels on the Spanish Mediterranean coast. Stages of the parasite were seen in various tissues but were most frequent in the intestinal epithelium and in the gonads. In addition to oral transmission, a transovarial route was indicated by the occurrence in developing ova. This is the first microsporan hyperparasite reported from a copepod (Vallmitjana and Durfort, 1976; Durfort and co-authors, 1980).

Apparently, *Mytilicola intestinalis* is confined to European waters. A single worm recovered by Pearse and Wharton (1938) from an oyster in Apalachicola Bay, Gulf of Mexico, and identified as *M. intestinalis* by C. B. Wilson, probably belongs to *M. porrecta*. The latter species was found in 101 of 241 ribbed mussels *Modiolus demissus* (= *Geukensia demissa*), in 4 of 22 bent mussels *Brachidontes recurvus* (= *Ischadium recurvum*) and in a single *Mercenaria mercenaria* from tidal marshes and bayous in Louisiana (USA) waters. All of 252 *Crassostrea virginica* living side by side with the

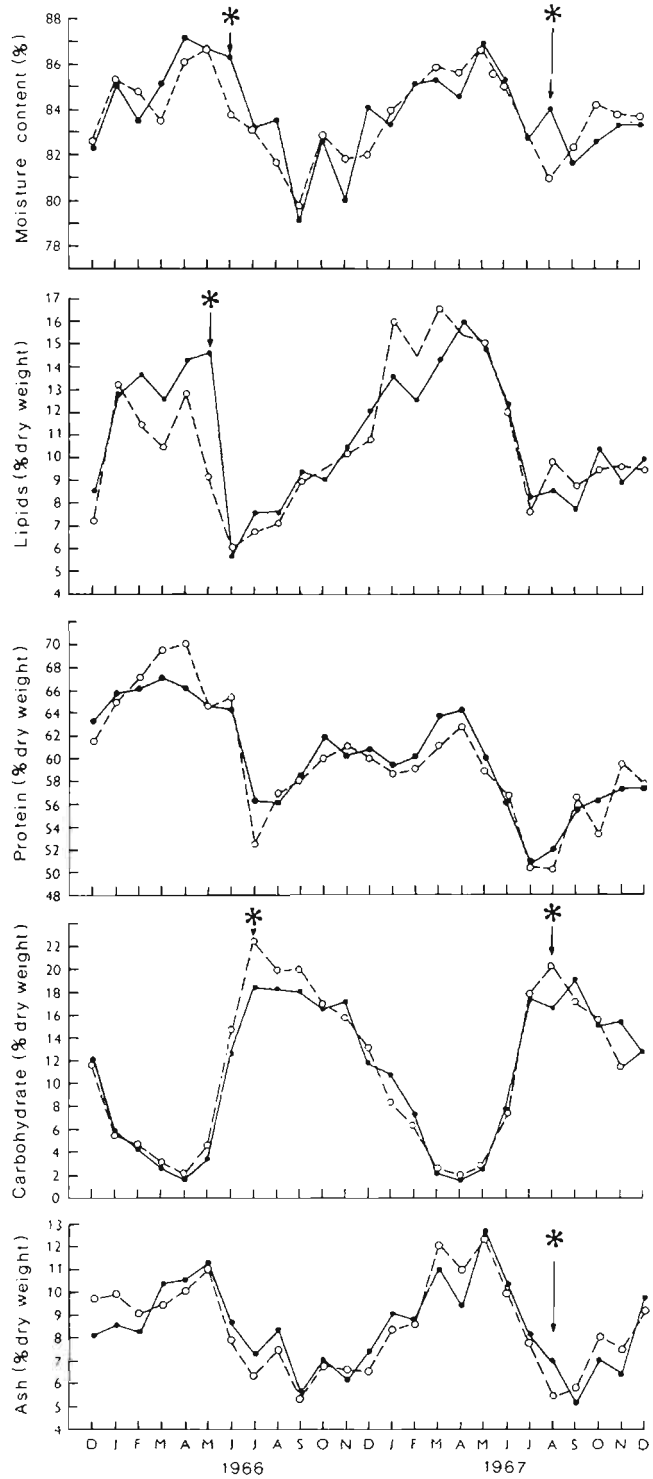

Fig. 13-176: *Mytilus edulis*. Seasonal variation in biochemical composition of healthy mussels (○) and of individuals infested with *Mytilicola intestinalis* (●). Asterisks: differences statistically significant at 5-% level. (After Williams, 1969c.)

infested *Geukensia* had no mytilicolids. The greatest number of copepods recovered from a single ribbed mussel was 15 (Humes, 1954a). Nothing is known about the relation between *M. porrecta* and its pelecypod hosts.

The dominant Pacific mytilicolid invading bivalves is *Mytilicola orientalis*, described from the gut of *Crassostrea gigas* and *Mytilus crassitesta* in the Inland Sea of Japan by Mori (1935). Individuals introduced into Puget Sound (Washington) with seed oysters obtained from Japan, were erroneously redescribed as *Mytilicola ostreae* by C. B. Wilson (1938). The parasite spread along the Pacific coast of North America where it now occurs in *Mytilus edulis, M. californianus, Crassostrea gigas, Ostrea lurida, Protothaca staminea, Saxidomus giganteus* and gastropods *Crepidula fornicata* (Odlaug, 1946; Chew and co-authors, 1964; Bernard, 1969; Johnson and Chew, 1969; Bradley and Siebert, 1978).

In Puget Sound, *Mytilicola orientalis* infestations were distinctly heavier in *Mytilus edulis* (range in samples: 42.5 to 73.6 %) than in *Ostrea lurida* (range: 1 to 9.2 %) (Odlaug, 1946). Similar conditions have been reported from Ladysmith Harbour, British Columbia (Canada), by Bernard (1969), and from Humboldt Bay, California (USA), by Chew and co-authors (1964), while Katkansky (1968b) and Sparks and co-authors (1968) found *Crassostrea gigas* from Humboldt Bay to be the most heavily infested species, followed by *M. edulis* and *O. lurida*.

Distinct adverse effects of *Mytilicola orientalis* on the condition index of *Crassostrea gigas* and *Ostrea lurida* have been reported by Odlaug (1946), Chew and co-authors (1965), Katkansky and co-authors (1967), Katkansky (1968b) and Sparks and co-authors (1968), but no relationship between infestation and oyster-growth reduction or mortality was established. Bernard (1969) was unable to detect a decrease in condition index in *Mytilicola*-infested *C. gigas* from British Columbia waters.

Recently, *Mytilicola orientalis* has been introduced into France with imported *Crassostrea gigas*. On some beds in the Arcachon region on the Atlantic coast, prevalences of up to 44 % were recorded in 1977, and up to 26 copepods have been recovered from individual hosts. *Ostrea edulis, Mytilus edulis* and *M. galloprovincialis* were also found to be infested, but to a lesser extent. The mytilids sometimes harboured concurrent infestations of *M. orientalis* and *M. intestinalis* (His, 1979).

Mytilicola orientalis causes metaplastic changes in the host gut comparable to those described by Moore and co-authors (1978) for *M. intestinalis*. In *Crassostrea gigas*, this metaplasia is particularly conspicuous in individuals recovered as gapers. Occasionally, the mucosa is completely eroded and an appendage of the copepod may be seen penetrating into the underlying connective tissue. Beneath the areas of epithelial metaplasia there appears to be a trend toward fibrosis of the underlying connective tissue, with the cells becoming more densely packed and containing less cytoplasm — a condition suggesting an attempt by the host to protect the underlying tissue by encapsulation of the parasite. When present in large numbers, *M. orientalis* may, like *M. intestinalis* in *Mytilus edulis*, cause occlusion of the gut lumen or distension of the gut wall. Heavily distended gut portions may, in turn, lead to the occlusion of adjacent blood vessels (Sparks, 1962). Katkansky and Warner (1968) reported on the unusual occurrence of *M. orientalis* in the digestive diverticula of *C. gigas*. Summer infestations as high as 14.4 % (69 of 479 oysters) were paralleled by 10.5 to 23.0 % infestations with copepods in the gut. Host response, indicated by extensive haemocytic infiltration, was sufficiently severe to cause resorption of the parasites.

Other species of *Mytilicola* reported from pelecypods include *M. fimbriatus* from Madagascan *Arca decussata* (Humes and Ho, 1970). Two further species, both assigned to the genus *Piratasta*, probably also belong to *Mytilicola*. *P. virgatula* has been found in *Laternula* (*Anatina*) sp. at the Siboga Expedition station No. 50 on the west coast of Flores (Leigh-Sharpe, 1934), and *P. brachidontis* occurred in *Brachidontes senhausi* from Lake Hamana, Siquoka Prefecture, Japan (Yamaguti, 1939). *Mytilicola mactrae* from the gut of *Mactra veneriformis* in Japan (Hoshina and Kuwabara, 1959) is, according to Humes and Ho (1970), probably identical with *M. orientalis*. Nothing is known about the relationships of these copepods with their pelecypod hosts.

Bresciani and Ockelmann (1966) described a strongly transformed copepod, which contrasts markedly with the above-mentioned forms. Named *Axinophilus thyasirae*, this species infests bivalves *Thyasira flexuosa* and *T. sarsi* in Scandinavian waters. Adult females, which measure about 2 mm in length, exhibit an almost complete lack of segmentation and absence of most of the limbs. The 1st and 2nd pairs of antennae are minute and very poorly segmented. Ventrally, in the place of the oral region, 2 horn-like lateral processes are found, which make contact with the host tissues. Eyes are absent and, except for those already mentioned, there are neither buccal nor any other appendages. The body is covered all over with microscopic papillae. The metasoma, about twice as long as the cephalic portion, carries a pair of lateral wing-like expansions. These contain the ovaries. The abdomen is long, fusiform, and gradually tapering posteriad. No segmentation is apparent and caudal rami are lacking (Fig. 13-177).

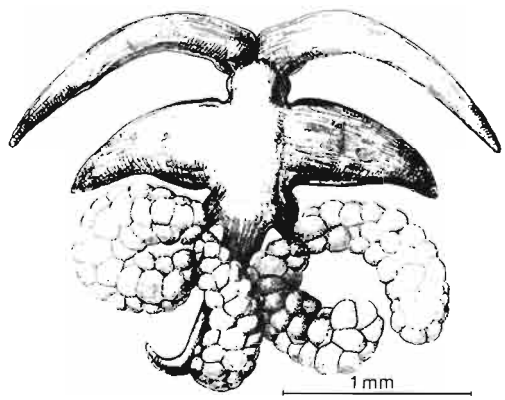

Fig. 13-177: *Axinophilus thyasirae* from bivalves *Thyasira flexuosa* and *T. sarsi*. (After Bresciani and Ockelmann, 1966.)

Except for the nauplii, all known stages of *Axinophilus thyasirae* were found attached to the anterior adductor muscle of the host. This position appeared to be very specific. The attachment is accomplished by the horn-like appendages, which are embedded in the host's tissues. Frequently, 2 to 3 copepods occurred in a single host, and up to 25 % of the clams were infested. In small *Thyasira* individuals, the parasites may considerably reduce the free space of the mantle cavity.

The male of *Axinophilus thyasirae* remains unknown. Bresciani and Ockelmann (1966) considered this genus distinct from all other known copepod genera. Possibly, it

even represents a new family. Although nothing has been reported concerning the feeding mechanism, it appears probable, from the brief description given, that this copepod feeds osmotrophically on the body fluids of its host. With respect to pathology, it seems likely that invasion by an adult female may significantly interfere with the proper function of the anterior adductor muscle of *Thyasira*.

Teredoika serpentina is a similarly extremely transformed parasitic copepod, which lives in the stomach of Mediterranean shipworms *Teredo utriculus*. The body of the female is composed of a central, vermiform part with 4 lateral, serpentine processes. There are no antennae, no mouthparts, and no legs but 2 ovisacs, which are 4-lobed in outline. Almost the entire body is filled with the ovaries (Fig. 13-178). The male is not known (Stock, 1960a, 1961).

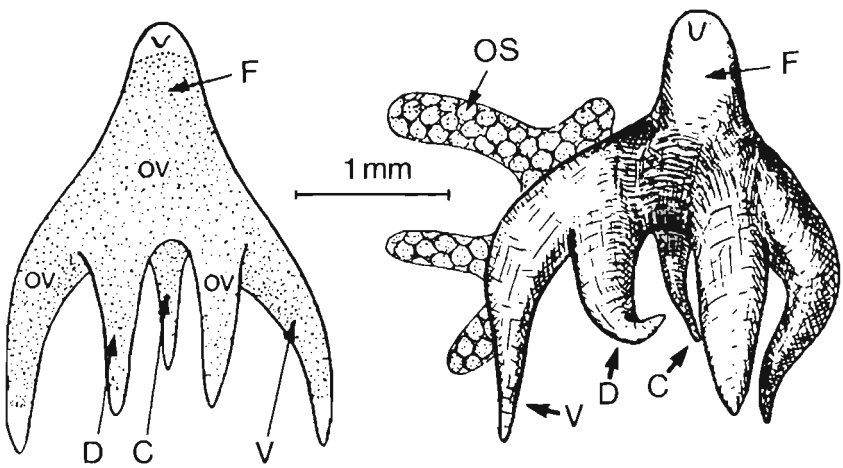

Fig. 13-178: *Teredoika serpentina* from stomach of *Teredo utriculus*. F, C frontal and caudal body ends; D, V dorsal and ventral lateral processes; OS ovisac; OV ovary. (After Stock, 1960a.)

Copepods living free in the mantle cavity (on the gills) of bivalves are usually morphologically untransformed or but slightly modified. Although most of these appear to be commensals, their attachment to the gill surfaces may inflict at least some degree of injury. The pathology associated with the presence of copepods within the mantle cavity has, however, scarcely been studied (Bocquet and Stock, 1963).

Harpacticoid copepods *Tisbe celata* have been found in the mantle cavity of 79 % of 265 *Mytilus edulis* from St. Andrews, New Brunswick, Canada. Numbers in individual hosts ranged from 1 to 41, with an average of 2 copepods per mussel. *T. celata* was not found in 203 *Mya arenaria* from the same area. It was concluded that *T. celata* is a parasite of the mussel rather than a free-living, accidentally introduced species, since it occurs only in *M. edulis* and then in relatively large numbers. The presence of immature stages indicates that *T. celata* breeds in the mussel. Other members of the genus *Tisbe* are free-living but a few, usually free-living forms, have also been found associated with marine invertebrates (Humes, 1954b).

A vast number of morphologically slightly or moderately transformed cyclopoid copepods have been recorded from the mantle cavity of marine bivalves. Only a few can be mentioned here. Further references may be found in the papers cited.

Clausidiids *Teredicola typicus* are associated with shipworms — wood-boring bivalves of the family Teredinidae — in Australian, New Zealand and Japanese waters (C. B. Wilson, 1944; M. S. Wilson, 1957; Humes and Turner, 1972; McKoy, 1975). Myicolids *Myocheres major* inhabit the mantle cavity of *Tagelus gibbus, Mya arenaria, Mercenaria mercenaria, Ensis directus, Spisula solidissima* and several other pelecypods from the Atlantic coast of North America (Humes and Cressey, 1960). Numerous further cyclopoid copepods, mostly members of the genera *Anthessius, Paranthessius, Lichomolgus, Myicola, Pseudomyicola, Modiolicola, Ostrincola, Herrmannella, Metaxymolgus, Gelastomolgus* and *Conchyliurus* associate with bivalves in various parts of the world (R. R. Wright, 1885; C. B. Wilson, 1932; Yamaguti, 1936; Pearse, 1947; Humes, 1953, 1958a, b, 1967, 1968a, b, 1970, 1972, 1973; J. A. Allen, 1956; Bocquet and Stock, 1957a, 1958a, b, 1959a, b; Gooding, 1957; Humes and Cressey, 1958; Illg, 1960; Stock, 1960a, b; Y. Kô, 1961, 1969; Bresciani and Lützen, 1962; Y. Kô and co-authors, 1962; Humes and Ho, 1965; Humes and Stock, 1965; Dethlefsen, 1972, 1974b; Ho, 1980; and others). Humes and Stock's (1973) revision of the family Lichomolgidae includes a host-associate list.

Few copepods associating with bivalves are host-specific, but some exhibit a distinct host preference. For example, J. A. Allen (1956) found *Myocheres inflata* in approximately 70 % of the *Lucina pennsylvanica,* but in only 10 % of the *Divaricella quadrisulcata* from Porgy Bay, North Bimini (Bahamas). *Myicola ostreae,* found on the gills of 40 % of *Crassostrea gigas* from Arcachon (France), also associates with *C. angulata* and *Ostrea edulis,* but was not found in *Mytilus edulis* and other commercially important bivalves (Comps, 1972b; His, 1979). Similar variations in host preference have been reported for numerous other associations.

Copepod prevalence in the mantle cavity of bivalves may vary greatly with the respective host and associate species involved, as well as with season and environmental factors. Bocquet and Stock (1957a) recorded clausidiids *Conchyliurus solenis* as being 'extremely abundant' in the mantle cavity of *Solen marginatus* from Finistère, French Atlantic coast. Almost every clam was infested. Infestation of *Mytilus edulis* from the German North Sea coast with *Modiolicola insignis* showed considerable local and seasonal variation. Highest prevalences — 60 to 90 % — occurred in the summer, lowest — 0 to 3 % — in the winter. Infestation rates varied between 0.15 and about 7 copepods per mussel (Dethlefsen, 1972, 1974b).

Some copepods invading molluscs have adapted well to the feeding methods of their hosts and are, therefore, restricted to specific niches. Lucine bivalves (family Lucinidae), for example, are unusual in that they build an anterior inhalant tube, which results in a new type of feeding mechanism (J. A. Allen, 1953). The anterior adductor muscle is elongate and ciliated for the transport of food particles to the mouth. Female copepods *Myocheres inflata* are invariably found curled around the ventral end of the anterior adductor of their hosts, *Lucina pennsylvanica* and *Divaricella quadrisulcata,* with their head toward the bivalve's mouth and with the entire animal lying over the ciliated tract. Never more than 1 female occurred in a single mollusc, and males were never found in this position on the adductor muscle but were always in the general mantle cavity (J. A. Allen, 1956). Other copepods, like *Modiolicola insignis* found in the mantle cavity of *Mytilus edulis,* appear to be more evenly distributed over the ciliated epithelial surfaces of their hosts (Dethlefsen, 1972). In most cases, however, the exact location of the copepods has not been determined.

Little information is available concerning the nature of most of the bivalve-copepod associations. The bulk of the above-cited papers is merely taxonomic; a few contain ecological notes. Despite the apparent lack of conclusive evidence, some authors prefer to label these associations as parasitic (Monod and Dollfus, 1932; Cheng, 1967). From a more conservative point of view, the majority of cyclopoid copepods associated with bivalves, whose mantle cavity affords excellent protection, appear to be commensals or semiparasites, feeding on mucus produced by the host's epithelia, as well as on food particles brought in by the filtering activity of the gills. Thus, the principal element of the mid-gut content of *Ostrincola koe, Modiolicola bifida* and *Conchyliurus quintus*, left overnight in beakers with sea water and excised gills of their host clam, *Tapes philippinarum*, consisted of protein containing sulphated mucosubstances, the histochemical properties of which were markedly in accordance with those of the mucus glands of the gills. In addition, a negligible amount of tissue or cellular gill debris was detected in the mid-gut content of the copepods (Yoshikoshi and Kô, 1974). *Ostrincola koe* could readily be reared using gills of *T. philippinarum* as food source for mucus (Kô and Yoshikoshi, 1974a, b).

Attachment and feeding activities of gill-inhabiting copepods have been found to be associated with local tissue damage. Thus, gill portions of *Crassostrea virginica* surrounding the attachment site of an unidentified copepod ("quite similar to a miniature *Ergasilus*") were strongly hypertrophied and exhibited an intense immune reaction (Quick, 1971).

Myicola ostreae causes gill lesions in *Crassostrea gigas* and *C. angulata*. Although damage done by a single copepod may be unimportant, high incidences (up to 40 % in *C. gigas* cultivated at Arcachon, France) may have some impact on oyster populations (Comps, 1972b; His, 1979). *Myicola metisiensis* and *Lichomolgus leptodermatus* have been found to be responsible for the production of local swellings and 'gall-like' tissue malformations on the gills of *Mya arenaria* and *Laevicardium crassum*, respectively (R. R. Wright, 1885; Bocquet and Stock, 1959a).

Myocheres major is a copepod frequently reported as a gill inhabitant associating with numerous bivalve species. Cheng (1967) found a single female in the stomach of an individual of *Mya arenaria* from Narragansett Bay, Rhode Island (USA). Similarly, *Pseudomyicola spinosus*, which is known from the mantle cavity of over 39 bivalve species from various parts of the world (Humes, 1968a), has been recovered from the intestinal tracts of *Mytilus galloprovincialis* from the French Mediterranean coast, of *Ostrea stentina* from the Gulf of Naples, and of *Crassostrea glomerata* from New Zealand (Korringa and Lambert, 1951; Stock, 1960a; Dinamani and Gordon, 1974). Both adult and early stages of *P. spinosus* were found on the palps and in the alimentary tract of *C. glomerata*, and there was evidence that the stages move freely between both sites. No damage to the gills was seen, but injury to the gut epithelium, comparable to that caused by *Mytilicola*, was apparent (Dinamani and Gordon, 1974). Kajihara and co-authors (1980) obtained experimental development of *P. spinosus* from the egg to the 6th copepodid stage on a diet consisting of small pieces of (unspecified) bivalve gill.

It appears, from the above observations, that other cyclopoid genera may display a behaviour similar to that of *Myocheres major* and *Pseudomyicola spinosus*. It seems also that our concept of the presumed non-pathogenicity of bivalve-invading Copepoda requires some revision.

Agents: Cirripedia

Barnacles, members of the family Balanidae, are the most conspicuous among the numerous fouling organisms frequently present on bivalve shells. Barnacles fouling the shells of calico scallops *Argopecten gibbus* may prevent complete valve closure so that predators can be admitted more easily (Allen and Costello, 1972). Wells and co-authors (1964) suggested that fouling organisms on scallops may impair mobility and thus reduce their ability to escape from predators. Barnacles sometimes cover the entire surface of the siphonal plates of *Tresus nuttalli* (Stout, 1970). The valves of *Mytilus edulis* from North Sea tidal flats may sometimes be covered with balanids to such an extent that the weight of the cirripeds exceeds by far that of the mussel. Poor condition and reduced growth of such individuals appear to be attributable to competition for food by the barnacles. Heavily encrusted mussels frequently had numerous metacercarial cysts of *Himasthla elongata* in their foot musculature (see section 'Agents: Trematoda'). It was concluded that the trematode infestation impaired the shell-cleaning behaviour normally saving healthy mussels from excessive fouling (Lauckner, pers. obs.).

Baumert (1924), Linke (1939) and Hertweck (1979) reported on balanids overgrowing the posterior shell portions of *Cardium edule*, which normally lives completely buried in the sediment. Fouling is restricted to cockles which do not, or — more correctly — are no longer able to bury. The specimens seen by the above authors have not been inspected for parasites, but it has been found by the reviewer that individuals of *C. edule* from Sylt, German North Sea coast, with *Balanus* spp. upgrowth on their posterior shell portions, were simultaneously heavily parasitized by *Himasthla elongata* metacercariae (Fig. 13-104). Such animals are sluggish and unable to bury completely. However, cockles with heavy *Balanus* upgrowth, recovered during July 1982 from Voslapp-Watt, Jadebusen (i.e., from the area from which Linke [1939] and Hertweck [1979] obtained part of their material), proved to be devoid of *Himasthla* metacercariae. Elevated cadmium levels in the tissues were believed to be responsible, at least in part, for the debilitation of these cockles (Lauckner, in preparation).

Malacolepas conchicola, a nude pedunculate cirriped, was found attached to the inner surface of the valves of *Cucullaea labiata* from Tanabe Bay (Japan). The peduncle of this strongly aberrant cirriped (Fig. 13-179) bears a basal attachment process enclosed by a calcareous tube, 6.8 to 24.6 mm in length and 1.5 to 3.5 mm in diameter, and resembling that of a serpulid annelid. Histological examination revealed that the tube is of host origin, obviously secreted by the bivalve in response to the irritation. There can be little doubt that the vital functions of *C. labiata* harbouring such growths are grossly impaired. Individuals of *M. conchicola* were also recovered from *Venerupis* (*Tapes*) *mitis*, but this host did not secrete a tube around the base of the cirripeds' peduncle (Hiro, 1933).

The morphologically most strongly transformed cirripedian associates of marine bivalves are members of the Acrothoracica. These burrowing barnacles are found in a wide variety of calcareous substrates including limestone, corals, bryozoans, chitons, gastropods and pelecypods (Tomlinson, 1969a). Species of *Weltneria, Lithoglyptes* and *Kochlorine* have been found in the shells of *Tridacna, Chama* and other bivalves. Initial penetration of the substratum by the acrothoracican cypris larva, which lacks abrasive teeth, is apparently the result of chemical dissolution. Adult females excavate their burrows largely by abrasion, which is accomplished by chitinous teeth extending outward from the mantle

surface. That mechanical abrasion accounts for most of the burrowing is attested by the piles of shell powder lying along the burrow aperture on the surface of the host shell in undisturbed individuals (Tomlinson, 1969a, b). However, enzymes may assist substrate penetration. Carbonic anhydrase has been detected in the mantle of *Trypetesa nassarioides*. Its concentration is very low during the resting phase, but increases conspicuously when the cirriped is excavating shell material. Alkaline phosphatase was also found, but its relation to the burrowing process has not yet been established (Turquier, 1968).

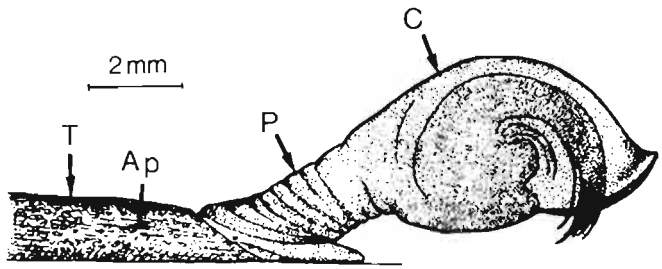

Fig. 13-179: *Malacolepas conchicola* from valve of *Cucullaea labiata*. Ap attachment process, C capitulum, P peduncle, T calcareous tube. (After Hiro, 1933.)

Since acrothoracican cirripeds are soft-bodied animals, which lack shelly plates of their own, burrowing is essential for the members of this order. Only the females excavate burrows. The motile dwarf males are partially buried within a pocket of the female's mantle tissue. The families of the Acrothoracica can readily be separated by burrow shape. These cirripeds can at most be considered a very modest shell-weakening pest, and in general do little if any harm to their bivalve hosts (Tomlinson, 1969b).

Agents: Amphipoda, Isopoda

A few amphipods are known to inhabit the mantle cavity of marine bivalves. All except one species are clearly commensal, obtaining protection and getting probably most of their food through the feeding activities of the host. Gammarids *Cardiophilus baeri*, reported from the mantle cavity of Caspian *Cardium baeri*, however, may represent some stage intermediate between commensalism and parasitism since it exhibits several morphological adaptations indicative of a semi-parasitic way of life. Its mouthparts are rather different from the normal pattern characteristic of the family, i.e., the palps of the maxillulae, as well as the dactyli of the maxillipedal palps, are greatly reduced, and the strongly hooked dactyli of the pereiopods are well suited for attachment to host tissues. Vader (1972) has reviewed the published records of known amphipod-mollusc associations.

Epicaridium larvae of an unidentified isopod species have been recovered from the mantle cavity of 2 of 124 *Mytilus californianus* from Windmill Beach, Bodega Head, California (Temnikow, 1974). It appears that these have accidentally been introduced into the mollusc via the respiratory current. Bivalves are not normal hosts for isopods.

Agents: Decapoda

Numerous brachyuran crabs, mostly members of the family Pinnotheridae, live symbiotically in the mantle cavity of marine bivalves in various parts of the world. Abundance and species diversity of the 'pea crabs', as they are called, is highest in tropical and subtropical waters and decreases significantly with geographical latitude, particularly on the northern hemisphere. Few species have been reported from Europe. Among the numerous American brachyurans associating with marine invertebrates, several have been reported from commercially important bivalves. Diseased conditions associated with the presence of pinnotherid crabs in the mantle cavity include emaciation, reduced filtering capacity and damage to gills, palps and mantle.

The body of literature on Pinnotheridae is immense; only limited reference can be made here to the most pertinent publications. For general information on the taxonomy of this group consult Rathbun (1918), Schmitt (1921), Wells (1928) and Schmitt and co-authors (1973). Wells (1940) studied the ecology of pinnotherid crabs from the North American Pacific coast, and Hyman (1924) described the experimental development of the larvae of several North American species and presented a key to the known zoeas. Major sources for information on Indopacific, East Asian and Australian pinnotherids are the publications of Bürger (1895), Tesch (1918), Balss (1922), Yokoya (1928), Shen (1932), Sakai (1934, 1939, 1976), Scott (1961), Bennett (1964) and Pregenzer (1979b).

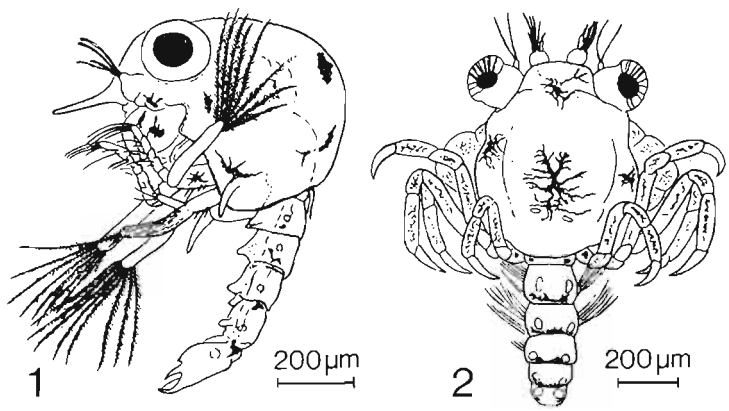

Fig. 13-180: *Pinnotheres pisum*. 1: Third zoea; 2: megalopa. (After Atkins, 1955a.)

The most thoroughly studied Pinnotheridae are those invading commercially exploited bivalves on the North American and European Atlantic coasts. The accumulated information on these forms has been summarized by Cheng (1967) and Silas and Alagarswami (1967). Additional important papers have been published subsequently. Pinnotherids associating with marine bivalves are mainly members of the genera *Pinnotheres, Fabia* and *Pinnixa*. In brief, the general pinnotherid life-cycle pattern may be outlined as follows: The larval life comprises several successive planktonic zoeal instars and a single megalopa stage. The first true crab stage invades the mantle cavity of bivalves and undergoes several pre-hard moults. The resulting hard stages of both sexes (erroneously designated 'Stage I') may or may not leave the host and copulate. Males die at this

stage, while the females re-invade bivalves, undergo 4 post-hard moults and reach sexual maturity at Stage V. All post-hard females, which never again leave the host, have thin, flexible exoskeletons.

In European waters, *Pinnotheres pisum* is the most common bivalve-invading crab. Aspects of its biology have been studied exhaustively by Coupin (1894), Orton (1920), Atkins (1926, 1955, 1958, 1960), Lebour (1928a, b), G. Williams and Needham (1939), Needham (1950), Berner (1952), Møller Christensen (1959, 1962), Houghton (1963), Huard and Demeusy (1966a, b, 1968) and Seed (1969b). *P. pisum* appears to have an unusual 2-host life cycle. After having passed through 4 planktonic zoeal and a single megalops stage (Lebour, 1928a, b; Atkins, 1955a; Fig. 13-180), it metamorphoses into the first true crab stage (Fig. 13-181, 1), about 0.5 to 0.75 mm in carapace width, which invades individuals of *Spisula solida*. In the mantle cavity of this regular initial host, *P. pisum* undergoes several successive pre-hard moults (Fig. 13-181, 2) until it transforms into the 2 to 7 mm long hard-shelled stage. It is at this stage that the crabs change hosts, copulate and invade *Mytilus edulis* and other bivalve species of the epifauna, such as *Modiolus modiolus*. Only the pereiopods of the first crab and the hard-shelled stages have swimming hairs. The other stages never leave their hosts (Møller Christensen, 1959).

Investigators prior to 1959 have consistently failed to discover pre-hard stages of *Pinnotheres pisum* because they never inspected bivalves other than mussels. This led to the erroneous assumption that the hard-shelled crab is the invasive stage (Atkins' Stage I). Møller Christensen (1962) later found an ovigerous female in *Spisula solida*. The author assumes that this small soft-shelled individual, which had a carapace width of about 4.5 mm, somehow got 'trapped' within the mantle cavity of the primary host, where it had no chance to reach the normal size of an adult female. Nevertheless, it obviously was able to reach maturity and to produce eggs, although in numbers far below average. Numerous *Spisula* clams opened since never yielded a single further post-hard *Pinnotheres* individual. In addition to *Spisula*, pre-hard and hard-stage *P. pisum* were also found in species of *Glycymeris, Cardium, Laevicardium* and *Mactra*, all of which are suspension-feeding members of the infauna of sand or gravel bottoms (Møller Christensen, pers. comm.). Huard and Demeusey (1966a, b) recorded first-stage crabs in *Mytilus edulis* from Luc-sur-

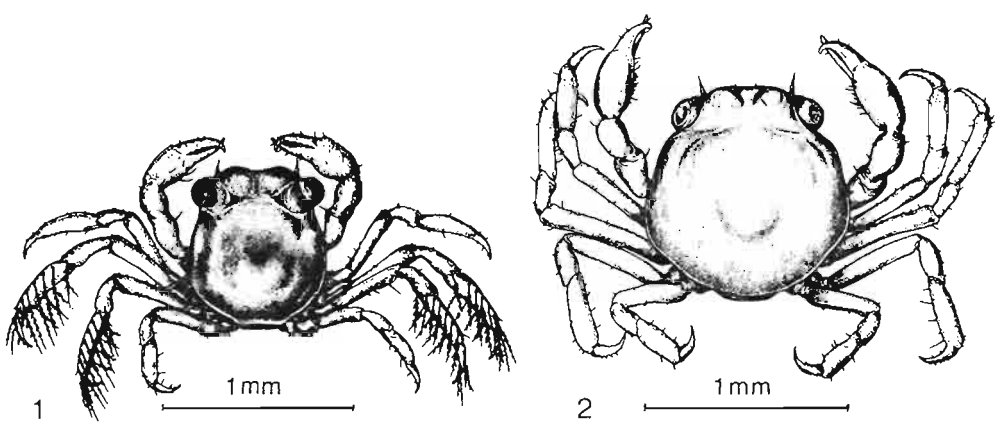

Fig. 13-181: *Pinnotheres pisum* from *Spisula solida*. 1: First crab stage; 2: pre-hard stage. (After Møller Christensen, 1959.)

Mer (France). The authors concluded that host change is not an essential step in the life cycle of *P. pisum*.

'Pinnotheres pisum', reported from outside Europe, represent other species of that genus. In New Zealand waters, for instance, records of *'P. pisum'* comprise *P. novaezelandiae* and, less frequently, *P. schauinslandi* (Scott, 1961; Bennett, 1964).

On the North and South American Atlantic coasts, *Pinnotheres maculatus* associates with at least 14 host species, 10 of which are bivalves including *Mytilus edulis, Mya arenaria, Argopecten irradians concentricus, A. gibbus, Placopecten magellanicus* and *Modiolus modiolus* (McDermott, 1961; Wells and co-authors, 1964; A. B. Williams, 1965; Broom, 1976). Aspects of the biology of *P. maculatus* have been studied by Rathbun (1918), Hyman (1924), Welsh (1932), Sastry and Menzel (1962), Pearce (1964), Costlow and Bookhout (1966), Eidemiller (1970), Kruczynski (1973) and Derby and Atema (1980). From this information, the life cycle of *P. maculatus* may be summarized as follows: Ovigerous females occur from late May to mid-June. The eggs hatch in August, giving rise to 5 subsequent zoeal stages followed by the megalops; all are planktonic. During mid-September, the megalops metamorphoses into the (invasive) first true crab stage. After several moults within the host, accompanied by size increase, a hard-crab instar results. At this stage, both sexes leave the mollusc to engage in copulatory swarming in the open water. After this, the females re-invade molluscs and undergo 4 post-hard moults until they attain the Stage-V (adult) form. At all post-hard stages the females' carapaces are soft and poorly calcified. Hence, the life cycle of *P. maculatus* resembles that of *P. pisum*, except that it involves a fifth zoeal stage and that the hard-stage crab does not change hosts.

The second common New World pinnotherid species is *Pinnotheres ostreum*. The 'oyster crab', as it is called, occurs primarily in the mantle cavity of *Crassostrea virginica* but has also been recorded from other bivalves, as well as from tubes of the annelid *Chaetopterus variopedatus*. Aspects of its biology and distribution have been studied by Birge (1882), Rathbun (1918), Hyman (1924), Stauber (1945), Sandoz and Hopkins (1947), Flower and McDermott (1952), Hopkins (1956d), Møller Christensen and McDermott (1958), Haven (1959), Gray (1961), McDermott (1961, 1962), Wells (1961), Beach (1969), Sandifer and Van Engel (1970), Sandifer (1972) and others. Stauber (1945) believed that *P. ostreum* invades oysters at the hard-shelled stage, but Møller Christensen and McDermott (1958), who elucidated the full life cycle of the oyster crab, showed that, as in the other pinnotherid species, the first-crab instar is the invasive stage. The hard-shelled crabs of both sexes do not engage in copulatory swimming. Instead, only the males leave the oyster in search for females in another host. Males do not develop beyond the hard stage measuring 1.4 to 4.6 mm in carapace width, but disappear after copulation with 1 or more females. The latter undergo several moults within the mantle cavity to reach sexual maturity at Stage V. Mature females have a carapace width of 4.4 to 15.1 mm.

Invasion of *Crassostrea virginica* by first-crab-stage individuals of *Pinnotheres ostreum* takes place in mid-August. The crabs prefer 0-group oysters (76.6 % infestation) over yearlings (54.6 %) and older ones (21.5 %), but survival is higher in larger (older) hosts, as indicated by the subsequent occurrence of hard or post-hard crabs in the 3 groups (3.1, 47.3 and 90.0 %, respectively) (Møller Christensen and McDermott, 1958). Up until the hard stage, young *P. ostreum* apparently utilize a wider variety of hosts than do adult females. In *Mytilus edulis*, oyster crabs presumably develop only to the hard swimming

stage. The crabs leave the mussels during the late fall, probably in search of some other host. Mature females have never been seen in mussels but may be found in jingle shells *Anomia simplex* (McDermott, 1961, 1962; Sandifer and Van Engel, 1970; Sandifer, 1972).

Pinnotheres hickmani, previously described as *Fabia hickmani,* is the most abundant pinnotherid presently known in Australia. Its life cycle involves *Mytilus planulatus* as host. Although pre-hard, hard and post-hard females, as well as hard males occur in the mussel, no pre-hard males were found. Hence, the existence of an additional host can, at present, not be ruled out (Guiler, 1950; Pregenzer, 1978, 1979a, b).

Among the other members of the family Pinnotheridae with a known life history, the North American west coast species *Fabia subquadrata* has a cycle similar to that of *Pinnotheres maculatus.* Its main molluscan host is *Modiolus modiolus* although it may occasionally occur in *Mytilus edulis, M. californianus* and *Saxidomus* sp. (Pearce, 1966a). In contrast, the life cycles and reproductive biology of *Pinnixa faba* and *P. littoralis* differ markedly from the previously described pinnotherid cycles. Both do not have an 'anomalous', free-swimming instar midway in their developmental cycle, and both have 2 annual reproductive periods, one lasting from late spring until summer and the second from winter to early spring. The crabs invade their host, *Tresus capax,* in pairs, one female and one male maturing in anyone clam. Occasionally, 1 to 5 immature crabs, ordinarily of the same species as the adults, accompany the pair (Pearce, 1966b). Occurrence in pairs, invariably consisting of one female and one male, has also been reported for *Fabia tellinae.* Its host is *Tellina magna* in the Gulf of Mexico (S. P. Cobb, 1973).

The incidence of pinnotherid infestation in bivalves may be very high locally. Up to 92 % of the *Crassostrea virginica* from Delaware Bay (USA) were found to be parasitized by the hard-shelled stage (erroneously designated 'Stage-I crabs') of *Pinnotheres ostreum,* and up to 33.3 % harboured Stage-V individuals (mature females). One oyster carried as many as 262 hard-shelled crabs (Stauber, 1945). In Virginia waters, prevalences of *P. ostreum* in *C. virginica* varied locally from less than 1 % to over 80 %, with an average of 30 to 40 % (Sandoz and Hopkins, 1947). Pearce (1964) reported 97.6 % of 1,820 *Mytilus edulis,* collected over a 13-month period near Woods Hole, Massachusetts (USA), to harbour *P. maculatus.* Populations of *Tresus capax* from Puget Sound, Washington, were 100 % infested with *Pinnixa faba* and *P. littoralis* (Pearce, 1966b).

Of 80 window-pane oysters *Placuna placenta* from Kakinada Bay on the east coast of India, 63 (78.8 %) harboured a single individual of *Pinnotheres* sp., and 1 oyster had 2 crabs (Bhavanarayana and Devi, 1974). *P. placenta* has not previously been reported as host for pinnotherids. Single adult (mostly female) *Pinnotheres villosulus* occurred in 85 (67.5 %) of 126 *Pinctada maxima* from Torres Straits, North Queensland, Australia (Dix, 1973). *Mytilus edulis* from Botany Bay, New South Wales (Australia), had an approximately 50 % infestation of *P. hickmani* (Pregenzer, 1979a). Lower incidences were recorded for *P. novaezelandiae* in green-lipped mussels *Perna canaliculus* from New Zealand. Of 4,625 mussels collected in 94 monthly samples from 7 sites, between 0 and 12 % harboured crabs (Hickman, 1978). Up to 76 % of the *Tapes philippinarum* from Fukuoka (Japan) have been found to harbour *Pinnotheres latissimus.* Usually, 1 or 2 crabs occurred in a single clam, but up to 34 individuals have been counted. As a rule, only one was a mature female, while the remainder were all small-sized, hard-shelled males (Ohshima, 1927, 1937).

Prevalences of considerable variation, but frequently of up to 100 %, have also been reported for *Pinnotheres pisum* in *Mytilus edulis* from British waters (Houghton, 1963; Seed, 1969b). *Cardium edule* from Southampton were about 50 % infested with this species. Most of the crabs were males (max. 48.6 %), while max. 10 % were females. Males were most numerous in July and females in February (Barnes, 1973).

There appears to be a definite correlation between host size and the size of its pinnotherid inhabitant, larger mussels harbouring larger crabs. Such a relationship has been demonstrated for *Mytilus edulis – Pinnotheres pisum, Crassostrea virginica – P. ostreum* and most of the other associations (Atkins, 1926; Møller Christensen and McDermott, 1958; Houghton, 1963; Seed, 1969b). Also, larger hosts are usually more frequently infested than smaller individuals. Within the mantle cavity, the crabs normally lodge in the widest portion, which indicates that the available space is the limiting factor (Pearce, 1966a). In mussels from Langstone Harbour, Portsmouth (England), the percentage infestations in the size groups 30 to 90 mm were 0, 4.3, 9.4, 5.1, 13.5, 23.9 and 57.1 %, respectively (Houghton, 1963).

Usually, bivalves from greater depths are more heavily infested with pinnotherids than individuals from shallower water. Of 550 *Mytilus edulis* collected from 6 intertidal sampling stations in the vicinity of Woods Hole, Massachusetts, only 2 contained *Pinnotheres maculatus,* whereas 421 mussels from 2 subtidal stations yielded 238 specimens. Only 1 % of the mussels growing near the surface on a piling harboured crabs, while those from 10 m depth were 40 % infested (Kruczynski, 1974). Houghton (1963) and Seed (1969b) observed a similar correlation between tidal height and *P. pisum* infestation in *M. edulis* from British waters. Longer submersion times of mussels living deeper on the shore may increase the crabs' chance to find a host. On the other hand, extended periods of valve closure, associated with cessation of feeding and concomitant anaerobic host respiration in mussels occurring at higher tidal levels may interfere with survival of crabs in such hosts.

In the subtidal zone, other factors such as prevalence of invasive first-crab individuals and hard-shelled crabs may determine the pattern of infestation. Thus, Pearce (1966a) found *Modiolus modiolus,* taken at 30 to 60 m depth in waters of the San Juan Archipelago, Washington (USA), to be consistently more than 80 % infested with *Fabia subquadrata,* while horse mussels from 200 m or greater depths rarely had more than 2 % and frequently less than 1 % infestations.

With the unique exception of *Pinnixa faba* and *P. littoralis,* none of the bivalve-invading pinnotherids appear to be host-specific, at least not during their entire life. Host specificity, if it occurs, is most pronounced in the adult female. Males, and in particular immature first-stage instars and hard-shelled crabs, are mostly less host-dependent. Some, like *Fabia subquadrata,* associate with a number of bivalve species, as well as with ascidians, but can also live independently without a host (Wells, 1928, 1940). Many pinnotherids have been taken from half a dozen or more different hosts, sometimes representing members of different invertebrate phyla (Møller Christensen and McDermott, 1958). There are 14 known species for *Pinnotheres maculatus.* Of these, 10 are bivalves (A. B. Williams, 1965). *P. pisum* has mostly been reported from *Mytilus edulis,* but may also be found in *Modiolus, Ostrea, Cardium, Parvicardium* and *Tapes.* Although Barnes (1973) recovered *P. pisum* from approximately 50 % of *C. edule* from Southampton (England) waters, he did not specifically report it from sympatric individuals of *C. lamarcki.*

Whether these findings constitute a case of host-symbiote incompatibility, is doubtful. They probably reflect differences in the ecology of the 2 congeneric cockles. *Cardium lamarcki* has weaker ciliary currents and, hence, filters lesser amounts of water than *C. edule* (Lauckner, 1972). It also normally occurs higher on the shore and, as a rule, pinnotherid infestation decreases with height above low-water mark in relation to the duration of shell closure during air exposure. Seed (1969b, 1976) reported high prevalences (up to 100 % in large hosts) of *Pinnotheres pisum* in *Mytilus edulis* from British waters but found sympatric 'Padstow-type' mussels (= *M. galloprovincialis*) to be only occasionally and lightly infested. Again, subtle differences in host ecology and behaviour appear to be factors determining the presence or absence of *P. pisum,* since Berner (1952) found Mediterranean *M. galloprovincialis* to be abundantly infested with *P. pisum.* The importance of microecological factors certainly accounts for Berner's observation that previously uninfested mussels from the lagoon Étang de Thau became almost invariably parasitized upon transplantation into waters of the Gulf of Marseille. Experiments conducted by Johnson (1952), Sastry and Menzel (1962) and Derby and Atema (1980) demonstrated that pinnotherids are chemotactically attracted by compatible hosts.

With respect to host specificity, *Pinnixa faba* and *P. littoralis* constitute a remarkable exception. Both species inhabit the mantle cavity of *Tresus capax.* In Puget Sound, Washington (USA) waters, 100 % of the available hosts were found infested. This suggests that host-population size is the factor limiting the population size of *Pinnixa.* None of the *T. nuttalli* living side by side with *T. capax* were found to harbour *P. faba* or *P. littoralis,* which indicates strict host specificity (Pearce, 1966b).

There is considerable controversy as to whether pinnotherids are commensals or parasites. In her monograph, Rathbun (1918) considered the majority of these crabs as commensals. Hopkins (1957a) characterizes the Pinnotheridae as "bordering on parasitism". He (p. 414) also stated that

> "it might be supposed that an organism which robs its host of nourishment must be harmful in some degree, even if the host shows no apparent effect, but under favorable conditions it is probably not difficult for the host to compensate or even overcompensate for the loss by ingesting more food. Under conditions of food scarcity the same parasite might become harmful".

Modern workers tend to regard at least some of the better-known pinnotherids as parasites in the broadest sense of being 'associates that do harm to their hosts'. According to Cheng (1967, p. 315), "the nature of the relationship between pinnotherid crabs and their hosts is at best uncertain".

Coupin (1894, 1895) found that *Pinnotheres pisum* feeds on phytoplankton collected by its host, thereby refuting the previous assumption that the crab selectively diverts the zooplankton items from the material retained by the gills while leaving the plant fraction to the mollusc. He regarded *P. pisum* as a true parasite. Orton (1920), who observed the crabs' manner of feeding through 'windows' cut into the valves of infested oysters, confirmed Coupin's findings. Stauber (1945) described an identical feeding behaviour for *P. ostreum* living in the mantle cavity of *Crassostrea virginica,* and Pearce (1966a) observed *Fabia subquadrata* to feed on mucous food strings picked from the ctenidial food grooves of its host, *Modiolus modiolus.* S. A. Campbell (1969) extracted plant pigments from the gut contents of *P. pisum* living in the mantle cavity of *Mytilus edulis,* which indicates ingestion of algae. *P. maculatus* accumulates radioactivity when present in

Argopecten irradians and *A. gibbus* fed *Nitzschia closterium* labelled with ^{14}C and in *Mytilus edulis* fed labelled *Thalassiosira pseudonana*. Between 28 and 40 % of the initial radioactivity were recovered from hosts containing clawed crabs, and between 2 and 7 % were taken up by the crabs, presumably via host-produced food strings. On the other hand, clawed *P. maculatus* kept in finger bowls outside the host accumulated 21 % of the initial radioactivity, which suggests that the crabs are capable of straining food algae directly from suspension. Gut analyses confirmed that the crustaceans ingested diatoms. No radioactivity above background was found in crabs with amputated chelae from both bivalve hosts and finger bowls (Kruczynski, 1975).

Although these experiments did not clearly show whether *Pinnotheres maculatus* obtains its food from the host's food strings, from faeces or pseudofaeces, or directly from suspended phytoplankton, there was no indication that the crabs feed on host tissues. There are also no observations suggesting that any other pinnotherid feeds directly on host tissues. Therefore, these crustaceans are definitely not parasites in the strict sense. On the other hand, their mere presence within the mantle cavity of bivalves may inflict a variety of functional and morphological host responses ranging from slight irritation to severe structural alterations and manifest pathology.

Functional alterations include a reduction in the filtering capacity of crab-containing bivalves. Although Kruczynski (1975) stated that *Argopecten irradians concentricus, A. gibbus* and *Mytilus edulis*, experimentally infested with *Pinnotheres maculatus*, resume normal filtration rates 3 to 5 days after implantation of crabs into the mantle cavity, Pregenzer (1979a) demonstrated reduced neutral red clearance rates in *M. edulis* harbouring *P. hickmani*.

Impairment of filtration activity may be due, in part, to the constant mechanical irritation of the gill cilia by the body of the crab, which leads to temporary cessation of ciliary activity. Also, the laminar water flow across the gill membranes is certainly massively disturbed by the presence of such large 'foreign bodies' in the mantle cavity. The major impairment, however, seems to result from extensive destruction, by the pinnotherids, of large areas of gill tissue.

Using Orton's (1920) method of cutting 'windows' into one of the valves, Pearce (1966a) studied the feeding behaviour of *Fabia subquadrata* (previously known as *Pinnotheres concharum*) in *Modiolus modiolus*. The adult female occupies the anterior half of the mussel's mantle cavity, with its abdomen placed against a pair of demibranchs. This position is maintained by the insertion of the dactyls of the pereiopods into the gill filaments and/or mantle tissues. Prolonged maintenance of the feeding position results in extensive ctenidial erosion of the entire gill portion underlying the crab. This portion is eventually destroyed. Damage to the palps consisting of size reduction and deformation is also seen. The lesions appear to be caused mainly by the constant contact of the chelae against the gills and palps, and to a lesser extent by the action of the pereiopod dactyls. Once the entire underlying gill has been eroded away, the crab attaches to the mantle, which may result in the formation of blisters or cyst-like protuberances. The latter anomaly was found in 55 % of the mussels harbouring Stage-V crabs. Damage caused by the much smaller Stage-I form, which move about more extensively within the mantle cavity, is not restricted to the area beneath the crab but is found along the entire gill margin.

Gill and palp lesions similar to those described by Pearce (1966a) have been observed in *Crassostrea virginica* harbouring *Pinnotheres ostreum* or *P. maculatus*, in *Meretrix casta*

containing *Pinnotheres* sp. and in *Pinctada maxima* infested with *P. villosulus* (Stauber, 1945; Sandoz and Hopkins, 1947; McDermott, 1961; Silas and Alagarswami, 1967; Dix, 1973). Although Stauber (1945) and Pearce (1966a) demonstrated that not only the large Stage-V crabs but also the much smaller Stage-I individuals can inflict severe damage on the molluscan gills, Møller Christensen and McDermott (1958) concluded that *P. ostreum* exerts no (visible) influence on oysters in its first year of life but that it probably does so in many cases in its second and third years. Of 1,502 crab-bearing *C. virginica* examined by these authors, about 50 % had light, 40 % had moderate and 9 % had heavy gill damage, whereas only about 1 % had no discernibly diseased gills.

Gill damage and palp erosion have also been observed in *Mytilus edulis* harbouring *Pinnotheres pisum* (Seed, 1969b). Up to 44 % of the mussels examined by Atkins (1931a) had abnormal gills, but deviations from normal ctenidial morphology also occurred in individuals devoid of pea crabs. Wright (1917) found adult female *P. pisum* to exert considerable pressure upon portions of the mantle lobes and on the shell beneath. In some instances, this pressure had apparently caused the nacreous shell layer to become dissolved away. Rapid wound healing and regeneration of gill tissue was believed to keep pace with *P. ostreum*-caused lesions in *Crassostrea virginica* if crab numbers were not exceedingly high (Stauber, 1945). Atkins (1931c) pointed out that *M. edulis* could not invariably repair gill damage, but that a narrow deep gap in the gills was always filled more rapidly than a broad shallow one. *Pinnotheres*-caused lesions are predominantly of the latter type.

Stauber (1945) believed that high prevalences of *Pinnotheres ostreum* in populations of *Crassostrea virginica* have contributed to unusually high oyster mortalities in Chesapeake Bay in the winter of 1941/42. Sandoz and Hopkins (1947) were unable to trace deaths of *C. virginica* to *P. ostreum* invasion in Virginia although they stated that the presence of crabs in the mantle cavity tended to keep oysters in relatively poor condition. Overcash (1946) and Haven (1959) found the condition indices of oysters with crabs to drop to 82.3 or 71.7 %, respectively. Poor condition of oysters from Chesapeake Bay, infested with *P. ostreum*, made spawning and stripping of sexual products difficult. Progeny of these oysters also acquired serious pea crab infestations and, although early development appeared normal, stunted growth was apparent in infested oysters at 2 years of age. Retarded oysters frequently harboured large crabs (Andrews and co-authors, 1968).

Infestation with *Pinnotheres maculatus* was found to cause similar stunting in *Argopecten irradians concentricus*. Bay scallops harbouring adult female pea crabs were initially slightly smaller and tended to weigh less than non-infested ones. Mean tissue-dry weights per shell height were significantly lower ($p < 0.05$) for infested individuals. Growth measurements conducted on 3 size groups of caged scallops over a 3-month period revealed that, in the small and medium-sized groups, infested individuals grew significantly less ($p < 0.05$) than healthy ones. No such difference was apparent in the 2 groups of large scallops (Kruczynski, 1972; Fig. 13-182).

Emaciation is also typical of *Pinnotheres pisum*-infested *Mytilus edulis* (Berner, 1952; Hancock, 1965). It is most pronounced in larger mussels, the mean tissue-dry weight of 9.5 cm long individuals being only 81.6 % of that of non-infested mussels (Seed, 1969b). In contrast, Wright (1917) stated that pea crabs never, or at the most very rarely, occur in poorly nourished mussels. But, as the individuals inspected by him came from areas where the mussels were well nourished and making rapid growth, the great food availability may

have enabled the parasitized individuals to compensate for the *Pinnotheres*-caused energy loss.

Concomitantly with the tissue-dry weight loss of *Pinnotheres pisum*-infested *Mytilus edulis*, Seed (1969b) observed an average 6.5 % increase in shell-dry weight. Konishi (1977) found that the shells of *Mytilus coruscus* (= *M. crassitesta*) from Usu, Hokkaido (Japan), infested with *P. sinensis*, were considerably thicker than those of crab-free individuals, the mean ratios of thickness to length being 0.402 and 0.340, respectively. Whether there is a concomitant increase in absolute shell weight, has not been reported.

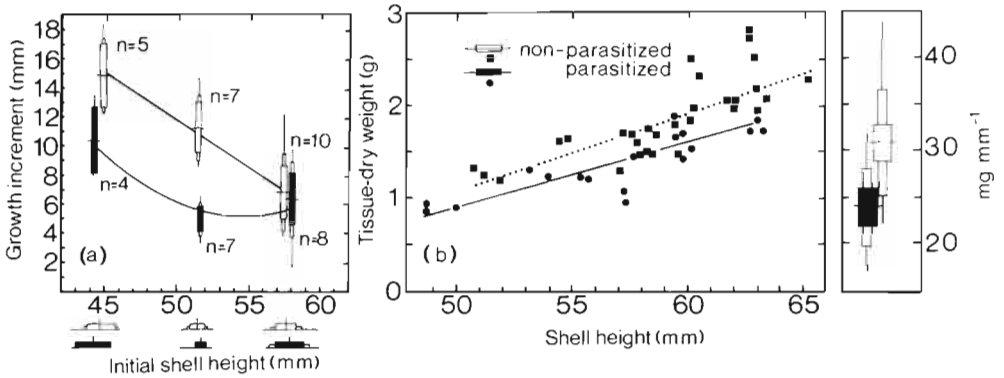

Fig. 13-182: *Argopecten irradians concentricus*. Effect of *Pinnotheres maculatus* on shell growth and body weight. (a) Shell growth as a function of initial shell height in 3 size groups of parasitized and non-parasitized scallops; (b) tissue-dry weight in relation to shell height and parasitization. Bar diagrams: horizontal line = mean; wide rectangles = standard errors; narrow rectangles = standard deviation; vertical line = range. (After Kruczynski, 1972.)

Berner (1952) accused food deprivation, brought about by the presence of large (10 to 13 mm) females of *Pinnotheres pisum* within the mantle cavity of French-coast *Mytilus edulis*, of causing a delay in gonad maturation in the host. Seed (1969b) did not notice a similar effect in British mussels, but Kruczynski (1972) suspected that depressions in the gonads of *Argopecten irradians concentricus* from North Carolina, presumably produced by mechanical pressure exerted upon the reproductive tissues by large female *P. maculatus*, may cause a decreased production of eggs or sperm. Gonad depression was reported from *Pinnotheres*-infested Indian *Meretrix casta* by Silas and Alagarswami (1967), and Awati and Rai (1931) found the presence of pea crabs in oysters to induce sex change toward maleness. Of 794 uninfested Indian *Saccostrea cucullata*, 41.7 % were males, 56.4 % were females, and 2.9 % hermaphrodites. In contrast, 82.6 % of the oysters harbouring *Pinnotheres* sp. were males, 10.4 % were females, and 7.0 % were hermaphrodites. Since female oysters can be induced experimentally to change sex by mere starvation, the authors concluded that the change in the sex ratio in *S. cucullata* may be the result of reduced food intake by crab-infested oysters. As shown by Amemiya (1929) and Coe (1934), the sex ratio in oysters is definitely influenced by environmental conditions. Excision of gill tissue from *Crassostrea gigas* causes growth-rate decrease and an increase in the proportion of males (Amemiya, 1935; Egami, 1953). This supports Awati and Rai's (1931) contention that the change in sex ratio in *Pinnotheres*-infested *S. cucullata* is

caused by food deprivation rather than by chemical parasite–host interaction. Drastic changes in the sex ratio of oysters, such as those reported by Awati and Rai (1931), may possibly affect oyster recruitment.

Apparently, pinnotherid inquilinism does not interfere with infestation of bivalve hosts by other arthropod parasites. Of the *Tapes philippinarum* from Fukuoka (Japan), inspected by Ohshima (1937), 76 % harboured up to 7 individuals of *Pinnotheres latissimus,* and 33 % of the clams were simultaneously infested with pycnogonids *Nymphonella tapetis.* Green-lipped mussels *Perna canaliculus* have been reported as hosts for *P. novaezelandiae* and larval trematodes *Cercaria haswelli* (Hickman, 1978). It was not stated whether both parasites are mutually exclusive.

Hyperparasitization of bivalve-associated pinnotherids has been reported on several occasions. The fungus *Leptolegnia marina,* a member of the Saprolegniaceae, was found to infect *Pinnotheres pisum* living in the mantle cavity of *Mytilus edulis* from Padstow, England. The mycelium is normally found in the gills but may also penetrate deeply into the body of the pea crab, surrounding the organs and occasionally extending into the appendages, mouth parts and even the eyestalks. The presence of *L. marina* is mostly indicated some days before death of the crab either by opaque white patches showing through the exoskeleton, or more rarely by opaqueness of the gills, though *P. pisum* may die of the disease without external signs. Eggs and embryos were also affected. The agent was believed to be identical with the fungus producing 'foot disease' in *Barnea candida* and *Cardium echinatum* (Atkins, 1929, 1954a; see section 'Agents: Fungi'). In addition to *L. marina,* 2 other fungi are known to infect the egg-masses of *P. pisum.* These are *Pythium thalassium* (Peronosporales, Pythiaceae) and *Plectospira dubia,* a presumed saprolegniacean (Atkins, 1954b; 1955b). The latter has subsequently been included in the newly created family Haliphthoraceae (Saprolegniales) as *Atkinsiella dubia* (Vishniac, 1958).

Coccidians, named *Aggregata coelomica,* frequently occur in *Pinnotheres pisum* infesting *Mytilus edulis* on the French coast of the English Channel. Léger (1901), who mistook them for gregarines, saw different developmental stages of the sporozoan within the intestinal lumen, in connective tissues underlying the intestine, and in the body cavity. *A. coelomica* does not appear to cause gross pathological changes in the crab host. Ryder (1883) and Beach (1969) found individuals of *Pinnotheres ostreum,* living in the mantle cavity of *Crassostrea virginica,* to support colonies of vorticellid ciliates *Zoothamnium* sp. on their carapace and legs.

Females of *Pinnotheres ostreum* inhabiting the mantle cavity of *Crassostrea virginica* may harbour, in their branchial chambers, unidentified parasitic nemerteans. Of 125 crabs obtained from oysters at Beaufort, North Carolina, 33 were hyperparasitized. The crab population consisted of 86 mature females with 5.5 to 13.6 mm carapace width and 39 juveniles, 7 of which were males. Thirty-two (37.2 %) of the mature females were infested, while only 1 immature female harboured a single immature worm. Over 50 % of the infested crabs had nemerteans in both chambers, with a maximum of 21 worms in one crab, 9 of which were mature red females. Large numbers of egg sacs, cemented to the pleopods of the ovigerous crabs, were produced under these conditions. A single female nemertean in a gill chamber of *P. ostreum* causes comparatively little disturbance but heavy infestations, in which all of the gills may be covered with the worms and their sheaths, result in deformed and sometimes abortive gills. The latter may be due to an interference with normal moulting. No doubt gas exchange is impaired to some extent in

heavily affected gills (McDermott, 1967). The unidentified nemertean has previously been recorded from *Pinnixa chaetopterana* (McDermott, 1966). From external examination, worms taken from *P. ostreum* resembled *Carcinonemertes* (Beach, 1969).

Pinnotheres pisum infesting *Mytilus edulis* is frequently hyperparasitized by entoniscid isopods *Pinnotherion vermiforme*. The presence of adult female isopods in the crabs causes partial to almost complete atrophy of the gonad. Infested males are distinctly larger than uninfested individuals and exhibit changes of secondary sex characters toward femaleness. *P. pisum* from mussels in the Camel estuary, Padstow (England), were 27.7 % hyperparasitized, and of crabs from Luc-sur-Mer on the French coast of the English Channel, about 5 % had *P. vermiforme* infestations (Giard and Bonnier, 1889; Mercier and Poisson, 1929; Atkins, 1933b).

Pinnaxodes mutuensis occurring in *Modiolus modiolus difficilis* from Usu, Hokkaido (Japan), were found to be heavily hyperparasitized by rhizocephalans *Sacculina* sp. or epicaridean isopods. Four out of 5 males, which carried a *Sacculina* infestation, were larger than normal males and showed external signs of feminization (Konishi, 1977). Similar parasite-induced morphological changes have also been observed in *Pinnotheres sinensis* and *P. cyclinus* infesting *Crassostrea gigas* and *Barbatia obtusoides*, respectively (Semitu, 1944; Suzuki, 1967).

The utilizability of anyone of these hyperparasites as a possible biological control agent for bivalve-invading pinnotherids has, apparently, not yet been tested. Loosanoff (1961, 1965) casually reported control of *Pinnotheres ostreum* on oyster beds treated with Sevin® and chlorinated benzenes, and suggested use of this insecticide for pea crab control. Following this proposal, Andrews and co-authors (1968) showed that oysters could be freed from pea crabs by exposing them to 10 mg l^{-1} technical Sevin® for 24 h. In one experiment, crabs were even killed with 1 mg l^{-1} Sevin dissolved in acetone. Oysters returned to the beds after treatment exhibited no differences from controls in growth and mortality during the subsequent year. No consideration was given to assaying oyster tissue for Sevin or its derivates since the method was intended for experimental purposes only.

Agents: Pantopoda

The sea spiders (Pantopoda or Pycnogonida) comprise a small class of arthropods with some 500 exclusively marine species, most of which associate with, and feed on, cnidarians. A nearly complete bibliography of pycnogonid literature is to be found in the papers of Helfer and Schlottke (1935), Hedgpeth (1947) and Stock (1956). King (1973) assembled a list of records of associations of larval and adult sea spiders with other invertebrates. The feeding mechanisms of pycnogonids have been studied in detail by Fry (1965). For general information on the class consult also King (1973, 1974), for pantopod-mollusc associations André and Lamy (1938). Adult pycnogonids are generally free-living. At least two larval forms are known to parasitize bivalves.

During routine examination of 144 individuals of *Tapes philippinarum*, collected from the shores of Fukuoka (Japan) in April 1926, 51 (35.4 %) of them were found to harbour, in the mantle cavity, immature pycnogonids in various stages of development. In total, 99 specimens were recovered. A second lot of 80 *T. philippinarum*, collected in August, yielded 8 very young stages in 6 hosts. The number of larvae per clam varied mostly from 1

to 3, but there were 5 parasites in 4 cases and 7 in another case. The young sea spiders were found to attach to the gills, visceral mass and mantle of *T. philippinarum* by means of their 3 anterior pairs of appendages, which are armed with chelae. The proboscis was seen to be inserted into the tissues of the host, which was highly suggestive of feeding. The largest individual measured about 7 mm in length, including the 2 mm long proboscis. The juvenile pantopods were believed to represent a new species and were named *Nymphonella tapetis* (Ohshima, 1927). It must be emphasized that identification of immature Pantopoda to species level is extremely difficult if not impossible.

Subsequently, however, free-living adult females and egg-carrying males of *Nymphonella tapetis* were recovered from sandy substrates off Fukuoka and Kyushu (Japan). Larvae were most common in *Tapes philippinarum* and less common in *Protothaca* (*Venus*) *jedoensis* from the same localities in late March until the beginning of June, infestation incidences approaching 45 %. Lesser infestations occurred in a second period, lasting from August to September. Life-cycle studies and studies of adult morphology confirmed *N. tapetis* being a new species. How the bivalves become infested with the eggs or early larvae could not be determined, and nothing has been reported on host pathology (Ohshima, 1933, 1935, 1937). LeCalvez (1950) reported free-living adult pantopods, believed to represent *N. tapetis*, from Banyuls, French Mediterranean coast. *N. lambertensis*, described from South African waters, is very closely related to *N. tapetis* and possibly deserves only subspecies rank (Stock, 1959). Juveniles invading bivalves have not been reported in these studies.

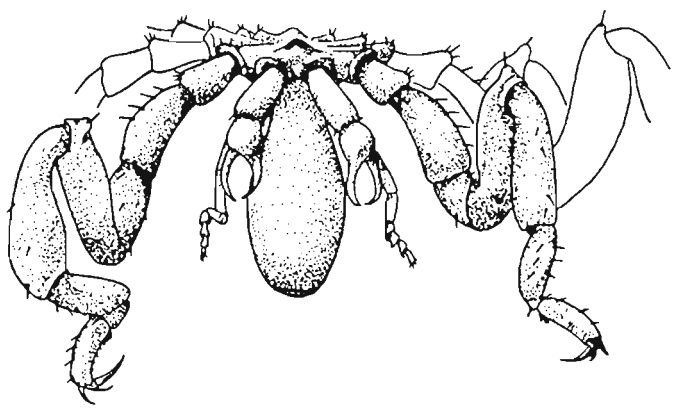

Fig. 13-183: *Achelia chelata*. Front view of adult, × 25. (After Hedgpeth, 1940.)

Mytilus californianus has been identified as host for immature and mature stages of *Achelia chelata* (Fig. 13-183). Sixteen of 32 mussels from Duxbury Reef, Marin County (California), harboured a total of 89 pantopods of both sexes, with numbers per host ranging from 1 to 21. Obvious destruction of the mussel's gill and gonadal tissues was evident. Damage to the visceral mass, foot and palps was also apparent in several hosts. In the individual exhibiting the greatest amount of damage, there was a complete loss of ctenidia and the major portion of mantle and gonadal tissue, accompanied by apparent atrophy of the entire animal (Benson and Chivers, 1960). *Achelia chelata* has repeatedly (also as *Ammothea euchelata*) been reported from California (Hilton, 1939; Hedgpeth,

1940, 1941; Ziegler, 1960), but has only been found to parasitize mussels just south of the Golden Gate in San Francisco and at Duxbury Reef in Marin County (Hedgpeth in Ricketts and Calvin, 1968).

Pantopod-lamellibranch associations may not be as rare as may be deduced from the sparse literature records. Linné (1767) was the first to cite a parasitic relationship between *Mytilus edulis* and *Phalangium grossipes.* Calman (1929) identified Linné's specimen as belonging to the common species *Phoxichilidium femoratum,* which normally associates with cnidarians (Helfer and Schlottke, 1935; Hedgpeth, 1948; Ziegler, 1960; King, 1973, 1974). Although some pantopods may display highly specific and different food preferences (Fry, 1965), others may not. In the laboratory, several specialized pycnogonids could not be maintained on food other than Hydrozoa. *Anoplodactylus petiolatus,* on the other hand, accepted small polychaetes and copepods as food and could be maintained on small pieces of *Mytilus edulis* meat for 9 months under laboratory conditions (Lotz, 1968).

ABNORMALITIES

Although bivalves are known for their large capacity of tissue repair, numerous structural abnormalities of both tissues and shell have been reported from this invertebrate group. Many of these lesions, which are the result of parasitic or microbial attack, have been described in the preceding sections of this chapter.

Various structural abnormalities in bivalves are known or suspected to be produced by environmental pollutants, such as heavy metals, crude oil, benzo[α]pyrene, nitroso compounds, organochlorides, and other chemicals (Sparks, 1972; Gardner and co-authors, 1975; Fries and Tripp, 1976; Hodgins and co-authors, 1977; Gardner, 1978; Khudoley and Syrenko, 1978; Sindermann, 1979a, b, 1980; Yevich and Barszcz, 1980; Murchelano and co-authors, 1980; consult these authors for further references). Being essentially non-biotic diseases, pollution-associated abnormalities will not be discussed here in detail.

Deviations from normal tissue structure may be classified grossly as hypertrophy, hyperplasia, metaplasia or neoplasia (for definition of terms see Vol. I, Chapter 2). However, distinction between these types of abnormalities occurring in molluscs is frequently difficult and sometimes impossible (Scharrer and Lochhead, 1950; Pauley, 1969; Sparks, 1969, 1972; Stewart, 1977).

Structural abnormalities in bivalves may involve both the shell and the soft parts. Malformations of the shell result from external mechanical impact or — more frequently — from disturbances of the proper function of the mantle, which secretes the shell material. Constant tissue irritation by ectoparasites may lead to recession of the mantle margin which, in turn, results in shell lesions and deformities (Fig. 13-161). Several types of shell abnormalities are produced by larval trematodes present in the extrapallial space or in the tissues of adductor muscles and siphons (see section 'Agents: Trematoda'). Beyond doubt, these ubiquitous metazoan bivalve parasites have been overlooked by many workers.

A gross deformity in the right valve of an individual of *Mya arenaria* from Marsh Island, Maine (USA), was diagnosed to result from mechanical injury during early life of the clam. It consisted of a tube-like fold of shell material, broadening toward, and projecting about 7 mm beyond, the valve margin (Morse, 1923). Another type of shell

abnormality was observed in soft-shell clams from Belfast Lough (Northern Ireland). It consisted of raised blisters, formed in the region of the central extrapallial space, and opening toward the posterior end of the valves. The anomaly occurred in 12 of 100 *M. arenaria*, and another dozen showed slight traces of the malformation. In the apparent absence of any macroscopic parasites, such as burrowing sponges or blister worms, the lesions are highly suggestive of massive invasion of the extrapallial space by trematode metacercariae. Fisher (1932), however, discussed the possibility of the existence of a distinct race of *M. arenaria* in that area.

Valve asymmetry is commonly observed in normally equivalve pelecypods, such as *Mytilus edulis*, *Barnea candida* and *Solen marginatus* (Pelseneer, 1920, 1923). A curiously shaped specimen of *Mya arenaria* with grossly asymmetrical valves has been described by Blake (1929). The author did not attempt to analyze the cause of this deformity but stated (p. 90):

> "One can easily contemplate the volition of some enthusiastic paleontologist who found such a shell in a fossil state, and it suggests a lesson to the hasty so-called species maker."

In fact, distorted or malformed bivalve shells have frequently puzzled the earlier conchologists. Among the *Cardium* spp. shell collections in the French Muséum National d'Histoire Naturelle, Paris, for instance, there are numerous malformed cockle shells, some of which have been described as varieties of *Cardium edule* or even as separate species. As indicated by the type material deposited by Chavan (1945) in the Paris Museum collections, many of the 5 subspecies and 46 varieties of *C. edule* described by him are actually pathologically modified shells. Some of these have distinctly been damaged mechanically and show various degrees of shell repair; others have probably been modified by trematode infestation (Lauckner, 1972).

Shuster (1966) described a 'uniquely shaped quahaug' *Mercenaria mercenaria* from Narragansett Bay, Rhode Island (USA), with distorted, asymmetrical valves. Reasoning about the origin of the — probably traumatic — malformation, the author concluded (p. 14):

> "The Narragansett Bay specimen resembles closely some ancient fossilized genera of non-burrowing mollusks, especially *Exogyra* and *Gryphaea*. Perhaps whatever happened to the present quahaug specimen triggered a latent genetic mechanism for shell shape that has been dominant in the oyster family for millions of years."

Another shell abnormality, distinct from those related to mechanical injury and other reported abnormal shell changes, has been observed in wild and hatchery-reared, bay-planted individuals of *Mercenaria mercenaria* from Great South Bay of Long Island, New York. It was not detected in other areas of Long Island sampled. The greatest incidence of shell-deforming disease occurred in newly planted, stunted, hatchery-reared stock. In adult clams, advanced lesions produced marked bilateral asymmetrical elevations and shell curvatures. Lesions of the inner nacreous layer, corresponding to the external deformities, consisted of elevated ridges and tough granular excavations.

Corresponding lesions of the secretory mantle comprised multiple sets of lobes and papillomatous and pedunculated outgrowths of the mantle epithelium, which appeared to be of a benign hyperplastic nature. In affected juvenile clams, lesions consisted of mantle atrophy, adductor muscle inflammation and degeneration, and an exudate of bacterial

masses and necrotic epithelial cells on the gill surface. Atrophy, fusion and atypical pigmentation were observed in the incurrent and excurrent siphons. Intranuclear inclusions of unknown identity occurred in both diseased and apparently healthy juvenile and adult clams (Leibovitz and co-authors, 1976b).

High percentages of *Ostrea edulis* grown in Maine (USA) exhibited unusual greenish deposits on the inner valve surfaces. These deposits, which were conchiolinous in nature, differed in structure from blisters typical of the oyster's response to parasites, in that they were firmly incorporated into the underlying shell layer. Oysters with such shell abnormalities occurred considerably less frequently (14 to 34 %) at colder, northerly sites than at southerly, estuarine sites (16 to 80 %). The deposits were distinctly concentrated around the adductor muscle scar and less significantly in the hinge area. The condition of the mantle and soft parts adjacent to the shell deposits appeared to be unaltered, but there was marked haemocytosis. Histological examination did not reveal any possible etiological agent (Logue, 1979).

Shell malformations were noted in the valves of approximately 15 % of *Crassostrea virginica*, collected along the east coast of the United States and the Gulf of Mexico. The abnormalities — which did not appear to be reactions to boring organisms, parasites and irritation caused by foreign bodies — consisted of massive globular pearls occupying the greater part of the shell space, of pedunculated pearls, of bridge-like structures comprising a series of calcified globular units, of calcified ridges over muscle scars with tufts of periostracum on top, of calcified cup-like cysts lined and covered with organic material, and of mantle recession resulting in excessive calcification of the valve margins (Galtsoff, 1969).

The author suggested that malformations of this type resemble calcifying neoplasia found in other animals. Since the soft tissues of the oysters so affected were not available, conclusions of this kind appear to be premature. The external characteristics of the shell lesions rather suggest interference of irritating or injurious organisms with normal shell secretion. Comps and co-authors (1973), who examined a cauliflower-like tumour on the mantle of an individual of *Crassostrea gigas*, observed calcified ridge-like outgrowths on the inner surface of both valves, situated adjacent to the site of the tumour. The growth appeared to be of connective-tissue origin and seemed to be benign, since no mitotic figures were observed. It was believed to result from a traumatic lesion, followed by excessive tissue proliferation. The calcareous ridges on the valves are comparable to some of the calcified structures described by Galtsoff (1969). If the latter author's interpretation of the *C. virginica* shell anomalies as 'calcifying neoplasia' are correct, these lesions could represent a later stage of an epithelial neoplasm comparable to that reported by Pauley and Sayce (1972; see section 'Neoplasia').

One of the most prominent properties of the molluscan body is the ability to precipitate calcium carbonate in the form of pearls. Studies on the mechanism and causes of their formation have provided the basis for much speculation. Although the occurrence of pearls in bivalves has been known for centuries, there are widely divergent opinions as to their origin. As early as 1856, de Filippi has identified larval trematodes, named *Distomum duplicatum*, as the causative agents of pearl formation in freshwater mussels *Anodonta cygnea*. Garner (1873) was the first to observe the occurrence of pearls in *Mytilus edulis* from British waters to be associated with the presence of "minute parasitical entozoa, fully developed distomes". His discovery initiated large-scale researches on the

occurrence and formation of pearls in mussels and other bivalves in Britain and France, beginning by the turn of the century.

Pearls or pearl-like calcareous concretions have subsequently been reported from European Atlantic and Mediterranean *Mytilus edulis, M. galloprovincialis, Cardium edule, C. lamarcki, C. glaucum, Macoma baltica, Donax vittatus, Tapes* spp., *Tellina* spp., *Ostrea edulis, Pinna nobilis, Pecten* sp., and other marine bivalves (d'Hamonville, 1894; Dubois, 1901a, b, 1903a, 1907, 1909; Jameson, 1902, 1903, 1912; Boutan, 1903, 1904; McIntosh, 1903; Herdman, 1904; Giard, 1907; Lebour, 1907c; R. G. Smith, 1907; Dollfus, 1912, 1923d; Alverdes, 1913; Cooper, 1932; Palombi, 1940; Cerruti, 1948; Götting, 1979a, b; Wachter, 1979; and others). Pearl formation in *M. edulis* and several other European species is believed to be due to *'Distomum margaritarum'* Dubois, 1901, a larval gymnophallid trematode of dubious identity, which actually encompasses a complex of species (see section 'Agents: Trematoda').

On rare occasions, pearls have also been reported from *Mytilus edulis* from the North American Atlantic coast. Stafford (1912) found "pearls of little or no value, but in considerable numbers", in mussels from Gaspé Bay, Canada. He simultaneously saw larval gymnophallids between the mantle and shell of the mussels. Linton (1915b), who, as a parasitologist, was very familiar with larval digeneans, found pearls in 2 *M. edulis* from Woods Hole, Massachusetts, but no metacercariae in over 100 carefully examined individuals. Scattergood and Taylor (1949) recorded the occurrence of pearls in various mussel populations along the northeast coast of the United States from Eastport, Maine, to Cape Cod, Massachusetts. Lutz and Hidu (1978) elaborated extensively on the quantitative relationship between mussel size and the number of pearls per host but did not positively identify larval gymnophallids as causative agents.

It appears that pearl formation in bivalves may result from causes other than larval trematode infestation. Jameson (1902, 1903) points out that, in *Modiolus modiolus,* pearls may form around sporozoan spores, an opinion shared by several other workers (see sections 'Agents: Apicomplexa' and 'Trematoda'). Dubois (1909) identified sporozoan spores as nuclei of pearls in *Pinna nobilis, Modiolus barbatus* and *Margaritifera vulgaris* (= *Pinctada margaritifera*) from the Gulf of Gabès, Tunisia. Microsporan spores or stages of other hyperparasites of metacercariae present in bivalves may also be involved in the process (Vol. I, p. 299; Vol. II, pp. 731 and 772). In pearl oysters of the genus *Margaritifera* (= *Pinctada*), pearl formation is believed to be induced by larval cestode infestation (see section 'Agents: Cestoda'). Herdman and Hornell (1906) identified a larval nematode as the nucleus of a pearl in a window-pane oyster *Placuna placenta* from the Gulf of Kutch, India. Against the background of this bulk of information one might cautiously conclude with Dubois (1901b) and Kunz (1923) that pearl formation in marine Bivalvia can have various causes.

Although most frequently occurring in bivalves, pearls have also been found in gastropods (R. G. Smith, 1907; Elliott, 1921; Kessel, 1937; for review consult Coomans, 1973; see also Vol. I, Fig. 12–44). The causes of these formations remain largely unexplained. However, pearl formation in freshwater snails *Biomphalaria glabrata* appears to have a genetic basis (Richards, 1970, 1972). The author (1972, p. 40) suggested "that genetics might be involved in pearl formation in some other mollusks".

Whatever the causes of pearl formation may be, it constitutes, in essence, a *malformation* resulting from the host's attempt to wall off an intruder which cannot be phagocytized

or eliminated. In the case of the pearl oyster, however, the outcome of this process may be a product of high commercial value. Therefore, the mechanism of pearl formation in molluscs and its artificial enhancement have attracted the interest of numerous scientists.

The process of 'nacrezation' (a term coined by Cheng, 1967, to describe the deposition of nacre around parasites which irritate or invade the mantle region) has been studied in great detail (e.g., Jameson, 1902, 1912; Boutan, 1904; Alverdes, 1913; Grégoire and co-authors, 1955; Nakahara, 1957; Tsujii, 1960; consult these authors for further references). *True nacreous pearls* consist of successively deposited layers of shell substance (i.e., conchiolin, in which the crystals of inorganic matter are deposited in the same manner as in the shell), enclosing a central nucleus, and formed in closed epithelial sacs embedded in the host tissue. *Blisters,* which appear as nacreous protuberances on the inner surface of the valves, result from the intrusion of foreign bodies between mantle and shell or from perforation of the valves by shell-burrowing organisms. In addition, calcareous *non-nacreous concretions* ('muscle pearls') may occur within connective tissues. Concretions not enclosed in epithelial sacs cannot acquire the structure of a true pearl (Jameson, 1902).

Calcareous non-nacreous concretions ('muscle pearls') may form in response to a variety of stimuli but are frequently produced by trematode metacercariae and form around nuclei resulting from precipitation of calcium carbonate from 'excretion granules', which are expelled from the excretory pore of migrating larval gymnophallids, or, more rarely, may form around dead metacercariae (see section 'Agents: Trematoda'). Precipitation of calcium carbonate from excretion granules may, on rare occasions, occur *within* the excretory vesicle of diseased metacercariae, which then may become the nucleus of a pearl (Vol. I, Fig. 10-16). In the case of *Meiogymnophallus minutus* (p. 722), however, pearl formation never ensues. This larval digenean is neither identical with *'Gymnophallus margaritarum'*, as believed by Cheng and Rifkin (1970), nor have the 'metacercarial sacs' of *M. minutus,* as figured by Bowers and James (1967; Fig. 13-118), anything to do with pearl sacs. Based on mere speculations and misinterpretations, Cheng and Rifkin's (1970) delineation of a nacrezation process in *Cardium edule,* involving *M. minutus* as a stimulant or pearl nucleus, is erroneous. Even dead *M. minutus* metacercariae have never been seen to induce the formation of solid concretions, not to mention true pearls.

In contrast to *Meiogymnophallus minutus, Gymnophallus gibberosus* is capable of inducing the formation of — sometimes pearl-like — non-nacreous calcareous concretions in *Cardium edule, C. lamarcki* and *Macoma baltica* from the German North and Baltic Sea coasts. A single migrating metacercaria may leave a 'comet tail' of concretions in its trail while penetrating the anterior adductor muscle (Fig. 13-116). Cavities produced by tissue-feeding *G. gibberosus* rapidly become filled with host-produced calcium-proteid material after the metacercariae have abandoned them. Presumably, transport of the organic calcium compounds into, and calcium carbonate precipitation within, the cavities is stimulated by host-tissue debris, metabolic waste products and excretion granules elaborated by the larval digeneans. Consonant with the dimension of the metacercariae, the resultant concretions assume the size and shape of the previously present worm, which led the early authors to assume that concretions or pearls are formed around living metacercariae. Upon acid dissolution of the calcium carbonate, however, merely transparent conchiolin coats remain, which enclose traces of amorphous, unidentifiable material (Fig. 13-117). Larger concretions within the tissues seriously interfere with the proper function of the adductor muscles in affected hosts.

Markowski (1936) described the progressive calcification of larval trematodes present in the mantle folds of *Macoma baltica*. Dead or decaying individuals of *Metacercaria mutabilis* (= *Lacunovermis macomae*) were initially walled off by an epithelial 'cyst' forming around the metacercariae. The 'cyst' then became calcified, showing a concentric structure. The observed process was, according to Markowski, strongly reminiscent of the mode of pearl formation, as described from other bivalves. Of 103 metacercariae dissected from individuals of *M. baltica*, 27 were calcified.

Mytilus edulis produces 2 types of pearls — spherical ones in the connective tissue of the gonads and the digestive gland, and ovoidal or pear-shaped ones attached to the inner surface of the valves (Fig. 13-184). The latter occur most frequently in the region of the posterior adductor muscle and along a narrow dorsal and ventral band. Most of these pearls have nuclei consisting of unidentifiable granular material, but others may reveal structures reminiscent of trematode metacercariae (Fig. 13-185) or 'ova' of unknown origin (Fig. 13-29; Götting, 1979a). Such foreign bodies initially become walled off by a fibrous capsule. Subsequently, amoebocytes surrounding the structure form a pearl sac (Fig. 13-186, PSE) with an inner surface rich in microvilli (MV). The pearl-sac epithelium secretes proteids and mucopolysaccharides (PM), which become condensed into concentrically and radially arranged conchiolin lamellae (CL) forming a network of pockets. Calcium-proteid complexes (CaP), synthesized outside the pearl sac, permeate through the pearl-sac epithelium and the conchiolin lamellae, and eventually cause the deposition of crystalline aragonite forming the pearl proper (Götting, 1979a, b). The chemical nature and structure of the conchiolin matrix and the calcium-binding polypeptides have been studied in great detail by Grégoire and co-authors (1955), Grégoire (1957, 1961), and Samata and Krampitz (1982).

The prevalence of pearl-bearers in populations of *Mytilus edulis* varies greatly between localities. McIntosh (1903), for example, found pearls in 45.1 % of 700 mussels collected from the estuary of the River Eden, Scotland. Similarly high prevalences have been recorded from other areas. Lutz and Hidu (1978) recovered between 2.2 and 27.7 pearls per host from mussels in Boothbay Harbor and Damariscotta River, Maine. Cooper (1932) counted a total of 93 pearls — the largest one measuring fully 3 mm in diameter — in a single *M. edulis* individual, 51 × 24 mm in shell dimension, from the N. E. Kent coast; and Götting (1979a) found 98 pearls (60 attached to shell, 38 free) in a single mussel of 70 mm shell length from Sylt, German North Sea coast.

The early authors' interest in pearl formation in *Mytilus edulis* has largely been stimulated by the — erroneous — belief that the mussel could be induced to produce pearls of commercial value, and proposals have been made in view of the artificial production of pearls in *M. edulis* (Boutan, 1903). Dubois (1903b) claimed to have enhanced pearl formation in *Pinctada margaritifera* by planting individuals into beds in the Mediterranean where trematode-infested *M. galloprovincialis* revealed to be pearl-carriers. However, mussel pearls are usually small (less than 1 to max. 3 mm) and, although being nacreous, they usually lack the lustre of true gem pearls. From the commercial standpoint, such 'druggist's pearls' in the mussel are detrimental rather than beneficial: In some areas, mussels may sometimes contain pearls in such numbers that they become unfit for the market (d'Hamonville, 1894; Dubois, 1901a, b).

In addition to being capable of pearl formation, molluscs have a tendency to deposit concretions in their excretory system (Potts, 1967). 'Kidney stones', composed mainly of

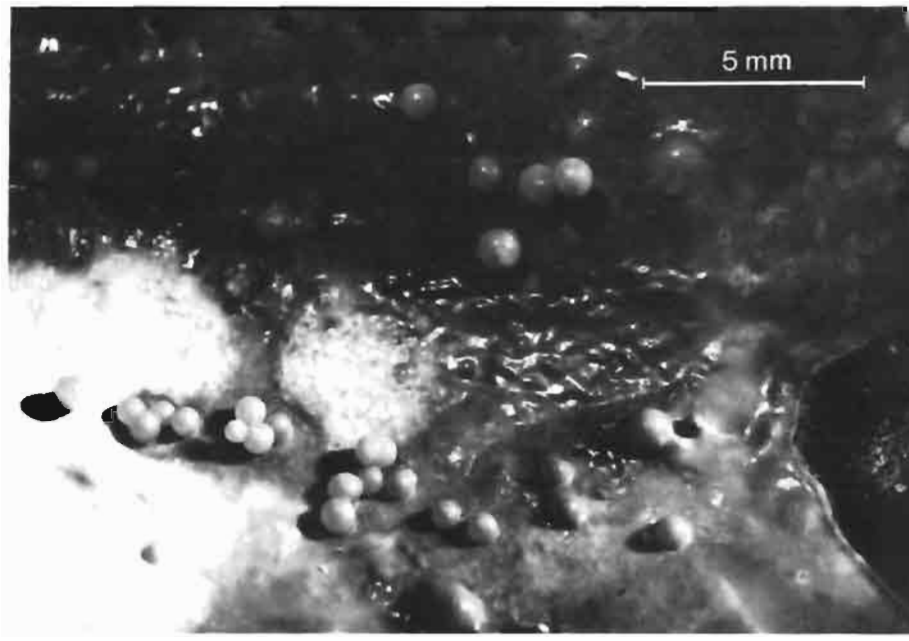

Fig. 13-184: *Mytilus edulis*. Pearls attached to inner valve surface. (After Götting, 1979a.)

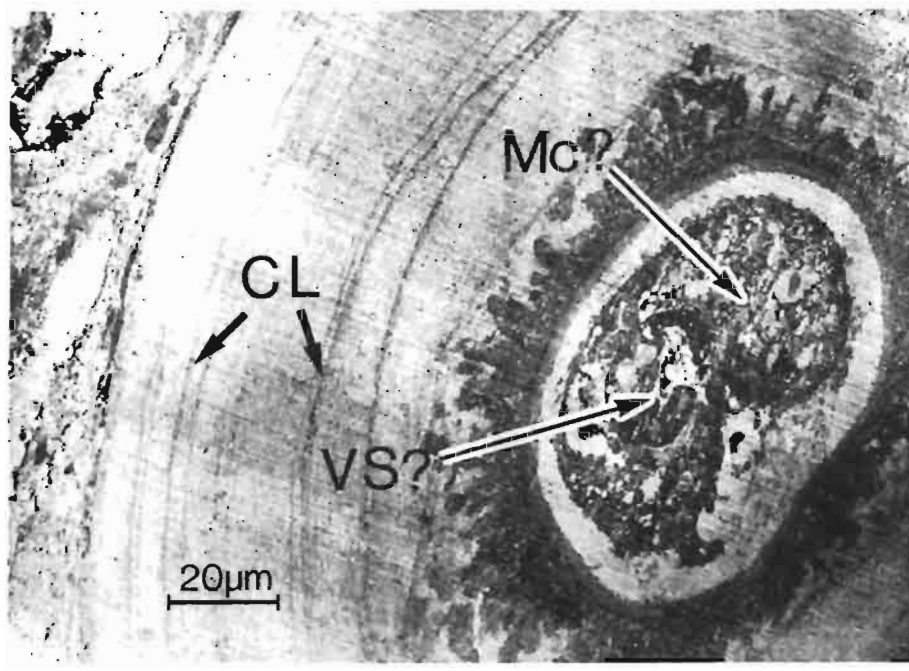

Fig. 13-185: *Mytilus edulis*. Free cyst pearl with nucleus containing presumed gymnophallid metacercaria. Ultrathin cut. CL concentric conchiolin layers of pearl, Mc? structure interpreted as enclosed metacercaria, VS? structure resembling ventral sucker of metacercaria. (After Götting, 1979a.)

amorphous calcium phosphate and lesser amounts of heavy metals bound in a mucopolysaccharide matrix, have been found in individuals of *Mercenaria mercenaria, Macrocallista nimbosa* and *Argopecten irradians* from the U.S. Atlantic coast. These renal concretions appear to be a normal product of the excretory process of molluscs living under reproductive, environmental or pollution stress (Doyle and co-authors, 1978; Gold and co-authors, 1982; Tiffany, 1982). Mauri and Orlando (1982) experimentally induced the formation of renal concretions in *Donax trunculus* from the northern Tyrrhenian Sea by exposing the test animals to manganese levels higher than those in natural sea water. The concretions, analyzed by X-ray microanalysis, showed a composition (P, Ca, Mn) similar to that of nephrolithes naturally occurring in *D. trunculus* from polluted coastal areas. These molluscan kidney stones can sometimes form in such large amounts that they almost completely fill the renal lumen.

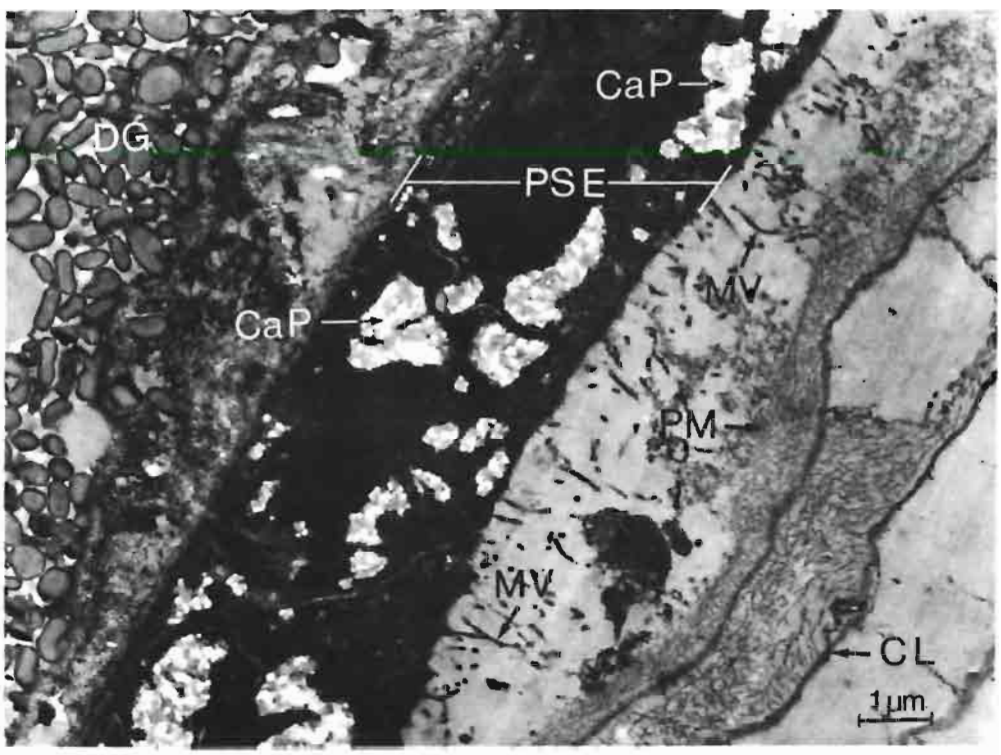

Fig. 13-186: *Mytilus edulis*. Mechanism of pearl formation. CaP Calcium-proteid complex, CL concentric conchiolin lamellae, DG digestive gland, MV microvilli, PM proteids and mucopolysaccharides, PSE pearl sac epithelium. For explanation see text. (After Götting, 1979a.)

Malformation of the soft parts of bivalves, presumably caused by mechanical injury, include partial or complete splitting of the foot. Pelseneer (1923) figured an individual of *Lucina lactea* with a bifid foot. The incision extended to about half of its length, leaving 2 tentacle-like, thin processes at the posterior end. Among hundreds of normal juvenile *Cardium edule* from Sylt (German North Sea coast), a single individual was found with a

bifurcate and atrophied foot (Fig. 13-187). The origin of the lesion remained unknown (Lauckner, unpubl.). Pelseneer (1923) described an individual of *Tapes pullastra* with a foot duplicity. The left foot was more fully developed in having 2 posterior retractor muscles, but lacked the left anterior retractor muscle. The right foot, which was of the same size as the left one, had no posterior and only a single (right) anterior retractor muscle. Several types of foot abnormality have been noted in *Mytilus edulis* from Padstow, England. That most frequently met with was a small foot-like outgrowth, or rudimentary accessory foot, originating from near the base of the primary foot, the latter showing no injury to its tip (Atkins, 1931b).

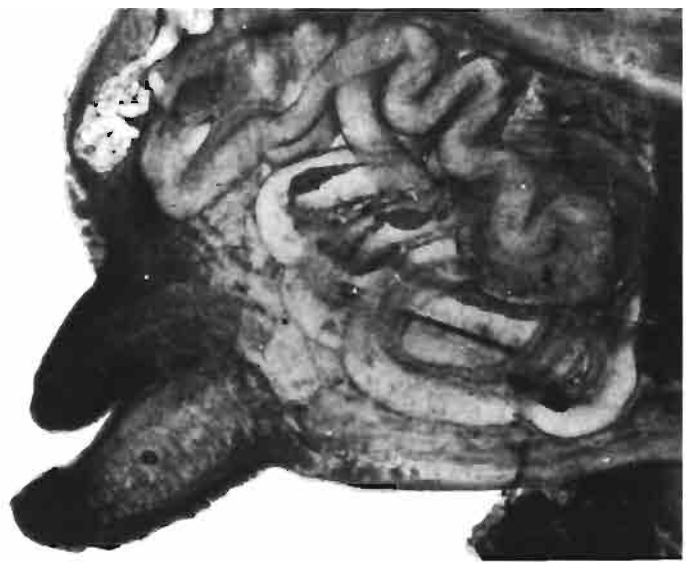

Fig. 13-187: *Cardium edule*. Abnormal bifurcate and atrophied foot of juvenile individual (10.5 mm shell length). (Original.)

Gunter (1958) and Pauley and Sayce (1967) described individuals of *Crassostrea virginica* and *C. gigas*, respectively, with abnormal bifurcated adductor muscles. A tumorous mass was growing from the anterior portion of the muscle in the *C. gigas* individual.

Abnormal siphonal structures have been observed in *Mya arenaria* and *Mercenaria mercenaria*. One soft-shell clam from Chesapeake Bay exhibited a supernumerary portion of the incurrent siphon. Terminal tentacles, similar to those surrounding the lumina of normal siphons, were absent from the tip of the process, which was a short blind sac anastomosed to the adjacent incurrent siphon. A second *M. arenaria* individual, originating from New Hampshire waters, had 3 normal-appearing siphonal orifices with surrounding tentacles. In sagittal section, the incurrent siphon was bifurcate, with connecting lumina and normal pigmentation. The *M. mercenaria* individual, collected in Narragansett, Rhode Island, waters, had a non-functional supernumerary, tentacled appendage on the incurrent siphon. The normal filtering activity of the clams did not appear to be affected adversely by the presence of these structures (Tubiash and co-authors, 1968).

Tissue discolouration in bivalves may result from infestation with protozoan or metazoan parasites. 'Green oysters' harbour endobiotic (parasitic) unicellular algae (see section 'Agents: Algae'). Protistan agents, presumably labyrinthomorphs, cause discolouration in oysters, termed 'Amber disease' (see section 'Agents: Labyrinthomorpha'). Infestation with trematode rediae or sporocysts frequently turns bivalves brilliant orange-red due to parasite-caused accumulation of carotenoids (see section 'Agents: Trematoda'). Pauley and Sayce (1967) observed an individual of *Crassostrea gigas* from Willapa Bay, Washington (USA), which was entirely black with the exception of the adductor muscle. The inside of the valves was also discoloured, and upon gross observation of a body cross-section, the animal was completely black. The cause of the discolouration was not determined. Brownish discolouration of tissues may result from hyperplasia of 'brown cells', which occurs in response to various diseases (Gutiérrez, 1977a). The latter author described several other histological abnormalities in *C. angulata*, including perivascular, connective-tissue and ctenidial haemocytic infiltration, arterial obliteration, thrombosis, ctenidial and connective-tissue hypertrophy, etc. Similar lesions and morphological anomalies have been observed in individuals of *C. virginica* and *Argopecten irradians* experimentally exposed to waste motor oil (Gardner and co-authors, 1975).

'Watery cysts', first noticed by Mackin (1962) in *Crassostrea virginica* from Louisiana waters, form in response to an unknown disease. The cysts are large conspicuous, bubble-like cavities, generally distended by internal pressure of accumulated fluid. Sections show these structures to contain a central granular material, usually arranged in concentric layers. Stained sections and smears of cysts revealed the presence of a very small bacillus. No attempts were made to cultivate the micro-organism, but it was believed to be the etiological agent of the 'watery-cyst' condition. Recovery from the presumptive infection appeared to take place by sloughing of the cyst contents.

Similar structures, described as 'multiple watery cysts', have been detected in *Crassostrea gigas* from Willapa Bay, Washington. Grossly, they appeared as circular domes, present anywhere on the oyster body, and ranging from 1 to 40 mm in diameter. They contained only clear, watery fluid and, in some instances, necrotic tissue debris but no bacteria. Histologically, the cysts were lined with an epithelium, varying from necrotic and unrecognizable to cuboidal and columnar cells with karyolytic nuclei. The tissue areas beneath the cysts exhibited haemocytic infiltration and edema. One oyster with watery cysts simultaneously had an unusual tissue proliferation believed to represent a ganglioneuroma. The cause of the neural lesions and their relation to the watery cysts are unknown (Pauley and Sayce, 1967; Pauley and co-authors, 1968; see section 'Neoplasia').

Non-neoplastic, inflammatory lesions, termed granulocytomas, have been found to induce atrophy and autolysis of the digestive gland of *Mytilus edulis*. The lesions were composed of eosinophilic granular haemocytes, small basophils and macrophages, all in varying stages of degeneration. The most prominent feature of the disease were swelling and coagulation of the cytoplasmic granules of the granulocytes to form amorphous, PAS-positive bodies, 8 to 10 μm in diameter. Pyknotic and karyolytic granulocyte nuclei were abundant, but the basophils and macrophages generally remained normal in appearance.

Development of the lesions appeared to originate in the vascular haemolymph sinuses of digestive-gland and mantle-connective tissues; they ranged in size from 150 to 1,500 μm in diameter. Digestive tubules adjacent to the lesions exhibited atrophic alterations, with resulting loss in cellular integrity and associated necrosis. In severe cases, the ducts and

tubules were decimated in number, and the digestive gland was then composed of a series of large lesions with a few remaining digestive ducts and tubules. No mitotic figures or multinucleate giant cells were visible, and there was no evidence of the presence of any microbial or protistan agent. Mussels from various sites around southern England and Wales were found to be affected by the disease, which was believed to result from chronic exposure to domestic and industrial waste products (Lowe and Moore, 1979; Green and Alderman, 1983).

Hyperplastic and metaplastic tissue changes, due to parasitic irritation, appear to be common in marine bivalves. Taylor (1966) found parasitic protozoans *Haplosporidium tumefacientis* to be the cause of tumefactions in the digestive gland and kidney of *Mytilus californianus*. Sparks (1962) observed metaplasia in the gut of *Crassostrea gigas*, induced by invasion of parasitic copepods *Mytilicola orientalis* (see section 'Agents: Copepoda'). High incidences of structural abnormalities of gill filaments and palps, including hyperplasia, have been reported from *M. edulis* from various localities in England (Atkins, 1931a, b). At least some of these appear to have been caused by parasitic invasion or irritation. Others bear a striking resemblance to the virus-induced 'gill disease' of oysters (p. 481). Gill hyperplasia may also result from the presence of pea crabs in the mantle cavity (see section 'Agents: Decapoda'), but the incidence of *Pinnotheres* infestations was generally low in the mussels inspected by Miss Atkins.

Of 20,000 *Crassostrea virginica*, collected by Harshbarger and co-authors (1979) from 23 areas in the Maryland portion of Chesapeake Bay, only a few had marked hyperplasia of the gill epithelium, and 4 showed hyperplasia of the mantle epithelium. The authors state that this is a condition commonly observed in oysters, but of unknown significance in bivalves. In freshwater fishes, gill hyperplasia is frequently associated with high levels of pesticides (Eller, 1975).

The most conspicuous and most suspect structural abnormalities in bivalves are tumours of the soft parts. Many of these were thought to represent neoplasia, until histological examination revealed them to be inflammatory responses or microbial or parasitic infestations. As pointed out, distinction between neoplasia, hyperplasia and response to injury or infestation is frequently difficult to determine confidently, mainly because there are few trenchant established criteria to diagnose neoplasia in the Mollusca. Presence of mitotic figures in sections of tumorous tissue is normally considered as indicative of neoplasia. As discussed below, occurrence of mitoses may have other reasons. The term 'tumour' is used here in its broadest, original sense, describing any swelling or abnormal mass of tissue (p. 863). Most bivalve tumours described are definitely non-neoplastic lesions. However, marginal cases exist, and it is not known whether hyperplastic (benign) proliferations in molluscs can develop into neoplastic (malignant) disorders, as can certain mammalian tumours.

The early literature on tumours of invertebrates has been reviewed by Scharrer and Lochhead (1950) and Dawe and Harshbarger (1969); that on neoplasia and tumour-like lesions in molluscs by Pauley (1969). This information has been updated by Sparks (1972), Harshbarger (1974) and Dawe and Homburger (1976).

Tumours occurring in marine bivalves have mostly been studied in commercially important species. The earliest report on abnormal growths in *Crassostrea virginica* is that by Ryder (1887), who observed a pedunculate structure of mesenchymal origin, about 25 mm long and 12.5 mm wide, soft and pliable and of nodular, polypoid appearance, in

the pericardial cavity of an oyster. The pericard was enlarged because the tumour had displaced the heart forwardly. Although the histology of the tumour was studied in detail, apparently no mitotic figures were seen. Another benign, nodular mesenchymal tumour, about 31 × 25 × 8 mm in dimension, has been found connected to the outer surface of the pericard of *C. virginica* by a narrow, delicate stalk. Both the tumour and the stalk were composed of connective-tissue cells and covered by a single layer of ciliated columnar cells (Smith, 1934).

Mix and Riley (1976, 1977) reported on a pericardial tumour in an Olympia oyster, *Ostrea lurida*, from Yaquina Bay, Oregon (USA), and Dinamani and Wolf (1973) described multiple tumours in the pericardial cavity of an individual of *Saccostrea cucullata*, collected from Georges River, New South Wales (Australia). The growths consisted of a pedunculate, cauliflower-like main tumour, 5 × 4.8 × 4.5 mm in size, 2 smaller tumours placed close together on the opposite wall of the pericard and near the ventricle, and a third tumour on the pericardial wall immediately below the ventricle. Mitotic figures were not positively identified.

Several apparently benign tumours have been observed in *Crassostrea gigas*. One of these abnormal growths, thought to be a hamartoma, was approximately 20 by 15 mm in size and lay just dorsal to the rectum. Microscopically, it consisted of a stroma of typical Leydig cells covered by an epithelium composed largely of deeply convoluted tall, columnar ciliated cells, but with some areas of typical mantle epithelium of low cuboidal cells. The tumour was connected to the body in the area of the rectum, from which it originated. There was a heavily vascularized area just beneath the epithelium, which contained large numbers of collagenous fibres (Sparks and co-authors, 1964a).

Upon gross inspection, another Pacific oyster appeared to have an identical lesion (Fig. 13-188, a). However, microscopic examination of the growth, which measured 24 × 18 × 16 mm, revealed that the apparent tumour was actually the expanded rectum, evidently obstructed by faecal impaction. Sectioned material showed a granular, eosinophilic, laminated mass surrounded by a band of necrotic haemocytes inside normal-appearing rectal epithelium (Sparks and co-authors, 1964b). A similar tumour-like expansion of the intestinal tract, growing out of the body, was observed in an individual of *Crassostrea gigas* from Willapa Bay, Washington (Pauley and Sayce, 1967).

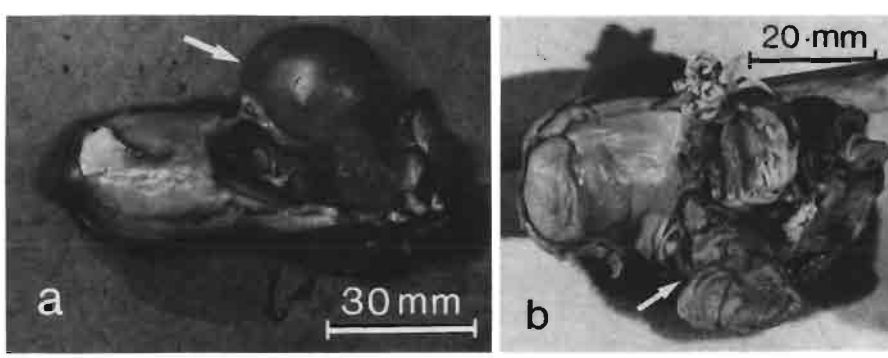

Fig. 13-188: *Crassostrea gigas*. (a) Individual with tumour-like faecal impaction (arrow); (b) pedunculated tumour (arrow) of mesenchymal origin (tumour pulled down to show ventral origin of lesion from body beneath the adductor muscle. (a after Sparks and co-authors, 1964b; b after Sparks and co-authors, 1969.)

An apparently benign, pedunculate mesenchymal *Crassostrea gigas* tumour, with an irregular, nodular surface, was noted by Sparks and co-authors (1969). It was crescent-shaped, 30 × 13 × 15–20 mm in size, and situated laterally and anterior to the adductor muscle, covering the pericardial area and protruding through the left side of the mantle (Fig. 13-188, b). No mitotic figures were seen. A similar large, pedunculate growth, attached to the mantle, was discovered in a Pacific oyster from Little Skokum Inlet, Puget Sound, Washington. It lacked mitotic figures and was interpreted as a benign vesiculo-epithelial polyp (Harshbarger, 1976).

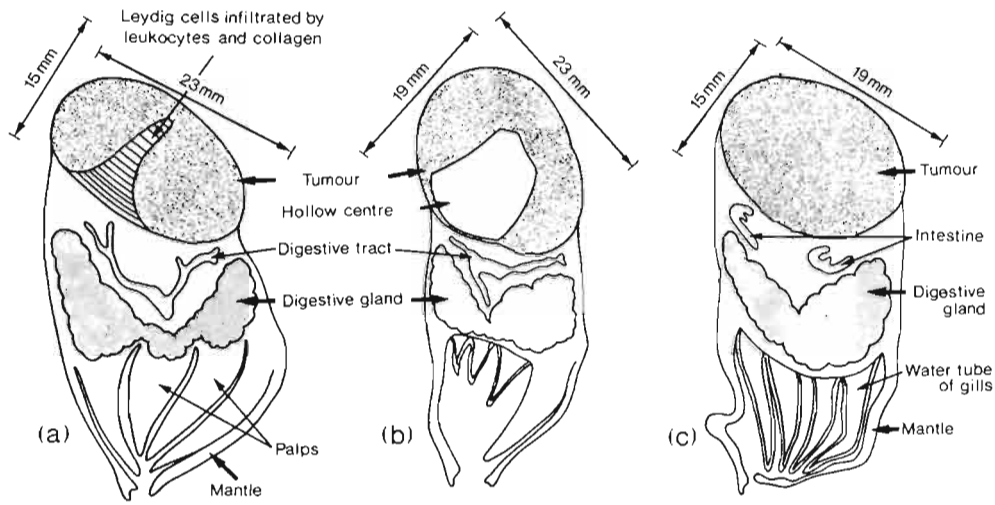

Fig. 13-189: *Crassostrea gigas*. Internal fibrous tumour. Cross section through anterior portion (a), mid-region (b) and posterior portion (c). (After Pauley and Sayce, 1968b.)

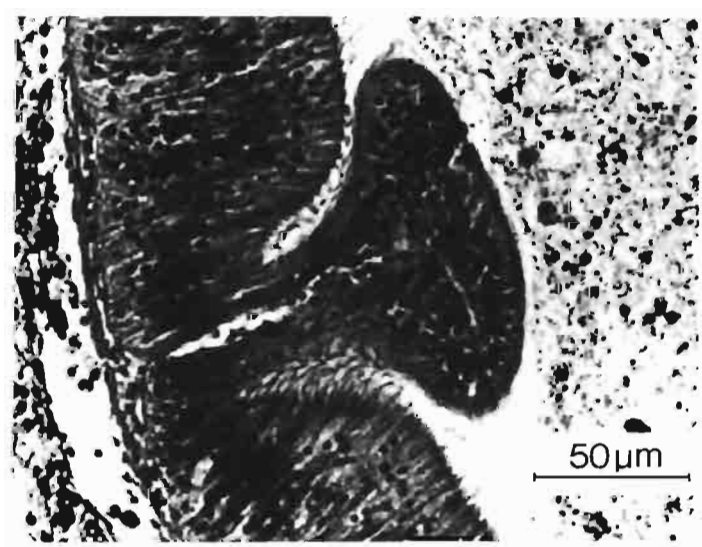

Fig. 13-190: *Ostrea edulis*. (a) Abnormal growth in posterior portion of intestine. (After Katkansky, 1968a.)

A smooth-textured, dark-green growth, 17 × 13 × 10 mm in dimension, has been found arising from the inside of the mantle of a Pacific oyster from Willapa Bay, Washington. A second specimen had a bifurcated adductor muscle, with a polypoid or papillary tumour, 25 × 10 × 10 mm in size, growing from the abnormal anterior portion of the muscle (Pauley and Sayce, 1967). Another *Crassostrea gigas* individual from Willapa Bay had an internal fibrous tumour, which was palpable as a large hard mass, measuring 52 × 23 × 19 mm, on the dorsal surface of the oyster's body. It had a hollow centre and appeared to be encased in a fibrous capsule. The growth seemed to originate from undifferentiated gonadal cells and had a well-defined border, with the tumorous mass apparently pushing the normal tissue aside rather than invading it. Since no mitotic figures were observed, the growth was considered benign (Pauley and Sayce, 1968; Fig. 13-189).

Two tumours of the digestive tract have been noticed in a European oyster, *Ostrea edulis*, from an experimental population in Drakes Estero, near San Francisco, California. One of these was mushroom-shaped, 0.15 × 0.12 mm in dimension, supported by a stalk, 0.04 mm in diameter, and consisting of slightly modified epithelial cells (Fig. 13-190). The other one, measuring 0.20 × 0.15 mm, lacked a well-defined stalk, but was characterized by prominent haemocytic infiltration. There was no indication of invasiveness (Katkansky, 1968a).

Reports on tumours of marine bivalves other than oysters are less numerous. A few abnormal growths have been detected in individuals of *Mya arenaria, Tresus nuttalli, Saxidomus giganteus, Mercenaria mercenaria, Mytilus edulis, Spisula solidissima* and *Pinctada margaritifera.*

Cauliflower-like papillary growths around the anal opening have been reported from about 2 % of *Mya arenaria* collected from Chesapeake Bay (Hueper, 1963). The condition

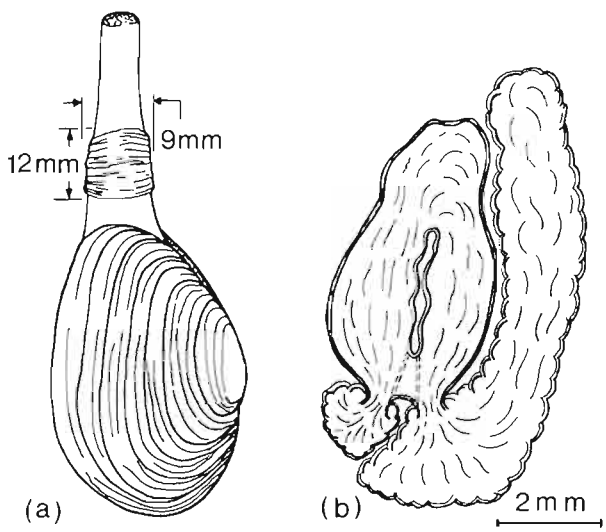

Fig. 13-191: *Mya arenaria.* (a) Gross appearance of tumour on siphons; (b) cross section of clam siphons and tumour arising from them. Dotted lines: epithelium surrounding cleft confluent with tumour and siphon lumen. (After Pauley and Cheng, 1968.)

was believed to have been caused by oil pollution. A re-examination of Hueper's microscopic slides showed that the lesions were non-invasive, papillary epithelial proliferations at the pedal orifice of the mantle, not near the rectum (Pauley, 1969).

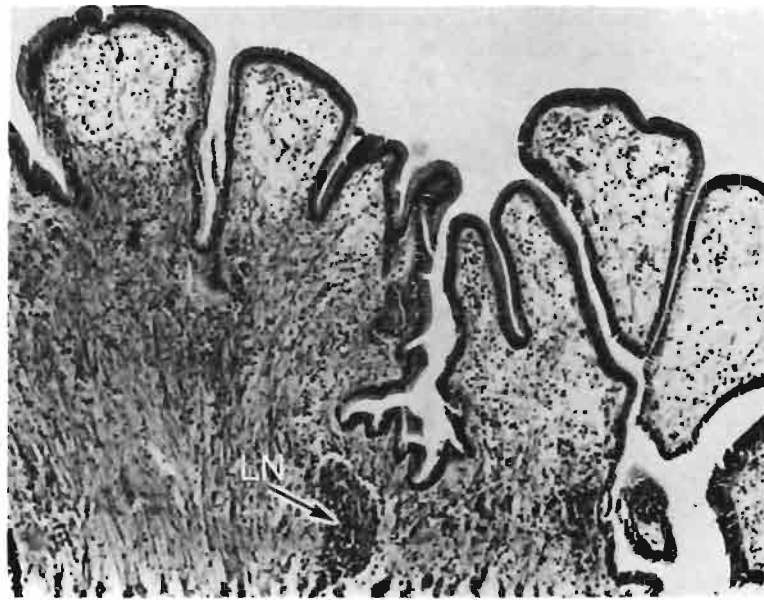

Fig. 13-192: *Mya arenaria.* Microscopic appearance of siphon tumour. Note folds in epithelial covering, edematous areas beneath surface, moderate haemocytic infiltration throughout lesion and nest of haemocytes (LN). Haematoxylin and eosin, × 70. (After Pauley and Cheng, 1968.)

A cylindrical growth of firm, muscular consistency and 4.0 × 1.3 mm in size, was found on the siphon of another soft-shell clam from Chesapeake Bay (Potter and Kuff, 1967). One out of 500 *Mya arenaria*, collected from the Tred Avon River estuary at Oxford, Maryland (USA), revealed a fungiform, wrinkled swelling, 12 × 9 × 6 mm in dimension, at the basal portion of the siphon (Figs 13-191 and 13-192). The lesion was seemingly benign, with no mitotic figures apparent on microscopic inspection (Pauley and Cheng, 1968).

Hillman (in Pauley, 1969) found 1 *Mya arenaria* with a large tumorous mass protruding from the mantle, but histological examination failed to reveal its nature. The growth contained sand, which may have stimulated or induced the lesion, or may have been there incidental to the primary tumour.

A papilloma-like tumour — similar to those reported from *Mya arenaria* by Potter and Kuff (1967) and Pauley and Cheng (1968) — was found on the siphon of a gaper clam *Tresus nuttalli* from Sequim Bay, Washington (USA). Detailed histological examination was impeded by severe autolytic changes that had occurred in the specimen prior to fixation. The cause of the tumour remained unknown, but it appeared to be a hyperplasia of muscle cells in the siphon. No mitotic figures were seen (DesVoigne and co-authors, 1970). Another gaper clam, bearing polypoid and papillary growths on the foot, was

collected off Marineland, Palos Verdes, California. Gross characteristics of the lesions were highly suggestive of neoplasia. Histological examination, however, revealed them to be inflammation and hyperplasia, associated with the presence of unclassified micro-organisms that were either primary or secondary invaders (Taylor and Smith, 1966).

Two apparently benign tumours of smooth-muscle origin have been recorded from the foot of butter clams *Saxidomus giganteus* from Sequim Bay, Washington (USA). The first, a large polypoid growth measuring 9 × 6 mm, was found on 1 of 183 clams. The other one, a similar but smaller, finger-like growth measuring 2.5 × 1 mm, was found among 394 *S. giganteus*. Both tumours were considered benign growths that lacked mitotic figures (Pauley, 1967a, 1969).

A surf clam *Spisula solidissima* from off Long Island, New York, was found with a multiple pedunculated tumour on the foot. The growth consisted of 6 firm smooth lobes, ranging from 1 to 2.5 cm in width and from 1 to 3 cm in height. Histologically, they were composed of well-differentiated smooth muscle cells that arose from normal foot muscle. The tumour was interpreted as a polypoid myoma (Leibovitz and co-authors, 1976a).

Two tumours of mesenchymal origin were found in 1 of 25 individuals of *Pinctada margaritifera*, collected from Centipede Reef, near Townsville, North Queensland (Australia). The growths arose from the visceral mass in the gut loop area and had normal low columnar ciliated and pigmented epithelium and associated secretory cells. Their stroma was richly vacuolarized, with well differentiated tissue of scattered muscle fibres in a collagenous matrix. No inflammation was evident; no mitotic figures were seen (Dix, 1972).

Although some of the above-described tumours have been designated neoplastic, there appears to be little evidence for such allocation, at least with respect to the stages described. It seems noteworthy that most of the tumorous bivalves appeared otherwise healthy and obviously suffered little detriment from their affections.

Neoplasia

Neoplasms ('new growths') may be defined as disturbances of growth characterized primarily by an *unceasing*, abnormal, excessive proliferation of cells. A tumour, in its general sense, is defined as any swelling or abnormal mass of tissue (Steinhaus and Martignoni, 1970). Tumours may result from non-neoplastic, controlled cell (tissue) proliferation (hyperplasia), or from uncontrolled proliferation (neoplasia). Through medical usage, the term 'tumour' is now — unfortunately — almost entirely restricted to neoplasia (Warren and Meissner, 1966; Sparks, 1972). In his review on neoplasia and tumour-like lesions in molluscs, Pauley (1969) frankly applied the term 'neoplasm' to non-neoplastic tumours.

Invertebrate tumour research is a fairly recent discipline. For long time it has been assumed that tumours cannot occur in animals at a phylogenetic level lower than the fishes. Engel (1930) elaborated extensively on the reasons why invertebrates are unable to develop cancer. However, with increasing insight it became apparent that invertebrates are quite susceptible to neoplasia (Vol. I, Chapters 6, 9, 10 and 12; for general review of literature on tumours in invertebrates consult Scharrer and Lochhead, 1950, and Sparks, 1969, 1972).

Tumour terminology has been developed almost exclusively for use in vertebrate

pathology and cannot, therefore, be applied to invertebrate disorders without modifications and adaptations. Neoplasia is easy to define theoretically, but sometimes difficult to diagnose with certainty histologically, particularly in lower invertebrates. There are large gaps in our knowledge about how invertebrate neoplasms conform to what is known about mammalian neoplasms, their morphological characteristics, biological course, relation to host-regulating mechanisms, and their transplantability and transmissibility. Because of the histological peculiarities of invertebrates, difficulties are encountered in the histological classification of their tumours. As a result, many so-called tumours in bivalves have subsequently been shown to be hyperplastic reactions to injury, rather than true neoplasms (Scharrer and Lochhead, 1950; Sparks, 1972; Cheng, 1976c; Stewart, 1976, 1977; see section 'Abnormalities').

Until about 1960, only a few hesitant attempts have been made in the study of neoplasms of poikilothermic animals (Schlumberger, 1957). Almost all neoplasms in bivalves have been discovered after 1960 (Harshbarger, 1977). In 1966, the 'Registry of Tumors in Lower Animals', residing at the Museum of Natural History, Smithsonian Institution, Washington, D.C., was founded under the auspices of John C. Harshbarger to facilitate the comparative study of tumorogenesis and related disorders in invertebrate and poikilothermic vertebrate animals. The approximately 1,500 accessions, received by the Registry during the period 1966–76, include 191 molluscan specimens with neoplastic and 97 specimens with non-neoplastic lesions (Harshbarger, 1969, 1977). Much of this material has been presented at various symposia and workshops (Dawe and Harshbarger, 1969; Farley, 1976c; Scarpelli and Rosenfield, 1976; Kraybill and co-authors, 1977). Recent research on molluscan neoplasia and related disorders has been stimulated by the question whether bivalves can be used as indicators of environmental carcinogens and radiation (Hueper, 1963; Mix and Sparks, 1970, 1971; Sparks, 1972; Mix, 1976b; consult these authors for further references). Thus far, ionizing radiation has not been found to produce neoplastic lesions in bivalves (Mix, 1976a). Numerous records seem to indicate a direct causal relationship between the occurrence of neoplasia in marine bivalves and environmental pollution and, in particular, with the presence, in water or sediment, of carcinogenic substances. However, neoplastic disorders have been recorded, sometimes with high prevalences, in oysters collected from areas remote from industrial or urban pollution (Wolf, 1969, 1971, 1976a; Pauley and Sayce, 1972). The suspected role of insecticides in the etiology of some of these lesions has not been established.

Antineoplastic substances have been isolated from various marine invertebrates, including *Mercenaria mercenaria*, *Crassostrea virginica* and *Macrocallista nimbosa* (Schmeer, 1964, 1966a, b, 1969; Schmeer and Huala, 1965; Schmeer and co-authors, 1966; Li and co-authors, 1968; Pettit and co-authors, 1970). Purified tissue extracts from these bivalves were active, *in vivo* and *in vitro*, against mammalian cancer. The role of such compounds in immunity or resistance of bivalves against neoplasia is unknown.

Neoplastic cells have 3 major cytodiagnostic characteristics. These are: (i) Anisokaryosis (nuclear polymorphism). Nuclei differ in size and shape and are usually larger than normal ones; the nuclear membrane is irregular. (ii) Nuclear hyperchromatism. Nuclei stain deeper than normal. (iii) Anisocytosis (cellular polymorphism). Truly neoplastic cells in bivalves share these diagnostic properties with mammalian cancerous cells. The phenomenon of metastasis is generally regarded as 'the hallmark of cancer' (Stewart, 1977). Although metastasizing tumours are comparatively rare in invertebrates, several

such cases have been reported from marine bivalves (Couch, 1969; Yevich and Barry, 1969; Barry and Yevich, 1972, 1975; Wolf, 1976a; Yevich and Barszsz, 1976, 1977). Frequent occurrence of mitoses is highly indicative of neoplasia and is particularly typical of haematopoietic neoplasms in bivalves, but mitotic activity may also be observed in regenerating molluscan tissues (Hillman, 1963; Sparks, 1976). Neoplasms observed in marine bivalves are of various tissue origins (Table 13-34).

Papillary mantle epitheliomas in various stages of development, displaying distinct signs of invasiveness and malignancy, have been found in more than 130 individuals of *Crassostrea commercialis* (= *Saccostrea cucullata*), collected over a 10-year period from non-cultivated stock in the Shoalhaven/Crookhaven estuary, New South Wales (Australia). Most of the tumours were either ovoidal or spherical, with deep macroscopic ridges, giving them a pedunculate, 'cauliflower-like' appearance. The size of the growths varied from 1 to 16 mm in diameter. One of these (Fig. 13-193) had a volume approximately one-eighth that of the body volume of the oyster from which it originated. Numerous oysters displayed 2 or more smaller lesions adjacent to the main tumour. Whether these represent metastases could not be determined with confidence; multicentric origin cannot be ruled out. Mitotic figures, normally very rare, were present in moderate-to-high numbers in all stages of division, indicating active proliferation and rapid growth. Oysters of both sexes were about equally affected (Wolf, 1969, 1971, 1976a). The author listed 3 possible explanations for the high tumour prevalence in oysters originating from the same shellbed: (i) Genetic changes producing a population of tumour-susceptible oysters, in which the growths appeared spontaneously; (ii) presence of an infectious agent, possibly a virus, localized in this particular shellbed; and (iii) ubiquitous presence of a causative agent, which only produces tumours in oysters rendered susceptible, as by mutation.

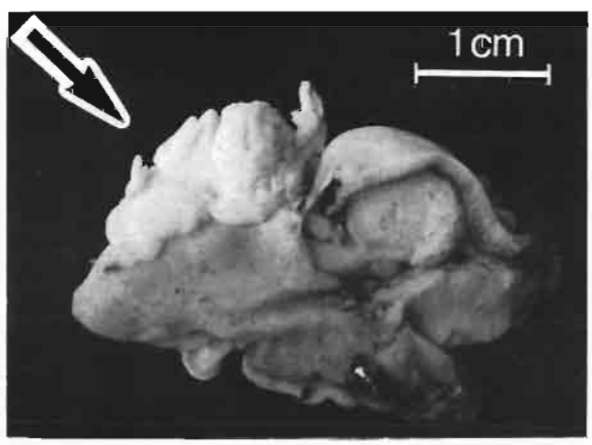

Fig. 13-193: *Saccostrea cucullata*. Unusually large neoplasm (arrow) arising from mantle. (After P. H. Wolf, 1971.)

Another epithelial neoplasm, recognizable as a rough, brown, irregularly indented, ulcerated area on the external mantle surface, has been detected in a single *Crassostrea gigas* from Willapa Bay, Washington (USA). The lesion, which measured 3.3 × 2.0 cm,

Table 13-34
Some records of neoplasia in marine bivalves (Compiled from the sources indicated)

Bivalve species	Classification, characterization and origin of neoplastic lesion	Locality	Prevalence	Time of collection	Source*)	Remarks
Crassostrea virginica	Germinoma (gonadal neoplasm)	Mispillion River, Delaware Bay	1 of 50	Sep 1969	*10*	Metastases in blood vessels, connective tissue and gastrointestinal epithelia
	Undifferentiated sarcomas; haematopoietic; epithelial; germinal neoplasms	Upper Chesapeake Bay and tributaries	19 of 19,500	1964–74	*16*	
	Undifferentiated sarcomas in perivascular and periintestinal connective tissue	Maryland portion of Chesapeake Bay	2 of 20,000	1974–77	*13*	
	Germinomas in gonadal follicles, spread to connective tissue and blood sinuses	Maryland portion of Chesapeake Bay	2 of 20,000	1974–77	*13*	Generalized haemocytosis
	Haematopoietic neoplasms	Harris Creek, Chesapeake Bay (Maryland)	1 of 5,000	1964–66	*6*	Focal neoplastic lesion in mantle
		U.S. Atlantic coast	5 cases	1960–67	*8*	Total of 30,000 *C. virginica* and *C. gigas* examined
		New Haven, Connecticut	1 of 1,400	June 1966	*15*	Lesions most abundant in gills
		Maryland portion of Chesapeake Bay	12 of 20,000	1974–77	*13*	Extensive tissue destruction
		Virginia portion of Chesapeake Bay	39 of 51,733	1964–73	*12*	
		Apalachicola Bay, Florida	1 of 373	?	*7*	Diseased individuals concurrently infested with *Perkinsus marinus*
Crassostrea gigas	Ganglioneuroma	Willapa Bay, Washington	1 case	?	*18*	
	Mantle epithelioma	Willapa Bay, Washington	1 case	June 1967	*17*	
	Haematopoietic neoplasm	Matsushima Bay, Japan	1 case	Aug 1966	*8*	
Saccostrea cucullata	Mantle epitheliomas	Greenwell Point, New South Wales (Australia)	over 130 cases	1965–75	*19*	All oysters collected from single, non-cultivated bed
Ostrea lurida	Haematopoietic neoplasms	Yaquina Bay, Oregon	about 12 %	1961–70	*11*	
Ostrea edulis	Haematopoietic neoplasms	Dubrovnik (Yugoslavia) and Ría de Noya (Spain)	20 to 35 %	?	*1*	Heavy mortalities
Mytilus edulis	Haematopoietic neoplasms	Yaquina Bay, Oregon	10 of 100	Sep 1968 and Febr 1969	*9*	Severe pathology
		Plymouth, England	16 of 994	1976–78	*14*	Potentially carcinogenic aromatic hydrocarbons detected in mussel-bed sediments
Mya arenaria	Neoplasms involving neurologic, connective- and glandular tissue in mantle	Upper Chesapeake Bay	1 of 1,400	1969–73	*16*	
	Germinomas	Long Cove, Searsport (Maine)	5.7 % of 3,213	1971–75	*21*	Metastases in 73 (2.27 %) of 3,213 cases

Table 13-34 (continued)

Bivalve species	Classification, characterization and origin of neoplastic lesion	Locality	Prevalence	Time of collection	Source*)	Remarks
Mya arenaria	Haematopoietic neoplasms	Maine to Rhode Island, 10 sampling stations	159 of 1,325	Jan to Sep 1976	*4*	Local variation from 0 to 64 %. Highest prevalences at oil-spill site
		Harpswell Neck, Maine	48 of 471	1972 and 1975	*21*	Extensive tissue destruction
		Laboratory stock	up to 70 % in laboratory containers	1975–80	*2*	Increased mortality in laboratory and field populations. Neoplasia shown to result from viral infection
Mercenaria mercenaria	Germinomas (gametoblastomas, ovarian tumours)	Narragansett Bay, Rhode Island	3 of 1,300	Summer 1968	*20*	Metastases in kidney
	Germinomas (gametoblastomas, ovarian and testicular neoplasms)	Narragansett Bay, Rhode Island	12 of 316 females, 2 of 223 males	Summers of 1969 and 1970	*3*	1 metastasizing ovarian neoplasm
Macoma baltica	Haematopoietic neoplasms	Chesapeake Bay, Maryland	up to 10 %	Sep 1969 to Oct 1970	*5*	Seasonal variation, with peaks in March/April
Macoma irus and *M. nasuta*	Haematopoietic neoplasms	Yaquina Bay, Oregon	5 %	?	*10*	

*) Sources: *1* Alderman and co-authors (1977); *2* Appeldoorn and Oprandy (1980); *3* Barry and Yevich (1972); *4* Brown and co-authors (1977); *5* Christensen and co-authors (1974); *6* Couch (1969); *7* Couch and Winstead (1979); *8* Farley (1969a); *9* Farley (1969b); *10* Farley (1976b); *11* Farley and Sparks (1970); *12* Frierman and Andrews (1976); *13* Harshbarger and co-authors (1979); *14* Lowe and Moore (1978); *15* Newman (1972); *16* Otto and Farley (1976); *17* Pauley and Sayce (1972); *18* Pauley and co-authors (1968); *19* Wolf (1976a); *20* Yevich and Barry (1969); *21* Yevich and Barszcz (1977).

was composed of proliferating, epithelium-forming, irregular gland-like or cystic structures containing deposits of conchiolin and mucin, and replacing the normal tissue. Mitotic figures were present but not numerous (Pauley and Sayce, 1972). Although, apparently, no calcareous ridges or abnormal depositions of calcium carbonate were formed on the inner surface of the shell, the authors observed PAS-positive, conchiolin-like deposits in parts of the tumour, which frequently appeared as organized spheroids containing concentrically arranged layers as in pearls. Since the tumour had retained the mucus- and conchiolin-secreting functions of the mantle epithelium from which it originated, one may speculate whether it would represent an early stage of 'calcifying neoplasia', as reported by Galtsoff (1969) from *C. virginica* (p. 850).

An unusual, apparently invasive growth infiltrating and virtually replacing areas normally occupied by connective tissue and digestive tubules, has been studied in a specimen of *Crassostrea gigas*. The tumour, which was — possibly coincidentally — associated with the presence of 'multiple watery cysts' (p. 857), consisted of collagen and nervous tissue and was accompanied by marked inflammatory reaction and edemas in several parts. There was also an increase in the number of 'brown cells', which typically occurs in oysters in periods of stress and disease (pp. 535 and 857). The lesions appeared to have characteristics of both malignant and benign growths. Although no mitotic figures were seen, the tumour was considered to be neoplastic and classified as a ganglioneuroma (Fig. 13-194; Pauley and co-authors, 1968; Sparks, 1972).

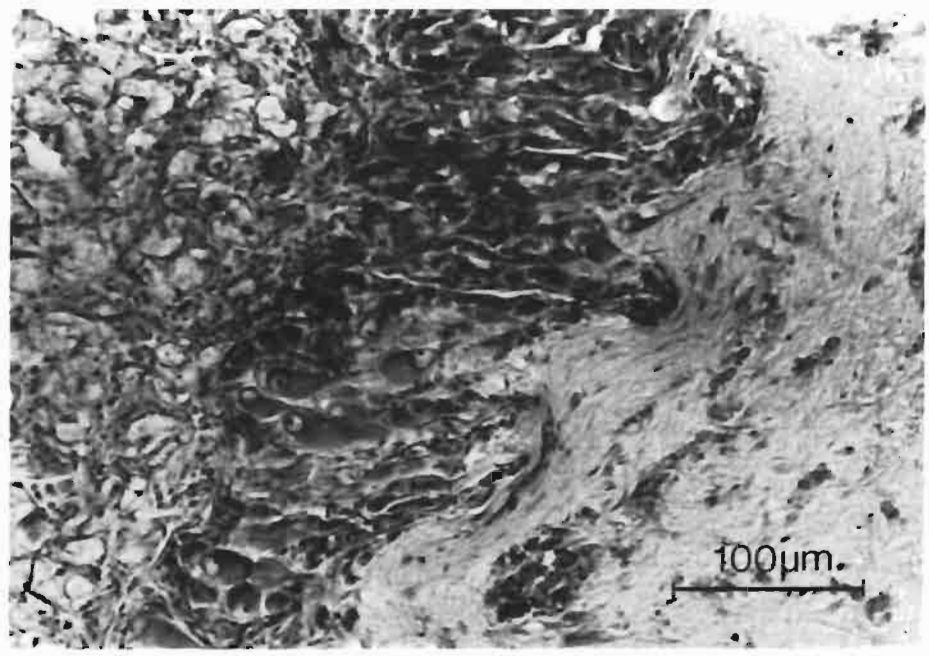

Fig. 13-194: *Crassostrea gigas*. Invasive nerve tumour (ganglioneuroma) in digestive-tubule area, which has replaced normal Leydig cells usually abundant in this area. Note normal Leyding tissue on left, extreme anisocytosis and anisokaryosis of tumorous nerve cells in centre, and abnormal amount of collagenous material on right. Masson's trichrome. (After Sparks, 1972.)

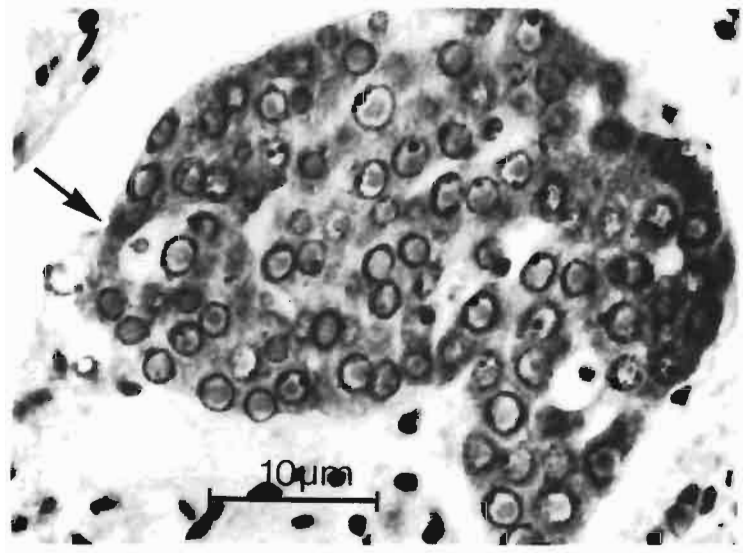

Fig. 13-195: *Mercenaria mercenaria*. Ovarian follicle filled with tumour cells apparently originating from germinal epithelium (arrow). (After Barry and Yevich, 1972.)

Germinomas — malignant tumours of the reproductive organs and of germ-cell origin — occur rather frequently in marine bivalves. Primary gonadal neoplasms (Fig. 13-195) have been detected in 12 of 316 female and in 2 of 223 male *Mercenaria mercenaria* from Rose Island, Narragansett Bay, Rhode Island (USA). In one of the females there was also an invasion of the red gland by large groups of neoplastic cells identical to those observed in the ovary. Furthermore, there was evidence of invasion of the heart muscle, as indicated by small foci of tumour cells and the presence of neoplastic cells lying free in the ventricle and pericardial cavity (Fig. 13-196). The ovarian follicles and genital ducts were also infiltrated by neoplastic cells.

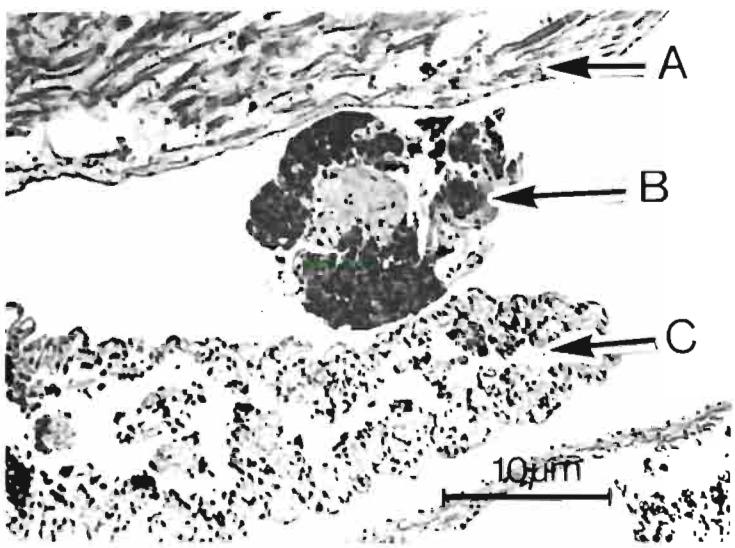

Fig. 13-196: *Mercenaria mercenaria*. Large group of neoplastic cells (B) in pericardial cavity between body wall (A) and heart auricle (C). (After Barry and Yevich, 1972.)

In the tumorous males, many testicular follicles were filled with large polyhedral cells containing vesicular nuclei with prominent red-staining nucleoli and clear pink cytoplasm. The neoplastic cells appeared to originate from the germinal epithelium of the testes and to proliferate into the testicular lumina. The same cells were also seen infiltrating the gonadal ducts. Extensive proliferation in some areas of the testes replaced the normal follicular architecture. Mitotic figures were evident in neoplastic cells. There was no invasion of surrounding tissues (Barry and Yevich, 1972).

The quahaugs had been collected during the summers of 1969 and 1970. Previously, 3 similar cases of primary ovarian neoplasms had been reported by Yevich and Barry (1969) in 1,300 *Mercenaria mercenaria* from Rhode Island waters, collected during summer 1968. In 1 of the 3 specimens, early invasion was observed in the kidney area, where definite clumps of tumour cells appeared in the supporting connective tissue. There was, at that time, no apparent relationship between incidence of tumorous quahaugs and water quality. Whether the apparent increase in incidence from 0.23 % in 1968 to 2.60 % in 1969/70 reflects an abrupt worsening of environmental conditions in the study area, has not been determined.

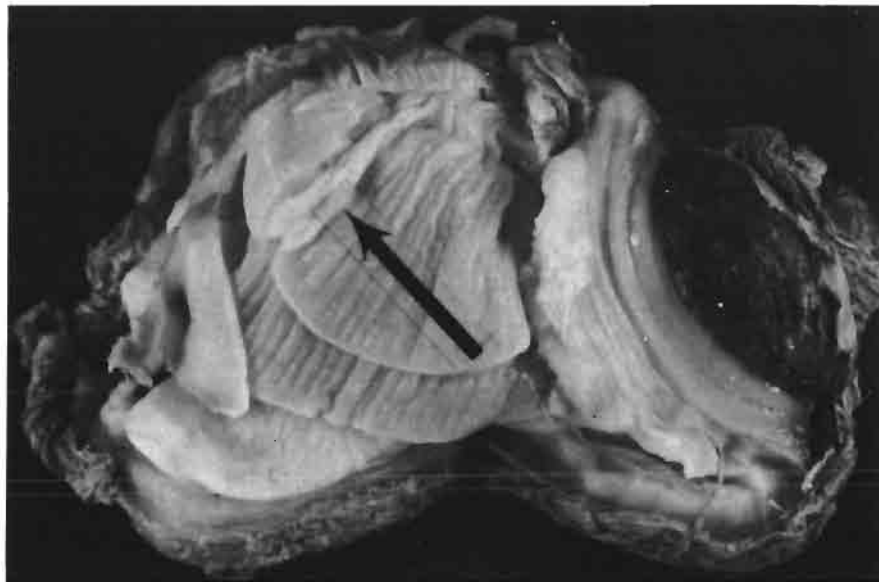

Fig. 13-197: *Mya arenaria.* Gross neoplastic gill lesion showing greyish-white mucosal surface (arrow). (After Barry and co-authors, 1971.)

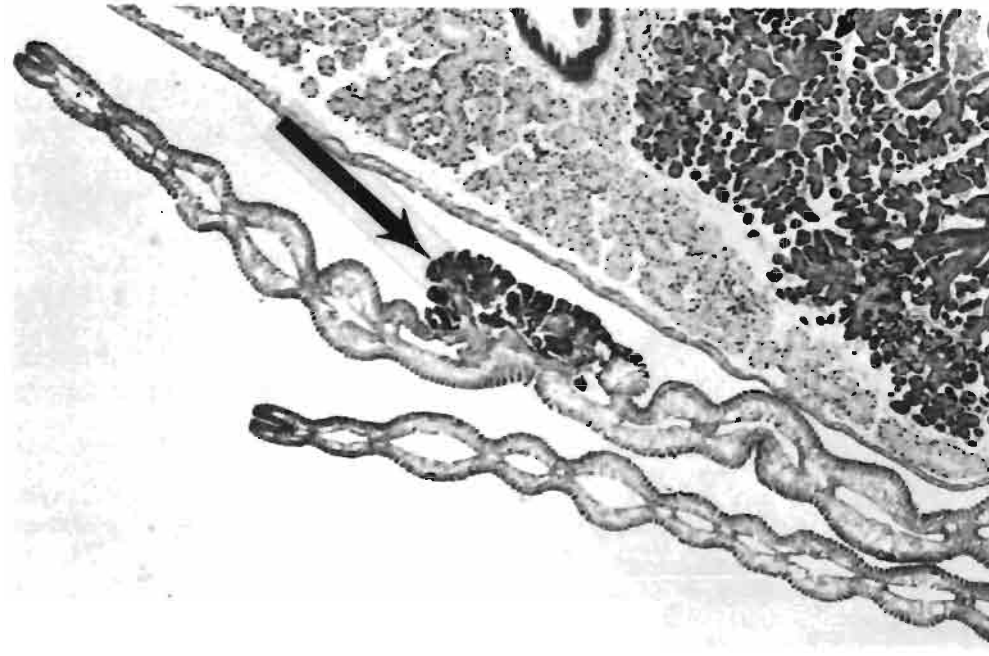

Fig. 13-198: *Mya arenaria.* Focal area of proliferative lesion on gill filament of inner lamella (arrow), outer lamella showing normal structure. H&E, × 39. (After Barry and co-authors, 1971.)

Water pollution has been suspected to influence the prevalence of neoplastic — particularly haematopoietic — lesions in marine bivalves. Of 940 *Mya arenaria*, collected from 4 geographic locations (Maine, Rhode Island, Maryland and California), 268 contained areas of hyperplasia; 194 showed hyperplasia of the gill filaments (Figs 13-197 to 13-199); and 143 exhibited atypical hyperplasia of the kidney epithelium, sometimes involving the entire organ, suggesting that normal function appeared improbable. Mitotic

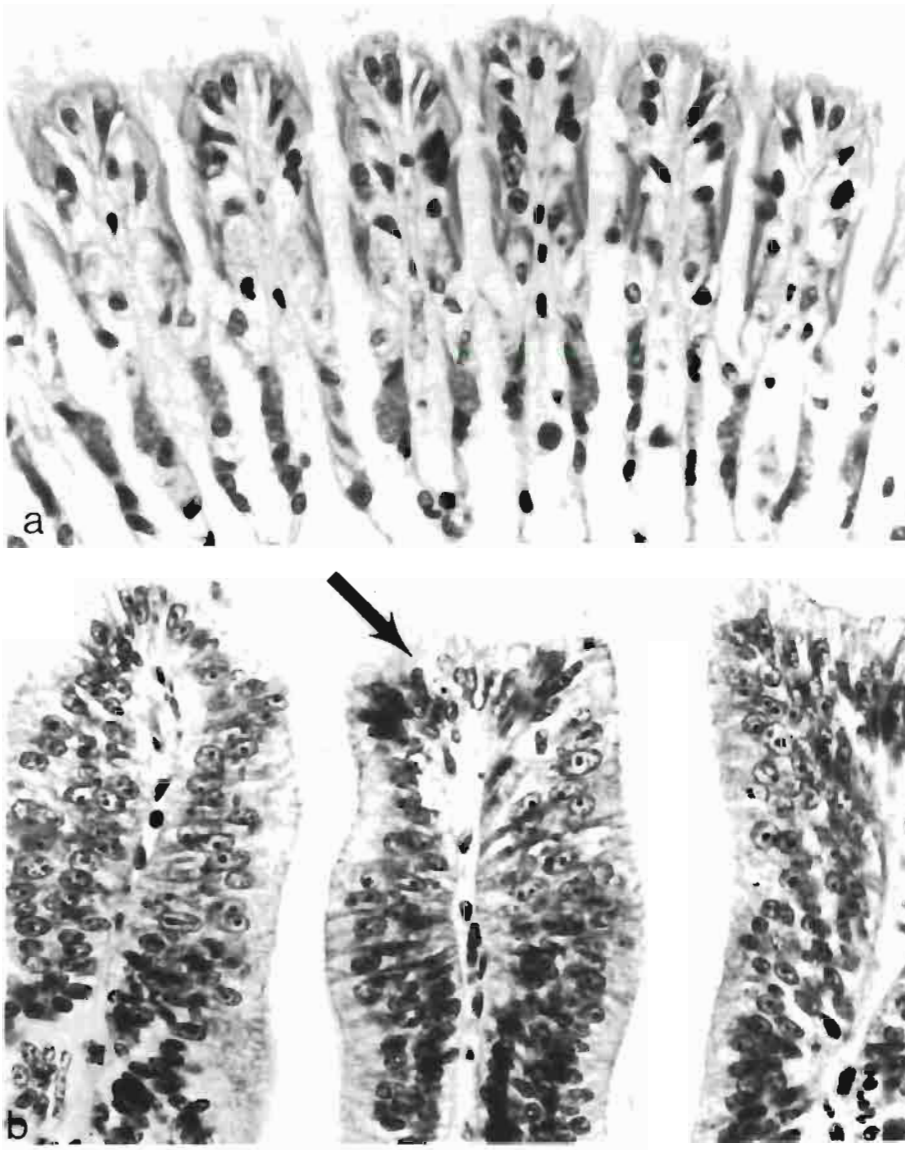

Fig. 13-199: *Mya arenaria*. (a) Normal gill filaments at higher magnification showing ciliated columnar epithelium. (b) Neoplastic gill filaments with increased number of cells containing large vesicular nuclei with prominent nucleoli. Note absence of cilia (arrow). H&E, × 1,875. (After Barry and co-authors, 1971.)

figures were abundant throughout the affected epithelia. Causative factors leading to such a high incidence of lesions in *M. arenaria* have not been identified, but since clams with the highest incidences were collected from localities surrounded by rural developments, it was speculated that the lesions might possibly result from man's impact on the coastal environment (Barry and co-authors, 1971). As shown by subsequent workers, the above-described 'hyperplastic' lesions in *M. arenaria*, as well as similar proliferative disorders discussed below, actually represent cases of haematopoietic neoplasia.

In July 1971, when approximately 25 % of the *Mya arenaria* population in Long Cove, Searsport, Maine (USA), had been killed by a March 1971 oil spill, collections of surviving clams for histological examination were made. The studies, continued for several years, revealed high incidences of gonadal neoplasms in clams from contaminated sites. Of a total of 2,056 soft-shell clams, collected from several stations in Long Cove, 124 (6.03 %) were found to be tumorous. The area of highest oil impact correlated with highest tumour incidence (up to 26.6 %) (Barry and Yevich, 1975).

Microscopic examination revealed neoplastic development, presumed to be of germ-cell origin, in the gonadal tissues of both male and female *Mya arenaria*. The lesions observed showed abundant mitotic figures and pleomorphic mononucleated, as well as multinucleated giant cells. Many of the nuclei were lobed and irregular, containing 2 or more prominent red-staining nucleoli, and were hyperchromatic due to the clumping of the nuclear chromatin. In some cases, the neoplasms had completely replaced normal gonadal tissue, making sexing of affected clams difficult or impossible. Some of the clams showed metastatic invasion of the interfollicular connective tissue of the gonadal area. Eventually, the normal architecture of such affected tissues was destroyed. Invasion of the body wall, efferent branchial blood vessels, epibranchial chamber, genital and urinary pores, kidney, pericardial wall, heart and red gland was also seen. In 1 individual, tumour cells had completely taken over the body, and the normal histology of the organs could not be determined any longer. At no time did any of the controls, collected from other areas, show indications of tumour formation (Barry and Yevich, 1975; Yevich and Barszcz, 1976, 1977).

Of a total of 3,213 *Mya arenaria* from Searsport, examined during the period 1971-75, 182 (5.66 %) had gonadal neoplasms, and of these, 73 (2.27 %) had metastases. Dissemination of the tumours occurred via 3 routes, i.e., (i) by direct extension or invasion through the walls of the follicles and gonadal duct into the visceral connective tissue; (ii) by neoplastic cells discharged through the genital pore into the epibranchial chambers, water tubes and gill ostia, which then colonized these sites; or (iii) by dissemination via sinuses and blood vessels within the visceral mass, through which neoplastic cells can move to other body organs. Small foci of neoplastic cells were found in blood vessels of the gill arches, with some cells lying free in the lumen and others being attached to the vascular wall. Apparently, the capillary gill bed trapped cells that had been shed into the blood sinuses. It was assumed that neoplastic cells clogging the capillaries of gill filaments may interfere with proper gas exchange (Yevich and Barszcz, 1977).

Although, initially, the gonadal neoplasms in *Mya arenaria* were believed to result from oil pollution, Yevich and Barszcz (1977) were not able to support this view. Exposure of soft-shell clams to several types of oil in the laboratory did not produce neoplasia. Appeldoorn and Oprandy (1980) found clams with low levels of neoplasia at both clean and polluted sites. Very low levels were encountered at some oil-polluted sites and

elevated levels were found at other such sites. The authors concluded that oil pollution was not a direct cause of neoplasia in *M. arenaria*. From 1967 to 1977, Yevich and his associates have examined more than 14,000 soft-shell clams, collected principally from coastal areas in Maine, Massachusetts, Rhode Island, Maryland and California. Some of these were from clean areas, others came from sites polluted by oil, heavy metals or other chemicals. No neoplasms were seen in *M. arenaria* from any of these areas other than the Long Cove oil-spill site and Harpswell Neck, Maine (Yevich and Barszcz, 1976, 1977).

A focal, tumour-like lesion was observed in 1 mature female out of approximately 5,000 *Crassostrea virginica* from Chesapeake Bay, Maryland. It replaced most of the normal connective tissue in the right mantle. A single type of cell predominated in the lesion; it was termed 'blastoid' because of its apparent lack of differentiation; it may be anaplastic or neoplastic. The cells ranged from 4.9 to 12.6 μm in diameter and had very large (3.5 to 10.5 μm) nuclei and scant amounts of cytoplasm. The nuclei were round, oval or bilobed, and had a conspicuous nucleolus. Rarely, binucleate blastoid cells were found. Mitotic figures were abundant; up to 7 were visible per oil-immersion field (Couch, 1969, 1970).

Haemolymph sinuses in the lesion were partially occluded by these abnormal cells, and large blastoid cells were found to occupy a perivascular focus in a blood sinus of the otherwise normal left mantle. Whether this was an independent focal lesion or a metastasis could not be determined, but the latter appears likely. Couch noted morphological resemblances of the lesions with certain mammalian reticular sarcomas, but was unable to trace the origin of the abnormal cells. The condition described by him, however, is consistent with haematopoietic neoplasia, as diagnosed by subsequent workers.

Concomitantly with Couch (1969, 1970), Farley (1969a, b) described several cases of similar lesions, diagnosed as haematopoietic or sarcomatoid, respectively, from *Crassostrea virginica, C. gigas* and *Mytilus edulis*. Of over 30,000 oysters, inspected during a 7-year period, 5 individuals of *C. virginica*, collected from different localities on the North American Atlantic coast, and 1 individual of *C. gigas* from Matsushima Bay (Japan), had the disease. The 6 oysters were distinctly moribund, exhibiting signs "indicative of fatal outcome if the disease had been allowed to run its course". The tissues of 4 of the oysters were in a 'watery condition', 3 had a pale digestive gland and mantle recession, and 4 had atrophied gonads (Farley, 1969a).

Microscopic examination revealed fibrosis and phagocytopenia in 4, and hyaline haemocytic infiltration in all individuals. Atypical haemocytes were common and showed changes comprising abnormally enlarged nuclei and unusual DNA patterns in pyknotic nuclei. Mitotically dividing hyaline haemocytes were common in invaded vesicular tissue. Atypical haemocytes in interphase contained abundant RNA in the cytoplasm. It was suspected that these presumptive haematopoietic neoplasms could be virus-induced. The extreme low incidence suggested a low infectivity rate or a strong innate resistance, possibly created or enhanced by the presence of mercenene or some other antineoplastic substance in the oysters, or by a rarity of transmissible stages of the presumptive virus (Farley, 1969a).

The diseased mussels described by Farley (1969b) came from a wild population in Yaquina Bay, Oregon. Of 43 *Mytilus edulis* collected in September 1968 and 75 specimens collected in February 1969, 3 and 7, respectively, exhibited sarcomatoid proliferative disorders. No gross abnormalities were recognizable externally, but microscopic examina-

tion revealed the presence of multiple local or diffuse lesions, consisting of abnormally large, undifferentiated cells, and varying from large tumorous masses in the mantle to small focal lesions in the connective tissue between the tubules of the digestive diverticula and in haemolymph spaces of the posterior adductor muscle.

The most striking atypical characteristic of the presumptive neoplastic cells was that both cell body and nucleus were distinctly larger than in normal haemocytes. The nuclei measured from 7 to 13 μm in diameter, as compared to 3 to 4 μm for normal haemocyte nuclei. The atypical cells had considerably more than the normal number of chromosomes, possibly polyploid sets, and their division was characterized by several aberrant features, such as division showing displaced groups of chromosomes and tripolar, as well as tetrapolar figures. Binucleate cells and abnormally shaped nuclei, some with multiple nucleoli, were also abundant. Mussels with large lesions or numerous atypical cells were in distinctly poor condition or even moribund, and gametogenesis was arrested (Farley, 1969b).

Of 471 *Mya arenaria* from Harpswell Neck, Maine, 48 (10.2 %) had similar haematopoietic neoplasms. Abnormal cells in the clams had large pleomorphic or lobed nuclei. Numerous and sometimes bizarre mitotic figures were seen in neoplastic cells in the gills, siphons, foot and connective tissues throughout the body. The abnormal cells were distinctly invasive, at times causing destruction of affected tissues and organs (Yevich and Barszcz, 1976, 1977). The authors were surprised by the fact that, at Harpswell Neck, Maine, soft-shell clams were only found with haematopoietic neoplasms, while individuals from Long Cove, Searsport, Maine, only had germinomas.

Similar haematopoietic neoplasms, somewhat resembling vertebrate leukemia, have been observed in *Crassostrea virginica* from the U.S. Atlantic and Gulf coasts (Farley and Sparks, 1970; Newman, 1972; Frierman, 1976; Frierman and Andrews, 1976; Otto and Farley, 1976; Couch and Winstead, 1979; Harshbarger and co-authors, 1979); in *C. gigas* from the U.S. Pacific coast (Farley and Sparks, 1970); in *Ostrea lurida* from the U.S. Pacific coast (Jones and Sparks, 1969; Mix, 1975, 1976a; Mix and co-authors, 1977); in *O. edulis* from France, Spain and Yugoslavia (Franc, 1975; Alderman and co-authors, 1977); in *Mytilus edulis* from the U.S. Pacific coast and from England (Mix and co-authors, 1977, 1979; Lowe and Moore, 1978; Green and Alderman, 1983); in *Mya arenaria* from the U.S. Atlantic coast (R. S. Brown and co-authors, 1976, 1977, 1979; Farley, 1976b; Appeldoorn and Oprandy, 1980; R.S. Brown, 1980); in *Macoma baltica* from the U.S. Atlantic coast (Christensen and co-authors, 1974; Farley, 1976a, b, 1977); and in *M. nasuta* and *M. irus* from the U.S. Pacific coast (Mix and co-authors, 1977) (Table 13-34).

In spite of intensive study, the true nature of these presumably neoplastic cells is not yet quite clear. Sparks (1972) first raised the question whether they could, in reality, be parasites. He further pointed out that the seasonal nature of the onset of the abnormalities, which has been observed in some of the above studies, is characteristic of virtually all known oyster epizootics of parasitic causation. The disease in *Ostrea lurida* from Yaquina Bay, Oregon, has been studied for several years. During early winter, the first stage of the disease invariably appears in a fairly large number of oysters. As the condition progresses through the intermediate, advanced and terminal stages, increasing numbers of oysters succumb. Those examined microscopically exhibit the characteristics of the proliferative disorder. Seasonality of onset and progression, as well as the apparent contagious nature of the epizootic, were highly suggestive of an infectious disease with possible parasitic

involvement. However, no protistan of known pathogenicity was ever found in the diseased oysters (E. J. Jones and Sparks, 1969; Sparks, 1972).

In a highly provocative paper, Mackin and Schlicht (1976) argued that the presumed neoplams of oysters and clams, described by Couch (1969), Farley (1969a, b), Christensen and co-authors (1974), and others, have the characteristics of *'Labyrinthomyxa patuxent'* (Fig. 13-20), a labyrinthulid protistan (see section 'Agents: Labyrinthomorpha'). Sparks (1972) has summarized these characteristics as: (i) Nuclear enlargement, (ii) unusual frequency of mitoses in stained preparations, some of which appear to be multipolar, (iii) excess of chromosome numbers over those in host cells, (iv) high frequency of lobed nuclei, (v) prominent nucleolus, sometimes 2 or more in a single nucleus, (vi) sparse but highly basophilic cytoplasm, and (vii) occurrence of bi- and multinucleate cells. It was assumed that no other known protistan displays these characteristics. Mackin and Schlicht (1976, p. 18) further conclude:

> "The cells, assumed to be of haemopoietic origin, also have certain other characteristics. These are as follows: 1) The mitoses are intranuclear, a characteristic of protistans; 2) the cells frequently become encysted; 3) they may be formed as endogenous buds; and 4) in addition to 'normal' mitoses, some of the mitotic figures are of the cruciform type characteristic of the Plasmodiophorales (see Karling, 1944) and the problematic *Phagomyxa algarum* (Karling, 1944), which the author believes is a species of *Labyrinthula* Cienkowski (1867).
>
> Only one group of the Protista combines all of the characteristics described for the assumed neoplastic diseases by the various authors. The list of characteristics was added to by the author from study slides contributed for use of participants at the shellfish pathology conference*). This group is the Labyrinthulales Cienkowski (1867). Plasmodiophorales apparently has all characteristics excepting only the capacity to reproduce by endogenous budding.
>
> Analysis of the papers of proponents of the neoplasm theory shows that their case is based on the fallacious assumption that mitoses observed in the enlarged cells are abnormal. Particularly singled out is the apparent presene of polycentric mitoses. These are, in fact, not polycentric. In the enlarged nuclei, the product of the first mitosis is two nuclei, both occupying the area of the original one nucleus, and flattened against each other (Fig. 13-20,c**). On initiation of the second division in the two daughter nuclei, division of the nucleoli precedes the formation of the equatorial plate. A peculiarity of the group is that the poles thus established by the nucleoli may lie at right angles to each other in the two daughter nuclei. In this manner simultaneous mitoses in the two daughter nuclei may be interpreted as one polycentric mitosis. Because the tetrad of small nuclei, still confined to the area of the original large nucleus, may initiate further crowded mitoses, the confusion is compounded.
>
> The burden of proof lies with the proponents of the 'neoplasm' theory. They must prove that carcinogenic or radiational damage to leucocytes of shellfish, or spontaneous changes in leucocytes, can produce alteration of chromosome number ending in a larger number by a significant margin. They must also prove

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* Scarpelli and Rosenfield (1976)

** Figure number of original paper changed to indicate numbers adopted in this chapter.

that leucocyte mitoses are intranuclear, that leucocytes may encyst, and that they may be multinucleate."

A disease involving 3 of 50 *Mya arenaria*, collected from Umpqua Bay, Oregon, closely fitted the above picture, as delineated by Mackin and Schlicht (1976). Histologically, the lesions resembled haematopoietic neoplasms, but appeared to be associated with the presence of small, difficult to see, protistan parasites. These were similar to *'Labyrinthomyxa patuxent'*, and the condition resembled leukemoid response in mammals (Farley, 1976b; Fig. 13-200). However, Farley stated that in neoplasias of mollusks, evidence of parasitic involvement is lacking, and anaplasia and mitotic activity are particularly evident in the haemic neoplasms.

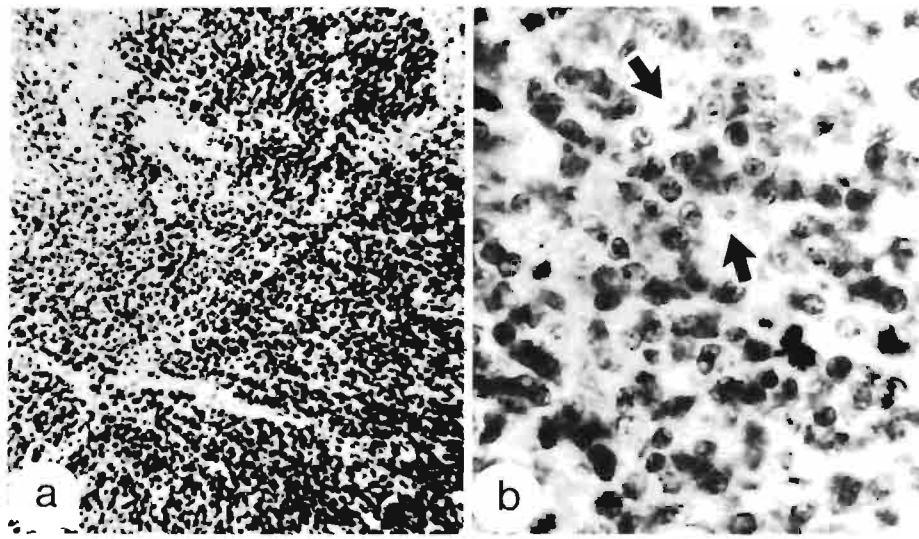

Fig. 13-200: *Mya arenaria*. (a) 'Leukemoid response' produced by unidentified protistan parasite (*Labyrinthomyxa patuxent*?). H&E stain, blue stain (FPM), × 250. (b) Protistan-like cells (arrows) staining differentially with Giemsa, × 1,000. (After Farley, 1976b.)

Similarly, Mix and co-authors (1977) emphasized that there is no support of the view that the large cells represent some form of unidentified protistan symbiote. Couch and Winstead (1979) reported on the concomitant occurrence of haematopoietic neoplasia and *Perkinsus marinus* infestation in an individual of *Crassostrea virginica* from Apalachicola Bay, Florida. Both protistan and neoplastic cells were observed in close proximity in the vesicular connective tissue of the oyster, but there was no morphological evidence that the two conditions are related.

Farley (1969a) suspected that haematopoietic neoplasms in *Crassostrea virginica* might be induced by viral infections. However, while studying haematopoietic neoplasms in *Macoma baltica* at the ultrastructural level, he (Farley, 1976a) found no signs of virus involvement in the neoplastic disease of this clam species.

Subsequently, Appeldoorn and Oprandy (1980) isolated virus-like particles from New England *Mya arenaria* suffering from haematopoietic neoplasia. No such particles occurred in healthy clams. The viral agent was found to have a form and properties similar

to other known cancer-causing viruses. Clams inoculated with purified viruses obtained from diseased cells became neoplastic within a short period of time. As a final step toward the satisfaction of Koch's postulates, viruses were found and reisolated from experimentally infected clams.

Appeldoorn and Oprandy's (1980) pioneering experiments were duplicated and verified by R. S. Brown (1980) and Oprandy and co-authors (1981). The latter authors showed the causative agent of haematopoietic neoplasia in *Mya arenaria* to be a B-type retrovirus (see section 'Agents: Virales'). R. S. Brown and co-authors (1976) and Cooper and co-authors (1982) described non-destructive *in vivo* bleeding techniques, involving (1) phase-contrast microscopy with fresh unstained haemocytes, and (2) bright-field microscopy with Giemsa-stained blood cells, for the diagnosis of haematopoietic neoplasms in soft-shell clams. They observed a positive correlation between the degree of tissue involvement and the number of circulating neoplastic cells. Depending on the percentage of abnormal cells present in the haemolymph, the lesions were graded from level 1 (light) to 5 (severe). In general, the accuracy of a single *in vivo* diagnosis varied from 66 to 71 % at level 1 and from 76 to 93 % at level 2, while it was 100 % at levels 3 to 5. The total percentage of clam neoplasms detected by the *in vivo* bleeding technique was 89 to 91 %, and the percentage of non-neoplastic clams detected was 95 %. R. S. Brown and co-authors (1976) have summarized the cytological characteristics of normal *M. arenaria* haemocytes and the 2 types of circulating neoplastic cells (Table 13–35). The occurrence of similar cells and neoplasms in other bivalve species has, thus far, not yet been found to be associated with, and induced by, viral agents.

As demonstrated by histological studies conducted during most of the above investigations, tissue destruction caused by infiltration of neoplastic cells is intense. Usually, earliest lesions appear to develop multifocally as local neoplasms in the gill epithelium,

Table 13–35
Mya arenaria. Cytological characteristics of normal haemocytes and neoplastic cells (After R. S. Brown and co-authors, 1976)

Blood Cell	Neoplastic Cell	
	Type II	Type I
Mean cell diameter = 6.0 μm, regular outline	Mean cell diameter = 6.6 μm, regular outline	Mean cell diameter = 8.3 μm, irregular outline
Well differentiated	Less differentiated	Anaplastic
Nucleus round, regular, densely basophilic, mean diameter 2.7 μm	Nucleus round, larger, homogeneous, clumped chromatin, basophilic, mean diameter 4.2 μm	Very large vesicular nucleus, lobed or binucleate, basophilic, mean diameter 6.5 μ
Nucleolus indistinct	Nucleolus distinct, smaller than Type I	Larger, prominent, eosinophilic nucleolus
No mitoses	Mitoses rare	Mitoses frequent
Abundant, homogeneous granular cytoplasm, brightly eosinophilic	Less cytoplasm, distinctly eosinophilic	Cytoplasm scant, pale
Nucleus: cytoplasm ratio 1 : 1.8	Nucleus: cytoplasm ratio 1 : 2.8	Nucleus: cytoplasm ratio 1 : 3.6

suggesting a target area in body surfaces exposed to the environment. As the disease progresses, normal tissues are gradually invaded and replaced rather than displaced by neoplastic tissue. Eventually, death ensues. Whether, under certain conditions, recovery of affected bivalves may occur, is not known.

Prevalences of haematopoietic neoplasms in bivalve populations and resultant mortalities may locally reach epizootic proportions (Table 13-34). Christensen and co-authors (1974), for example, observed dramatically increased death rates in tank-held neoplastic individuals of *Macoma baltica* previously collected from subtidal mud flats off Wells Point in Broad Creek, Maryland.

Field observations conducted by Appeldoorn and co-authors (1979) and Appeldoorn and Oprandy (1980) indicate that neoplasia could affect over 50 % of the individuals in *Mya arenaria* populations on the North American Atlantic coast from Cheasapeake Bay to Nova Scotia and reduce soft-shell clam production by as much as 20 %.

Frierman (1976) and Frierman and Andrews (1976) provided support of pathological evidence that haematopoietic neoplasms in *Crassostrea virginica* cause deaths of oysters. Two groups of inbred oysters exhibited particularly high prevalences of the disease. Thirty-one neoplasms were diagnosed in 369 oysters from these 2 lots. In susceptibles, lesions appeared at an age of 1 year with severe mortalities. In contrast, only 39 cases were recorded from 51,773 individuals examined from 1964 to 1973 in other progeny lots and native imports. The authors concluded that genetically susceptible races may provide excellent materials for studies of neoplasms in molluscs.

The fairly recent discovery of haematopoietic neoplasms in marine bivalves provides much space for speculations. Thus, Alderman and co-authors (1977) considered it possible that some of the unexplained mass mortalities of *Ostrea edulis*, as reported from some European oyster growing areas, may have been caused by haematopoietic neoplasia. Such abnormally high mortalities, ranging from 20 to 90 % of the population, have been experienced for at least 15 years in the Mali-Ston area near Dubrovnik (Yugoslavia) mainly during the period from April to October. Similar conditions have been recorded in the Ría de Noya, Galicia (Spain) since the introduction of *O. edulis* culture in that area, June to September mortalities reaching 60 to 80 % in recently imported French stocks.

Oysters from both Yugoslavia and Spain showed heavy levels of haemocytic infiltration of the connective tissue mainly of the digestive-gland tubules, gut and surface tissues. Infiltrating haemocytes, which had abnormally large nuclei, were found in 20 to 35 % of the oysters sampled. The connective tissues of affected individuals were in generally poor condition, but the gonads, in contrast, were often exceedingly full. Italian Adriatic oysters laid in the Ría de Noya were not reported as being subject to any mortality but, nevertheless, when examined showed up to 45 % infiltration by the abnormal giant-nucleus type of haemocyte.

Previously, E. J. Jones and Sparks (1969) had observed similar unusually large cells of (at that time) unknown origin and identity in the Leydig tissue of diseased individuals of *Ostrea lurida* from Yaquina Bay, Oregon. The appearance of these cells coincided with oyster mortalities in that area. A subsequent report (Farley, 1969b) provided additional information about similar cells in *Mytilus edulis* from the same area and ascertained their neoplastic nature.

Against the background of this information, it would be tempting to speculate on the possible involvement of neoplastic (haematopoietic) development in the recurrent but

largely unexplained *Crassostrea gigas* mass mortalities experienced in Hiroshima and Matsushima Bays, Japan (see section 'Agents: Bacteria'). Apparently, detailed studies of oyster-blood cell morphology have not been conducted during the mortality periods, but Fujita and co-authors (1953) briefly stated in the abstract of their Japanese-language paper that "many small and large round cells had invaded into organs of some individuals, especially to their ovaries, diverticulae and intestines and we found destroyed ovary cells to be phagocytized and tissues between lobes of diverticula almost in state of necrosis with many round cells invading around there, which is diagnosed to be possibly an acute inflammation". What, if these 'round cells' actually were abnormal (neoplastic) haemocytes? Similarly, Tamate and co-authors (1965) reported infiltration of *C. gigas* connective tissues by a "larger type of amoebocyte", and Numachi and co-authors (1965) observed "amoebocytes of large size" and morphologically different from normal ones, in enormous numbers in oysters undergoing high mortality. The nature and origin of these abnormal blood cells have not been determined.

Accumulating evidence indicates that proliferative disorders, and particularly haematopoietic neoplasia, are of common occurrence in marine bivalves. Although a — suspected — direct correlation between (oil-)pollution and neoplastic lesions has not definitely been established, their apparently increasing prevalence parallels the increasing deterioration of marine and estuarine ecosystems and directly reflects man's impact on the environment.

The discovery by Appeldoorn and Oprandy (1980) of the viral etiology of haematopoietic neoplasia in *Mya arenaria* and its transmissibility, as well as the development of simple techniques for diagnosing and inducing the diasease, has opened a new avenue of investigation, which could help to answer a great many of open questions.

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14. DISEASES OF MOLLUSCA: AMPHINEURA

G. LAUCKNER

The class Polyplacophora (chitons or coat-of-mail shells) in the molluscan subphylum Amphineura includes some 1,000 exclusively marine species. The pallial groove of chitons is an ideal environment for commensals, offering them excellent protection against desiccation and predation. Annelids of the polynoid genus *Arctonoë* are common associates of *Cryptochiton stelleri*. In the Monterey Peninsula area, California, for example, nearly 60 % of the *C. stelleri* population carry these worms (Webster, 1968). *Arctonoë* spp. have a considerable host range. Their relationship with the various invertebrates is clearly commensalistic (Davenport, 1950). The same holds true for the crustacean associates of chitons (see below), although their presence, on the gills, may inflict some degree of mechanical irritation or even damage. In spite of their great intertidal abundance, the placophorans appear to be relatively free from true metazoan parasites. Only a few protozoal but no microbial diseases have been recognized in these molluscs.

DISEASES CAUSED BY PROTISTANS

Agents: Apicomplexa and Ascetospora

Coccidians *Pseudoklossia chitonis* invade the digestive-gland cells of *Acanthochites* (= *Acanthochiton*) *fascicularis* on the English and French coasts of the English Channel. Almost all individuals taken at Plymouth were found infested, and most of these heavily (Debaisieux, 1919, 1922). Up to 12 macrogametes, 15 µm long, as well as numerous microgametes, may be seen in a single host cell. Fertilization and sporogony have not been observed but were presumed to take place in the intestine or outside the host. The morphology and life-cycle stages of the parasite were not described in detail but were said to be very similar to those of *Pseudoklossia patellae* parasitizing limpets *Patella vulgata* from the same localities (Vol. I, p. 317). In the older literature, stages of the haplosporidian*) *Haplosporidium chitonis*, later misnamed *Minchinia chitonis*, have been mistaken for those of the coccidian *Pseudoklossia chitonis*, and *vice versa* (Debaisieux, 1919, 1920, 1922). Pellérdy (1974) correctly lists *'Minchia chitonis'* (a misprint for *Minchinia*) as a synonym of *P. chitonis* Debaisieux, 1920, although he incorrectly credits Lankester (1885) instead of Labbé (1896) with the naming of the genus (see footnote on p. 964/965).

*) The classical Haplosporidia are now in order Balanosporida, phylum Ascetospora (see Table 13-3).

Acanthochiton fascicularis harbours another, yet poorly studied and imperfectly classified coccidian in the salivary gland. Young intracellular schizonts were about 3 μm in diameter, and fully developed ones measured up to 12 μm. These contained some 100 merozoites. Macrogametes, 20 × 15 μm, and spores, 20 × 10 μm in dimension, were also seen. The development of the parasite was believed to remind somewhat of that of eimeriid coccidians (Debaisieux, 1919). The agent was not named and, apparently, has not been restudied. No mention of it has been made in Pellérdy's (1974) monograph on Coccidia and coccidiosis.

Hatt (1931) listed *Chiton* (*Middendorfia*) *caprearum* as intermediate host for porosporid gregarines *Nematopsis legeri*, which also occur in a variety of gastropods (Vol. I, p. 317) and bivalves (p. 545). Decapod crustaceans have been identified as definite hosts of this sporozoan.

Balanosporidans *Haplosporidium chitonis* parasitize European Atlantic chitons *Craspidochilus* (= *Lepidochiton*) *cinereus*. The literature on this sporozoan is higly confused. Originally believed to be a coccidian, it was placed in the genus *Klossia* Schneider, 1875, as *K. chitonis* by Lankester (1885). Labbé (1896) described what he erroneously believed to be the same parasite (but which was in fact a species-mix) as *Minchinia chitonis*. Pixell-Goodrich (1915) showed that the sporozoan in *L. cinereus* belongs to the Balanosporida. Although she did not find the coccidial stages observed by Labbé (1896), she unfortunately and unjustifiedly adopted the generic name *Minchinia* for the balanosporidan in *L. cinereus*. Debaisieux (1919, 1920) discussed the errors made by Labbé (1896) and Pixell-Goodrich (1915) and correctly renamed the chiton parasite *Haplosporidium chitonis*. Unfortunately again, Sprague (1963), in his revision of the genus *Haplosporidium*, restored *Minchinia* Labbé, 1896, and listed *M. chitonis* as type species. The justification for Sprague's restoration of the genus *Minchinia* is open to serious debate (see footnote).

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1) Lankester (1885) described sporozoan spores found in an undetermined species of *Chiton* (probably *Lepidochiton cinereus*) from England, which he believed to be those of a coccidian of the genus *Klossia* Schneider, 1875.

2) Pixell-Goodrich (1915) demonstrated that the sporozoan in *Lepidochiton cinereus* is actually a haplosporidian (balanosporidan), not a coccidian.

3) Labbé (1896, 1899) discovered sporozoan stages in *Acanthochiton fascicularis* from Roscoff, France, which are clearly coccidians. He simultaneously observed spores (of a haplosporidian, later named *Haplosporidium chitonis*) in chitons (probably *Lepidochiton cinereus*) obtained from England. In combining the stages of both (quite unrelated) sporozoans, he pieced together a fictive life cycle and named the species-mix *Minchinia chitonis*. A protistan similar to '*M. chitonis*' was said by Labbé to occur in *Patella vulgata* and *Trochus* (= *Calliostoma*) sp.

4) Debaisieux (1919, 1920) showed that the haplosporidian spores observed by Lankester (1885) in chitons (*Lepidochiton cinereus*?) are identical to those found in the same host species (given as *Craspidochilus cinereus*) at Plymouth. Development and morphology of the parasite share generic characteristics with members of the genus *Aplosporidium* Caullery and Mesnil, 1899, (the name of which was changed into *Haplosporidium* by Lühe, 1900). The species was described as '*Haplosporidium* (*Minchinia*) *chitonis*'. Its correct designation is *Haplosporidium chitonis* (Lankester, 1885) Debaisieux, 1920 (Debaisieux' 1919 publication, in which the above name was proposed, does not contain a valid species description).

5) According to Debaisieux (1919, 1922), the protistan stages found by Labbé (1896) in *Acanthochiton fascicularis* and named *Minchinia chitonis* are actually coccidians, which share generic characteristics with the genus *Pseudoklossia* Léger and Duboscq, 1915. The species was described as *P. chitonis*. Its correct name is *Pseudoklossia chitonis* Debaisieux, 1920 (Debaiseux' 1919 publication in which the above name was proposed does not contain a valid species description).

Early stages of *Haplosporidium chitonis* infestation are easily diagnosed by gross inspection of the digestive gland of *Lepidochiton cinereus*. It is normally brownish, but in infested hosts it assumes a whitish colour, due to the presence of numerous colourless plasmodia and young sporocysts (Pixell-Goodrich, 1915). In heavy infestations, the late stages of the parasite cause a progressive darkening of the foot and gills, which is due to the large number of dark brown mature spores disseminated throughout these organs (Fig. 14-1). Fully formed sporocysts vary in size, with a diameter range of 18 to 50 μm, the smaller ones containing about 75 and the largest some 400 spores. Mature spores of *H. chitonis* are ovoidal, with a flattened pole covered by an operculum, and have tail-like projections at either end, which are extensions of the cytoplasmic envelope or extraspore cytoplasm covering the spore body and the operculum. Fresh mature spores are fairly uniform in size, 9 to 11 μm long and 6 to 8 μm wide, with projections measuring 30 to 42 μm long (Fig. 14-2).

Sporocysts in muscle tissue (Fig. 14-1, b) often appear as foci isolated from one another as if the parasites had invaded separate areas independently. In heavy infestations, large areas of muscle are replaced by spores and the digestive gland is destroyed (Fig. 14-1, d). The gills are also often invaded. No hypertrophy or cellular reactions are discernible. It seems, therefore, that the chiton has no control over the development of the parasite. The number of sporocysts decreases in direct proportion to the distance from the intestine and digestive gland, suggesting an invasion via the alimentary tract. It is remarkable that the host survives such heavy infestations when large areas of tissue are replaced by *Haplosporidium chitonis* sporocysts (Ball and Neville, 1979). Although mortality studies have not been conducted, it appears that the pathogenicity of *H. chitonis* is less severe than that of other species of *Haplosporidium* (*'Minchinia'*) parasitizing bivalves (Chapter 13).

The earliest intracellular stages of *Haplosporidium chitonis* observed in *Lepidochiton*

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6) To date, *Pseudoklossia chitonis* (Coccidia) has only been reported from *Acanthochiton fascicularis* but never from *Lepidochiton cinereus*. Evidently, *Haplosporidium chitonis* only occurs in *L. cinereus* but not in *A. fascicularis* (Debaisieux, 1919, 1920, 1922). It does neither occur in *A. crinitus* collected from the same regions in which *L. cinereus* is heavily infested (Ball and Neville, 1979). Strict host specificity is, therefore, indicated for both species.

7) As a consequence, *Acanthochiton fascicularis* is not available as type species for any known species of balanosporidan. The stages described by Labbé (1896) as *'Minchinia chitonis'*, for which *A. fascicularis* is the type host, are definitely coccidians, namely *Pseudoklossia chitonis* Debaisieux, 1920. Hence, *Michinia* has to be regarded as a *nomen nudum*.

8) Presently, the invalid genus *'Minchinia'* contains balanosporidans whose spores have an operculum which opens like a lid to permit escape of the amoeboid sporoplasm. In the 'type species', *M. chitonis*, the lid has a margin extending beyond the orifice which it covers and hanging over the rim of the body of the spore wall. The valid genus *Haplosporidium* contains several species in which the lid on the spore does not have an overhanging margin (Sprague, 1970a). Electron microscope studies have proven this distinction between the 2 genera invalid (Ormières and de Puytorac, 1968; Perkins, 1968, 1969; Rosenfield and co-authors, 1969; Sprague, 1970b; and others). Sprague (1970b, p. 328) concluded: "Now we must either distinguish them on another basis or transfer all operculate species to genus *Minchinia* and suppress *Haplosporidium*." In a subsequent paper, however, Sprague (1978) returned all balanosporidans previously transferred by him (Sprague, 1963) to *Minchinia*, back to the genus *Haplosporidium*. The problem has not yet been solved. In his latest comment on the 'Classification of the Haplosporidia', Sprague (1979) retained both genera in the family Haplosporidiidae in the newly established order Balanosporida, which replaces the well-known name Haplosporida. As shown above, *Michinia* is an invalid genus and can, therefore, neither be included in the Coccidia nor in the Balanosporida.

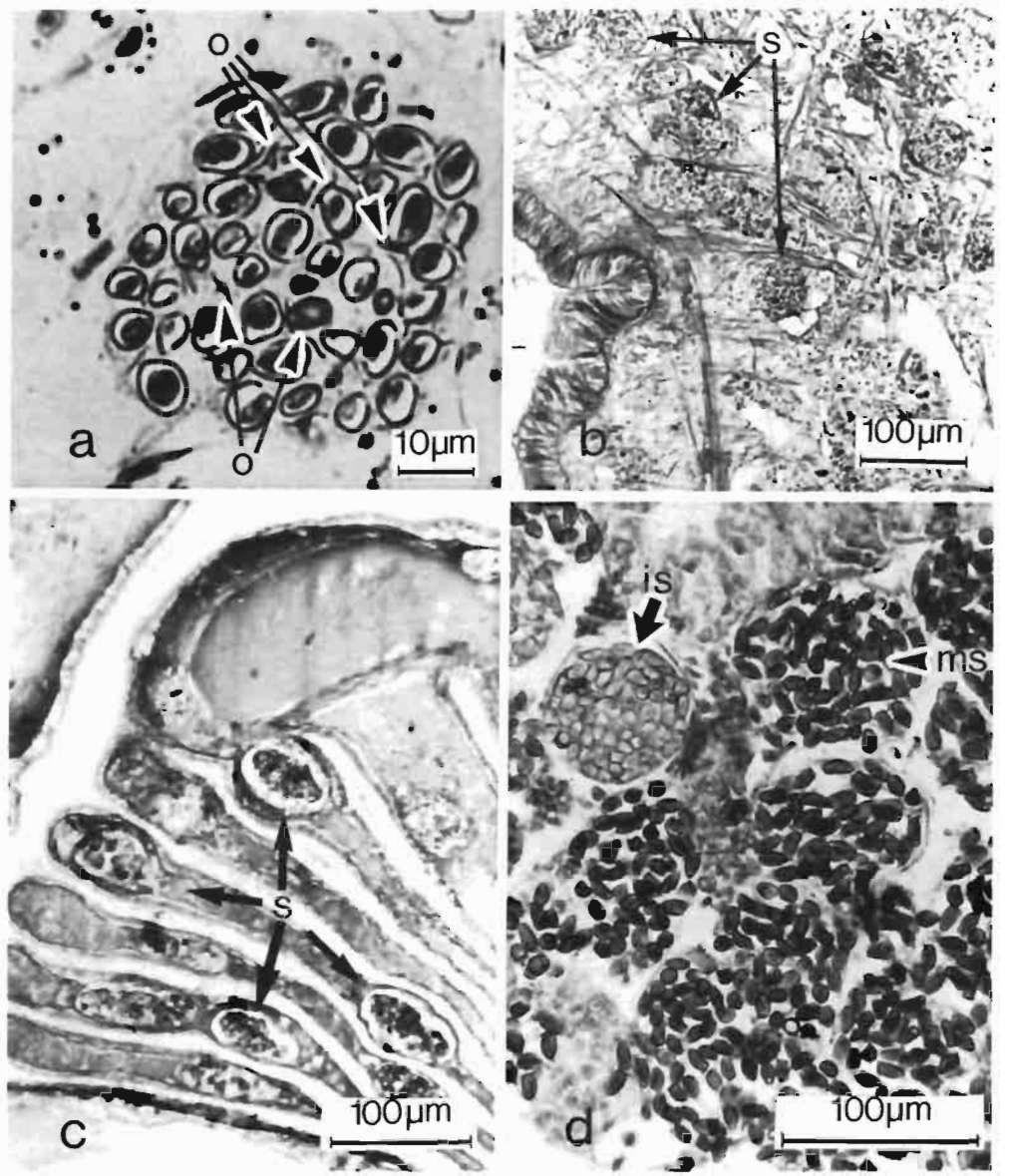

Fig. 14-1: *Haplosporidium chitonis* in *Lepidochiton cinereus*. (a) Section of sporocyst with mature spores. Note thick walls and opercula (o); (b) section showing sporocysts (s) in foot musculature; (c) sporocysts in gill filaments; (d) digestive gland with mature (ms) and immature (is) sporocysts. (After Ball and Neville, 1979.)

cinereus are spherical binucleate plasmodia, about 5 μm in diameter, which occur in epithelial cells of the digestive gland or gonad. Later stages are histozoic in the connective tissue of the digestive gland, the perivisceral region and other tissues. By growth and division of the nuclei, which usually remain paired, the binucleate plasmodia develop into multinucleate plasmodia. When the latter have reached their full size, the nuclei have

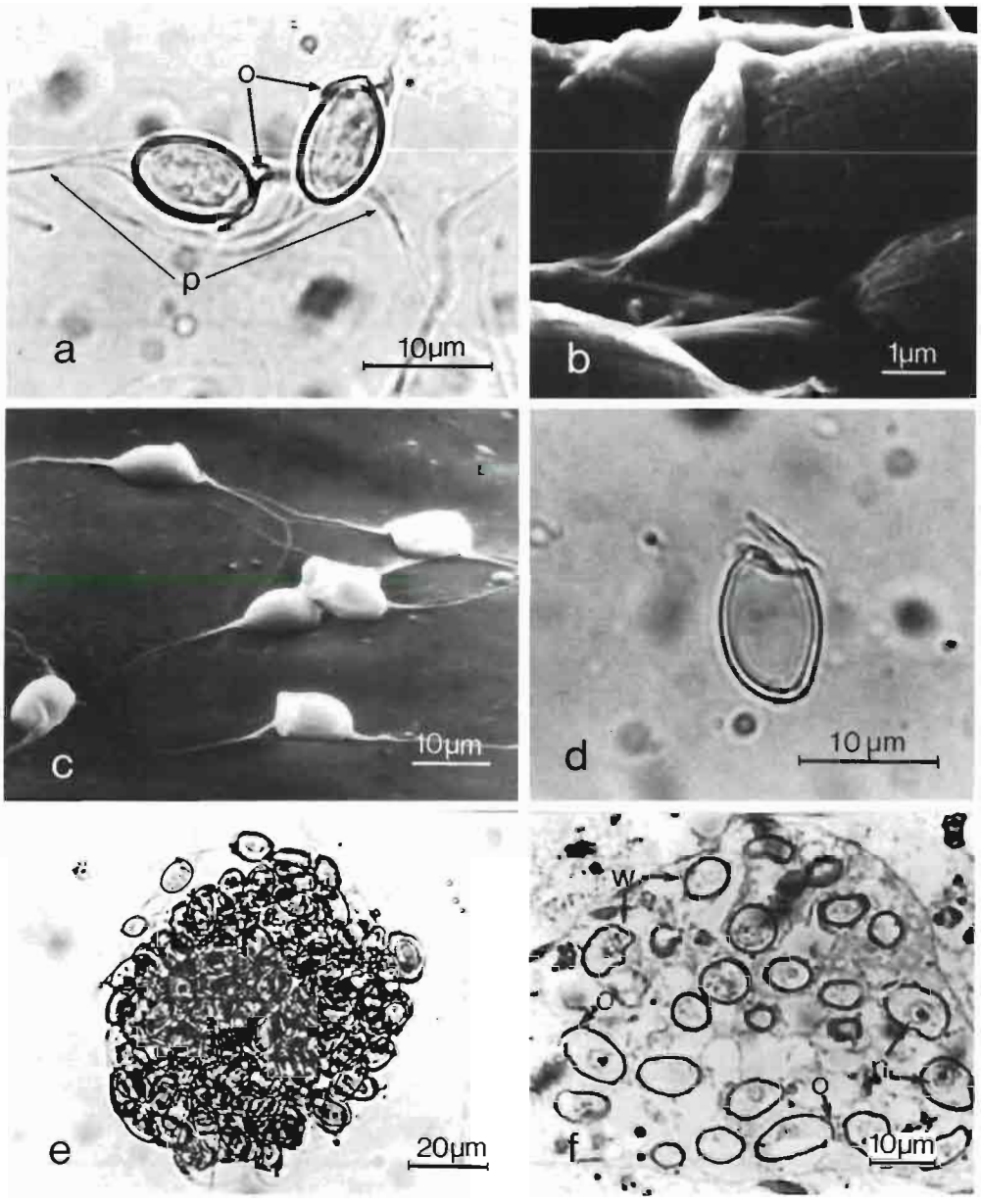

Fig. 14-2: *Haplosporidium chitonis* in *Lepidochiton cinereus*. (a) Mature spores showing operculum (o) and projections (p); (b) scanning electron micrograph of spores showing details of both ends; (c) entire spores; (d) squash preparation of spore with raised operculum; (e) sporocyst with mature spores; (f) sporocyst with immature spores showing thin wall (w), opercula (o) and nuclei (n). (After Ball and Neville, 1979.)

separated and grown considerably. Whether the enlargement is due merely to growth, or to fusion of the paired nuclei, is unknown. Division of the large nuclei results in the production of numerous smaller nuclei, which is followed by fragmentation of the plasmodium into smaller units containing 1, 2 or more nuclei. The binucleate forms may start the cycle again. It has been suggested that in some of the latter the nuclei may fuse and give rise to uninucleate sporoblasts. These develop directly into spores. The sporoblast stage appears to be very transitory, since it is very scarce in comparison with the abundance of the preceding and following stages.

In spite of the extensive studies on the life cycle of *Haplosporidium chitonis*, no sexual phase has yet been observed (Pixell-Goodrich, 1915; Debaisieux, 1920; King, 1926; Ball and Neville, 1979). The mode of spore escape from one host and infestation of new hosts is also unknown. Crabs, sea stars and blennies feed on chitons. The spores of *H. chitonis* pass through the intestinal tracts of these predators unchanged but are disseminated in this way. When chitons were exposed to free spores, numerous unhatched and unchanged spores, often enclosed in faecal pellets, were found in the intestine of several of these. In no case did the spores show any sign of opening. Whether their germination requires passage through an additional host, is unknown (Pixell-Goodrich, 1915).

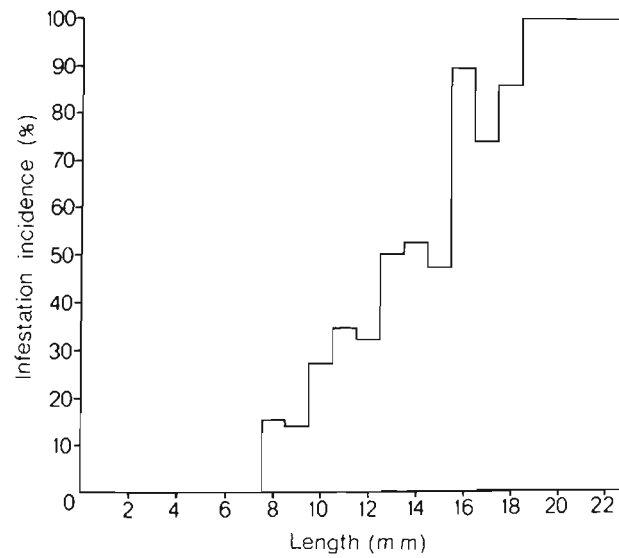

Fig. 14-3: *Lepidochiton cinereus.* Relation between frequency of infestation with *Haplosporidium chitonis* and length. (After Baxter and Jones, 1978.)

The vegetative and sporogonic stages of *Haplosporidium chitonis* appear to be restricted to *Lepidochiton cinereus*. *Acanthochiton crinitus*, occurring together with infested *L. cinereus*, never harbour the parasite (Ball and Neville, 1979). Caullery (1953) incorrectly lists *Chiton* (= *Acanthochiton*) *fascicularis* as additional host for *H. chitonis*. Vegetative sporozoan stages encountered in *A. fascicularis* actually represent *Pseudoklossia chitonis* (see above and footnote on p. 964/965), which, in turn, appears to be host-specific to *A. fascicularis*.

In *Lepidochiton cinereus* from Easthaven, Tayside (Scotland), *Haplosporidium chitonis* infestation increases rapidly with age after the first year of life. Uninfested individuals longer than 16 mm are rare (Fig. 14-3). The sporozoan significantly alters the chitons' growth rate. Uninfested individuals show a growth pattern which conforms to the von Bertalanffy (1934) growth equation. In infested chitons, on the other hand, it remains nearly linear after the first 4 months of life. The maximum recorded length of parasitized *L. cinereus* at Easthaven is 28.1 mm, considerably larger than the theoretical maximum of 20.0 mm for uninfested individuals. This suggests parasite-induced growth enhancement, which involves the whole animal because there are no differences in the shell and soft body relationships (Fig. 14-4; Baxter and Jones, 1978). Comparable reports on enhanced growth ('gigantism'; Vol. I, Chapter 12) in parasitized molluscs, as discussed by Cheng (1971), refer to shell growth rather than to that of the entire animal. Length comparisons of *L. cinereus* from Llanrhystyd (Wales), where 62 % of 52 chitons were found to be infested with *H. chitonis*, revealed no difference in growth pattern between parasitized and non-parasitized individuals (Ball and Neville, 1979).

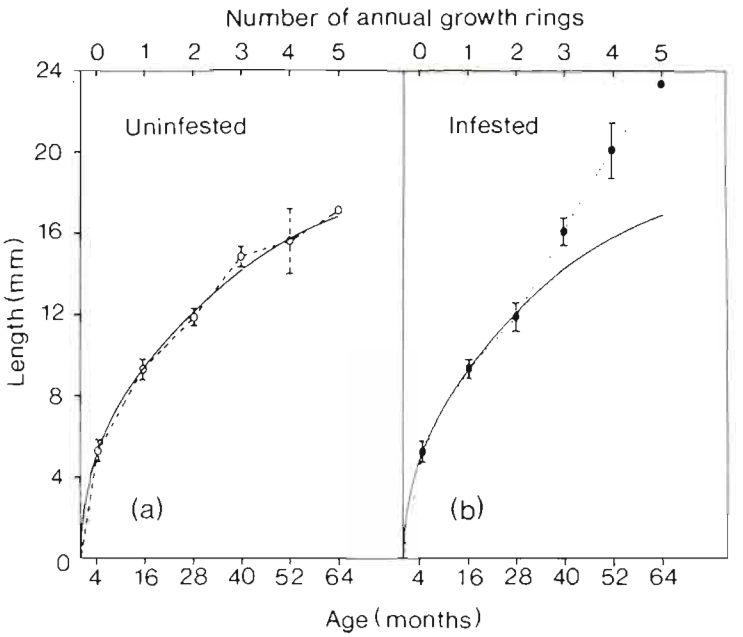

Fig. 14-4: *Lepidochiton cinereus*. Growth curves (broken lines) for uninfested (a) and *Haplosporidium chitonis*-infested individuals (b). Solid lines: growth curves predicted by von Bertalanffy growth equation; data given as year-class means and 95 % confidence limits, except where $n < 5$). (After Baxter and Jones, 1978.)

Pixell-Goodrich (1915), Debaisieux (1919, 1920) and King (1926) all obtained their infested *Lepidochiton cinereus* from Rum Bay near Plymouth, England. *Haplosporidium chitonis* was not found, at that time, in chitons from other places. Arvy (1957), however, made passing mention of the occurrence of *H. chitonis* in *Lepidopleurus* (= *Lepidochiton*) *cinereus* from Dinard, French coast of English Channel. In Rum Bay, Pixell-Goodrich (1915) found 85 (63 %) out of 135 chitons to be infested. No other study has been done

on this parasite until 1978 when Baxter and Jones reported high incidences and intensities of *H. chitonis* infestation in *L. cinereus* from Easthaven on the east coast of Scotland. Ball and Neville (1979) restudied the parasite and its distribution along the southwest, south and southeast coasts of the British Isles. During a 12-year investigation, a total of 249 individuals of *L. cinereus* from 11 different localities were examined and the prevalence of *H. chitonis*, as ascertained by the presence of mature spores, was recorded. The sources and number of hosts infested/number of chitons inspected were as follows: Port Erin, 1/18; Llanrhystyd, 32/52; Port Eynon, 3/5; Oxwich Bay, 0/27; Weymouth, 0/15; Wembury Bay, 2/6; Plymouth breakwater, 9/45; Black Rock, Brighton, 5/11; Peacehaven, 4/16; Robin Hood's Bay, 0/40; Dornoch Bay, Sunderland, 0/14. Only the highest incidence recorded by these authors (Llanrhystyd: 62 %) corresponds to the incidence found by Pixell-Goodrich (1915) in *L. cinereus* from Rum Bay. Ball and Neville (1979) concluded that, although *H. chitonis* does not appear to have increased its prevalence in *L. cinereus* populations, it might have increased in distribution during the past 64 years.

Agents: Ciliophora

Chiton olivaceus from the Gulf of Naples has been identified as host for thigmotrichous ciliates *Ancistrum cyclidioides* var. *lata*, all of 6 hosts examined being infested. Morphologically similar forms, all believed to be varieties of the same species, occur in a number of gastropods and bivalves (Issel, 1903). Nothing is known about the effect of *A. cyclidioides* on its hosts. Being an arhynchodine ciliate, it might be a harmless commensal. *A. isseli*, a related species occurring on the gills of *Modiolus modiolus*, however, has its food vacuoles usually filled with the yellow, granular pigment present in host cells (Kidder, 1933a, b). Whether this stems from the mollusc or from the food has not been determined. Pauley and co-authors (1966) regarded *A. mytili* and *A. caudatum* as potential pathogens in *Mytilus edulis* living under adverse environmental conditions.

Hypocomella katharinae, a rhynchodine ancistrocomid ciliate (Fig. 14-5), lives on the gills of chitons *Katharina tunicata* from the Cape Arago region, Oregon (USA). Of 43 chitons, 8 were found to be infested. When removed from the host, *H. katharinae* tends to be rather sluggish and to show relatively little ciliary activity, but an occasional individual

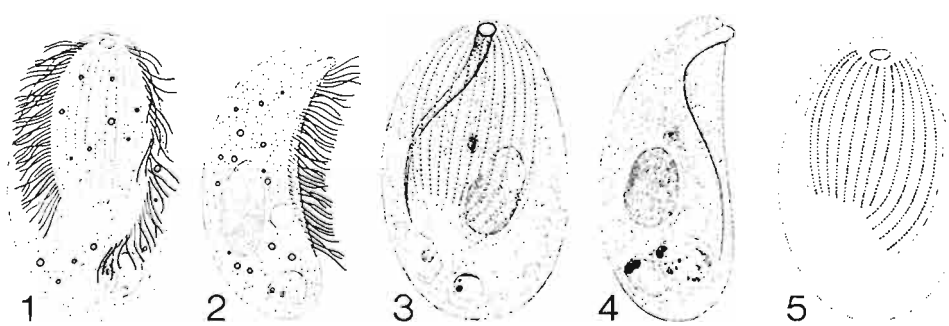

Fig. 14-5: *Hypocomella katharinae* from *Katharina tunicata*. Inferior (1) and lateral (2) aspect of living individuals; inferior (3) and lateral (4) view of specimens fixed in Hollande's fluid and stained with iron haematoxylin; 5: inferior aspect, Protargol. × 1,500. (After Kozloff, 1961.)

may exhibit weak swimming movements for a time (Kozloff, 1961). Although being labelled 'parasitic', nothing is reported on the pathology of this species. As a rhynchodine ciliate equipped with a suctorial tentacle, it is well adapted to a parasitic mode of life.

Peritrichs *Urceolaria korschelti* occur on the gills of *Lepidochiton cinereus* and *Chiton marginatus* (Zick, 1928; Kahl, 1934, 1935). On the basis of the descriptions given by Cuénot (1891), Caullery and Mesnil (1915) and Brouardel (1951), Fenchel (1965) was not able to distinguish it from *Leiotrocha patellae* (Vol. I, Fig. 12-5), originally described as *Trichodina patellae* from the gills of *Patella vulgata* at Roscoff, France, by Cuénot (1891). Fenchel (1965) reports *L. patellae* from a number of bivalves in Scandinavian waters (Chapter 13), and states that peritrichs assigned to this species embody in reality a complex of morphologically indistinguishable forms. *U. karyodactyla* occurs in the mantle cavity and on the gills of *Ischnochiton ruber* at Saint Andrews, New Brunswick (Canada). Cytoplasmic vacuoles contained various food items, notably diatoms (Laird, 1961). Kozloff (1961) found peritrichous ciliates of the genera *Trichodina* and *Scyphidia* on the gills of all of the 43 *Katharina tunicata* examined by him but did not study them in detail.

DISEASES CAUSED BY METAZOANS

Agents: Platyhelminthes

Chitons *Liolophura japonica* var. *tesselata* from Shimoda (Japan) are hosts for polycladid turbellarians *Stylochoplana parasitica*. In the summer, nearly half of the chitons are infested with 1 to 10 of these planarians, which inhabit the pallial groove. Worms removed from the host survive for a few days in sea water, but eventually disintegrate. During the summer and autumn, *S. parasitica* deposits eggs enclosed by cocoons, about 7 × 2 mm in size, on the host's hyponotum along the margin of the pallial groove. Larvae, hatching from the eggs in about 10 days, are strongly positively phototactic and leave the host immediately after liberation. *S. parasitica* appears to be fairly host-specific for *L. japonica* var. *tesselata*. On rare occasions, it was also found on *L. japonica* but never on sympatric *Acanthochiton* or *Ornithochiton* (Kato, 1935).

Polyplacophorans are not known to serve as first intermediate hosts for digeneans (Wright, 1966); there is a single report on the presence of metacercariae in members of this molluscan group. Larval *Proctoeces maculatus* occur free in the connective tissue of *Acanthochites* (*Acanthochiton*) *discrepens* from the Gulf of Marseille, France (Prévot, 1965). Metacercariae of this species commonly parasitize a number of marine invertebrates, mainly bivalves (Chapter 13). *P. maculatus* appears to be cosmopolitan in distribution.

No other records of trematode or cestode infestation in chitons have come to the revierwer's attention. Apparently, these hosts have not been examined adequately for parasitic flatworms.

Agents: Gastropoda

Pyramidellid snails are well-known parasites of gastropods, bivalves (Chapters 12 and 13) and other invertebrates. There are only 2 records of a pyramidellid associated with an amphineuran. This is *Odostomia chitonicola*, parasitizing chitons *Dinoplax gigas* on the

South African coast. Of 6 *D. gigas* taken near Port Edward, Natal, the largest individual (83 mm long) carried 180 *O. chitonicola*, attached to the dorsal surface of the girdle among the spines. The largest snail measured 1.4 mm, but most were young and some were extremely small.

Unfortunately, the pyramidellids were not discovered until after the hosts had been killed in alcohol and dried. Although, therefore, feeding of *Odostomia chitonicola* has not been observed, a parasitic relationship was (probably rightly) inferred. The presence of numerous very small individuals on the chiton suggests that their development is non-pelagic and that the larvae are fairly sedentary, starting to feed parasitically at an early stage.

About 15 juvenile snails were found on the girdle of another, 63 mm long host, and 2 more on a third, 46 mm long chiton. No pyramidellids were recovered from the remaining 3 *Dinoplax gigas* (66, 57 and 38 mm long). The scarcity or absence of *Odostomia chitonicola* on all but one of the chitons is noteworthy in view of the fact that all six were collected within a few feet of each other. Subsequently, 22 additional *D. gigas*, collected from 8 localities, were examined for parasites, but no pyramidellids were found (Robertson and Orr, 1961). The only other record of the occurrence of *O. chitonicola* on this polyplacophoran host is the original description of the species by Smith (1899), who obtained his specimens from *Chiton fossus* (= *Dinoplax fossus*, a form of *D. gigas*) at Unkomaas, Natal, 67 miles north of Port Edward, where Robertson and Orr (1961) have rediscovered *O. chitonicola.*

About 7 of 1,000 *Chiton tuberculatus* from the Bermuda Islands were found with oyster-drill holes in 1 or more valves. The individuals whose shells were so attacked were always still alive. The holes pierced merely the tegmentum, the dense, hard articulamentum being impervious to the oyster drill's efforts (Arey and Crozier, 1919).

Agents: Arthropoda

Only a few species of arthropods have been identified as associates of amphineurans. Most of these appear to be commensals. In some cases, the interrelationship has not been explored sufficiently enough to exclude parasitism.

Harpacticoid copepods, *Harpacticus* sp. and *Heterolaophonte* (?), clinging to the girdle of *Acanthopleura granulata* from Puerto Rico, are probably commensals, but studies on their feeding behaviour are lacking (Glynn, 1968). Copepods which are clearly parasitic have not been reported from polyplacophorans (Humes, 1958).

Hyalid amphipods *Parhyale hawaiensis* have been found in the inhalant chamber of the pallial groove and the inhalant and exhalant respiratory canals of the girdle of Puerto Rican *Chiton tuberculatus* (Glynn, 1968). After analyzation of published records of associations between amphipods and molluscs, Vader (1972) concludes that all of these appear to be commensalistic.

Similarly, isopod-chiton partnerships appear to be non-parasitic. Sphaeromatids *Exosphaeroma crenulatum* occupy the ctenidial grooves and the undersurface of the girdle of *Chiton tuberculatus* at Bermuda. As many as 20 or more of these crustaceans occurred on large 'hosts'. The association was considered more or less facultative, as *E. crenulatum* was also found free among the algae growing close to the chitons (Arey and Crozier,

1919). Puerto Rican sphaeromatids *Dynamenella perforata* inhabit mainly the inhalant chamber of the pallial groove of *Acanthopleura granulata* and *C. tuberculatus*. *Exosphaeroma* spp. associate with *C. tuberculatus* and *C. marmoratus*, and *Dynamenopsis dianae* with *C. tuberculatus*. The main role of the placophoran 'hosts' seems to provide shelter against desiccation. The prevalence of *D. perforata* on *A. granulata* ranged from 3 to 100 %, and up to 48 isopods were counted on a single chiton. The crustaceans deeply insert the head end of the body between the individual gill filaments, often as far medially as the exhalant chamber. The projecting rear portion of the pleotelson is frequently about the only part of the isopod visible when in this posture (Glynn, 1968). At least some degree of irritation — and possibly mechanical damage to the gill epithelium — may result from high isopod population densities. Similar associations between sphaeromatid isopods and intertidal gastropods have been reported by Nishimura (1976) from Japan, but there was no evidence of a parasitic relationship.

Pinnotherid crabs *Opisthopus transversus* inhabit the pallial groove of Californian giant chitons *Cryptochiton stelleri*. Mucus adhering to the crabs' carapaces presumably originates from the pallial mucous tracts on the inner walls of the pallial grooves and might serve as food for the inquilines (Webster, 1968). *O. transversus* is not known to harm its 'host', but judging from the effects of pinnotherid crabs on bivalves (Chapter 13), one may conclude that it causes at least some degree of irritation and mechanical damage to the pallial and ctenidial epithelium of *C. stelleri*. *O. transversus* also associates with a number of other invertebrates (Beondé, 1968).

A single female sea spider *Halosoma viridintestinale* has been recovered from the girdle spines of *Mopalia muscosa* collected near Bolinas, California (Ziegler, 1960). Although several pycnogonids are known as parasites of molluscs (Chapters 12 and 13), the above record is probably accidental since *H. viridintestinale* normally feeds on hydroids (Ricketts and Calvin, 1968).

Halacarids *Halixodes* (*Agaue*) *chitonis* occur, attached to the gills, on acanthochitonids *Cryptoconchus porosus* from New Zealand. The stages observed were hexapod larvae, young octopod nymphs, and second-stage nymphs with rudimentary genital organs. The adult male and female remain unknown (Brucker, 1897; André, 1931). Similar larval mites have been recovered from the mantle cavity of limpets in New Zealand (Stout and Viets, 1959).

ABNORMALITIES

The chiton shell normally consists of 8 plates (valves). Several types of deviation from the regular number and structure of the plates may occur. *Hypomery* (or hypomerism) is a term coined to describe the reduction in plate number (Fig. 14-6, 1). Seven-, 6-, 5- and 3-valved chitons have been reported, but 4-valved individuals have never been seen (Stearns, 1901; Sykes, 1901; Pelseneer, 1919, 1923; Taki, 1932; Langer, 1978). Most of the hypomeric chitons are 7-valved. In total, 35 cases of this kind have been described in the literature. In 20 of these, however, the apparent reduction in plate numbers is due to the coalescence of 2 neighbouring valves. Such abnormal individuals have repeatedly been described as new species or varieties under the species or subspecies name *septemvalvis*.

Six-valved chitons occur much less frequently (12 documented cases), and 5- and 3-

valved specimens are exceedingly rare (2 and 2 cases) (Taki, 1932; Langer, 1978). No case of hypermery, i.e., the occurrence of supernumerary shell plates, is yet known. Concomitant changes in the soft parts of hypomeric chitons include decrease in body length and reduction of the number of spicule bundles (in *Acanthochiton*), while the number of ctenidia is scarcely correlated with hypomery.

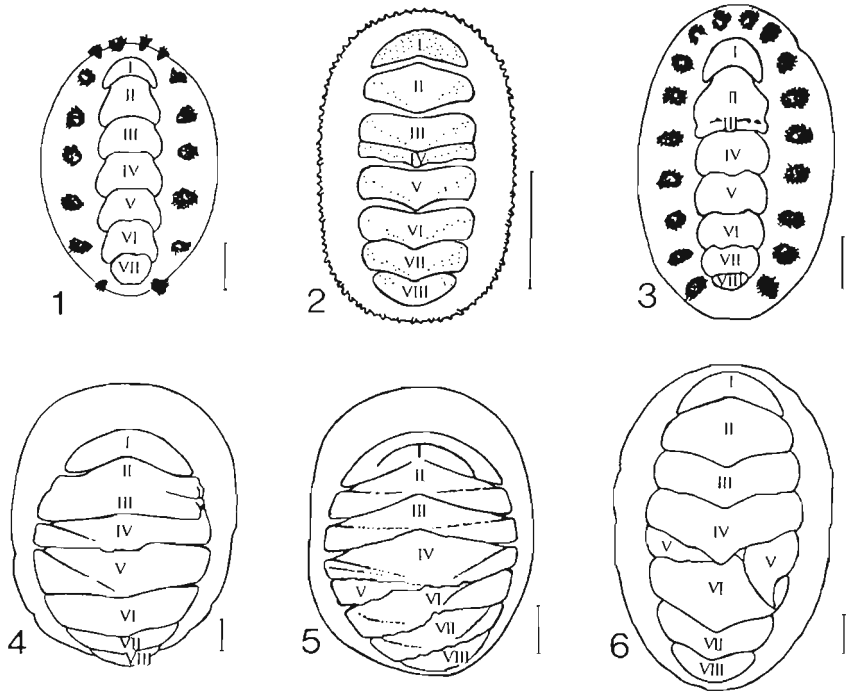

Fig. 14-6: Polyplacophora. Shell-plate abnormalities. 1: 7-valved *Acanthochiton rubrolineatus*; 2: *Liolophura japonica* with 1 fused valve; 3: *A. rubrolineatus* with 1 coalesced valve; 4, 5: *Placiphorella stimpsoni* with partially coalesced valves; 6: *L. japonica* with split valve. Bars: 5 mm. (After Taki, 1932.)

The term *coalescence* is applied to changes in which shell plates are partially atrophied and fuse with other plates (Fig. 14-6, 2 and 3). This type of abnormality is by far more common than hypomery (Crozier, 1919; Pelseneer, 1919, 1923; Berry, 1925; Taki, 1932). It appears likely that some of the reported 6-, 5- and 3-valved chitons are, in fact, specimens with 2 or more pairs of coalesced plates. Sometimes, valve fusion is incomplete (Fig. 14-6, 4 and 5). The third type of abnormality observed is *splitting*, which may occur in anyone of the 8 valves (Fig. 14-6, 6).

Disorders in the formation of polyplacophoran shell plates are rare. Thus, Pilsbry (in Taki, 1932) found only two 7-valved *Mopalia ciliata* among several thousand normal individuals. Crozier (1919) found 2 of over 2,100 *Chiton tuberculatus* from Cross Bay (Bermuda), to have coalesced plates, and Pelseneer (1919) reported one coalesced, four 7-valved and one 6-valved *Lepidochiton cinereus* from collections of several hundred individuals. The causes of these abnormalities are not known but some may be due to injury.

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15. DISEASES OF MOLLUSCA: SCAPHOPODA

G. LAUCKNER

The class Scaphopoda includes some 300 exclusively marine species, comprising very small (few millimeters) to moderately large (max. 12 cm) forms. Literature on scaphopods is scanty. Monographs on *Dentalium* have been published by Lacaze-Duthiers (1856-1957), Fol (1889) and Plate (1892). Although having dissected hundreds of scaphopods, none of these authors has reported anything on parasites. Boissevain (1904) mentioned the occasional occurrence of unidentified rediae in *D. entalis* from Naples (Italy), and Pelseneer (1906) briefly described larval trematodes from *D. tarentinum* (*D. vulgare*), but nothing new has been published on diseases or parasites of scaphopods until the late 1940's when Lucie Arvy commenced her investigations. In a series of papers she subsequently described several protozoan and one trematode parasite of *Dentalium*. No microbial diseases have as yet been reported from scaphopods.

DISEASES CAUSED BY PROTISTANS

Agents: Apicomplexa and Ascetospora

Of the only 3 known protozoans parasitizing scaphopods, 2 are apicomplexans and 1 is an ascetosporan (Table 13-3). An acephaline gregarine occurs in cells of the intestinal epithelium of *Dentalium entalis*. Its earliest discernible intracellular stages are ovoidal and situated near the basement membrane. Healthy host cells have a height of about 35 μm, but in those harbouring the gregarine, both the nucleus and the cytoplasm are grossly hypertrophied. When the growing parasite has reached a size of approximately 45 × 12 μm, displacement of the host-cell nucleus and atrophy of the entire cell ensue. Fully grown schizonts, ready to be discharged into the gut lumen, are 52 × 35 μm in size, with a nucleus of about 15 × 12 μm and a nucleolus 4 μm in diameter. The unnamed parasite was believed to be related to *Gonospora testiculi*, described by Tregouboff (1916, 1918) from prosobranchs *Cerithium vulgatum*, as well as to another undescribed gregarine infesting *Turritella communis*. Five of 132 *D. entalis* from Paramé (French coast of English Channel) were found to harbour this sporozoan (Arvy, 1957).

The second protozoan parasite of *Dentalium entalis*, reported by Arvy (1957), is a coccidian occurring in the epithelial cells of the digestive gland. Affected host cells become 'ballooned' to such an extent that they sometimes protrude three times their normal height from the epithelial surface. Such cells measure up to 68 × 33 μm, while normal ones are about 20 × 14 μm. Their nuclei are also strongly hypertrophied and pushed to the periphery of the affected cell by a number (probably 16) of oval bodies, 18 × 9 μm in

dimension (Fig. 15-1, 8 and 9) and developing in the cytoplasm. Smaller bodies, 12 × 4 μm in size (Fig. 15-1, 6), develop in cells hypertrophied to about 30-35 × 15-20 μm. The former bodies are believed to represent macromerozoites and the latter micromerozoites. They originate from macroschizonts (Fig. 15-1, 7) and microschizonts (Fig. 15-1, 1 to 5). Upon rupture of the enclosing host cell, the liberated micro- and macromerozoites float free in the lumen of the digestive gland and, in massive infestations, also occur in the gut lumen. The latter may also contain stages representing the sequence of spore formation (Fig. 15-2, 7 to 12). The above observations suggest an unusual development of the coccidian. Although sexual stages were not seen, a hypothetical life cycle was outlined. Schizogony, giving rise to micro- and macromerozoites, clearly takes place in the epithelial cells of the digestive gland. Gamogony was believed to occur in the lumen of the digestive gland and sporogony in the lumen of the intestine (Fig. 15-2). At

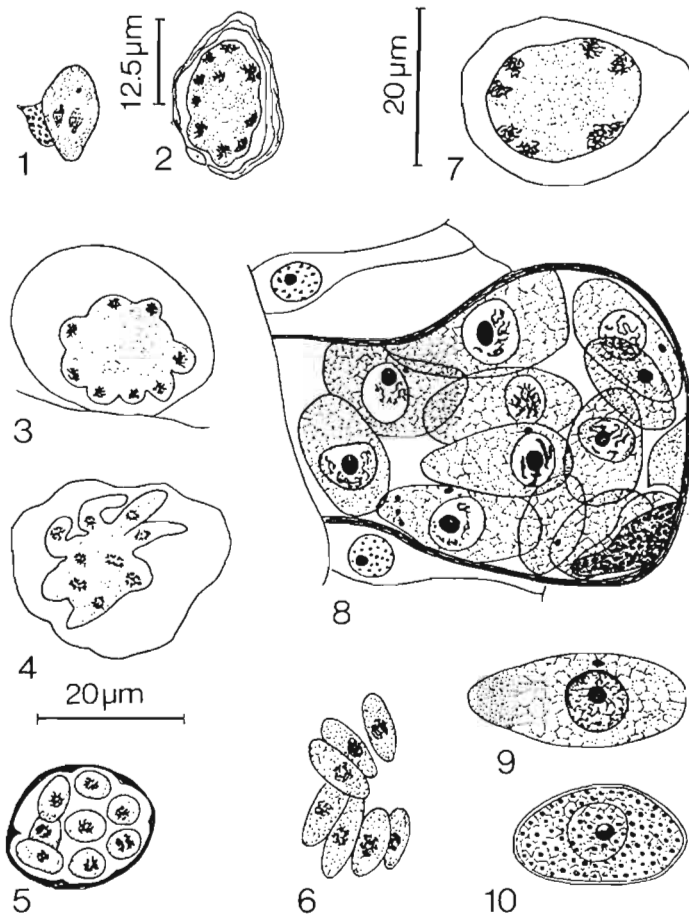

Fig. 15-1: Stages of unidentified coccidian from digestive gland of *Dentalium entalis*. Presumed development of micromerozoites (1-6) and macromerozoites (7-9). 1: Earliest intracellular stage with 2 mitotic figures; 2: microschizont with multiple mitoses; 3-5: differentiation of micromerozoites; 6: micromerozoites; 7: macroschizont; 8: differentiation of macromerozoites; 9: macromerozoite; 10: macrogamete (?). (After Arvy, 1957.)

Paramé, 66 (50 %) of 132 *D. entalis* were found to be infested with this sporozoan. Of these, 2 simultaneously harboured a balanosporidan, *Haplosporidium dentali*, and 4 had triple infestations involving *Cercaria prenanti*, *H. dentali* and the coccidian. Heavy infestation with the schizogonic stages of the latter caused massive destruction of large areas of digestive-gland epithelium. The coccidian was not named, and Arvy (1957) recommended restudy and classification of the parasite by protozoologists. However, this has apparently not been done to date. Although making ample reference to coccidians described from marine invertebrates, Pellérdy (1974) does not even mention Arvy's species in his monograph on Coccidia and coccidiosis.

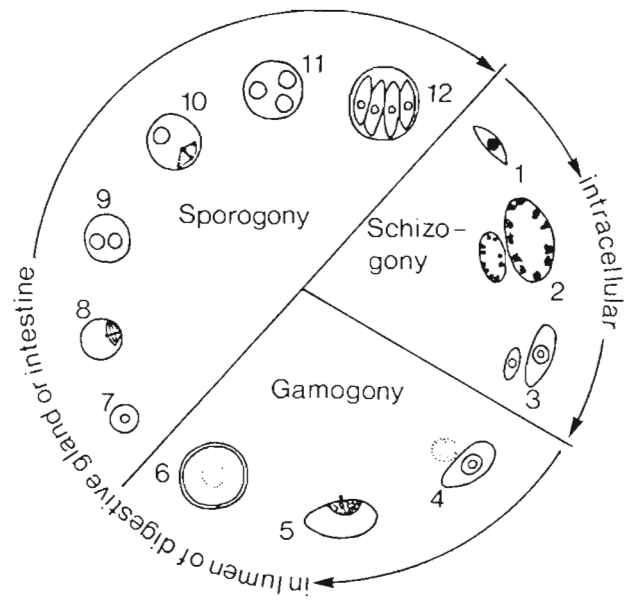

Fig. 15-2: Presumed life cycle of unidentified coccidian from digestive gland of *Dentalium entalis*. 1: sporozoite invading host cell; 2: development of micro- and macroschizonts; 3: micro- and macromerozoites; 4: formation of micro- and macrogametes; 5: zygote formation; 6: oocyst; 7-11: sporogony; 12: mature spore containing 4 sporozoites. (After Arvy, 1957; modified.)

The third protozoan parasitizing *Dentalium entalis* is a balanosporidan. Arvy (1949a, 1957) described it as *Haplosporidium dentali*, but Sprague (1963) transferred it to the genus *Minchinia*, under which generic name it has since been listed. Here it is retransferred to *Haplosporidium* because *Minchinia* must be regarded as invalid (for justification see Chapter 14 and footnote on p. 964/965). Plasmodia of *H. dentali*, 2.5 to 3 μm in size, and spores, 5 to 7 × 3 to 4 μm, occur mainly in the connective tissue surrounding the intestine. In heavily infested hosts, the intestinal epithelium appears to be separated from the underlying connective tissue by an almost continuous layer of spores and plasmodia in various stages of development. The digestive-gland, nervous and gonadal tissues of *D. entalis* may also be affected. *H. dentali* rarely invades the musculature proper, but tissue breakdown may occur where masses of parasities infiltrate the superficial portions of the foot, mantle, and dorsal retractor muscles.

Of 132 *Dentalium entalis* dredged off Paramé, 36 were found to be infested with *Haplosporidium dentali*. Of these, 22 simultaneously harboured rediae of *Cercaria prenanti*. The theoretically expected frequency of double infestations is only E = 9, the difference being statistically highly significant ($\chi^2 = 17.36$, $p < 0.001$; for calculation see Vol. I, p. 352). Two of the 132 *D. entalis* were simultaneously infested with *H. dentali* and the unnamed coccidian. The theoretically expected frequency of doubly occurrences, in this case, is E = 18. Again, the difference is statistically highly significant ($\chi^2 = 13.34$, $p < 0.001$). In conclusion, double infestations involving *H. dentali* and *C. prenanti* occur much more frequently, and those involving *H. dentali* and the coccidian much less frequently than expected to occur by chance alone. The observed number (O = 4) of triple infestation with *H. dentali, C. prenanti* and the coccidian, on the other hand, did not differ significantly from expectation (E = 4.5).

DISEASES CAUSED BY METAZOANS

Metazoan parasites of scaphopods appear to be extremely rare. Representatives of various agent groups, commonly associated with gastropods, pelecypods or cephalopods, apparently do not attack the Scaphopoda. Copepod parasites, as an example, are unknown in this molluscan class (Humes, 1958).

Agents: Trematoda

Tailless cercariae, named *Cercaria dentalii*, have been recovered from a single preserved individual of *Dentalium tarentinum* (= *D. vulgare*). The larvae develop in extremely long (1.5 cm) tubiform sporocysts (Pelseneer, 1906). Palombi (1941), who refound the parasite in *D. entalis* (*D. dentale*) from the Gulf of Naples, identified it as the cercaria of *Ptychogonimus megastoma*. Its unencysted metacercaria occurs in brachyuran crabs (Vol. III) and the adult in elasmobranchs (Dollfus, 1937).

Cercaria prenanti develops in rediae, up to 2,550 × 600 μm in dimension, in the gonads of *Dentalium entalis*. The cercaria is a typical cystophorous hemiuroid larva. Arvy (1949a, b, 1957) provided a detailed description of its development in the scaphopod host. There is remarkably little tissue reaction against the rediae, but the effects on the host are severe. Heavy infestation invariably causes complete castration. Of 132 *D. entalis* dredged off Paramé, 33 were infested with *C. prenanti*. Of these, 22 simultaneously harboured balanosporidans *Haplosporidium dentali*, 2 an unidentified coccidian and 1 an unidentified gregarine (see above). Four had triple infestations of *C. prenanti, H. dentali* and the coccidian.

Ching (1960) reported what she believed to be the same species of cercaria from 2 of 42 *Dentalium dalli* dredged mainly from East Sound, Friday Harbor (Washington). One scaphopod harboured 42 rediae, the other 34. The infestation covered the digestive gland region entirely and appeared to have castrated the hosts.

The further life-cycle stages of *Cercaria prenanti* remain unknown. Ching (1960) exposed copepods to cercariae, but the crustaceans did not become infested. However, larvae excised from rediae instead of naturally emerged individuals have been used in

these experiments. Arvy (1957, 1972a, b) attributed the rarefaction of *Dentalium entalis* in the Dinard (France) region during the 1940's and 1950's to heavy *C. prenanti* infestations. These declines in population densities were paralleled by similar extinctions, in the Dinard region, of opisthobranchs *Philine aperta* and bivalves *Pandora albida* also infested with cystophorous hemiuroid cercariae (Arvy, 1951a, b; Arvy and Gaillard, 1956).

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