DIAGNOSIS AND CONTROL OF MARICULTURE DISEASES IN THE UNITED STATES

Middle Atlantic Coastal Fisheries Center
National Marine Fisheries Service
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NATIONAL MARINE FISHERIES SERVICE
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FOREWORD

A preliminary draft of this handbook of diagnosis and control of diseases in mariculture was made available at the fifth annual meeting of the World Mariculture Society in January, 1974. A request was made at that time for additional unpublished information that could be incorporated in a definitive printed handbook to be distributed at the 1975 meeting of the Society. Response to the request has been good, and a number of research people have provided as yet unpublished information and given permission to have it incorporated in the handbook for purposes of completeness. Inclusion of such information in this handbook should not be construed in any way as interfering with the publication priorities of individual authors.

In a field such as mariculture diseases, which is undergoing rapid expansion, a handbook of this kind becomes out of date very quickly. Ideally, the document should be revised and updated at least every two years. Revision should begin the day after publication of the first edition. To that end, we in the Middle Atlantic Coastal Fisheries Center would appreciate reprints, photographs and comments, as well as suggestions for changes in format, corrections, or additions -- anything which might improve the next edition.

The handbook is a summarization of the work of a great numbere of people. To stay within reasonable page numbers, and within the format chosen, much valuable detailed information has had to be excluded. Hopefully, the references provided with each section will help to atone for such omissions.

The concept of the handbook was developed in early discussions with Dr. Aaron Rosenfield, Mrs. Helen Lang and Mr. Haskell Tubiash -- all of the Oxford Laboratory of the Middle Atlantic Coastal Fisheries Center. Mrs. Lang, Librarian, and Mr. Tubiash, Microbiologist, both contributed substantially to the preliminary draft, and Mrs. Lang has continued her detailed literature searches for relevant data. The present document should thus be considered as a contribution by this Center and thus by the National Marine Fisheries Service to the expanding interest in mariculture in the United States.

I would like to express my personal thanks to the disease specialists who graciously and willingly contributed material -- some of it unpublished -- for inclusion in this volume, and to Mrs. Kathe Melkers, for her assistance in preparing the several drafts and revisions of this work.

Carl J. Sindermann
Highlands, New Jersey
December, 1974

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INTRODUCTION

This handbook of diagnosis and control of diseases in mariculture has been assembled by the Middle Atlantic Coastal Fisheries Center, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, U. S. Department of Commerce. It has developed from an obvious need for compilation and summarization of information about diseases important to mariculture in the United States, and from a stated request by the World Mariculture Society at its second annual meeting for the production of such a document.

To be fully effective, it was felt that the handbook must meet a number of criteria:

(1)e It should contain concise summaries of all informatione presently available -- published and unpublished -- aboute significant diseases of mariculture animals;

(2)e It should be adequately illustrated, with both figures ande photographs of diagnostic features of each disease;e

(3)e It should be updated periodically -- probably every twoe years;

(4)e It should be freely available to anyone with an intereste in mariculture; and

(5)e It should contain key references, as well as a list of namese and addresses of people with disease expertise.e

The handbook is limited to those disease entities known at present or assumed to be of significance to mariculture in the United States. Undoubtedly, as mariculture in this country and elsewhere expands, new or presently unsuspected disease entities will emerge and assume positions of significance. While emphasis in this document is on pathogens, it should be clearly recognized that poor water quality and inadequate nutrition are often basic determinants of disease outbreaks and should be of primary concern in disease control. Disease is an expression of a complex interaction of host, pathogen, and environment -- and the environment (as well as the host) may be highly abnormal in many mariculture situations. It is important to distinguish between what we can label primary pathogens, such as Gaffkya, which can kill even when other environmental factors are adequate -- and adventitious or opportunistic pathogens, which kill when other physiological or environmental factors are poor or marginal. Included in the latter category would be many of the vibrios, pseudomonads and aeromonads. What we refer to as disease is often a reflection of one or more marginal environmental factors: nutrition, water quality, oxygen, temperature, salinity, and high bacterial populations.

It is abundantly clear that at present the halophilic vibrios constitute the most serious bacteriological problems of mariculture species -- fish and shellfish -- as evidenced by many reports in the literature. It should be noted that the pasteurellas are also important. Pasteurella piscicida has been implicated as the etiological agent of a massive mortality of white perch (Roccus americanus) in Chesapeake Bay in 1963; Pasteurella plecoglosacida has been identified as the cause of summer epizootics in pond cultured ayu (Plecoglossus altivalis) in Japan; Pasteurella piscicida infections were held responsible for serious mortalities in yellowtail farms of Japan; and a Pasteurella sp. was implicated in an extensive mortality of menhaden (Brevoortia tyrannus) and mullet (Mugil cephalus) in Texas waters in 1968.

Virus diseases have not yet assumed the dominant role in mariculture that they now have in fresh-water fish culture, but there are several reports published within the past several years of viruses in oysters and crustaceans -- reports which suggest future problems in mariculture.

Protozoan diseases, often but not always associated with crowding and poor water quality, have been identified as important in culture of certain marine species. These include several ciliates, certain dinoflagellates, and a number of sporozoans.

Worm diseases, with the possible exception of those produced by monogenetic trematodes, have not yet appeared to be significant problems in mariculture. This is probably due in large part to their complex life cycle and the difficulty in completing such cycles in culture systems. One general point is that diseases may emerge in culture situations which are normally benign or unknown in natural populations.

Diseases and abnormalities due to environmental contaminants and nutritional deficiencies have been recognized, and will be important until adequate and defined diets, as well as effective control of water quality, become realities. This handbook deals principally with infectious diseases, but contaminants and inadequate nutrition must also be recognized as important problem areas. Brief statements about contaminant-induced and nutritional-deficiency diseases are included near the end of this handbook.

Mariculture species included in this handbook are limited to those currently receiving significant attention. There are other species which are receiving more limited attention, and which might be included in future revisions. Possible candidates would be abalone, mullet, Dungeness crabs, scallops, and catfish reared in brackish water.

The fungus Lagenidium has been recognized in Dungeness crab larval cultures, a Pasteurella sp. has been reported to cause mortalities in mullet, scallop larvae are subject to vibrio infections, abalone survival is affected by nematode infestations, and mortalities attributed to Aeromonas infections have occurred in catfish reared in brackish water.

It should be pointed out too that many other parasites and disease conditions have been described for the species of animals selected for inclusion in this handbook. They have been excluded because (1) they have not been identified as a present or potential problem in mariculture, or (2) they have been described too vaguely to constitute reasonable disease entities based on what we know. In category 2 would be "amber" disease and "mycelial" disease of oysters, "leopard spot" disease of lobsters, and others. We still have much to learn about the diseases that are included. For some the etiological agent is unknown, and for others we may find that several agents are involved.

SOURCES OF INFORMATION ABOUT DISEASES OF MARINE ANIMALS

There are two principal sources of mariculture disease information: (1) published technical literature and (2) direct contact with active research groups and individual investigators.

The published technical literature on diseases of marine animals is in many forms and in many stages of complexity. The most comprehensive bibliography of diseases in marine animals available today contains 5,230 citations. This is probably at most about 1/5 of all available papers -- making a very rough total of some 25,000 papers published on the subject. Of these, possibly 10% or 2,500 papers have some relevance to diseases and parasites which may be of significance to mariculture. Books in English, German, Japanese, and other languages are available in increasing numbers. Some translations into English are appearing. Review articles are also available in quantity, as are original papers concerned with one or more specific disease problems. In a special category is the Leetown (Eastern Fish Disease Laboratory) Leaflet Series (concerned largely with fish disease problems in fresh water, but exemplary of possibilities for coverage of diseases of marine species). Unfortunately, for the generalist, the marine disease literature is dispersed and unwieldly, and is often a source of frustration rather than enlightenment. As a starting point, a basic list of general disease references is attached as Appendix I and some specific references are included with the sections on particular diseases.

Those engaged in mariculture development, pilot production or production usually do not have time to wade through this vast and highly dispersed literature to find answers to particular disease problems of the moment. Such individuals would far prefer either (1)eto call or write someone who can supply firsthand informatione that will help solve their problem, or (2) (and far less desirable) turn to a manual or handbook such as this one that will provide the information they need. Direct contact with individuals or groups actively involved in ongoing research on mariculture diseases is often productive of information relevant to problems of the moment, provided the mariculturist with the problems knows where to turn for such information. The numbers of individuals and groups with major commitments to marine disease research are increasing yearly, often with financial encouragement from Sea Grant or enlightened state natural resource directors. A list of disease research groups is attached as Appendix II.

CRUSTACEAN DISEASES

Crustacea which are attracting the most attention in the United States today as mariculture species are penaeid shrimps, lobsters, fresh-water shrimps, and some of the crabs. Commercial scale production of shrimps has already been achieved, although there are still many shortcomings in available technology. Culture of other species is still in the experimental, developmental, or pilot plant stages, with much still to be learned about inexpensive defined diets, maintenance of water quality, larval survival, and disease control.

Almost without exception, the infectious disease problems that have surfaced thus far in crustacean culture are microbial in origin -- bacteria, fungi, and protozoa all seem of significance, with bacteria well in the lead. Among the bacteria, the shell-destroying forms and the halophilic vibrios are noteworthy. The viruses are waiting in the wings -- with one virus disease recently reported in shrimps and another in crabs.

An interesting and somewhat unique situation with certain Crustacea, not precisely mariculture, but subject to extremes of disease potential, is that of short-term holding of individuals for market. This is accomplished in lobster pounds or live cars, and

crab shedding tanks. Some of the most serious diseases of lobsters -- gaffkaemia and shell disease -- cause mortalities in such concentrated and artificially-held populations. Shell disease, ciliate disease, and gray crab disease manifest themselves in shedding tanks. Much can be learned about the role of pathogens in mariculture populations through studies of these artificially-held animals.

REFERENCES:

Anderson, J. I. W. and D. A. Conroy. 1968. The significance of disease in preliminary attempts to raise Crustacea in sea water. Proc. 3rd Sympos., Comm. Off. Int. Epizoot. (Stockholm) 1968.

Sprague, V. and J. Couch. 1971. An annotated list of protozoan parasites, hyperparasites, and commensals of decapod Crustacea. Jour. Protozool. 18: 526-537.

Shrimp Diseases

Because of major interest and investment in penaeid shrimp mariculture, research and development by states, universities and federal agencies has resulted in development of an appreciable body of information about shrimp diseases that are now or may become problems for successful culture ventures. Twelve diseases are summarized here:

(1)e Virus disease;e

(2)e Vibrio (V. parahemolyticus) disease;e

(3)e Vibrio (V. alginolyticus) disease;e

(4)e Brown spot disease;e

(5)e Filamentous bacterial disease;e

(6)e Larval mycosis (Lagenidium);e

(7)e Fungus (Fusarium) disease;e

(8)e Milk or cotton shrimp disease;e

(9)e Microsporidiosis of reproductive organs;e

(10)e Ciliate (Zoothamnium) disease;e

(11)e Black gill disease;e

(12)e Muscle necrosis.e

It should be pointed out that many other parasites of shrimps from natural waters have been identified -- particularly protozoa, worms, and crustaceans -- but these have not yet been demonstrated to be of significance to mariculture populations. Some of these may emerge as problems in the future however.

It should also be pointed out that certain of the organisms responsible for so-called "diseases" (such as ciliate disease) are facultative pathogens or ectocommensals, and are able to prosper when culture conditions are less than optimal.

Some of the diseases reported here (such as black gill disease) are very incompletely described, and the causative agent (if a single entity exists) is as yet unknown. In at least some instances -- and "black gill disease" is a good example -- what are described as disease entities are probably not entities at all, but complexes of generalized disease signs which may result from a number of causes, infectious and non-infectious.

KEY REFERENCES:

Hutton, R. F., F. Sogandares-Bernal, B. Eldred, R. M. Ingle and K. D. Woodburn. 1959. Investigations on the parasites and diseases of saltwater shrimps (Penaeidae) of sports and commercial importance to Florida. Fla. State Bd. Conserv., Mar. Lab. Tech. Ser. 26, 38 pp.

Kruse, D. N. 1959. Parasites of the commercial shrimps, Penaeus aztecus Ives, P. duorarum Burkenroad, and P. setiferus (Linnaeus). Tulane Stud. Zool. 7: 123-144.

Lightner, D. V. (in press). Some potentially serious disease problems in the culture of penaeid shrimp in North America. Proc. U.S.-Japan Natural Resources Program, Symposium on Aquaculture Diseases, Tokyo, 1974.

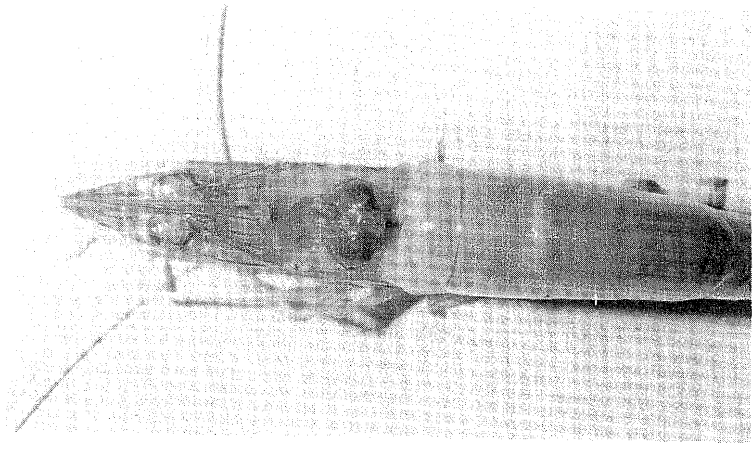
Overstreet, R. M. 1973. Parasites of some penaeid shrimps with emphasis on reared hosts. Aquaculture 2: 105-140.

Sprague, V. 1970. Some protozoan parasites and hyperparasites in marine decapod Crustacea. In. Snieszko, S. F. (ed.). A symposium on diseases of fishes and shellfishes. pp. 416-430. Pub. No. 5, Amer. Fish. Soc., Wash., D. C.

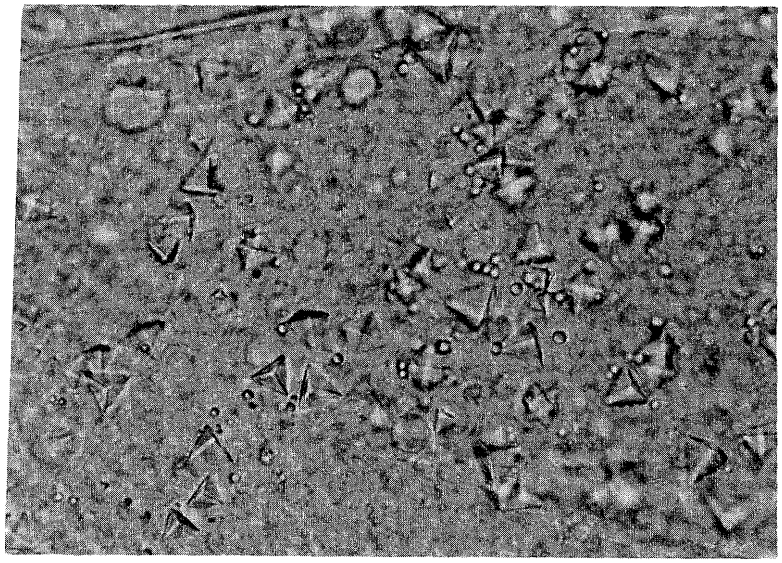
Sprague, V. and J. Couch. 1971. An annotated list of protozoan parasites, hyperparasites, and commensals of decapod Crustacea. J. Protozool. 18: 526-537.

Villella, J. B., E. S. Iversen and C. J. Sindermann. 1970. Comparison of the parasites of pond-reared and wild pink shrimp (Penaeus duorarum Burkenroad) in South Florida. Trans. Amer. Fish. Soc. 99: 789-794.

(1) VIRUS DISEASE OF SHRIMPS



Dorsal view of pink shrimp showing hepatopancreas -- the organ infected by Baculovirus penaei. Photograph supplied by John A. Couch, Gulf Breeze Environmental Research Laboratory.



Fresh squash of pink shrimp hepatopancreas showing many polyhedral inclusion bodies (PIB's). Note characteristic pyramidal or tetrahedral forms of PIB's. Photograph supplied by John A. Couch, Gulf Breeze Environmental Research Laboratory.

COMMON NAME: Virus disease

SPECIES AFFECTED: Pink shrimp, Penaeus duorarum

GROSS SIGNS: None

CAUSE: Virus of the Baculovirus group, designated Vaculovirus penaei

METHOD OF DIAGNOSIS: Electron microscopy of absorptive hepatopancreas tubule cells. Rod-shaped viral particles associated with nuclear hypertrophy and chromatin diminution. Large crystalline inclusion body distorts nuclear membrane.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Present in feral and experimental pink shrimp from Florida. Stress (exposure to Aroclor, Mirex, crowding) seems to enhance viral development in experimental studies.

EFFECT ON HOST: Cytopathology limited to certain hepatopancreatic cells; no gross effects described.

TREATMENT: None reported.

PREVENTIVE MEASURES: None reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Cedar Key-Pensacola section of the Florida coast.

KEY REFERENCES:

Couch, J. A. 1974. Free and occluded virus similar to Baculovirus, in hepatopancreas of pink shrimp. Nature 247 (5438): 229-231.

Couch, J. A. and D. R. Nimmo. 1974. Ultrastructural studies of shrimp exposed to the pollutant chemical, Polychlorinated Biphenyl (Aroclor 1254). Bull. Soc. Pharm. Envir. Pathol. 2: 17-20.

(2) VIBRIO (V. PARAHEMOLYTICUS) DISEASE OF JUVENILE AND ADULT SHRIMPS

VIBRIO (V. PARAHEMOLYTICUS) DISEASE OF SHRIMPS

COMMON NAME:	Vibrio (V. parahemolyticus) disease
SPECIES AFFECTED:	Pink shrimp, Penaeus duorarum Brown shrimp, Penaeus aztecus White shrimp, Penaeus setiferus
GROSS SIGNS:	Shrimp uneasy, jumped hitting the cover of the aquarium, dropped to bottom, laid on their sides, jumped again. Dead within 3 hours; often die in upright position. Hemolymph clots slowly and becomes turbid; body muscles may become milky; hemocyte numbers may be reduced; often a pronounced flexure at third abdominal segment.
CAUSE:	Bacterium, Vibrio parahemolyticus -- possibly an exotoxin resulting from it. (Other halophilic vibrios may affect shrimp also).
METHOD OF DIAGNOSIS:	Suspected hemolymph samples streaked on blood agar and standard methods agar, isolates cultured on brain heart infusion agar.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

The pathogen produced high mortalities in brown shrimp caught in Galveston Bay and maintained in aquaria. Isolates from thesee mortalities were not pathogenic for post-larval brown shrimp or juvenile pink shrimpe when tested at another laboratory. Isolatese have been made from pond-reared shrimpse and bait shrimps. Also isolated from whitee shrimp caught in Galveston Bay.e

EFFECTS ON HOST:	Behavioral abnormalities and rapid death.e
TREATMENT:	None reported.e
PREVENTIVE MEASURES:	Water sterilization and filtration; avoid use ofe contaminated natural food; avoid excessivee handling.e

VIBRIO (V. PARAHEMOLYTICUS) DISEASE OF SHRIMPS

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

Ubiquitous in marine waters -- isolated from sediments, crabs, oysters. Also causes enteric disturbances in humans.

NOTE:

A recent paper by Delves-Broughton (1974) reports in vitro sensitivity of V. parahemolyticus and V. anguillarum to Furanace -- a new broad spectrum chemotherapeutic developed in Japan by Shimuzu and Takase (1967). Furanace was found to be non-toxic at treatment levels to the fresh-water shrimp Macrobrachium rosenbergi.

Another vibrio, V. panulirus, has been reported from pond cultured shrimp (Penaeus japonicus) in Japan, where it causes blackening of gills (Kusuda and Watada, 1969).

KEY REFERENCES:

Delves-Broughton, J. 1974. Preliminary investigations into the suitability of a new chemotherapeutic, Furanace, for the treatment of infectious prawn diseases. Aquaculture 3: 175-185.

Kusuda, R. and A. Watada. 1969. A new pathogenic bacterium, belonging to the genus Vibrio, isolated from diseased spiny lobster and prawn. Res. Repts. Köchi Univ. 18(8): 77-79.

Shimizu, M. and Y. Takase. 1967. A potent chemotherapeutic agent against fish diseases, 6-hydroxymethyl 2-(2-5-nitro-2-furyl)vinyl) pyridine, P-7138. Bull. Jap. Soc. Sci. Fish. 33: 544.

Vanderzant, C., R. Nickelson and J. C. Parker. 1970. Isolation of Vibrio parahemolyticus from Gulf Coast shrimp. J. Milk Food Technol. 33: 161-162.

Vanderzant, C., E. Mroz and R. Nickelson. 1970. Microbial flora of Gulf of Mexico and pond shrimp. J.eMilk Foode Technol. 33: 346-350.

Vanderzant, C., R. Nickelson and P. W. Judkins. 1971. Microbial flora of pond-reared brown shrimp (Penaeus aztecus). Appl. Microbiol. 21: 916-921.

Vanderzant, C., B. F. Cobb, C. A. Thompson and J. C. Parker. 1973. Microbial flora, characteristics, and shelf life of four species of pond-reared shrimp. J. Milk Food Technol. 36: 443-446.e

(3) VIBRIO (V. ALGINOLYTICUS) DISEASE OF SHRIMPS

VIBRIO (V. ALGINOLYTICUS) DISEASE OF SHRIMPS

COMMON NAME: Vibrio (V. alginolyticus) disease of shrimp

SPECIES AFFECTED: White shrimp Penaeus setiferus and brown shrimp Penaeus aztecus. Pink shrimp also reported to be susceptible.

GROSS SIGNS: Shrimp become lethargic and disoriented. Experimental infections cause flexure of abdomen at 3rd segment, opaque white abdominal musculature, red discoloration of pleopods and pereiopods, and failure of blood to clot. Infected individuals often die in upright position.

CAUSE: Bacterium, Vibrio alginolyticus (and probably V. anguillarum as well).

METHOD OF DIAGNOSIS: Isolation of bacteria from moribund shrimps with characteristics of V. alginolyticus (culture heart hemolymph on trypticase-soy agar with 2% salt).

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

V. alginolyticus isolated, inoculated, re-isolated from brown shrimp. Experimentalo infections obtained principally by intra-muscular inoculation of bacterial suspensions.o Feeding of isolates to experimental shrimpo seldom produced disease. Natural infectionso thought to occur through ruptures in the cuticle.o Mass mortalities in aquarium-held larvaeo reported. Major losses also in tank-heldo juveniles and adults. Reported to have causedo two mass mortalities in brown shrimp on theo Texas coast. In tanks, outbreaks usually followo handling of shrimps.o

EFFECTS ON HOST: Mortalities in larvae, juveniles, and adults -- in some instances up to 100% of tank-held populations.

VIBRIO (*V. ALGINOLYTICUS*) DISEASE OF SHRIMPS

TREATMENT: Terramycin added to food at minimum rate of 360 mg/kg body weight/day was lowest reported level at which survival was improved.

PREVENTIVE MEASURES: Not reported, but minimal handling of shrimps is suggested, as well as protection from injury to the cuticle, and overcrowding.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: Texas coast.

NOTE: Chan and Lawrence (1974) reported effectiveness of oxytetracycline-oleandomycin combinations in reducing bacterial populations in larval shrimp cultures. Mysis to 10-day post-larval stages tolerated the antibiotics well, but nauplei and protozoea stages gave evidence of declining oxygen consumption at dose levels of 62.5 µg oxytetracycline +25 µg oleandomycin/ml of sea water.

Another vibrio, *V. anguillarum*, was reported (Lewis, in press) to cause death in adult brown shrimp after injection of cultures, and was isolated from bacteremic shrimps from Galveston Bay and from hatchery-reared shrimp (Lightner and Lewis, in press). The three vibrios, *V. parahemolyticus*, *V. alginolyticus* and *V. anguillarum* may or may not produce clearly distinguishable disease entities in shrimps. Methodology for separating the vibrio species has been outlined by Lewis (1973). The picture is complicated by the suggestion of Lightner (in press) that an *Aeromonas* sp. may cause a disease syndrome in shrimps which is similar to that produced by vibrios. Probably the important observation is that vibrios constitute serious sources of infection for shrimps, but other related bacteria may produce similar disease signs.

VIBRIO (V. ALGINOLYTICUS) DISEASE OF SHRIMPS

KEY REFERENCES:

Chan, E. S. and A. W. Lawrence. (in press). Effect of antibiotics on the respiration of brown shrimp larvae to post-larvae, and bacterial populations associated with shrimp. Proc. 5th Ann. Workshop, World Mariculture Soc., Charleston, S. C., 1974.

Corliss, J. (in press). Effect of terramycin on growth and survival and its use in disease control in P. aztecus. Proc. 5th Ann. Workshop, World Mariculture Soc., Charleston, S. C., 1974.

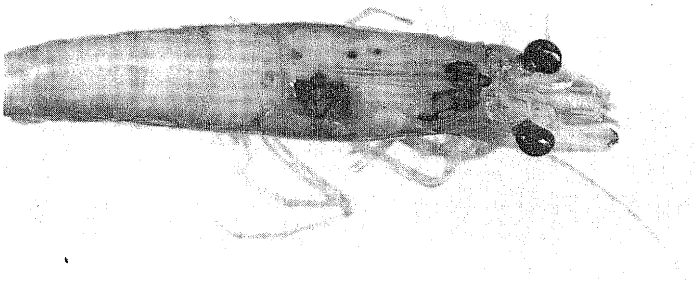
Lewis, D. H. 1973. Predominant aerobic bacteria of fish and shellfish. Texas A&M Univ., Sea Grant Pub. No. 401, 102 pp.

Lewis, D. H. (in press). Response of brown shrimp to infection with Vibrio sp. Proc. Fourth Ann. Workshop, World Maricult. Soc.

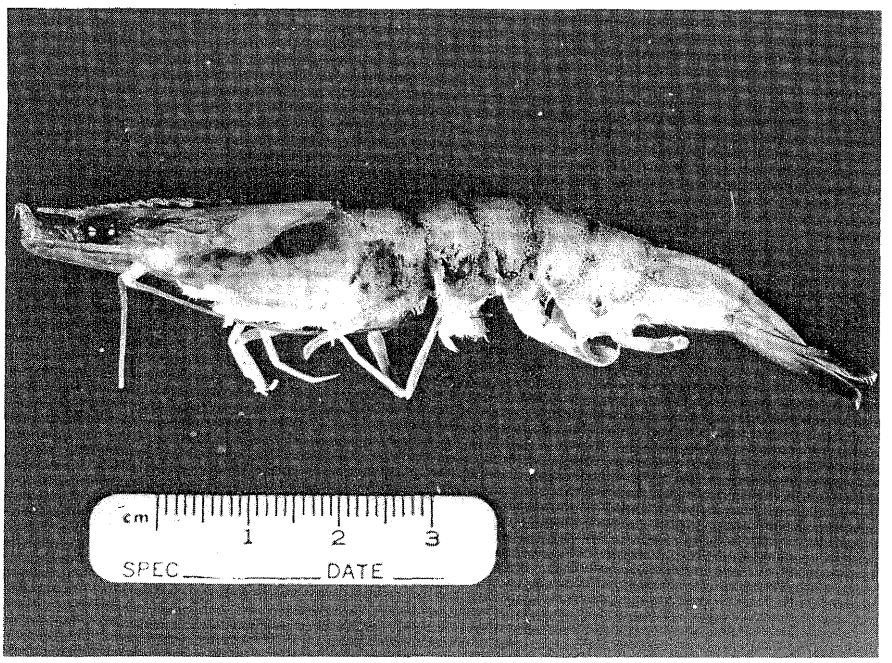
Lightner, D. V. and D. H. Lewis. (in press). Preliminary notes on a septicemic bacterial disease syndrome of penaeid shrimp. Proc. AIBS Symposium on Diseases of Crustaceans (1972).

(4) BROWN SPOT DISEASE

OF SHRIMPS



Brown spot disease of shrimp. Photograph supplied by Robin M. Overstreet, Gulf Coast Research Laboratory.



Brown spot disease of California brown shrimp. Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.

BROWN SPOT DISEASE OF SHRIMPS

COMMON NAME: Brown spot disease (also known as burned spot disease, shell disease and rust disease).

SPECIES AFFECTED: Pink shrimp, Penaeus duorarum
White shrimp, Penaeus setiferus
Brown shrimp, Penaeus aztecus
California brown shrimp, Penaeus californiensis

GROSS SIGNS: Brownish eroded areas on exoskeleton, often beginning as small circular spots.

CAUSE: Several species of chitin-destroying bacteria, probably with secondary bacterial and fungal invaders. Bacterial genera associated with spots include Benekea, Vibrio and Pseudomonas. Vibrio anguillarum isolated as causative agent in Penaeus californiensis.

METHOD OF DIAGNOSIS: Brown spots, often with white margins and depressed centers on exoskeleton; sometimes with necrosis of underlying tissues; bacterial isolates include chitin-destroying organisms.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Chitin-degrading bacteria are ubiquitous in the marine environment and are a normal part of the microbial flora of crustaceans -- living and dead. Increase in bacterial numbers may be favored by certain holding and grow-out situations. Injury to exoskeleton may provide route of entry. In some instances tank-held populations may be infected rapidly -- producing up to 100% infection, with mortalities due to gill destruction.

EFFECT ON HOST: Progressive destruction of exoskeleton, providing route of entry for secondary pathogens. May result in death, due to secondary invaders. An epizootic of shell disease occurred in California brown shrimps in raceways at Puerto Peñasco, Mexico, apparently caused by Vibrio anguillarum. Mortalities of 1-5% per day were observed.

TREATMENT: Disease effects eliminated at molting, except when underlying tissues are damaged by secondary invaders.
In the case of V. anguillarum-caused shell disease in California brown shrimp, mixtures of malachite green and formalin at .05 to 1 ppm and 20 to 75 ppm respectively, were found effective in reducing losses. Also, Terramycin (20 g/45 kg ration fed for 14 days) also seemed effective in preliminary studies.

PREVENTIVE MEASURES: Adequate water filtration and sterilization; remove infected and dead individuals; prevent injuries which probably serve as primary portals of entry.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Generalized occurrence in marine waters of the world; disease has been found in natural populations in tank and pond-reared shrimp, and in many other crustaceans.

NOTE: Fontaine (in press) referred to two types of shell disease in shrimps -- brown or black shell disease caused by chitinoclastic bacteria, and white shell disease possibly caused by a fungus similar to Atkinsiella dubia Sparrow.

Dark lesions of the exoskeleton of Hawaiian fresh-water shrimps, Atya bisulcata, have been recognized recently by J. G. Chan (personal communication). About 20% of individuals sampled from various streams were affected, and Dr. Chan considers the lesions similar to those in penaeid shrimps.

KEY REFERENCES:

Cook, D. W. and S. R. Lofton. 1973. Chitinoclastic bacteria associated with shell disease in Penaeus shrimp and the blue crab (Callinectes sapidus). J. Wildl. Dis. 9: 154-159.

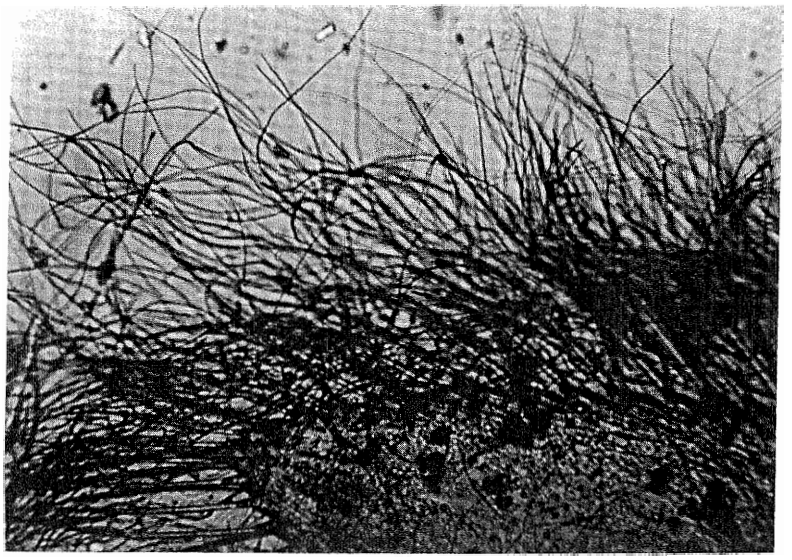
Fontaine, C. T. (in press). Studies on diseases of aquatic animalse at the Gulf Coastal Fisheries Center. Proc. Reg. Sympos. Diseases of Aquatic Animals, LSU (1974).e

Lightner, D. V. (in press). Some potentially serious diseasee problems in the culture of penaeid shrimp in North America. Proc. U.S.-Japan Natural Resources Program, Symposium on Aquaculture Disease, Tokyo, 1974.

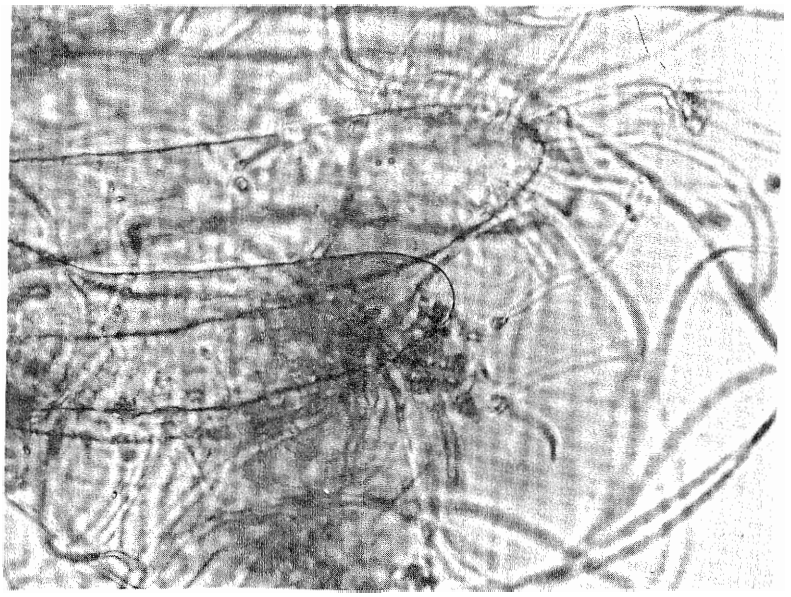
Overstreet, R. M. 1973. Parasites of some penaeid shrimps with emphasis on reared hosts. Aquaculture 2: 105-140.

Rosen, B. 1970. Shell disease of aquatic crustaceans. In. Snieszko, S. F. (ed.). A Symposium on diseases of fishese and shellfishes. pp. 409-415, Spec. Pub. No. 5, Amer. Fish. Soc., Wash., D. C.

(5) FILAMENTOUS BACTERIAL DISEASE OF SHRIMPS



Filamentous bacteria on surface of shrimp larva. Photograph supplied by S. K. Johnson, Texas A&M University.



Leucothrix infestation of brown shrimp larva. Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.

FILAMENTOUS BACTERIAL DISEASE OF SHRIMPS

COMMON NAME: Filamentous bacterial (Leucothrix)disease

SPECIES AFFECTED: Brown shrimp, Penaeus aztecus
White shrimp, Penaeus setiferus
Mexican white shrimp, Penaeus vannamei
California brown shrimp, Penaeus californiensis

GROSS SIGNS: Filamentous growth, often on appendages on post larvae.

CAUSE: Filamentous bacteria of genus Leucothrix, and possibly other genera of filamentous bacteria.

METHOD OF DIAGNOSIS: Direct microscopic examination of fresh mounts, growth on non-selective media, followed by reinoculation into culture water.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Infestations often, but not always, associated with poor water quality.

EFFECT ON HOST: Can produce mortalities of post larvae with heavy infestations. Adult shrimp can be killed by inoculation of cultured bacteria. Mortality of shrimps with heavy infestations of gills usually occurs during or immediately following molting.

TREATMENT: Potassium permanganate (5 to 10 ppm in 1 hour static treatments) effective for 5 to 10 days.

PREVENTIVE MEASURES: Maintenance of good water quality.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Texas shrimp culture ponds, Mississippi.

NOTE: There is little question that filamentous bacteria can cause problems in shrimp culture. In most instances specific identifications have not been made, and it is even possible that blue-green algae may be involved.

FILAMENTOUS BACTERIAL DISEASE
●F SHRIMPS

Fontaine (in press) described "brown gill disease" of brown shrimp which he attributed to mats of filamentous algae on gills which accumulated detritus and led to suffocation.

Ishikawa (1966, 1967) reported a disease in cultured post-larval and adult Kuruma-prawn caused by a bacterium resembling Leucothrix mucor. Heavy infestation of gills apparently affected respiration and caused high mortality. The colorless filamentous microorganism densely covered the body and gills of many postlarvae.

Other ectocommensals can be found on gills and body surfaces of cultured shrimp, especially when water quality is poor. Overstreet (1973) reported the blue-green algae Schizothrix calcicola from Alabama and Louisiana shrimps, and the encrusting hydroid Obelia bicuspidata from Alabama brown shrimps.

KEY REFERENCES:

Barkate, J. A., C.sR. Laramore, Y. Hirono, and H. Persyn. (in press). Some marine microorganisms related to shrimps diseases. Proc. 5th Ann. Workshop, World Mariculture Soc., 1974.

Fontaine, C. T. (in press). Studies on diseases of aquatic animals at the Gulf Coastal Fisheries Center. Proc. Reg. Sympos. Diseases of Aquatic Animals, LSU (1974).

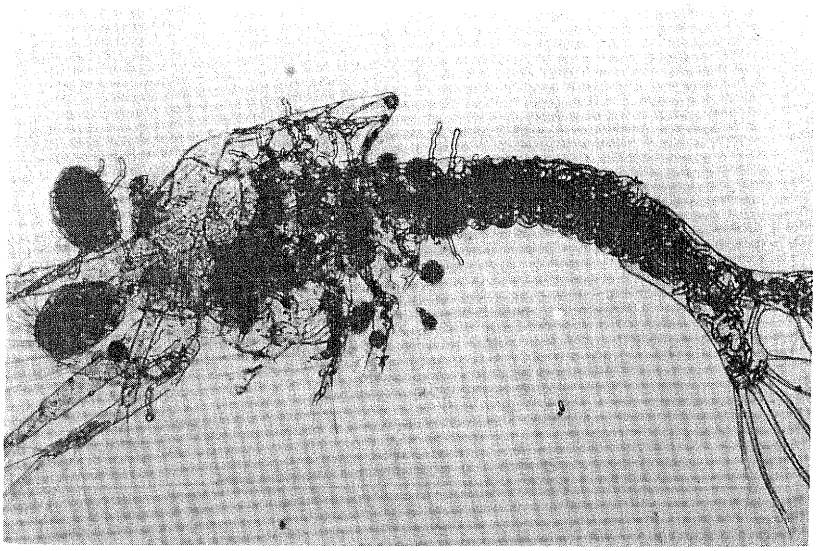
Ishikawa, Y. 1966. A disease of young cultured Kuruma-prawn Penaeus japonicus Bate (In Japanese). Bull. Fish Expt. Sta. Okayama Pref. 1966. pp. 5-9.

Ishikawa, Y. 1967. On the filamentous bacteria which grow on the gills of cultured Kuruma-prawn (In Japanese). Fish Pathol. 2: 68-72.s

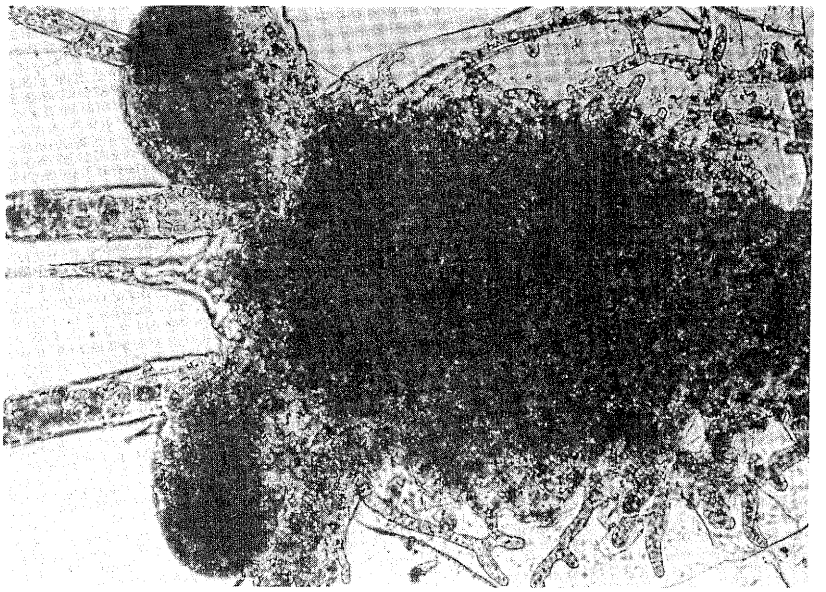
Johnson, S. K. 1974. Ectocommensals and parasites of shrimp from Texas rearing ponds. Texas A&M University, Sea Grant Report No. TAMU-SG-74-207, 20 pp.

(6) LARVAL MYCOSIS

OF SHRIMPS



Lagenidium infection of white shrimp larva. Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.



Lagenidium mycelium in tissues of white shrimp larva. Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.

LARVAL MYCOSIS
OF SHRIMPS

COMMON NAME: Larval Mycosis

SPECIES AFFECTED: White shrimp, Penaeus setiferus
Brown shrimp, Penaeus aztecus

GROSS SIGNS: Systemic infection of larvae, with extensive, highly-branched fungal mycelium throughout body, yellowish green in color, with numerous oil droplets.

CAUSE: Fungus Lagenidium sp.

METHOD OF DIAGNOSIS: Isolates grown on Sabouraud agar or broth; sporulation induced by transfer of cultured mycelium to sterile sea water.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Fungal mycelium gradually invades and replaces all tissues of larval shrimp, infected individuals become immobile and settle to bottom of tank. Sporogenesis then begins with formation of exit tubes and release of free-swimming biflagellate zoospores. Zoospores settle and encyst, then send germ tube into larvae. Epizootics have been produced experimentally in brown shrimp larvae.

EFFECT ON HOST: Mortalities produced rapidly in hatchery tanks among larvae up to first mysis stage only. Mortalities may reach 100% within 2 days.

TREATMENT: Malachite green reported as effective at .001 to .006 ppm., but toxicity to larvae not yet fully tested (Bland, in press).

PREVENTIVE MEASURES: Chlorination and filtration of water reported as an effective control measure.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Reported from a Texas shrimp laboratory and hatchery. Other species of Lagenidium occur in eggs of blue crabs, in barnacles, and other crustaceans.

NOTE: This fungus is probably identical to the Lagenidium reported by Cook (1971) from brown shrimp larvae in Texas. Cook also reported a fungus resembling Dermocystidium in brown shrimp larvae, and another unidentified fungus which caused mortalities in hatchery-reared juvenile brown shrimp.

KEY REFERENCES:

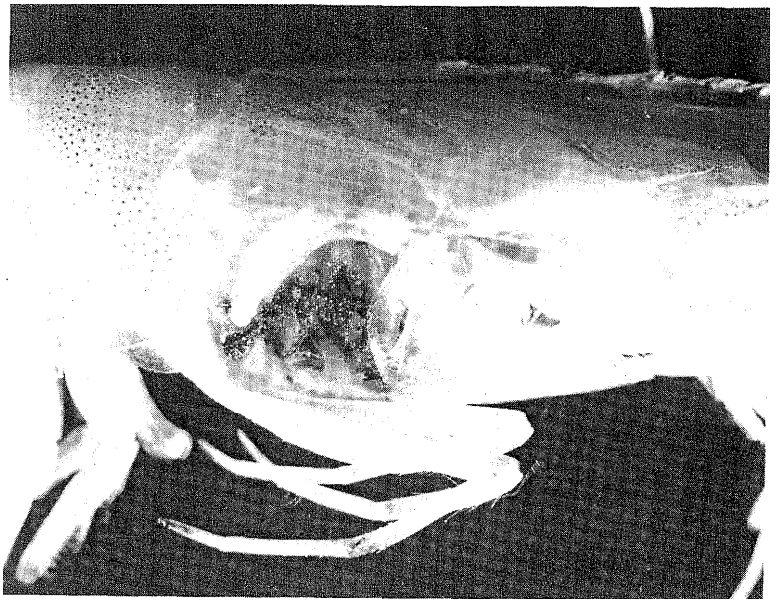
Anonymous (Cook, H. L.). 1971. Fungi parasitic on shrimp. FAO Aquaculture Bull. 3(4): 13.

Barkate, J. A., C. R. Laramore, Y. Hirono and H. Persyn. (in press). Some marine microorganisms related to shrimp diseases. Proc. 5th Ann. Workshop, World Maricult. Soc., Charleston, S. C., 1974.

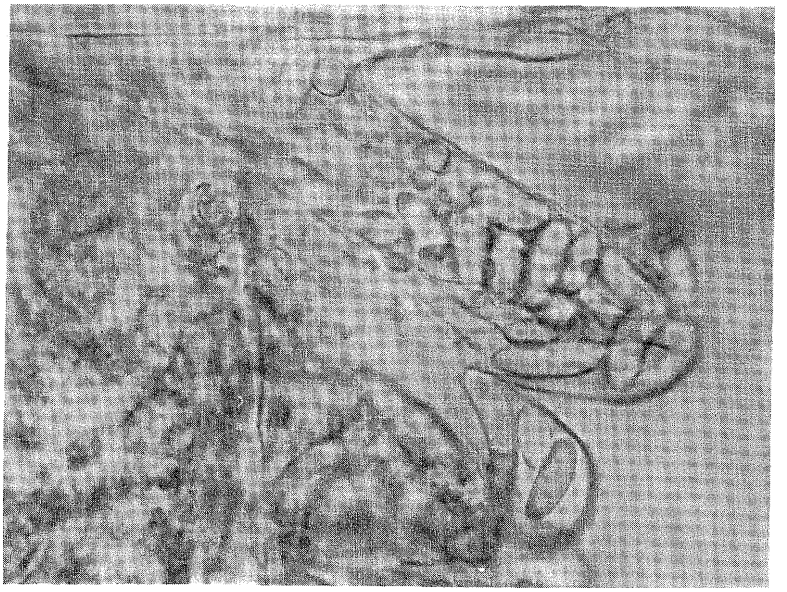
Bland, C. E. (in press). A survey of fungal diseases of marine organisms with emphasis on current research concerning Lagenidium callinectes. Proc. Gulf Coast Regional Symposium on Diseases of Aquatic Animals, Baton Rouge, La., 1974.

Lightner, D. V. and C. T. Fontaine. 1973. A new fungus disease of the white shrimp Penaeus setiferus. J.eInvert. Pathol. 22:e 94-99.

(7) FUNGUS (FUSARIUM) DISEASE OF SHRIMPS



Fusarium in California brown shrimp. Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.



Fusarium spores in gills of California brown shrimp (post mortem). Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.

FUNGUS (FUSARIUM) DISEASE OF SHRIMPS

COMMON NAME: Fungus (Fusarium) disease of shrimps.

SPECIES AFFECTED: Pink shrimp, Penaeus duorarum
California brown shrimp, Penaeus californiensis

GROSS SIGNS: Black gills (in P. californiensis)

CAUSE: Fungus Fusarium sp. (possibly several species of Fusarium and other fungi may be involved as well).

METHOD OF DIAGNOSIS: Fresh mounts of infected tissue with hyphae. Characteristic boat-shaped macroconidia formed. Isolation on Sabouraud agar, with formation of macro and micro conidia, and production of brown diffusible pigment.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Infection may begin at several loci and spread slowly, eventually affecting up to 10% of the body. Infected individuals made up only small proportion of total population of aquarium-held shrimps in one study.

EFFECT ON HOST: Reported as the cause of a severe epizootic in California brown shrimp of 10 cm being held in raceways at Puerto Peñasco, Mexico, with up to 100% incidence in one raceway, and with approximately 90% mortality in that raceway. The fungus typically affected gills, basal segments of walking legs, and body wall behind the gills.

TREATMENT: Malachite green (.05 to .1 ppm) for 24 hours effective against exposed spores and hyphae, but internal hyphae and spores not affected.

PREVENTIVE MEASURES: Not reported. Elimination of sources of spores and destruction of infected individuals suggested.

FUNGUS (FUSARIUM) DISEASE OF SHRIMPS

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

Reported thus far only from aquarium-held pink shrimp in Texas, and from California brown shrimp in raceways in Mexico.

NOTE:

A Fusarium sp. was reported by Egusa and Ueda (1972) as causing a black gill disease in Penaeus japonicus.

Hatai et al. (1974) tested the effectiveness of 40 chemicals against the infection, and found several (especially Nystatin and Azalomycin F) to be effective.

KEY REFERENCES:

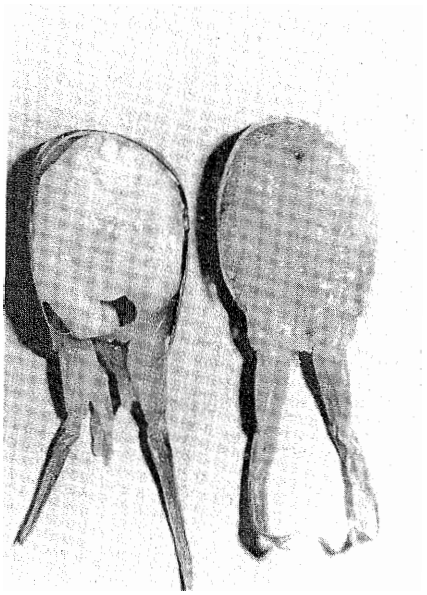
Egusa, S. and T. Ueda. 1972. A Fusarium sp. associated with black gill disease of the Kuruma Prawn, Penaeus japonicus Bate. Bull. Jap. Soc. Sci. Fish. 38: 1253-1260.

Hatai, K., K. Nakajima and S. Egusa. 1974. Effect of some fungicides on black gill disease of Kuruma prawn, Penaeus japonicus, caused by Fusarium sp. (in Japanese). Fish. Pathol. 8: 156-160.

Johnson, S. K. 1974. Fusarium sp. in laboratory-held pink shrimp. Texas A&M University, Texas Agricultural Extension Service, Fish Disease Diagnostic Laboratory, Publication No. FDDL-51. (May, 1973): 1 p.

Lightner, D. V. (in press). Some potentially serious disease problems in the culture of penaeid shrimp in North America. Proc. U.S.-Japan Natural Resources Program, Symposium on Aquaculture Diseases, Tokyo, 1974.

(8) MILK OR COTTON DISEASE OF SHRIMPS



Cotton shrimp (left). Photograph supplied by Robin M. Overstreet, Gulf Coast Research Laboratory.

MILK OR COTTON DISEASE OF SHRIMPS

COMMON NAME: Milk shrimp, Cotton shrimp

SPECIES AFFECTED: Pink shrimp, Penaeus duorarum
White shrimp, Penaeus setiferus
Brown shrimp, Penaeus aztecus

GROSS SIGNS: Opaque white areas in abdominal muscles, often extensive, sometimes with blue-black color on back and sides of shrimp. Also invades digestive tract and heart.

CAUSE: Several microsporidan protozoans: Nosema nelsoni, Pleistophora sp., Thelohania duorara.

METHOD OF DIAGNOSIS: Gross signs provide a good clue. Blue-black pigmentation especially found with Pleistophora sp. infections. Microscopic examination of fresh squashes from infected muscles will disclose multitudes of characteristic microsporidan spores. Polar filaments extruded from fresh spores with mechanical pressure; stained spores used for identification.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Transmission probably by ingestion of spores, or of intermediate hosts which have fed on spores. Multiple infections can occur. Found in bait and adult shrimp.

EFFECT ON HOST: Infected individuals can be weakened or killed, especially if other environmental stresses exist. Infection seems to inhibit normal migration.

TREATMENT: None reported.

PREVENTIVE MEASURES: Destroy infected individuals; avoid contact of infected brood stock or infected egg-bearing females with offspring; sterilize tanks.

MILK OR COTTON DISEASE
OF SHRIMPS

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Gulf of Mexico and South Atlantic Coast of United States.

KEY REFERENCES:

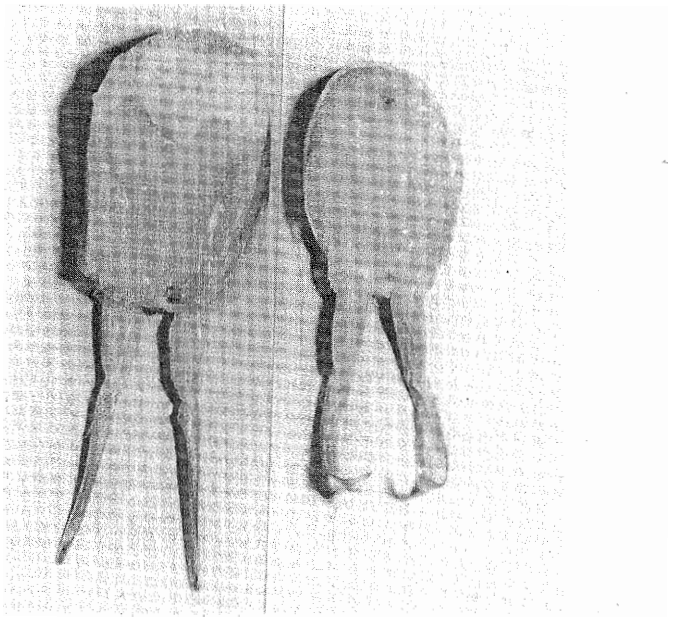
Overstreet, R. M. 1973. Parasites of some penaeid shrimps with emphasis on reared hosts. Aquaculture 2: 105-140.

Sprague, V. 1950. Notes on three microsporidian parasites of decapod Crustacea of Louisiana coastal waters. Occ. Pap. Mar. Lab. La. State Univ. No. 5, 7 pp.

Sprague, V. 1970. Some protozoan parasites and hyperparasites in marine decapod Crustacea. In. Snieszko, S. F. (ed.). A symposium on diseases of fishes and shellfishes. pp. 416-430, Spec. Pub. No. 5, Am. Fish. Soc., Wash., D. C.

(9) MICROSPORIDIOSIS OF REPRODUCTIVE ORGANS OF SHRIMPS

MICROSPORIDIOSIS OF
REPRODUCTIVE ORGANS
OF SHRIMPS



Microsporidan infection of shrimp gonad (left).
Photograph supplied by Robin M. Overstreet,
Gulf Coast Research Laboratory.

MICROSPORIDIOSIS OF REPRODUCTIVE ORGANS OF SHRIMPS

COMMON NAME: Microsporidiosis of Reproductive Organs

SPECIES AFFECTED: White shrimp, Penaeus setiferus (on rare occasions found in other species).

GROSS SIGNS: Opaque white areas -- often extensive -- in cephalothorax, reproductive organs, blood vessels and digestive tract, along dorsal midline of shrimp.

CAUSE: Microsporidan protozoan Thelohania penaei.

METHOD OF DIAGNOSIS: Differentiated from other microsporidan infections by being concentrated in dorsal areas of shrimp, and not usually invading muscles of tail. Characteristic microsporidan spores of two size groups seen in microscope preparations. T. penaei appears to be a parasite of smooth muscle while other microsporidans of shrimps typically invade body muscles.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: This may have been the disease reported by Viosca (1945) as destroying the reproductive organs of 90% of the white shrimp sampled on the Louisiana coast in 1919. Transmission of infection may be by infected eggs, or by shrimps eating spores or intermediate hosts which had eaten spores.

EFFECT ON HOST: Infected shrimp can be castrated, weakened, or killed. Infection renders shrimp more vulnerable to other environmental stresses.

TREATMENT: None reported.

PREVENTIVE MEASURES: Destroy infected individuals; avoid contact of infected brood stock with offspring; sterilize tanks.

MICROSPORIDIOSIS OF
REPRODUCTIVE ORGANS
OF SHRIMPS

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Gulf of Mexico and South Atlantic coast of United States.

NOTE: Shrimp affected by gonadal microsporidiosis sometimes called "roe" shrimp, and sometimes "cotton" or "milk" shrimp also.

KEY REFERENCES:

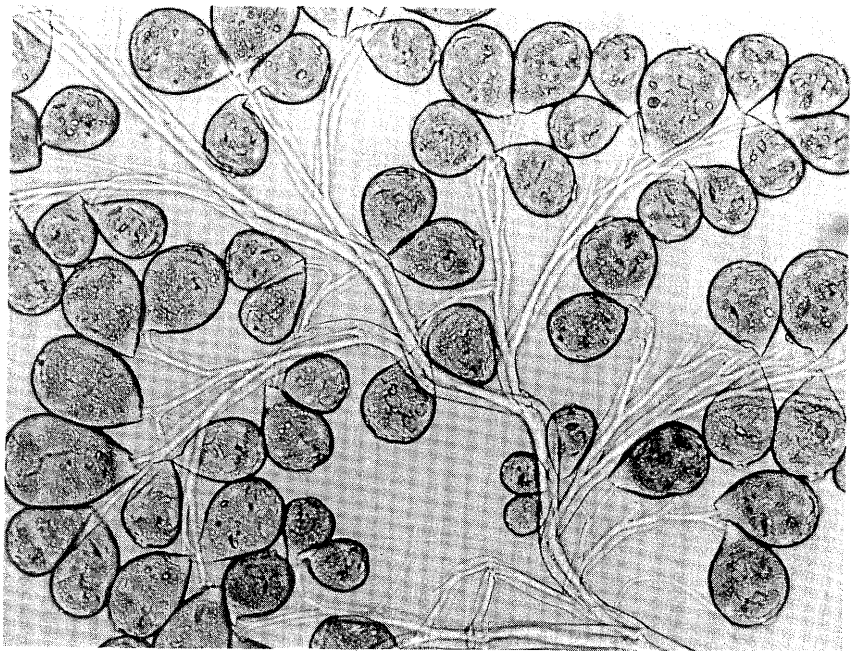
Overstreet, R. M. 1973. Parasites of some penaeid shrimps with emphasis on reared hosts. Aquaculture 2: 105-140.

Sprague, V. 1950. Notes on three microsporidian parasites of decapod Crustacea of Louisiana coastal waters. Occ. Pap. Mar. Lab. La. State Univ. No. 5, 7 pp.

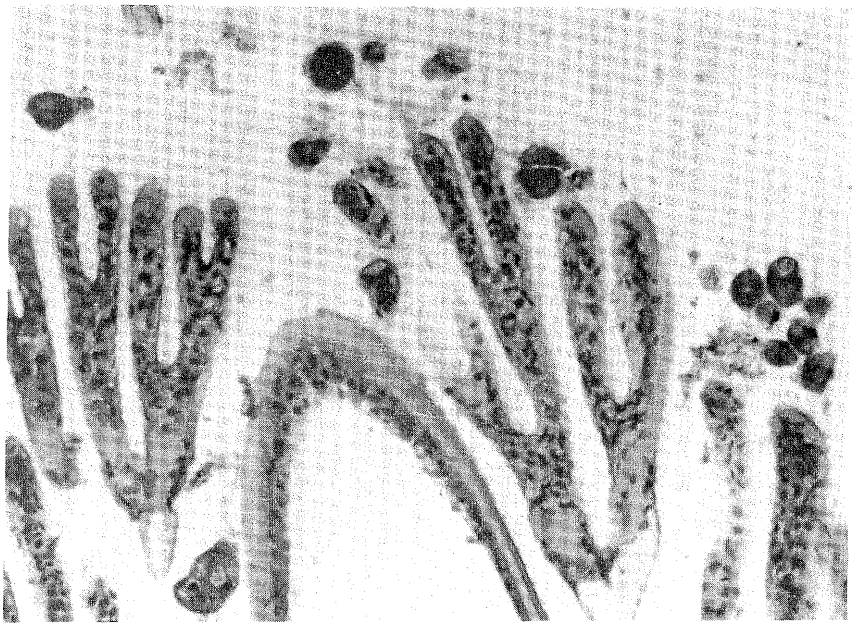
Sprague, V. 1970. Some protozoan parasites and hyperparasites in marine decapod Crustacea. In. Snieszko, S. F. (ed.). A symposium on diseases of fishes and shellfishes. pp. 416-430, Spec. Pub. No. 5, Am. Fish. Soc., Wash., D. C.

Viosca, P. 1945. A critical analysis of practices in the management of warm-water fish with a view to greater food production. Trans. Amer. Fish. Soc. 73: 274-283.

(10) CILIATE (ZCOTHAMNIUM) DISEASE OF SHRIMPS



Zoothamnium colony from gills of brown shrimp.



Histological section of gills of white shrimp with Zoothamnium infestation. Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.

CILIATE DISEASE
OF SHRIMPS

COMMON NAME: Ciliate disease

SPECIES AFFECTED: Pink shrimp, Penaeus duorarum
White shrimp, Penaeus setiferus
Brown shrimp, Penaeus aztecus

GROSS SIGNS: Heavy infections may produce a fuzzy mat on gill surfaces, and occasionally on eyes, appendages, and carapace. Heavily infected shrimps may exhibit generalized disease signs of lethargy, white discoloration of abdomal muscles, dorsal flexure of abdomen, and redness of appendages.

CAUSE: Stalked peritrichous ciliate protozoan Zoothamnium sp. (and possibly other ciliates, particularly of the genera Epistylus and Acineta). Zoothamnium occurs most frequently on the gills, whereas Epistylus and Acineta usually occur on the body surfaces and appendages.

METHOD OF DIAGNOSIS: Fresh mounts of gill material examined microscopically at low magnification disclose characteristic stalked colonial organisms with connecting roots.

LIFE HISTORY, BICLOGY, EPIZOOTICLOGY:
Infestations spread by free-swimming stages called telatrochs. One of most common organisms found on pond-reared shrimp.

EFFECT ON HOST: Heavy infestations of Zoothamnium may cause mortalities in shrimp culture ponds -- particularly among young individuals, and particularly when infestations are heavy and oxygen levels are low. Light infestations do not seem to affect growth of large shrimp. Primary effect is interference with gill gas exchange.

TREATMENT: 25 ppm formalin dip found successful by Johnson et al (in press). The organism was reported to be a low salinity form with an optimum at 10-12 parts per thousand. Raising salinity to 20 ppt reported to be an effective treatment (Fontaine, in press).

PREVENTIVE MEASURES: Rigid sanitary control of culture water.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: Gulf of Mexico and South Atlantic Coast of United States (also reported from the Mexican shrimp Penaeus vannamei and P. occidentalis.

NOTE: Zoothamnium does not cause tissue damage, but the related loricate peritrich Lagenophrys can cause extensive tissue damage to gills -- producing leucocytic infiltration and melanin deposition. A "scab" is formed which sloughs off with the parasite. Lagenophrys has been reported from the body surfaces of white shrimp (Johnson, 1974). Ciliates (probably Epistylus) also occur on cultured fresh-water shrimps, Macrobrachium rosenbergii, sometimes forming a mat on the exoskeleton.

KEY REFERENCES:

Anonymous. 1973. Protozoan and nematode parasites of Mexican shrimp. FAO Aquaculture Bull. 5(3-4): 17.

Fontaine, C. T. (in press). Studies on diseases of aquatic animals at the Gulf Coastal Fisheries Center. Proc. Gulf Coast Regional Symposium on Diseases of Aquatic Animals, Baton Rouge, La., 1974.

Johnson, S. K. 1972. Epistylus sp. infestations on penaeid shrimp. FAO Aquaculture Bull. 4(4): 15.

Johnson, S. K. 1974. Ectocommensals and parasites of shrimp from Texas rearing ponds. Texas A&M University, Sea Grant Publication No. TAMU-56-74-207, 20 pp.

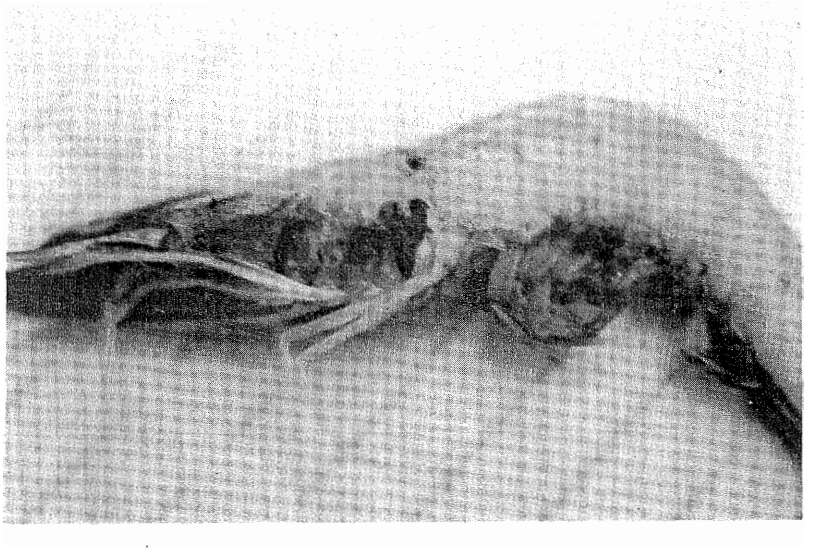
Johnson, S. K., J. C. Parker and W. H. Holcomb. (in press). Control of Zoothamnium on penaeid shrimp. Proc. Fourth Annual Workshop, World Mariculture Soc. (1973, Mexico City).

Overstreet, R. M. 1973. Parasites of some penaeid shrimps with emphasis on reared hosts. Aquaculture 2: 105-140.

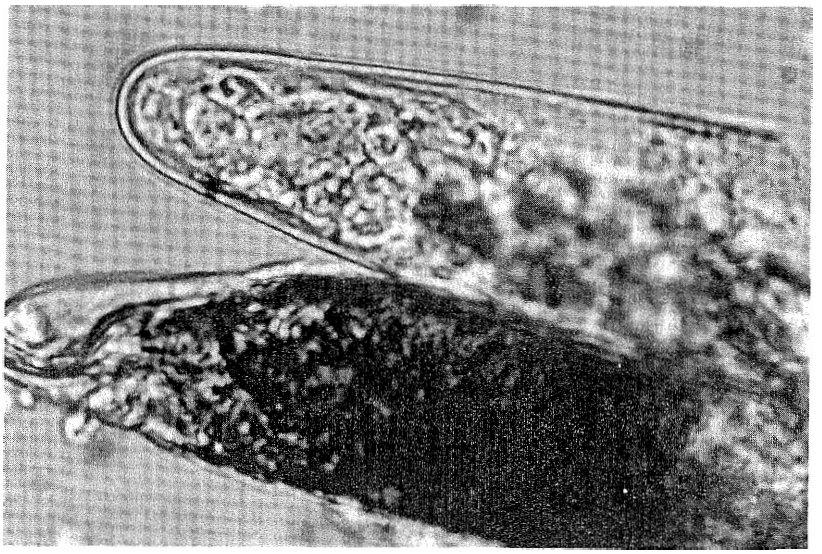
Villella, J.oB., E. S. Iversen and C. J. Sindermann. 1970. Comparison of the parasites of pond-reared and wild pink shrimp (Penaeus duorarum Burkenroad) in South Florida. Trans. Am. Fish. Soc. 99: 788-794.

(11) BLACK GILL DISEASE

OF SHRIMPS



Black gill disease of shrimp. Photograph supplied by S. K. Johnson, Texas A&M University.



Close-up of black gills of shrimp. Photograph supplied by S. K. Johnson, Texas A&M University.

BLACK GILL DISEASE OF SHRIMPS

COMMON NAME: Black gill disease

SPECIES AFFECTED: Mexican white shrimp, Penaeus vannamei

GROSS SIGNS: Brownish discoloration and atrophy of tips of gill filaments. In advanced cases most of the filaments are affected and the gills take on a blackened gross appearance.

CAUSE: Unknown. Attempts to isolate bacteria and fungi were unsuccessful. (A black gill disease of Japanes shrimps (P. japonicus) was reported by Egusa and Ueda (1972) as being caused by a fungus Fusarium sp.). Ishikawa (1968) also described gill blackening in Japanese shrimps.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Disease is progressive, beginning with involvement of only a few gill filaments.

EFFECT ON HOST: Not described.

TREATMENT: Not described.

PREVENTIVE MEASURES: Not described.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: Described thus far only from Texas rearing ponds, but seen by others from other shrimp rearing areas.

NOTE: Blackened gills are common in Crustacea from the badly degraded areas of the New York Bight. Blackening may be an accretion of sediments, material deposited by an organism, or intracellular pigment in the gills. Concomitant with discoloration, high prevalences of ciliates have been observed.

BLACK GILL DISEASE OF SHRIMPS

A "black spot gill syndrome" of northern shrimp, Pandalus borealis, has recently been described by Rinaldo and Yevitch (1974). The disease, of unknown etiology, destroys gill tissue, and is recognizable by macroscopic black spots on gills. A similar condition was reported earlier (Uzmann and Haynes, 1968) in the closely related pandalid shrimp Dichelopandalus leptocercus.

Brownish or black gills may be a generalized pathological sign, produced by pigment deposition as an aftermath of tissue destruction. Possible association with chitin-destroying bacteria exists.

KEY REFERENCES:

Egusa, S. and T. Ueda. 1972. A Fusarium sp. associated with black gill disease of the Kuruma Prawn, Penaeus japonicus Bate. Bull. Jap. Soc. Sci. Fish. 38: 1253-1260.

Ishikawa, Y. 1968. Observations on the gill blackening of cultured Kuruma-prawn, Penaeus japonicus Bate. (In Japanese). Fish. Pathol. 3: 34-38.

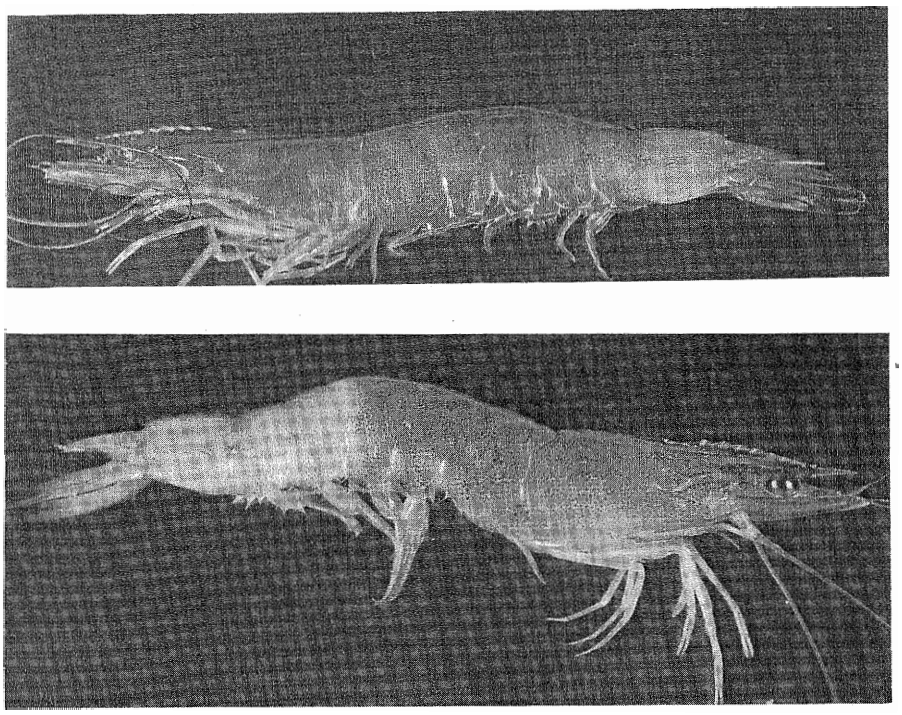
Johnson, S. K. 1974. Ectocommensals and parasites of shrimp from Texas rearing ponds. Texas A&M University, Sea Grant Publication No. TAMU-SG-74-207, 70 pp.

Rinaldo, R. G. and P. Yevich. 1974. Black spot gill syndrome of the northern shrimp Pandalus borealis. J. Invert. Pathol. 24: 224-233.

Uzmann, J. R. and E. B. Haynes. 1968. A mycosis of the gills of the pandalid shrimp, Dichelopandalus leptocerus (Smith). J. Invert. Pathol. 12: 275-277.

(12) MUSCLE NECROSIS

OF SHRIMPS



Muscle necrosis of brown shrimp. Early (above) and advanced (below). Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.

MUSCLE NECROSIS
OF SHRIMPS

COMMON NAME: Muscle Necrosis

SPECIES AFFECTED: Brown shrimp, Penaeus aztecus

GROSS SIGNS: White patches develop in abdominal segments, rapidly expanding all along the abdomen, resulting in necrosis and death in 24 hours unless stress conditions corrected.

CAUSE: Overcrowding, low oxygen pressure, sudden salinity-temperature fluctuations. Some secondary bacterial infections seen.

METHOD OF DIAGNOSIS: Gross observation; histologic examination disclosing degenerating striated muscle and absence of microsporidan parasites.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Related to sudden and severe environmental change. May be produced instantaneously, and may be reversible in initial stages if stress factors reduced promptly.

EFFECT ON HOST: Muscle necrosis often followed by death.

TREATMENT: Reduce stress conditions.

PREVENTIVE MEASURES: Avoid overcrowding, high water temperature, low oxygen levels, excessive handling.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
The condition thus far has been reported only from the Gulf of Mexico region.

NOTE: This condition is sometimes called "tail rot" by shrimp fishermen. A similar opacity of abdominal muscles has been seen in cultured fresh-water shrimps, Macrobrachium rosenbergii.

KEY REFERENCES:

Anonymous. 1971. "Necrosis" in shrimp. FAO Aquaculture Bull. 3(3): 11.

Anonymous. 1971. Necrosis in shrimp. FAO Aquaculture Bull. 4(1): 3.

Rigdon, R. H. and K. N. Baxter. 1970. Spontaneous necrosis in muscles of brown shrimp, Penaeus aztecus Ives. Trans. Amer. Fish. Soc. 99: 583-587.

Fresh-water Shrimp Diseases

Within the past five years, attention in a number of countries, including the United States, has been focused on several species of fresh-water shrimps belonging to the genus Macrobrachium. Because of their outstanding mariculture potential, research and development (as well as pilot scale production) ventures have been organized in Florida, Hawaii, the West Indies, Central America, and elsewhere. Because spawning and larval development take place in saline water, these shrimps logically become part of mariculture.

Thus far only three ill-defined diseases -- one probably bacterial, one possibly caused by a fungus, the third attributed to a protozoan -- have been recognized as present problems. Published information about even these diseases is very limited.

Fujimura (1972) reported heavy mortality of larvae due to water pollution from decomposition of uneaten food. Mortality was heaviest during the last stages of larval development. Mortality was also related to overexposure to sunlight, which reduced feeding.

KEY REFERENCES:

Fujimura, T. and H. Okamoto. 1972. Notes on progress made in developing a mass culturing technique for Macrobrachium rosenbergii in Hawaii. In. Pillay, T.V.R. (ed.). Coastal Aquaculture in the Indo-Pacific Region, pp. 313-327. Fishing News (Books) Ltd. London. 497 pp.

Ling, S. W. 1967. The general biology and development of Macrobrachium rosenbergii (deMan). FAO Sci. Conf. Biol. and Culture of Shrimps and Prawns. Mexico City.

(1) BLACK SPOT DISEASE

OF FRESH-WATER SHRIMPS

BLACK SPOT DISEASE OF
FRESH-WATER SHRIMPS

COMMON NAME: Black Spot Disease

SPECIES AFFECTED: Fresh-water shrimps, Macrobrachium vollenhovenii and M. rosenbergii

CAUSE: Not described for M. vollenhovenii but probably chitin-destroying bacteria or fungi involved. Chitinoclastic bacteria tentatively identified as Benekea sp. isolated from M. rosenbergii.

GROSS SIGNS: Progressive erosion of exoskeleton, beginning as small brown to black lesions.

METHOD OF DIAGNOSIS: Not described; gross signs only, for M.svollenhovenii. Chitin culture mediums for M. rosenbergii.s

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Not described.

TREATMENT: Furanace tested, with variable results.

PREVENTIVE MEASURES: Not described.

EFFECT ON HOST: Infection eroded exoskeleton and attacked underlying tissues. Areas most affected were gill filaments, ventral abdominal muscles, telson, and walking legs. In final stages, shrimp lie on sides with movements restricted to pleopods and gill covers.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
England, Liberia.

NOTE: A similar condition has been seen in English prawn, Palaemon serratus by Anderson and Conroy, 1968, and was related by them to infection by a species of the fungus Pythium. Another similar condition in brown shrimp from Alabama was reported by Barkate (1972) Gram-negative bacilli, but no fungi, were isolated.

KEY REFERENCES:

Barkate, J. A. 1972. Preliminary studies of some shrimp diseases. Proc. 3rd Ann. Workshop, World Mariculture Soc. pp. 337-346.

Delves-Broughton, J. 1974. Preliminary investigations into the suitability of a new chemotherapeutic, Furanace, for the treatment of infectious prawn diseases. Aquaculture 3: 175-185.

Miller, G. C. 1971. Commercial fishery and biology of the fresh-water shrimp Macrobrachium in the lower St. Paul River, Liberia, 1952-55. U. S. Dept. Commerce, NOAA, NMFS, Spec. Sci. Rept. - Fish. No. 626.

(2) FUNGUS DISEASE OF FRESH-WATER SHRIMP LARVAE

FUNGUS DISEASE OF
FRESH-WATER SHRIMPS

COMMON NAME: Fungus disease of fresh-water shrimp larvae.

SPECIES AFFECTED: Fresh-water shrimp, Macrobrachium rosenbergii

GROSS SIGNS: Small opaque whitish patches occur first at base of appendages and in tail of larvae, then spread throughout entire body.

CAUSE: Considered to be a fungus infection by Ling (1969) but not further identified.

METHOD OF DIAGNOSIS: Not described. Gross appearance only.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY.
Not described.

EFFECT ON HOST: Produces sporadic heavy larval mortalities.

TREATMENT: Not described.

PREVENTIVE MEASURES: Infected larvae, or entire infected batches of larvae, should be removed and destroyed. Troughs and tanks should be cleaned and disinfected. Water should be filtered.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Thus far only reported from Malaysia, but since M. rosenbergii has been transferred to other countries, including the United States, the disease may be found elsewhere.

KEY REFERENCES:

Fujimura, T. 1966. Notes on the development of a practical mass culture technique of the giant prawn, Macrobrachium rosenbergii. Paper presented to the Indo-Pacific Fisheries Council, 12th Session. IPFC/C66/WP47, 4 pp.

Ling, S. W. 1969a. The general biology and development of Macrobrachium rosenbergii (deMan). FAO Fish. Rep. 3(57): 589-606.

Ling, S. W. 1969b. Methods of rearing and culturing Macrobrachium rosenbergii (deMan). FAO Fish. Rep. 3(57): 607-619.

(3) PROTOZOAN DISEASE OF FRESH-WATER SHRIMPS

PROTOZOAN DISEASE OF
FRESH-WATER SHRIMPS

COMMON NAME: Protozoan disease

SPECIES AFFECTED: Fresh-water shrimp, Macrobrachium rosenbergii

GROSS SIGNS: Not described.

CAUSE: Protozoan (not further identified).

METHOD OF DIAGNOSIS: Not described.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Not described.

EFFECT ON HOST: High mortalities reported.

TREATMENT: Malachite green (0.2 ppm for 1/2 hr daily); formalin (200 ppm for 1/2 hr daily); copper sulfate (0.4 ppm for 6 hrs, repeated at 24 hr intervals).

PREVENTIVE MEASURES: Not described.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: Known from Malaysia (Ling, 1969) and Hawaii (Fujimura, 1966).

KEY REFERENCES:

Fujimura, T. 1966. Notes on the development of a practical mass culture technique of the giant prawn, Macrobrachium rosenbergii. Paper presented to the Indo-Pacific Fisheries Council, 12th Session, IPFC/C66/WP47, 4 pp.

Ling, S. W. 1969. Methods of rearing and culturing Macrobrachium rosenbergii (deMan). FAO Fish. Rep. 3(57): 607-619.

Blue Crab Diseases

Annual production of blue crabs, Callinectes sapidus, on the United States East and Gulf coasts is highly variable from year to year, apparently because of variation in survival of year classes. This fact, combined with increasing price and market demand for crabs, makes the blue crab an excellent potential mariculture species. Highest value is placed on soft-shelled crabs, and an elaborate system of holding (shedding) tanks has been devised to provide such crabs.

Diseases of blue crabs have been recognized from captive populations in shedding tanks, as well as from natural populations. Thus far seven disease entities, of potential mariculture significance have been reported:

(1)e Vibrio disease;e

(2)e Shell disease;e

(3)e Egg fungus disease;e

(4)e Nosema disease;e

(5)e Gray crab disease;e

(6)e Ciliate disease; ande

(7)e Hematodinium disease.e

Other protozoan, worm, and crustacean parasites of blue crabs are known, but they seem less likely to cause significant problems in mariculture.

(1) VIBRIO DISEASE OF

BLUE CRABS

VIBRIO DISEASE OF
BLUE CRABS

COMMON NAME: Vibrio disease

SPECIES AFFECTED: Blue crab, Callinectes sapidus

GROSS SIGNS: Lethargic, weak individuals; mortalities, often extensive subsequent to onset of disease signs.

CAUSE: Bacterium Vibrio parahemolyticus.

METHOD OF DIAGNOSIS: Large numbers of bacteria in hemolymph; suspected samples streaked on blood agar and standard methods agar; isolates cultured on brain heart infusion agar. Isolates capable of producing mortality within a few hours after inoculation.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Isolates of V. parahemolyticus have been made from sea-water, marine sediments and fish and shellfish from several locations in the world, and the organism (and its closely related variants) is probably in and on healthy animals, as well as sick ones.

EFFECT ON HOST: May contribute to mortalities of crabs held in shedding tanks, since the organism has been isolated from lethargic and moribund crabs being held in commercial shedding tanks. Mortalities in some tanks were in excess of 50%.

TREATMENT: None reported.

PREVENTIVE MEASURES: Water sterilization and filtration.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Ubiquitous in marine environment. Isolated from blue crabs from Chesapeake Bay.

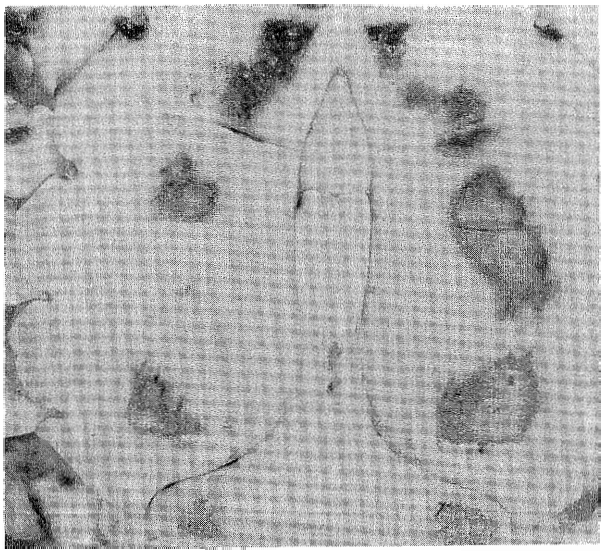
VIBRIO DISEASE OF
BLUE CRABS

NOTE: V. parahemolyticus is known as the causativee agent of a type of food poisoning that is especially common in the Orient, where raw seafood is an important dietary element. Itse occurrence as a pathogen of humans in thee United States as related to blue crabs ise limited thus far to an outbreak caused bye contamination of cooked crab meat by drippingse from live crabs.e

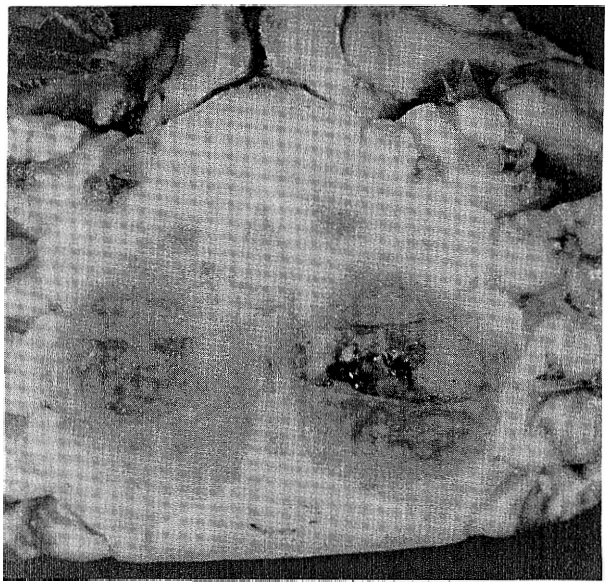
KEY REFERENCES:

Krantz, G. E., R. R. Colwell and E. Lovelace. 1969. Vibrio parahemolyticus from the blue crab Callinectes sapidus in Chesapeake Bay. Science 164: 1286-1287.

(2) SHELL DISEASE OF BLUE CRABS



Shell disease of blue crab (early). Photograph supplied by Robin M. Overstreet, Gulf Coast Research Laboratory.



Shell disease of blue crab (advanced). Photograph supplied by Robin M. Overstreet, Gulf Coast Research Laboratory.

SHELL DISEASE OF BLUE CRABS

COMMON NAME: Shell disease

SPECIES AFFECTED: Blue crab, Callinectes sapidus

GROSS SIGNS: Necrotic lesions of the exoskeleton. In early stages as numerous punctiform brown marks with reddish-brown depressed centers. At later stages, marks join to form irregular areas with a deep necrotic center. Darkly colored lines appear surrounding the necrotic areas. Once the epicuticle is breached, the calcified chitin is susceptible to attacks by chitinoclastic bacteria, also fungi.

CAUSE: Chitin destroying bacteria, probably of several genera.

METHOD OF DIAGNOSIS: Isolates from lesions capable of digesting chitin in culture.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Organisms are ubiquitous in the marine environment, using the chitin matrix as a source of energy, carbon, and nitrogen. Not immediately fatal. Disease does not appear to invade the soft tissue underlying the shell. Definitely contagious; increase with temperature. Up to 50% of blue crabs sampled from natural waters may be infected.

EFFECT ON HOST: Extensive erosion of exoskeleton may provide route of entry for secondary invaders.

TREATMENT: By surviving and achieving ecdysis, the crab can rid itself of the disease, as the new shell is not infected.

PREVENTIVE MEASURES: Infected crabs should be removed from holding facilities as soon as possible.

SHELL DISEASE OF
BLUE CRABS

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

Ubiquitous in marine environment. Causes similar shell diseases in lobsters, king crabs, shrimps, other crustacea.

NOTE:

Shell disease has recently been recognized in Dungeness crabs, Cancer magister, from Washington eby J. G. Chan (personal communication).e

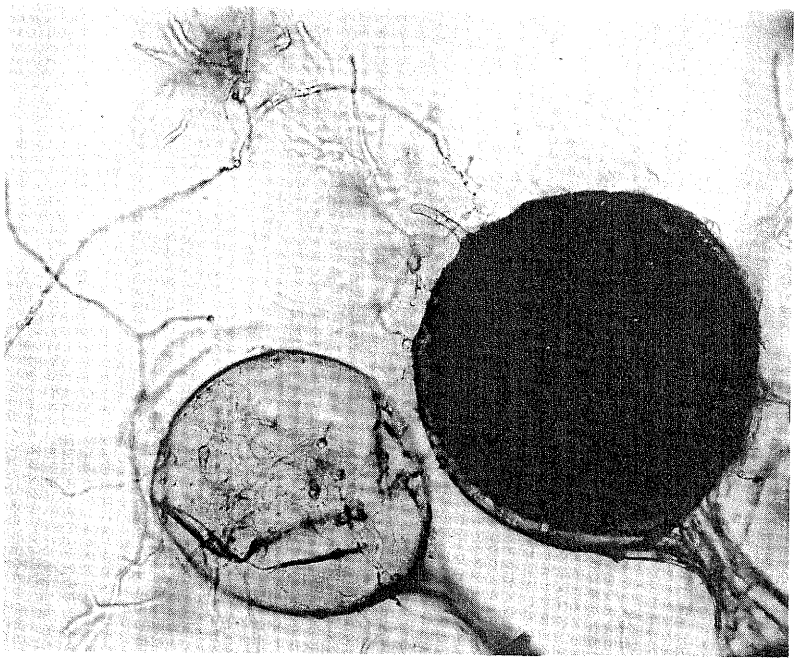
KEY REFERENCES:

Cook, D. W. and S. R. Lofton. 1973. Chitinoclastic bacteria associated with shell disease in Penaeus shrimp and the blue crab Callinectes sapidus. J. Wildl. Dis. 9: 154-159.

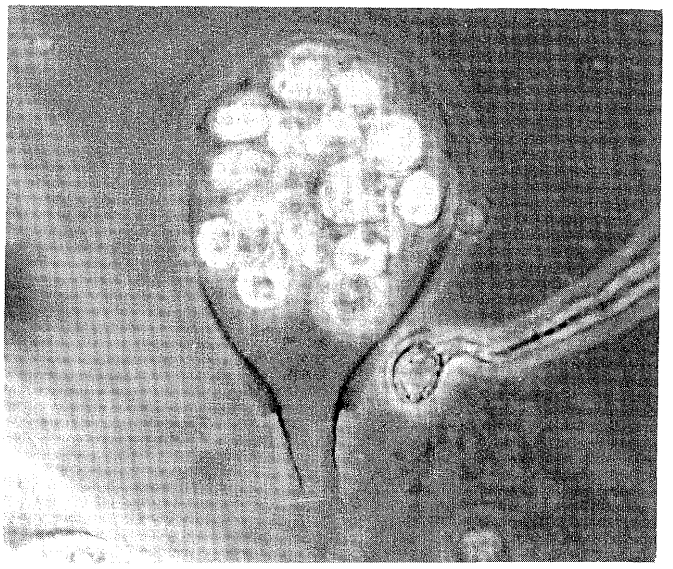
Rosen, B. 1967. Shell disease of the blue crab, Callinectes sapidus. J. Invert. Pathol. 9: 348-353.

Rosen, B. 1970. Shell disease of aquatic crustaceans. In. Snieszko, S. F. (ed.). A symposium on diseases of fishes and shellfishes. pp. 409-415, Spec. Pub. No. 5, Amer. Fish. Soc., Wash., D. C.

(3) FUNGUS DISEASE OF BLUE CRAB EGGS AND LARVAE



Lagenidium growing from blue crab eggs. Photograph supplied by Charles E. Bland, East Carolina State University.



Lagenidium sporulation. Photograph supplied by Charles E. Bland, East Carolina State University.

FUNGUS DISEASE OF BLUE CRAB EGGS AND LARVAE

COMMON NAME: Fungus disease of blue crab eggs and larvae

SPECIES AFFECTED: Blue crab, Callinectes sapidus

GROSS SIGNS: Invade ova and immature embryonic stages only. Heavily infected eggs can be recognized by their smaller size and greater opacity. Diseased portion of sponge assumes a brown color on yellow-orange sponges and a grayish color on brown and black sponges. Sponge usually smaller than in uninfected crabs. Invasion by fungus restricted to periphery of sponge.

CAUSE: Fungus Lagenidium callinectes.

METHOD OF DIAGNOSIS: Low power microscopic examination with brilliant illumination. Eggs filled with and surrounded by hyphae of the fungus. Fungus readily isolated on agar medium.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

Zoospores of fungus settle on eggs, germinate on surface sending in a germ tube which develops into a branched, sparingly septate mycelium. Each segment may become a zoosporangium. Develops in salinities of 5 to 30 ppt. Contagious and spreads rapidly in the peripheral eggs but does not penetrate into the interior of the egg mass.

EFFECT ON HOST: Infected eggs do not hatch. Very heavy infections involve up to 25% of eggs in the egg mass. In hatchery tanks, zoea larvae which have hatched from uninfected eggs, may become infected if fungus spores are present. Infected larvae weaken and become unable to swim.

FUNGUS DISEASE OF BLUE CRAB EGGS AND LARVAE

TREATMENT: Commercially available fungicides (Tribasic copper sulfate, Benlate, Dyrene, Captan, Manzate 200, Difolatan, and Dithane M45) have been tested. Minimum lethal doses have been determined for some: Tribasic copper sulfate, 159 ppm active component; Benlate, 28 ppm active component; Dyrene, 13 ppm active component; and Captan, 3.2 ppm active component.

PREVENTIVE MEASURES: Remove from culture system and destroy female crabs with infected sponges, since under crowded conditions Lagenidium may spread rapidly to uninfected sponges.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: East and Gulf coasts of United States. Recently reported prevalence of over 95% in blue crabs from North Carolina.

NOTE: Experimental infections obtained in oyster crabs (Pinnotheres ostreum) and mud crabs (Neopanope texiana). The fungus has been found in Chelonibia patula (a barnacle occurring on the carapace of blue crabs, oyster crabs and mud crabs). A Lagenidium sp. has also recently been reported from Dungeness crab larvae on the United States west coast.

KEY REFERENCES:

Bland, C. E. and H. V. Amerson. 1973. Observations on Lagenidium callinectes: isolation and sporangial development. Mycologia 65: 310-320.s

Couch, J. N. 1942. A new fungus on crab eggs. J. Elisha Mitchell Scientific Society 58: 158-163.

Johnson, T. W. Jr. 1970. Fungi in marine crustaceans. In. S. F. Snieszko, (ed.). A symposium on diseases of fishes and shellfishes. Amer. Fish. Soc. Spec. Publ. No. 5, 405-408, Wash., D. C.

Johnson, T. W. Jr. and R.eR. Bonner. 1960. Lagenidium callinectes Couch in barnacle ova. J.e Elisha Mitchell Sci.e Soc. 76: 147-149.

Newcombe, C. L. and M. R. Rogers. 1947. Studies of a fungus parasite that infects blue crab eggs. Turtox News 25(9).

Rogers-Talbert, R. 1948. The fungus Lagenidium callinectes Couch (1942) on eggs of the blue crab in Chesapeake Bay. Biol. Bull. 95: 214-228.

Ruch, D. G. and C. E. Bland. 1973. Preliminary results concerning the efficacy of selected fungicides in controlling Lagenidium callinectes, parasitic on blue crab ova. Sea Grant Reprint No. 37, North Carolina State Univ., Raleigh, N. C.

(4) NOSEMA DISEASE OF BLUE CRABS

NOSEMA DISEASE OF BLUE CRABS

COMMON NAME: Nosema disease

SPECIES AFFECTED: Blue crab, Callinectes sapidus

GROSS SIGNS: In terminal stages, sick crabs occur in shallow water; movements are sluggish, carapace appears dirty, often with rusty spots, and sometimes overgrown with algae. Muscles have opaque white appearance, and have coarse fibrous texture.

CAUSE: Microsporidan protozoan, Nosema michaelis.

METHOD OF DIAGNOSIS: Characteristic oval spores, 2.2x1.7 μ in necrotic muscle tissue.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Not described.

EFFECT ON HOST: Causes lysis of muscles and may kill infected crabs, especially if they are under stress.

TREATMENT: None reported.

PREVENTIVE MEASURES: None reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: East coast of United States.

NOTE: Another microsporidan, Nosema sapidi, also occurs in blue crabs, but is apparently less pathogenic than N. michaelis. It can cause muscle degeneration, but host activity is not noticeably affected. Spores of N. sapidi are larger than those of N. michaelis (3.5x2.1 μ as compared with 2.2 μ x 1.7 μ for N. michaelis). A third microsporidan Pleistophora cargoi has also been reported from the muscles of blue crabs in Maryland.

Fishermen refer to the disease caused by Nosema as "sick crab" disease.

KEY REFERENCES:

DeTurk, W. E. 1940. The parasites and commensals of some crabs of Beaufort, North Carolina. Thesis, Duke University (unpublished).

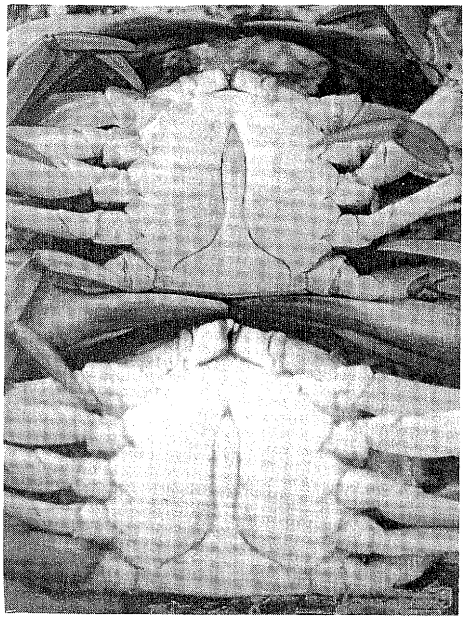
Sprague, V. 1965. Nosema sp. (Microsporida, Nosematidae) in the musculature of the crab Callinectes sapidus. J. Protozool. 12: 66-70.

Sprague, V. 1966. Two new species of Plistophora (Microsporida Nosematidae) in decapods, with particular reference to one in the blue crab. J. Protozool. 13: 196-199.

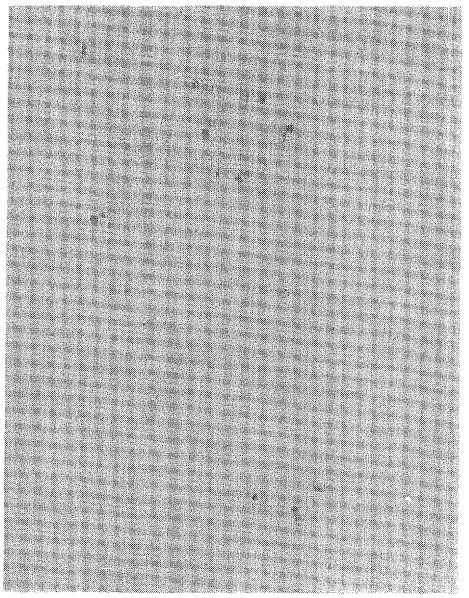
Sprague, V. 1970 Some protozoan parasites and hyperparasites in marine decapod Crustacea. In. Snieszko, S. F. (ed.). A symposium on diseases of fishes and shellfishes. pp. 416-430, Spec. Pub. No. 5, Amer. Fish. Soc., Wash., D. C.

Weidner, E. H. 1970. Ultrastructural studies of microsporidan development. 1. Nosema sp. Sprague, 1965, in Callinectes sapidus Rathbun. Z. Zellforsch. 105: 33-54.

(5) GRAY CRAB DISEASE OF BLUE CRABS



Gross signs of gray crab disease. Infected (above) and normal (below).



Characteristic appearance of Paramoeba in stained hemolymph smears from infected blue crab.

GRAY CRAB DISEASE OF BLUE CRABS

COMMON NAME: Gray crab disease (Paramoeba disease)

SPECIES AFFECTED: Blue crab, Callinectes sapidus

GROSS SIGNS: Lethargy; grayish discoloration of the ventral side of the crab in heavy infections. Hemolymph may have milky appearance.

CAUSE: An amoeba, Paramoeba perniciosa, which destroys the blood cells of the crab and lyses skeleton muscles in advanced infections.

METHOD OF DIAGNOSIS: Amoebae from blood of infected crabs observed with phase contrast, either alive or fixed in 5% formalin sea-water and stained with dilute methylene blue. Permanent smears made, fixed in Bouin's, Davidson's or Hollands solutions and stained with iron hematoxylin or Giemsa. Paramoeba characteristically has two nucleus-like bodies readily visible in stained material.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Can occur in epizootic proportions. One occurred in Chincoteague Bay, Virginia (Newman and Ward, 1973) which affected 17% of the crab population. The original description of the disease (Sprague and Beckett, 1966) was developed from an outbreak in crabs held in shedding tanks, where mortalities were estimated at 20-30%. Implicated in extensive crab mortalities in 1968 on the Georgia and South Carolina coasts.

EFFECT ON HOST: Infections usually fatal, within a very short period, especially for "peeler" crabs in holding tanks, but also for "hard" crabs.

GRAY CRAB DISEASE OF
BLUE CRABS

TREATMENT: None reported.

PREVENTIVE MEASURES: None reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Middle and South Atlantic coast of United States, limited to higher salinities.

KEY REFERENCES:

Newman, M. W. and G. E. Ward. 1973. An epizootic of blue crabs, Callinectes sapidus, caused by Paramoeba perniciosa. J.eInvert. Pathol. 22: 329-334.e

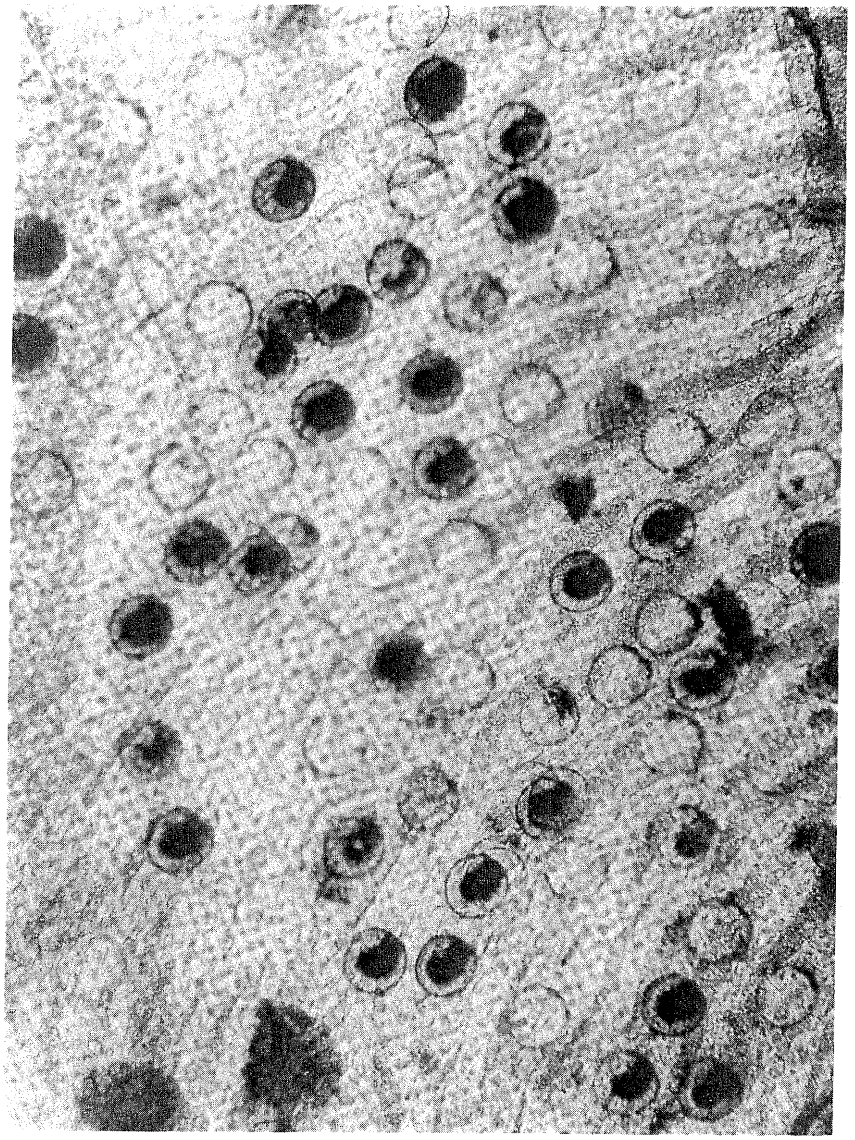
Sawyer, T. K. 1969. Preliminary study on the epizootiology ande host-parasite relationship of Paramoeba sp. in the blue crab, Callinectes sapidus. Proc. Nat. Shellfish. Assoc. 59: 60-64.

Sprague, V. and R. L. Beckett. 1966. A disease of blue crabse (Callinectes sapidus) in Maryland and Virginia. J. Invert. Pathol. 8: 287-289.

Sprague, V. and R. L. Beckett. 1968. The nature of the etiological agent of "gray crab" disease. J. Invert. Pathol. 11: 503.

Sprague, V., R. L. Beckett and T. K. Sawyer. 1969. A newe species of Paramoeba (Amoebida, Paramoebidae) parasitice in the crab Callinectes sapidus. J. Invert. Pathol. 14: 167-174.

(6) CILIATE DISEASE OF BLUE CRABS



Lagenophrys infestation of blue crab gills. Photograph supplied by John A. Couch, Gulf Breeze Environmental Research Laboratory.

CILIATE DISEASE OF BLUE CRABS

COMMON NAME: Ciliate disease

SPECIES AFFECTED: Blue crab, Callinectes sapidus (also found on grass shrimps, Palaemonetes sp.).

GROSS SIGNS: Heavily parasitized crabs have less vitality, movements are sluggish, and they are the first to die in holding tanks. Gills often heavily surfaced by lorica (tests) containing the ciliates, many of which can be seen to be undergoing binary fission.

CAUSE: Peritrichous ciliate protozoan Lagenophrys sp.

METHOD OF DIAGNOSIS: Branchial chambers of fresh crabs placed in petri plates filled with sea-water, lamellae teased from branchiae. Unstained specimens of ciliates collected from lamellae surfaces.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
No evidence that parasite gains nourishment from host, but presence could interfere with normal diffusion of gases across the gill membrane, if present in sufficient numbers.

EFFECT ON HOST: May contribute to mortalities in shedding tanks, because of reduction in gass exchange across gill surfaces.

TREATMENT: Weak formalin dips have been tried with some success.

PREVENTIVE MEASURES: Avoid crowding, low oxygen, and high temperatures in holding tanks.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Middle Atlantic and Gulf of Mexico coast of United States (and probably elsewhere).

CILIATE DISEASE OF
BLUE CRABS

NOTE: The stalked peritrichous ciliate, Epistylus has also been reported to occur on gill lamellae of blue crab.

KEY REFERENCES:

Couch, J. A. 1966. Two peritrichous ciliates from the gills of the blue crab. Chesapeake Sci. 7: 171-172.

Couch, J. A. 1967. A new species of Lagenophrys (Ciliatea: Peritrichida: Lagenophrydiae) from a marine crab, Callinectes sapidus. Trans. Am. Microsc. Soc. 86: 204-211.

(7) *HEMATODINIUM* DISEASE OF BLUE CRABS

HEMATODINIUM DISEASE OF BLUE CRABS

COMMON NAME: Hematodinium disease

SPECIES AFFECTED: Blue crab, Callinectes sapidus

GROSS SIGNS: Crabs moribund; die quickly, rarely surviving transport following capture. Hemolymph is milky appearing and contains few if any hemocytes.

CAUSE: Parasitic dinoflagellate, Hemotodinium sp.

METHOD OF DIAGNOSIS: Moribund crabs have milky-appearing hemolymph with few if any hemocytes but with massive populations of non-motile uninucleate dinoflagellate cells. Occasional multinucleate motile forms seen in hemolymph. Parasites found in blood vessels and sinuses, which may be occluded; also found in connective tissue, antennal gland, hepatopancreas, Y organ, and hematopoetic tissue.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Modes of infection or transmission unknown.
Flagellated stages not reported.
Peak prevalence of 30% found in autumn.

EFFECT ON HOST: Can cause fatal infections.

TREATMENT: None reported.

PREVENTIVE MEASURES: None reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
South Atlantic and Gulf of Mexico coast of United States from North Carolina southward.

KEY REFERENCES:

Couch, J. A. (Personal communicatior 74).

Newman, M. W. and C. A. Johnson III ı press). A fatal disease of blue crabs (Callinectes ius) caused by ae parasitic dinoflagellate, Hematodi: sp. J. Parasit.e

Newman, M. W. and C. A. Johnson III. 1973. A fatal disease of blue crabs caused by parasitic dinoflagellates. 1973 Wildlife Disease Conference, Univ. Conn., p. 19 (Abstract).

Lobster Diseases

Market demand for lobsters, Homarus americanus, is high, and natural populations on the United States east coast seem to be exploited already at maximum sustainable yields. For these reasons, as well as the high unit value of lobsters, research and development projects are underway in several locations. The work is a logical extension of lobster hatching, carried out in the early decades of this century to produce larvae for release in the natural environment -- but it goes far beyond this, to control of the entire life cycle of the animal under culture conditions.

Thus far, six diseases have been reported to be of significance in holding or rearing lobsters:

(1) Gaffkaemia;

(2) Shell disease;

(3) Filamentous bacterial disease of larvae;

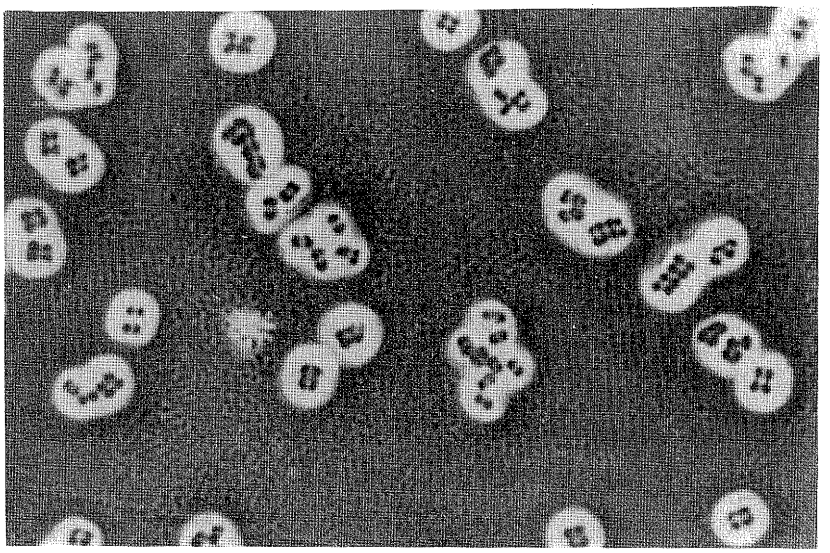
(4) Fungus (Haliphthoros) disease of larvae;

(5) Fungus (Fusarium) disease of juveniles; and

(6) Ciliate disease.

This list is too short, and undoubtedly other organisms -- fungi, viruses, and protozoa -- will appear. Early hatchery efforts (in the first few decades of this century) were impeded by a suctorian protozoan, Ephelota gemmipara, which destroyed lobster eggs in Norwegian hatcheries, and histriobdellid annelid worms, which reduced success of hatching. The same annelid was recently reported from New England lobsters. Still other parasites are known from lobsters, but their potential role in lobster culture is less obvious.

(1) GAFFKAEMIA OF LOBSTERS



Stained hemolymph smear from infected lobster, showing typical Gaffkya tetrads (modified from Stewart and Rabin, 1970).

GAFFKAEMIA OF
LOBSTERS

COMMON NAME: Gaffkaemia (Red Tail)

SPECIES AFFECTED: American lobster, Homarus americanus

GROSS SIGNS: "Sometimes a pink discoloration of the ventral side of the abdomen" (Snieszko & Taylor, 1947). "...No obvious external signs of disease even in heavily septicemic lobsters until shortly before death when they lie quietly with their chelae extended" (Rabin, 1965). Blood clotting eliminated in advanced infections.

CAUSE: Systemic bacterial disease -- caused by the micrococcus Gaffkya homari (recently renamed Pediococcus homari, and still more recently called Aerococcus viridans (var.) homari.

METHOD OF DIAGNOSIS: Blood smears and blood cultures examined for typical tetrads of the pathogen. Blood pink, less viscid, and usually with much-prolonged clotting time. In advanced cases, number of blood cells sharply reduced.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Bacterial disease evidently transmitted from lobster to lobster; heaviest mortalities in holding tanks and pounds. Organisms can be found in slime on lobster cars, in mud of pounds and in sea-water. A portal of entry -- usually an injury of some kind -- is considered necessary for infection.

EFFECT ON HOST: Mortalities within a few days following first appearance of disease signs -- often extensive in captive populations in lobster pounds or live cars.

TREATMENT: Sulfonamides have been found effective in natural infections. Penicillin and Streptomycin treatment has been reported successful. A.e viridans was found to be resistant to Furanacee in in vitro tests. The antibiotic Vancomycin wase found to be effective in early stages of infectione if given at high levels (25 mg/kg lobster), withe slow clearance rate.e

PREVENTIVE MEASURES: Treatment of tidal pounds with calcium hypochlorite reduces populations of the pathogen in bottom mud, and subsequent losses of lobsters. Chlorine can be used in tanks, but they must then be flushed thoroughly. Recently a degree of resistance was induced experimentally in lobsters by using avirulent strains of the pathogen, by using formalin-killed pathogens or by a vaccination procedure using a low dose of the antibiotic Vancomycin followed in 24 hours by injection of live pathogens. Prophylactic immunization by these techniques may become methods of choice in lobster culture.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: United States east and west coast, Canadian east coast, Europe (in Homarus gammarus).

NOTE: Infections in lobsters are almost invariably fatal; there is little indication of host immune response to infection. The crab, Cancer irroratus, can be infected experimentally, but disease is milder, and mean time to death is longer (42 days vs 18 days in lobsters). Crabs may be reservoirs of infection for lobsters. Experimental infections have also been obtained in blue crab, Callinectes sapidus. Chronic disease resulted, with mortalities occurring from 2 to 6 weeks after inoculation. Experimental infections have also been obtained (by injection) in California spiny lobsters, Panulirus interruptus.

KEY REFERENCES:

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Rabin, H. and J. T. Hughes. 1968. Studies on host-parasite relationships in gaffkemia. J. Invert. Pathol. 10: 335-344.

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Stewart, J. E. and B. M. Zwicker. (in press). A comparison of various vaccines for inducing resistance in the lobster (Homarus americanus) to the bacterial infection, gaffkemia. J. Fish. Res. Bd. Canada.

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Tubiash, H. S. and G. E. Krantz. Experimental bacterial infection of the blue crab, Callinectes sapidus. Bacteriological Proc. 1970, G80.

(2) SHELL DISEASE OF LOBSTERS

SHELL DISEASE OF
LOBSTERS

Pitting and sculpturing of carapace of lobster caused by chitin-destroying bacteria.

SHELL DISEASE OF LOBSTERS

COMMON NAME:	Shell disease
SPECIES AFFECTED:	American lobster, Homarus americanus
GROSS SIGNS:	Pitting and sculpturing of the exoskeleton. Initial lesions occur on walking legs. Large areas of shell of tail and carapace may be eroded, exposing soft inner layer, in advanced stages.
CAUSE:	Chitin-destroying, gram-negative bacteria, probably of several genera.
METHOD OF DIAGNOSIS:	Bacteria isolated and cultured, tested against chitin strips.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

Method of infection unknown, but lodging of bacteria in pores and ducts of the shell has been proposed as a route of invasion. Disease develops rather slowly, at least 3 months to advanced stages, dormant below 35°F but active at 40°F or over. Spread through physical contact, possibly sea-water and mud.

EFFECT ON HOST:	Erosion of gill chitin can lead to mortalities when lobsters are exposed to other stresses.
TREATMENT:	Lobsters that have been infected and recover after molting do not get reinfected if kept under strictly sanitary conditions.
PREVENTIVE MEASURES:	Sanitation of all lobster storage areas to eliminate breeding places of bacteria. Stock pounds when temperature is under 45°F. Remove dead lobsters and destroy by burying or burning. Careful chlorination of tanks.

SHELL DISEASE OF
LOBSTERS

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

Ubiquitous distribution in marine waters.

KEY REFERENCES:

Hess. 1937. A shell disease in lobsters (Homarus americanus) caused by chitinovorous bacteria. J. Biol. Board Can. 3: 358-362.e

Sawyer, W. H. Jr. and C. C. Taylor. 1949. The effect of shell disease on the gills and chitin of the lobster (Homarus americanus). Maine Dept. Sea Shore Fish. Res. Bull. 1, 10 pp.

Taylor, C. C. 1948. A study of lobster shell disease with observations and recommendations. Maine Dept. Sea Shore Fish. Bull. (no date or number).

(3) FILAMENTOUS BACTERIAL DISEASE OF LOBSTER LARVAE

FILAMENTOUS BACTERIAL DISEASE OF LOBSTER LARVAE

COMMON NAME: Bacterial (Leucothrix) disease of lobster larvae

SPECIES AFFECTED: American lobster, Homarus americanus

GROSS SIGNS: Massive filamentous mat often covering entire exoskeleton.

CAUSE: Bacterium Leucothrix mucor.

METHOD OF DIAGNOSIS: Gross examination, bacterial isolation.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Not reported.

EFFECT ON HOST: Infected larvae molt incompletely and die during or immediately after molt; older lobsters not as severely affected. Larval mortalities reported to have reached 90%.

TREATMENT: Streptomycin added to water in rearing tanks every 3 days.

PREVENTIVE MEASURES: Not reported, but may be associated with poor water quality.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Reported from experimental tanks at Univ. of California (Davis) and Bodega Bay Marine Station, also at California State University at San Diego. Normally this bacterial genus is associated with eggs of fish.

NOTE: A species of Leucothrix was also reported by Anderson and Conroy (1968) from the surface of eggs of the English prawn, Palaemon serratus, and apparently interfered with normal hatching. The bacterium also occurred on pleopods of egg-bearing females. Disinfectant baths for eggs were recommended.

Leucothrix also occurs on cultured penaeid shrimp larvae in southern United States. A filamentous bacterium, possibly Leucothrix mucor, was reported (Johnson, 1974) on the

gills and appendages of brown and white shrimp in Mississippi and adjacent areas -- and from P. vannamei, P. aztecus, P. setiferus, P. stylorostris from rearing ponds in Texas. A filamentous bacterium (not further identified, but probably also Leucothrix) was reported by Barkate et al. (in press) from post-larvae of penaeid shrimps held in laboratory tanks. Inoculation of isolates killed shrimps within 48 hours, but exposure of healthy post-larvae with sediment from infected tanks did not produce infections.

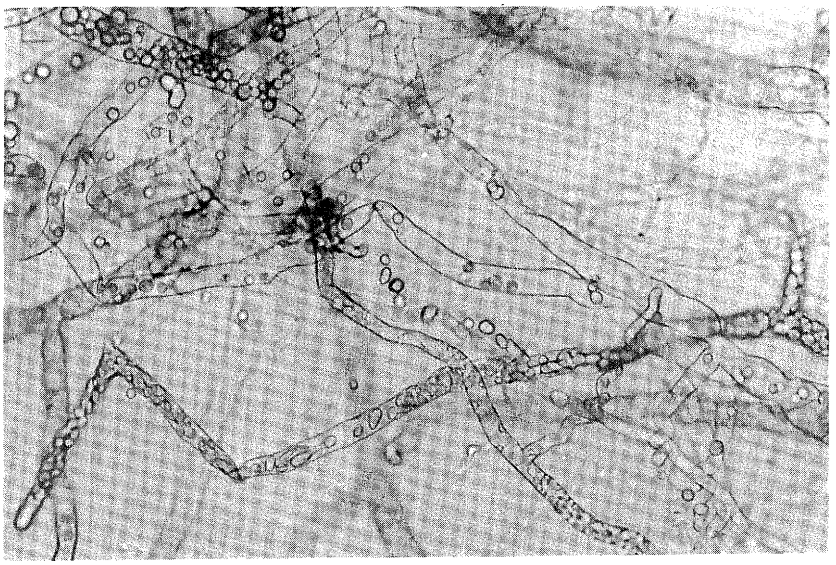
KEY REFERENCES:

Anderson, J. I. W. and D. A. Conroy. 1968. The significance of disease in preliminary attempts to raise Crustacea in sea-water. Proc. 3rd Symp. Mond. Comm. Office Intern. Epizoot. Etude Maladies Poissons, Stockholm (1968) Separate No. 3, 8 pp.

Anonymous, 1972. Rx for lobsters. Sea Grant '70's 3(4): 2.

(4) FUNGUS (HALIPHTHOROS) DISEASE OF LOBSTER LARVAE

FUNGUS (HALIPHTHOROS) DISEASE OF LOBSTER LARVAE



Haliphthoros from lobster larvae. Hyphae, spores and sporangia. Photograph supplied by Charles E. Bland, East Carolina State University.

FUNGUS (HALIPHTHOROS) DISEASE OF LOBSTER LARVAE

NOTE: This entire section is drawn from an as yet unpublished manuscript graciously supplied by Dr. Robert Shleser, Bodega Marine Laboratory, University of California, so that material could be incorporated into this handbook.

COMMON NAME: Fungus disease of lobster larvae and early juveniles.

SPECIES AFFECTED: American lobster, Homarus americanus, and European lobster, Homarus gammarus.

GROSS SIGNS: Dark red-brown "scabs" of host response to mycelial invasion of muscle tissue beneath the carapace and extending down to basal segments of appendages.

CAUSE: A fungus tentatively identified as Haliphthoros sp., with highly branched non-septate mycelium.

METHOD OF DIAGNOSIS: Reported to be easily isolated on corn meal agar from "scabs" or surrounding tissues. Growth occurs on surface and into agar.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Fungus may be chitinoclastic or may enter as secondary invader through breaks in exoskeleton. Most common point of entry seems to be the inner wall of the gill chamber, andain some instances the infection seems limited to this area. Infection thought to be spread from one animal to another by spores, which were observed in fecal material.

EFFECT ON HOST: Produces mortality in larvae, and in juveniles under 27 mm. Most infected animals die during molt, apparently because of adhesions produced by the "scabs" between old and new exoskeletons, mechanically preventing successful ecdysis. Experimental exposures of larvae resulted in mortalities of up to 46% within 3 weeks, with initial mortalities in less than one week.

FUNGUS (HALIPHTHOROS) DISEASE OF LOBSTER LARVAE

TREATMENT: Furanace tried, but found ineffective.

PREVENTIVE MEASURES: Strict cleansing of system (and probably discard of entire batch of larvae).

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE CRGANISM:

Reported thus far only from experimental tanks in Bodega Bay Laboratory (California). Haliphthoros has been reported from other crustaceans from Martha's Vineyard (Massachusetts) which has been a source of adult lobsters for west coast studies. (There is, however, no direct evidence that imported lobsters are the source of infection).

NOTE: Several other fungi have been reported from lobsters. Sindermann (1970) briefly discusseda an unidentified fungus causing "mottling disease"a in lobsters (H. americanus) from Maine.a Sordi (1959) described a fungus disease ofa H. gammarus held in sea-water tanks in Italy,a and identified the fungus Ramularia branchialisa from extensive gill lesions. Recently,a mortalities of larval lobsters have also beena attributed to Lagenidium infections.

KEY REFERENCES:

Lightner, D. V. and C. T. Fontaine. (in press). A mycosis of the American lobster (Homarus americanus) caused by a Fusarium sp. J. Invert. Pathol.

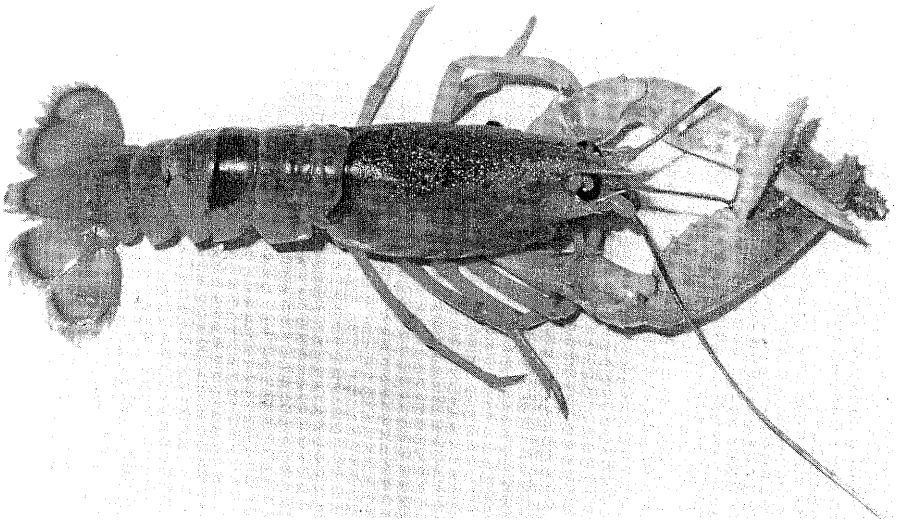
Nilson, E. H. and W. S. Fisher. (Personal communications).

Shleser, R. (unpublished manuscript "A fungal disease of lobsters" made available to the author on April 11, 1974, for inclusion of material in this handbook).

Sindermann, C. J. 1970. Principal diseases of marine fish and shellfish. Acad. Press, N. Y., 369 pp.

Sordi, M. 1959. Micosi dei Crostacei decapodi marini. Riv. Parassitol. 19: 131-137.

(5) FUNGUS (FUSARIUM) DISEASE OF JUVENILE LOBSTERS



External signs of Fusarium infection of juvenile lobster. Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.

Fusarium macroconidia in lobster gills. Photograph supplied by Donald V. Lightner, Gulf Coastal Fisheries Center.

FUNGUS (FUSARIUM) DISEASE OF JUVENILE LOBSTERS

NOTE: This entire section is drawn from a paper by Lightner and Fontaine (J. Invert. Pathol., in press) made available in advance of publication by the authors so that material could be incorporated into this handbook.

COMMON NAME: Fungus (Fusarium) disease of juvenile lobsters

SPECIES AFFECTED: American lobster, Homarus americanus

GROSS SIGNS: Affected individuals have "black spots" of various dimensions on exoskeleton and brownish discoloration of gills.

CAUSE: Imperfect fungus Fusarium sp.

METHOD OF DIAGNOSIS: Isolation of fungus from infected gills on sabouraud dextrose agar or in fluid thioglycollate medium. Mycelium produces purplish-brown diffusible pigment. Macroconidia canoe-shaped, with 3-5 cells (usually 4).

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Hyphae and conidia develop on and in gill lamellae and in cuticle of the exoskeleton. Hyphae frequently protrude through tips of gill lamellae. Host hemocytic response results in encapsulation of hyphae in subcuticular tissues, often with melanin deposition.

EFFECT ON HOST: According to A. Gmeiner (Woodside, N.Y.), who originally observed the disease, the earliest sign of infection is the appearance of white spots on the exoskeleton 6-10 days after molt. Spots turn orange, then black. Infected lobsters do not survive the next molt; death occurs just before or during molting. Mortalities attributable to the disease in one experimental rearing operation reached 35%. Destruction of gills by the fungus is considered to be a principal cause of death.

FUNGUS (FUSARIUM) DISEASE OF JUVENILE LOBSTERS

TREATMENT: None reported.

PREVENTIVE MEASURES: None reported, but strict attention to water quality and filtration, combined with isolation of infected individuals, may reduce effects of the disease.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: Reported thus far in lobsters only from one experimental rearing operation at Woodside, N. Y. Lobsters had been obtained as fourth-stage larvae from the Massachusetts Lobster Hatchery on Martha's Vineyard.

NOTE: Fusarium has also been reported from Kuruma prawn, Penaeus japonicus, in Japan, and from the pink shrimp, Penaeus duorarum, in Texas. The isolate from lobsters is similar to that from kuruma prawn, in which it causes a disease labelled "black gill disease."

KEY REFERENCES:

Lightner, D. V. and C. T. Fontaine . (in press). A mycosis of the American lobster (Homarus americanus) caused by a Fusarium sp. J. Invert. Pathol.

(6) CILIATE (ANOPHRYS) DISEASE OF LOBSTERS

CILIATE DISEASE OF LOBSTERS

COMMON NAME: Ciliate (Anophrys) disease of lobsters

SPECIES AFFECTED: American lobster, Homarus americanus

GROSS SIGNS: Unusual mortalities in lobster holding tanks and pounds; no gross signs reported.

CAUSE: Holotrich ciliate protozoan, Anophrys sp.

METHOD OF DIAGNOSIS: Examination of fresh hemolymph or stained hemolymph smears for presence of ciliates. Hemolymph may be cloudy or milky in heavy infections, due to density of ciliates (reported at up to 160,000 per cubic millimeter of hemolymph).

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

Route of invasion unknown, but may be through breaks in exoskeleton. Ciliates multiply in hemolymph, devouring amebocytes of the lobster, and eventually becoming the only formed element in the blood.

Anophrys may be normal inhabitant of seawater, and may be facultatively parasitic.

The biology of the same or a closely related species from shore crabs (Carcinus maenas) in France includes conjugation and encystment on death of the host. Some indications of individual variations in resistance were found, and some crabs recovered.

EFFECT ON HOST: Destroys blood cells and produces mortalities within 6 weeks, possibly because of anemia and asphyxiation, followed by severe bacteremias. Clotting ability of hemolymph and wound repair are inhibited. Dead lobster larvae were also reported to be infected with large numbers of Anophrys, devouring flesh as well as blood cells, but it was not determined whether the ciliates caused death or invaded after death.

TREATMENT: None reported.

PREVENTIVE MEASURES: None reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Lobster-holding tanks at St. Andrews, New Brunswick, Canada. The same or similar organisms have been described from Europe, both as free-living forms and as parasitic forms from shore crabs. Two species, Anophrys magii and A. sarcophaga have been described; they may or may not be the same species.

NOTE: Experimental infections were obtained in normal lobsters and in several species of crabs by injecting infected lobster hemolymph.

KEY REFERENCES:

Aiken, D. E., J. B. Sochasky and P. G. Wells. 1973. Ciliate infestation of the blood of the lobster Homarus americanus. Int. Council Expl. Sea, Shellfish and Benthos Com., Rept. No. CM1973/K:46, 2 pp.

Bang, F. B. 1970. Disease mechanisms in crustacean and marine arthropods. In. Snieszko, S. F. (ed.). A symposium on diseases of fishes and shellfishes. pp. 383-404. Amer. Fish. Soc. Spec. Pub. No. 5, 526 pp.

Bang, F. B., J. Audouin and M. Leglise. 1972. Ciliate infection of the blood of the edible crab, Cancer pagurus, in holding tanks in Brittany, France. J. Invert. Pathol. 20: 226-227.

Poisson, R. 1930. Observations sur Anophrys sarcophaga Cohn (=A. maggii Cattaneo) infusoire holotriche marin et sur son parasitisme possible chez certains Crustaces.

Sprague, V. 1970. Some protozoan parasites and hyperparasites in marine decapod Crustacea. In. "A Symposium on Diseases of Fishes and Shellfishes" (S. F. Snieszko, Ed.) p. 416-430. Amer. Fish. Soc. Spec. Pub. No. 5, 526 pp.

MOLLUSCAN DISEASES

Microbial diseases dominate the pathology scene with mollusks. Hatchery problems involving vibrios and fungi have been identified in the published literature on oyster culture, while a protozoan and a fungus -- Minchinia nelsoni and Labyrinthomyxa (=Dermocystidium) marina -- have long dominated certain oyster grow-out areas in the Middle Atlantic states and the Gulf of Mexico respectively. Both these organisms still cause substantial losses of oysters, although the epizootic of Minchinia has abated since the mid-1960's in the Middle Atlantic states.

On the United States west coast, where seed oysters have been imported from Canada and Japan for many decades, sporadic mortalities occur in grow-out areas, but the possible disease entities that might be involved are ill-defined. A bacterium causing a focal necrosis has been described from Japanese seed and from the parent stock in Japan; and a disease of possible protozoan etiology is enzootic in stocks in British Columbia and Washington. The real impact of these organisms on oyster populations is unknown.

The diseases reported for clams are remarkably similar to those described for oysters. Bacterial and fungal diseases of larvae are similar, and the fungus Labyrinthomyxa parasitizes many bivalve molluscs, including clams and oysters.

REFERENCES:

Sprague, V. 1970. Some protozoan parasites and hyperparasites in marine bivalve molluscs. In. S. F. Snieszko (ed.). A symposium on diseases of fishes and shellfishes, pp. 511-526. Amer. Fish. Soc. Spec. Pub. No. 5, 526 pp.

Oyster Diseases

Oysters are probably the only marine animals being produced on a large scale at a profit in United States mariculture today. Although much of this country's production still comes from natural populations, there are substantial ongoing mariculture efforts, at various levels of sophistication, ranging from transfer of naturally caught seed to growing areas, to the oyster hatcheries of Long Island. Disease has been a factor in production from natural and cultured populations. Epizootics with accompanying mass mortalities have affected natural and cultivated beds during recent decades. Research by many groups -- state, federal, and university -- has developed much information about a number of serious pathogens of oysters. Included in this summary are the following ten oyster diseases:

(1)e Virus disease;e

(2)e Bacillary necrosis of larvae;e

(3)e Focal necrosis;e

(4)e Larval mycosis;e

(5)e Fungus disease;e

(6)e Delaware Bay disease;e

(7)e Seaside disease;e

(8)e Mytilicola (red worm) disease;e

(9)e Malpeque Bay disease;e

(10)e Denman Island disease.e

Other parasites and disease conditions exist, but either their potential role in oyster mariculture is not apparent, or information about them is still too vague. Malpeque Bay disease and Denman Island disease are still of uncertain etiology, but are included because they have been implicated in oyster mortalities.

REFERENCES:

Sprague, V. 1971. Diseases of oysters. Ann. Rev. Microbiol. 25: 211-230.

(1) VIRUS DISEASE OF OYSTERS

COMMON NAME: Virus Disease

SPECIES AFFECTED: American oyster, Crassostrea virginia

GROSS SIGNS: Pale digestive gland in infected individuals; sporadic mortalities.

CAUSE: Herpes type virus -- hexagonal, 70-90 mm in diameter, with single coat, some with dense nucleoid.

METHOD OF DIAGNOSIS: Electron microscopy of suspected tissues, examining for intranuclear inclusion bodies.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Apparently enzootic in the population of oysters studied. Experimental stocks transferred to heated effluent of power generating plant in Marsh River, Maine. The higher ambient temperatures (28-30°C) in the effluent (compared with 12-18°C in natural waters) apparently induced higher mortalities and higher virus prevalences.

TREATMENT: None reported, but return to ambient environmental temperatures could retard infection and mortalities.

PREVENTIVE MEASURES: None reported, but selection of stocks not infected with the virus might be an approach.

KNOWN GEOGRAPHIC RANGE OF ORGANISM:
At present reported only from relic population in Piscataqua River in Maine.

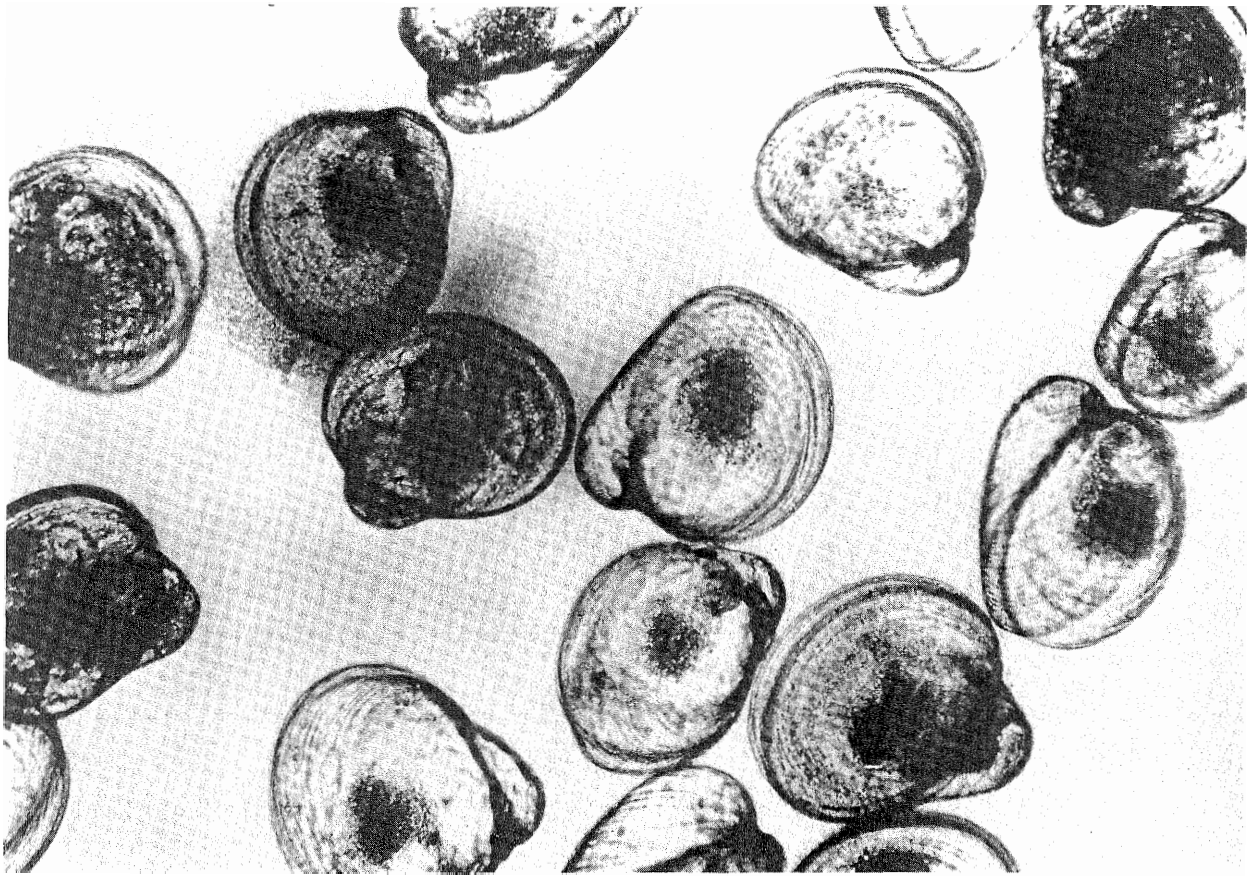
NOTE: A second virus disease of oysters, called "ovacystis disease" has been reported briefly by Farley (1973). A characteristic of the disease is massive hypertrophy of genital epithelial cells containing large feulgen-positive granular masses in the nucleus. Electron microscopy revealed extracellular and nuclear arrays of icosahedral particles, with features resembling those of papovaviruses. The infection seems to be salinity-dependent, with a peak at about 14 ppt. Infection experiments have not reproduced the disease.

KEY REFERENCES:

Farley, C. A. 1973. Virus diseases of oysters. 1973. Wildlife Disease Conf., Meeting Abstract, p. 19.

Farley, C. A., W. G. Banfield, G. Kasnic Jr., and W. S. Foster. 1972. Oyster herpes-type virus. Science 178: 759-760.

(2) BACILLARY NECROSIS OF OYSTER LARVAE



Bacillary necrosis of oyster larvae, showing typical bacterial swarming. Photograph supplied by Haskell S. Tubiash, Middle Atlantic Coastal Fisheries Center.

BACILLARY NECROSIS OF OYSTER LARVAE

COMMON NAME: Bacillary necrosis

SPECIES AFFECTED: American oyster, Crassostrea virginica, larvae

GROSS SIGNS: Settling and decrease in larval motility. High, sudden mortalities.

CAUSE: Vibrio anguillarum, V. alginolyticus and other marine Vibrio spp; possibly aeromonads and pseudomonads also.

METHOD OF DIAGNOSIS: Direct microscopic examination of live affected larvae. Swarming vibrios are diagnostic. Bacteriological culture and experimental challenge with isolates.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Vibrios believed to be enzootic, causing overt infection and mortality when critical infective level is reached because of adverse environmental conditions. Entry via esophagus. Proliferation throughout the tissues with lysis and necrosis of tissues. Course of experimental infections is rapid, with disease signs apparent 4 to 5 hours after exposing larval cultures to pathogens. Deaths begin at 8 hours and complete mortality of culture population by 18 hours.

EFFECT ON HOST: Mortality, usually complete, in larval cultures.

TREATMENT: Combistrep - 50 to 100 ppm.
Other antibiotics suggested: Chloramphenicol (10 ppm), polymyxin B, erythromycin, and neomycin.

PREVENTIVE MEASURES: Improve water quality and general sanitation.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

Reported from United States east coast. Larvae of hard clams, American and European oysters proven susceptible; as are probably most bivalve mollusks.

NOTE:

Mortalities with characteristics similar to those caused by bacillary necrosis occurred in larval cultures of Ostrea edulis in Conway, Wales, in 1968 and 1969. Pathogens isolated were pseudomonads, and were pathogenic for larvae of Ostrea edulis, Mytilus edulis, Venerupis decussata, and Crassostrea gigas, but not for juveniles of O. edulis.

KEY REFERENCES:

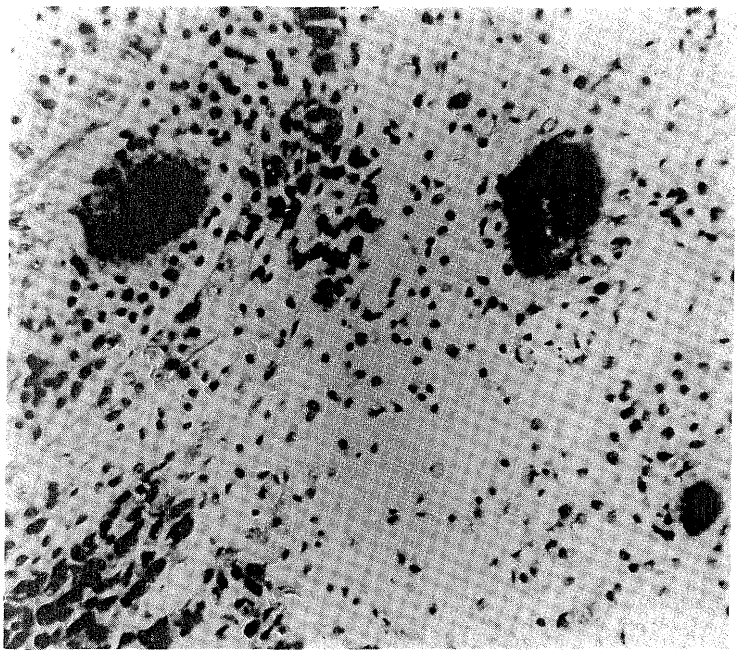
Guillard, R. L. 1959. Further evidence of the destruction of bivalve larvae by bacteria. Biol. Bull. 117: 258-266.

Helm, M. M. and F. M. Smith. 1971. Observations on a bacterial disease in laboratory-cultured larvae of the European flat oyster Ostrea edulis L. Internat. Council Expl. Sea, Shellfish and Benthos Committee, Rept. No. CM1971/K:10, 9 pp.

Tubiash, H. S., P. E. Chanley and E. Leifson. 1965. Bacillary necrosis, a disease of larval and juvenile bivalve mollusks. I. Etiology and epizootiology. J. Bacteriol. 90: 1036-1044.

Tubiash, H. S., R. R. Colwell and R. Sakazaki. 1970. Marine vibrios associated with bacillary necrosis, a disease of larval and juvenile bivalve mollusks. J. Bacteriol. 103: 272-273.

(3) FOCAL NECROSIS OF OYSTERS



Focal necrosis in tissues of Pacific oyster.

FOCAL NECROSIS OF OYSTERS

COMMON NAME: Focal necrosis

SPECIES AFFECTED: Pacific oyster, Crassostrea gigas

GROSS SIGNS: Pale digestive gland, gaping, sporadic mortalities in seed and adult oysters.

CAUSE: Unidentified gram-positive bacterium.

METHOD OF DIAGNOSIS: Histological examination of oyster tissues discloses multiple abscesses with concentrations of gram positive bacteria.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
The disease was reported in up to 20% of cultured oyster stocks in Matsushima Bay, Japan, and was subsequently found in seed and market-sized oysters in the State of Washington. Its association with repeated oyster mortalities in northern Japan and in Washington waters is suspected, but unconfirmed.

TREATMENT: None reported.

PREVENTIVE MEASURES: None reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Matsushima Bay, Japan
Willapa Bay, Washington

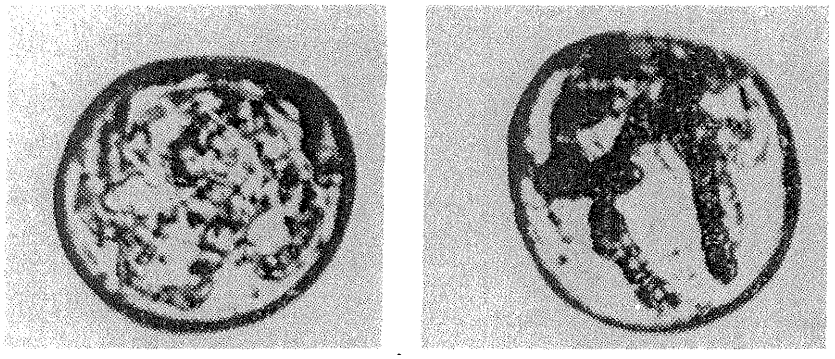
KEY REFERENCES:

Numachi, K., J. Oizumi, S. Sato and T. Imai. 1965. Studies on the mass mortalities of the oyster in Matsushima Bay. III. The pathological changes of the oyster caused by gram positive bacteria and the frequency of their infection. (In Japanese, with English summary). Bull. Tohuku Reg. Fish. Lab. 25: 39-48.

Sindermann, C. J. 1970. Disease and parasite problems in marine aquiculture. In. W. J. McNeil, ed. Marine Aquiculture, pp. 103-134. Oregon State University Press, Corvallis, Oregon.

Sindermann, C. J. and A. Rosenfield. 1967. Principal diseases of commercially important marine bivalve Mollusca and Crustacea. U. S. Fish Wildl. Serv., Fish. Bull. 66: 335-385.

(4) LARVAL MYCOSIS OF OYSTERS



Young oyster larvae infected with Sirolpidium (neutral red stain). (Modified from Davis et al., 1954).

LARVAL MYCOSIS OF OYSTERS

COMMON NAME:	Larval mycosis
SPECIES AFFECTED:	Oyster, Crassostrea virginica, larvae
GROSS SIGNS:	Growth ceased; rapid mortality in larval populations. Larvae may be observed in various stages of disintegration with the fungus quite apparent in their interior. Juveniles also found infected.
CAUSE:	Systemic fungus invasion by Sirolpidium zoophthorum.
METHOD OF DIAGNOSIS:	Microscopic examination of larvae unstained or stained with neutral red or lactophenol cotton blue. The fungus acquires a deeper stain than larval tissues if living larvae are kept in a solution of neutral red in sea water, a characteristic to be used both for detecting the first appearance of the fungus, also as a tag to study the progress of an outbreak.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

Presence of fungus apparently of enzootic nature, on occasion acquires epizootic proportions, with a small number surviving as if having acquired immunity. Fungus is transmitted by zoospores which emerge through the tip of the exit tube that the sporangium puts forth to the exterior of the infected larva. Within the larvae, the fungus develops as a contorted, looped, and sparsely branched mycelium. Zoospores infect other larvae.

TREATMENT:	None reported. Entire larval population of infected batch should be discarded and containers sterilized.
PREVENTIVE MEASURES:	Filtration and ultraviolet treatment of seawater help in preventing outbreaks.

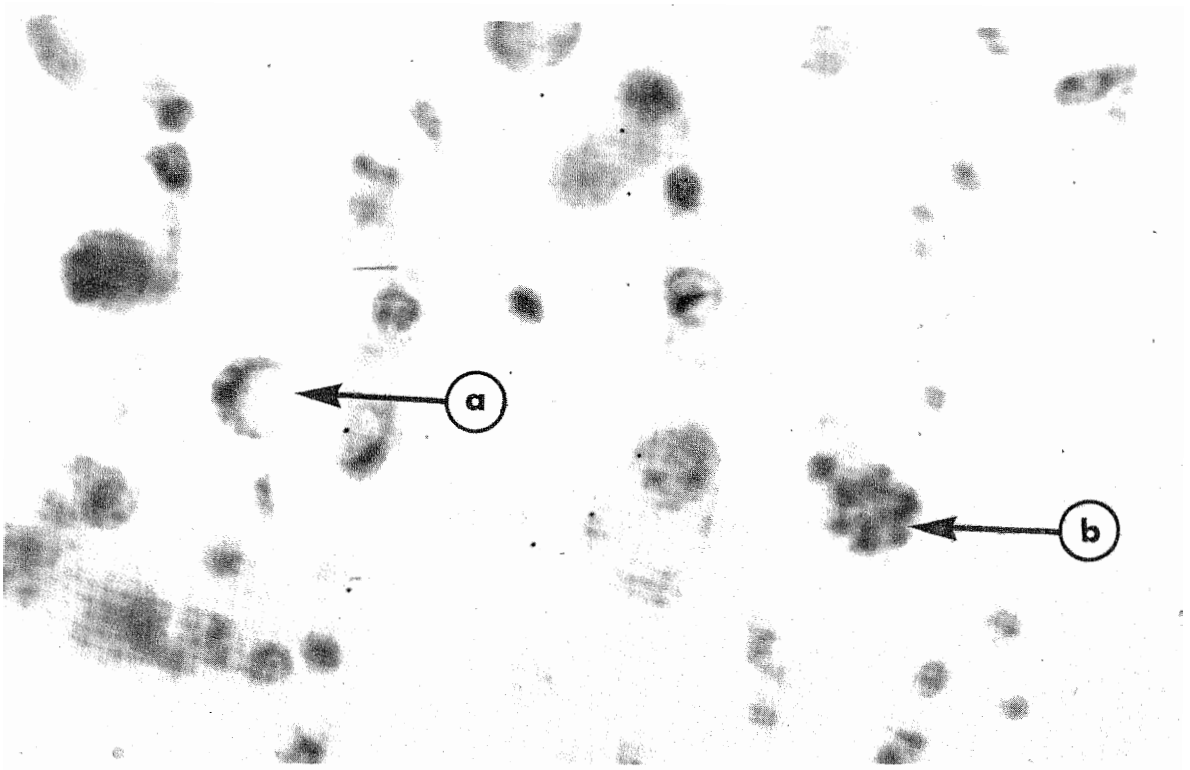
KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
East coast of United States. Also affects hard clam (Mercenaria mercenaria) larvae.

KEY REFERENCES:

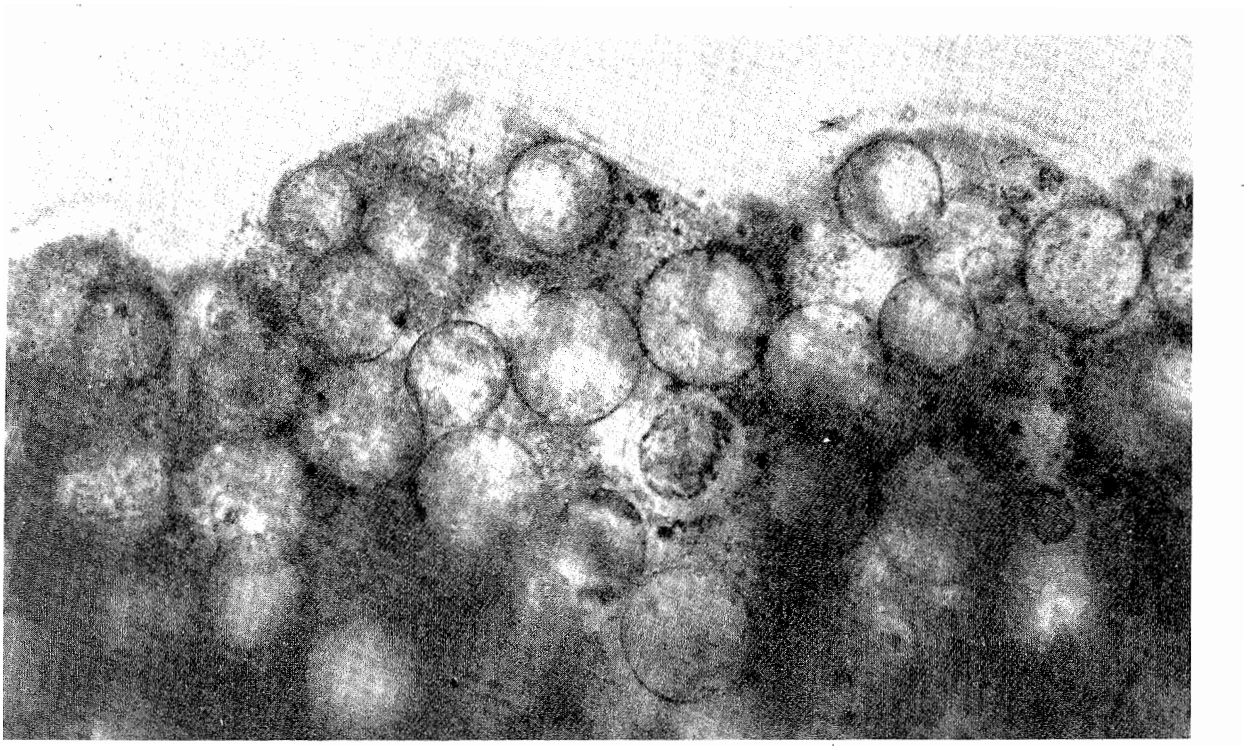
Davis, H. C., V. L. Loosanoff, W. H. Weston and C. Martin. 1954. A fungus disease in clam and oyster larvae. Science 120: 36-38.

Vishniac, H. S. 1955. The morphology and nutrition of a new species of Sirolpidium. Mycologia 47: 633-645.

(5) FUNGUS DISEASE OF OYSTERS



Life history stages of Labyrinthomyxa in tissues of American oysters.



Labyrinthomyxa spores in oyster tissues after thioglycollate culture (Modified from Ray, 1954).

FUNGUS DISEASE OF OYSTERS

COMMON NAME: Oyster fungus disease

SPECIES AFFECTED: American oyster, Crassostrea virginica (also found in Ostrea frons, O. equestris and O. lurida).

CAUSE: Systemic fungus invasion by Labyrinthomyxa marina (=Dermocystidium marinum).

GROSS SIGNS: Severe emaciation, gaping, mortalities. Digestive diverticulum pale.

METHOD OF DIAGNOSIS: Culture of oyster tissues in fluid thioglycollate medium, followed by iodine staining to disclose fungus spores. Histological examination of fixed and stained oyster tissues may also disclose characteristic stages of the organism.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

Prevalence limited to salinity and temperature requirements: 15 ppt salinity required, temperatures above 25°C. Transmitted from oyster to oyster, but may be further spread by scavengers. Prevalence increased by crowding. Motile biglagellate life cycle stages described as method of transmission.

The disease is enzootic in oysters of the Gulf and South Atlantic coasts, but prevalences may occasionally approach epizootic levels. For example, one survey by Ray (1966) disclosed prevalences of up to 100% in samples from the Gulf of Mexico. An outbreak of L. marina occurred recently in oysters (C. virginica) in Hawaii. From 90 to 99% of the Pearl Harbor oyster population was destroyed.

EFFECT ON HOST: Invasion thought to take place through the gut epithelium, possibly through the mantle. Tissues are invaded and damaged, and multiple abscesses are formed. Oyster seed shows geographic differences in susceptibility to the fungus. Virginia seed most susceptible; South Carolina the least--of the samples tested.

TREATMENT: Light infections of L. marina in laboratory populations of oysters may be controlled by the antifungal agent Cycloheximide (actidione) in continuous treatment of 1 ug/ml week. Use in natural populations would be questionable.

PREVENTIVE MEASURES: Control density of planting in enzootic areas. Plant seed in early autumn, harvest in late spring.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: United States east and Gulf coasts, from Massachusetts to Texas.

NOTE: A low-salinity form of this fungus probably infects the soft clam, Mya arenaria.

KEY REFERENCES:

Andrews, J. D. and W. G. Hewatt. 1957. Oyster mortality studies in Virginia. II. The fungus disease caused by Dermocystidium marinum in oysters of Chesapeake Bay. Ecol. Monogr. 27: 1-26.

Beckert, H., D. G. Bland and E. B. May. 1972. The incidence of Labyrinthomyxa marina in Alabama. Alabama Mar. Res. Bull. No. 8, pp. 18-24.

Kern, F. G., L. C. Sullivan and M. Takata. 1973. Labyrinthomyxa-like organisms associated with mass mortalities of oysters, Crassostrea virginica, from Hawaii. Proc. Nat. Shellf. Assoc. 63: 43-46.

Mackin, J. G. 1952. Oyster disease caused by Dermocystidium marinum and other microorganisms in Louisiana. Publ. Inst. Mar. Sci. Univ. Texas. 7: 132-229.

Mackin, J. G., H. M. Owen and A. Collier. 1950. Preliminary note on the occurrence of a new protistan parasite, Dermocystidium marinum n. sp. in Crassostrea virginica (Gmelin). Science 111: 328-329.s

Mackin, J. G. and S. M. Ray. 1966. The taxonomic relationship of Dermocystidium marinum, Mackin, Owen, and Collier. J. Invertebr. Pathol. 8: 544-545.

Perkins, F. O. and R. W. Menzel. 1967. Ultrastructure of sporulation in the oyster pathogen Dermocystidium marinum. J.eInvert. Pathol. 9: 205-229.e

Perkins, F. A. and R. W. Menzel. 1966. Morphological and cultural studies on a motile stage in the life cycle of Dermocystidium marinum. Proc. Nat. Shellf. Assoc. 56: 23-30.

Quick, J. A. Jr. 1972. Fluid thioglycollate medium assay of Labyrinthomyxa parasites in oysters. Fla. Dept. Nat. Resources, Leaflet Ser. Vol. 6, Part 4, No. 3, 11 pp.

Quick, J. A. Jr. and J. G. Mackin. 1971. Oyster parasitism by Labyrinthomyxa marina in Florida. Fla. Dept. Nat. Resources, Prof. Pap. Ser. No. 13, 55 pp.

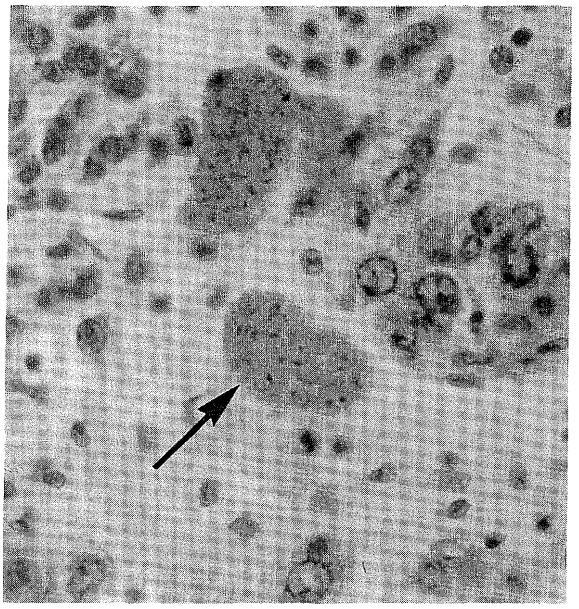
Ray, S. M. 1966a. A review of the culture method for detecting Dermocystidium marinum, with suggested modifications and precautions. Proc. Nat. Shellf. Assoc. 54: 55-69.

Ray, S. M. 1966b. Cycloheximide: inhibition of Dermocystidium marinum in laboratory stocks of oysters. Proc. Nat. Shellf. Assoc. 56: 31-36.

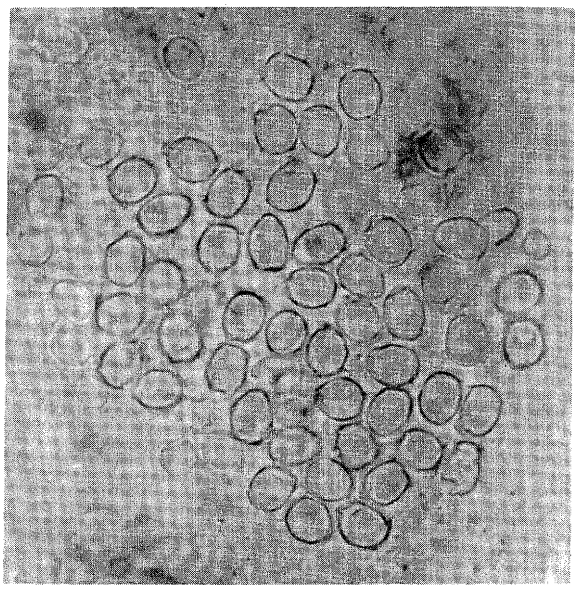
Ray, S. M. 1955. Dermocystidium marinum, a parasite of oysters. Exper. Parasitol. 4: 172-200.

Ray, S. M. 1954. Biological studies of Dermocystidium marinum, a fungus parasite of oysters. Rice Inst. Pamphlet Monogr. in Biology, Spec. Issue, 114 pp.

(6) DELAWARE BAY DISEASE OF OYSTERS



Plasmodium of Minchinia nelsoni in stained histological section of the American oyster.



Spores of Minchinia nelsoni in fresh mount.

DELAWARE BAY DISEASE OF OYSTERS

COMMON NAME:	Delaware Bay disease
SPECIES AFFECTED:	American oyster, Crassostrea virginica
GROSS SIGNS:	Summer mortalities; mantle recession; emaciation. Pale diverticulum; gaping; occasionally with pustules on inner shell.
CAUSE:	Haplosporidan protozoan, Minchinia nelsoni
METHOD OF DIAGNOSIS:	Careful histological examination of fixed and stained tissues, disclosing characteristic multi-nucleate plasmodia and (rarely) characteristic spores in digestive diverticula.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

Salinity dependent. Range from New England to North Carolina. Thought to enter oyster through gill epithelium and to gradually proliferate throughout oyster. Plasmodial forms predominate, with spores rare. Recover and resistance occur. Complex life cycle proposed; existence of reservoir hosts likely.

EFFECT ON HOST:	One of the most significant causes of mortalities in oyster beds of the Middle Atlantic states in the 1960's. Now enzootic with some residual high prevalences in localized areas.
TREATMENT:	Transfer of infected stocks to low salinity suggested.
PREVENTIVE MEASURES:	Grow-out should be in low salinity areas (below 15 o/oo) during epizootic periods. Early exposure of spat to disease proposed as a control method. Development of resistant stocks proposed. Oysters (including seed) should not be transferred to areas where disease is epizootic.

DELAWARE BAY DISEASE
OF OYSTERS

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

East coast of United States, Massachusetts to North Carolina. A similar haplosporidan has been recognized in plasmodial stages in Pacific oysters, Crassostrea gigas, from Korea, Taiwan and the State of Washington. Spores in the same size range as those of M.snelsoni were found in one Korean seeds oyster and one sporulating infection wass found in a moribund C. gigas from Humboldts Bay, California. The plasmodial stage of as haplosporidan parasite was recently reporteds from native oysters (Ostrea lurida) froms Oregon.s

KEY REFERENCES:

Andrews, J. D. 1966. Oyster mortality studies in Virginia. V.sEpizootiology of MSX, a protistan pathogen of oysters.s Ecology 47: 19-31.s

Andrews, J. D. 1968. Oyster mortality studies in Virginia. VII.s Review of epizootiology and origin of Minchinia nelsoni.s Proc. Nat. Shellf. Assoc. 58: 23-36.s

Andrews, J. D. and M. Frierman. 1974. Epizootiology of Minchinia nelsoni in susceptible wild oysters in Virginia, 1959 to 1971. J. Invert. Pathol. 24: 127-140.

Andrews, J. D. and J. L. Wood. 1967. Oyster mortality studies in Virginia. VI. History and distribution of Minchinia nelsoni, a pathogen of oysters, in Virginia. Chesapeake Sci. 8: 1-13.

Couch, J. A., C. A. Farley and A. Rosenfield. 1966. Sporulation of Minchinia nelsoni (Haplosporida, Haplosporidiidae) in Crassostrea virginica (Gmelin). Science 153: 1529-1531.

Farley, C. A. 1968. Minchinia nelsoni (Haplosporida) disease syndrome in the American oyster Crassostrea virginica. J. Protozool. 15: 585-599.s

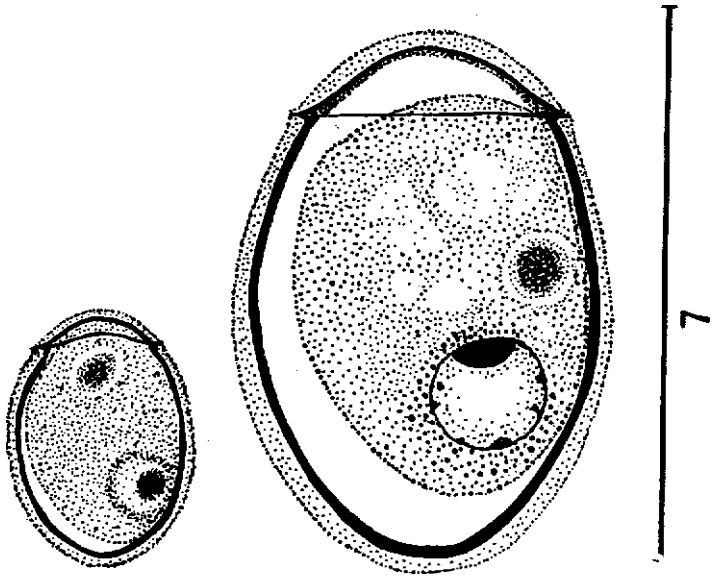
Farley, C. A. 1967. A proposed life cycle of Minchinia nelsoni (Haplosporida, Haplosporidiidae) in the American oysters, Crassostrea virginica. J. Protozool. 14: 616-625.

Haskin, H. H., L. A. Stauber and J. G. Mackin. 1966. Minchinia nelsoni n. sp. (Haplosporida, Haplosporidiidae) causative agent of the Delaware Bay oyster epizootic. Science 153: 1414-1416.

Katkansky, S. C. and R. W. Warner. 1970. Sporulation of a haplosporidan in a Pacific oyster (Crassostrea gigas) in Humboldt Bay, California. J. Fish. Res. Bd. Canada 27: 1320-26.

Mix, M. C. and V. Sprague. 1974. Occurrence of a haplosporidan in native oysters (Ostrea lurida) from Yaquina Bay and Alsea Bay, Oregon. J. Invert. Pathol. 23: 252-254.

(7) SEASIDE DISEASE OF OYSTERS



Comparative sizes of spores of Minchinia nelsoni (right) and M. costalis (left). (Modified from Couch, 1967).

SEASIDE DISEASE OF OYSTERS

COMMON NAME: Seaside disease

SPECIES AFFECTED: American oyster, Crassostrea virginica

GROSS SIGNS: Seasonal spring mortality, with sharp peak; mantle recession, emaciation, pale diverticulum, gaping.

CAUSE: Minchinia costalis, haplosporidan parasite.

METHOD OF DIAGNOSIS: Histological examination. Appearance of the spores differentiate from Minchinia nelsoni; spores are distributed in the tissues, rather than being confined to digestive diverticula, as these are in M. nelsoni infections. Spores smaller and more common than M. nelsoni spores. Sporulates May to July.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Found thus far only in seaside bays of Maryland and Virginia, and in Delaware Bay, where it causes mortalities in planted and natural beds. High salinity -- over 15 o/oo.

EFFECT ON HOST: Causes mortalities from mid-May to early July, with sharp peak and short duration.

TREATMENT: Not reported.

PREVENTIVE MEASURES: Management of shell stocks; removal of stocks from enzootic areas to low salinity waters; possible development of disease-resistant stocks; quarantine against planting oysters from enzootic areas in any other high-salinity growing area.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: Known thus far only from Delaware Bay and Seaside bays of Maryland and Virginia.

KEY REFERENCES:

Andrews, J. D., J. L. Wood and H. D. Hoese. 1962. Oyster mortality studies in Virginia. III. Epizootiology of a disease caused by Haplosporidium costale Wood and Andrews. J.eInsect Pathol. 4: 327-343.e

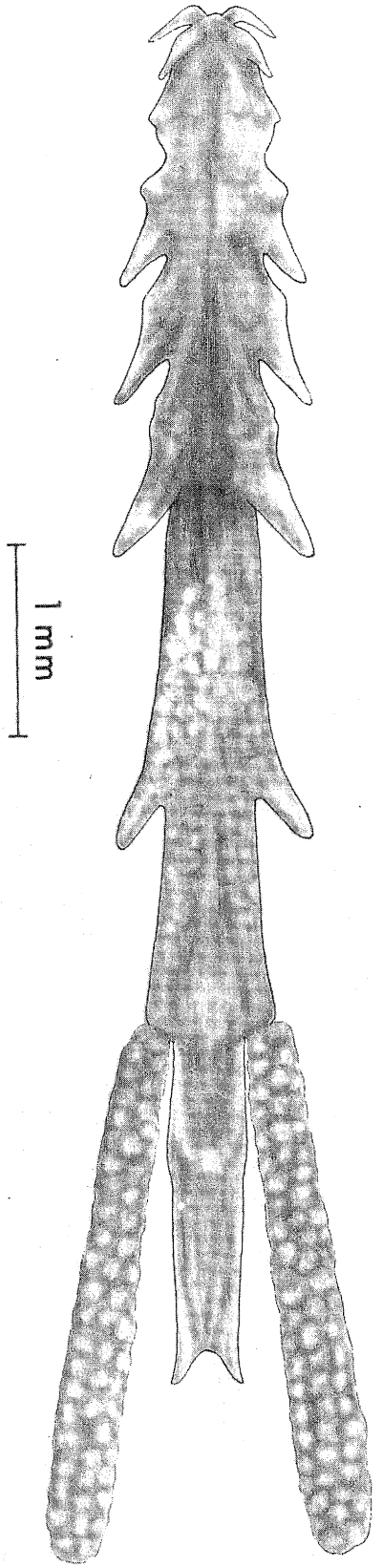
Couch, J. A. 1967. Concurrent haplosporidian infections of the oyster, Crassostrea virginica (Gmelin). J. Parasitol. 53: 248-253.e

Couch, J. A. and A. Rosenfield. 1968. Epizootiology of Minchinia costalis and Minchinia nelsoni in oysters introduced into Chincoteague Bay, Virginia. Proc. Nat. Shellfish. Assoc. 58: 51-59.

Wood, J. L. and J. D. Andrews. 1962. Haplosporidium costale (Sporozoa) associated with a disease of Virginia oysters. Science 136: 710-711.

(8) MYTILICOLA (RED WORM) DISEASE OF OYSTERS

MYTILICOLA
DISEASE OF OYSTERS



Mytilicola or "red worm" dissected from host.

MYTILICOLA DISEASE OF OYSTERS

COMMON NAME: Mytilicola (red worm) disease

SPECIES AFFECTED: Pacific oyster, Crassostrea gigas, and Olympia oyster, Ostrea lurida.

GROSS SIGNS: Poor growth; poor condition and sporadic mortalities; dissection of oysters disclose one or more reddish worm-like copepods in digestive tract.

CAUSE: Parasitic copepod, Mytilicola orientalis.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Probably imported from Japan to the United States west coast with early shipment of C. gigas seed. Now enzootic there, where it also occurs in O. lurida.

EFFECT ON HOST: Causes poor growth and condition; causes extensive tissue damage in the gut; cane result in sporadic mortalities.e

TREATMENT: Not reported.e

PREVENTIVE MEASURES: Not reported.e

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: West coast of United States (Introduced from Japan with seed oysters).

NOTE: M.e orientalis has also been reported frome mussels in Japan and Washington.e

KEY REFERENCES:

Chew, K. K., A. K. Sparks and S. C. Katkansky. 1965. Preliminary results on the seasonal size distribution of Mytilicola orientalis and the effect of this parasite on thee condition of the Pacific oyster, Crassostrea gigas. J.eFish. Res. Bd. Canada 22: 1099-1101.e

Odlaug, T. O. 1946. The effect of the copepod, Mytilicola orientalis, upon the Olympia oyster, Ostrea lurida. Trans. Amer. Microsc. Soc. 65: 311-317.

Sparks, A. K. 1962. Metaplasia of the gut of the oyster Crassostrea gigas (Thunberg) caused by infection with the copepod Mytilicola orientalis Mori. J. Insect Pathol. 4: 57-62.

(9) MALPEQUE BAY DISEASE OF OYSTERS

MALPEQUE BAY DISEASE
OF OYSTERS

COMMON NAME: Malpeque Bay disease

SPECIES AFFECTED: American oyster, Crassostrea virginica

GROSS SIGNS: Extreme weight loss of meats; stunted shell growth; yellow-green pustules (in early stages of epizootic); spawning failure; high mortalities.

CAUSE: Unknown infectious agent; possible virus. Unidentified bacteria isolated but pathogenicity not demonstrated.

METHOD OF DIAGNOSIS: Gross examination.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Extremely persistent, highly infectious agent. High mortalities of harvested shell stock in storage. Apparent development of resistance in stocks subject to high mortalities over several years.

EFFECT ON HOST: Not reported.

PREVENTIVE MEASURES: Rigid quarantine on transfer of live oysters from epizootic areas. Use of disease-resistant stocks apparently developed naturally as a result of early mortalities for restocking affected oyster-producing beds.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Appears host specific for Crassostrea virginica in the Gulf of Saint Lawrence.

KEY REFERENCES:

Drinnan, R. E. and L.eA. England. 1965. Further progress in rehabilitating oyster stocks. Fish. Res. Bd. Canada, Gen. Ser. Circ., Biol. Sta. St. Andrews, N. B. 48: 1-4.

Logie, R. R. 1956. Oyster mortalities, old and new, in the Maritimes. FRB Can. Progr. Rep. Atl. Coast Sta. 65: 3-11.

Logie, R. R., R. E. Drinnan and E. B. Henderson. 1960. Rehabilitation of disease-depleted oyster populations in eastern Canada. Proc. Gulf Carib. Fish. Inst. 13th Ann. Sess. pp. 109-113.

Needler, A. W. H. and R. R. Logie. 1947. Serious mortalities in Prince Edward Island oysters caused by a contagious disease. Trans. Roy. Soc. Can. 3(V), 41:73-89.

(10) DENMAN ISLAND DISEASE OF OYSTERS

DENMAN ISLAND DISEASE OF OYSTERS

COMMON NAME: Denman Island disease

SPECIES AFFECTED: Pacific oyster, Crassostrea gigas

GROSS SIGNS: Deep pustules on body and mantle surfaces; pus-filled sinuses.

CAUSE: Unknown, although a peculiar cell type (microcells) may be a life history stage of a pathogen.

METHOD OF DIAGNOSIS: Gross signs; microscopic examination of fixed and stained tissues discloses so-called "microcells" intracellularly and extracellularly.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Causes periodic mass mortalities at Denman Island, B. C., among oysters of older age groups, which are otherwise in excellent condition.

TREATMENT: None reported.

PREVENTIVE MEASURES: None reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
The disease is known only from Denman Island, B. C.

NOTE: This disease in C. gigas bears some resemblance to Malpeque Bay disease in C. virginica, in that "microcells" have been seen in Malpeque Bay oyster tissues. Further studies are needed to establish the nature of both diseases, or their identity.

KEY REFERENCES:

Quayle, D. B. 1961. Denman Island disease.
Fish. Res. Bd. Canada, Ms. Rept. Ser. (Biol.) 713, 9 pp.

Sprague, V. 1971. Diseases of oysters. Ann. Rev. Microbiol.
25: 211-230.

Clam Diseases

Hatching, rearing and limited production of hard clams, Mercenaria mercenaria, occurs in this country on a small scale. Other clams, especially those from the Pacific coast, have attracted research attention. Increasing production of surf clams, Spisula solidissima, from natural beds, and the existence of large unexploited populations of mahogany quahogs, Arctica islandica, make clam mariculture a difficult choice at present, except for specialty production such as small raw clams for consumption on the half shell.

Clam hatching and rearing attempts have encountered two diseases -- bacillary necrosis and larval mycosis -- which aree similar to those which affect oyster larvae. A variety of clame parasites exists, but none have been clearly shown to be detrimentale to culture in the United States.e

(1) BACILLARY NECROSIS OF CLAM LARVAE

BACILLARY NECROSIS OF
CLAM LARVAE

COMMON NAME: Bacillary necrosis

SPECIES AFFECTED: Hard clam, Mercenaria mercenaria, larvae and juveniles.

GROSS SIGNS: Settling and decrease in larval motility. High, sudden mortalities.

CAUSE: Vibrio anguillarum, V. alginolyticus and other marine Vibrio spp; possibly aeromonads and pseudomonads also.

METHOD OF DIAGNOSIS: Direct microscopic examination of live affected larvae. Swarming vibrios are diagnostic. Bacteriological culture and experimental challenge with isolates.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Vibrios believed to be enzootic, causing overt infection and mortality when critical infective level is reached because of adverse environmental condition. Entry via esophagus. Proliferation throughout the tissues with lysis and necrosis of tissues. Course of experimental infections is rapid, with disease signs apparent 4 to 5 hours after exposing larval cultures to pathogens. Deaths begin at 8 hours and complete mortality of culture population by 18 hours.

EFFECT ON HOST: Mortality, usually complete, in larval cultures.

TREATMENT: Combistrep - 50 to 100 ppm.
Other antibiotics suggested: Chloramphenicol, polymyxin B, erythromycin, neomycin.

PREVENTIVE MEASURES: Improve water quality and general sanitation.

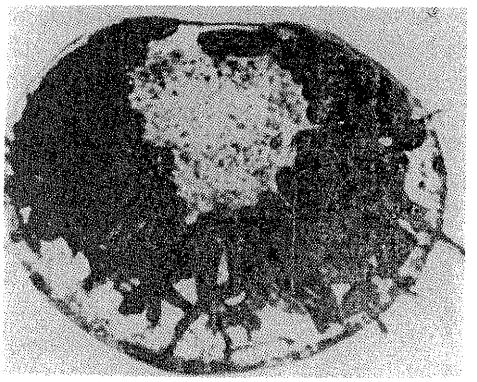
KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Reported from United States east coast.
Larvae of hard clams, American and European oyster proven susceptible; as are probably most bivalve mollusks.

KEY REFERENCES:

Tubiash, H. S., P. E. Chanley and E. Leifson. 1965. Bacillary necrosis, a disease of larval and juvenile bivalve mollusks. I. Etiology and epizootiology. J. Bacteriol.e 90: 1036-1044.e

Tubiash, H. S., R. R. Colwell and R. Sakazaki. 1970. Marine vibrios associated with bacillary necrosis, a disease of larval and juvenile bivalve mollusks. J. Bacteriol.. 103: 272-273.e

(2) LARVAL MYCOSIS OF CLAMS



Clam larvae infected with Sirolpidium (cotton blue stain).
(Modified from Davis et al., 1954).

LARVAL MYCOSIS OF CLAMS

COMMON NAME: Larval Mycosis

SPECIES AFFECTED: Hard clam, Mercenaria mercenaria, larvae

GROSS SIGNS: Growth ceased, rapid mortality in larval populations. Larvae may be observed in various stages of disintegration with the fungus quite apparent in their interior. Juveniles also found infected.

CAUSE: Systemic fungus invasion by Sirolpidium zoophthorum.

METHOD OF DIAGNOSIS: Microscopic examination of larvae unstained or stained with neutral red or lactophenol cotton blue. The fungus acquires a deeper stain than larval tissues if living larvae are kept in a solution of neutral red in sea water, a characteristic to be used both for detecting the first appearance of the fungus, also as a tag to study the progress of an outbreak.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

Presence of fungus apparently of endemic nature, on occasion acquires epidemic proportions, with a small number surviving as if having acquired immunity. Fungus is transmitted by zoospores which emerge through the tip of the exit tube that the sporangium puts forth to the exterior of the infected larvae. Within the larvae, the fungus develops as a contorted, looped, and sparsely branched mycelium. Zoospores infect other larvae.

TREATMENT: Not described. Entire larval population of infected batch should be discarded and containers sterilized.

PREVENTIVE MEASURES: Filtration and ultraviolet treatment of seawater suggested.

LARVAL MYCOSIS OF CLAMS

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

East coast of United States. Also affects oyster (Crassostrea virginica) larvae.

KEY REFERENCES:

Davis, H. C., V. L. Loosanoff, W. H. Weston and C. Martin. 1954. A fungus disease in clam and oyster larvae. Science 120: 36-38.

Vishniac, H. S. 1955. The morphology and nutrition of a new species of Sirolpidium. Mycologia 47: 633-645.

FISH DISEASES

A number of marine and anadromous fish species are in experimental or pilot plant stages of culture -- and one, salt-water rearing of Pacific salmon, is approaching the production stage in a few ventures. Other species in favor at present are pompano, striped bass, Atlantic salmon, and even mullet.

Salt-water rearing of salmon has had to contend with some of the classic diseases of fresh-water salmon hatcheries -- furunculosis and kidney disease -- which are continuing problems in the salt-water environment. Additionally, halophilic vibrios (particularly Vibrio anguillarum) have emerged as serious mortality factors in cage, tank, and pond salt-water culture of salmon. Several disease research groups are occupied with vibriosis, using such approaches as oral immunization and development of resistant stocks.

Intensive culture, using silo techniques, has been tried experimentally with pompano, Atlantic salmon, and striped bass. Disease has not yet emerged clearly as a major mortality factor, although there are sporadic mass mortalities and continuing background mortalities that do not seem attributable to nutritional or environmental deficiencies.

Salmon Diseases

Salt-water rearing of salmon (Pacific and Atlantic species) has been attempted on both coasts of United States in the past several years. At present, in addition to government and university experimental facilities, there are five commercial salt-water salmon rearing operations in the Pacific Northwest, and two in Maine. Floating net enclosures are generally used, although tanks and net-enclosed portions of estuaries have been considered.

Since salmon spawning and early development takes place in fresh water, some of the important fresh-water diseases - furunculosis and kidney disease -- can be retained, can prosper, and can in some instances be transferred in salt water. Additionally, salmon in salt water are seriously affected by vibrio infections -- in fact such infections and resultant mortalities are at present one of the principal deterrents to economically successful large-scale culture. Prophylactic immunization (oral or by inoculation) with killed vibrios, combined with terramycin added to moist pellet diet, currently controls outbreaks to some extent.

The disease of salmon reported to be of significance in salt-water rearing include:

(1)e Vibriosis;e

(2)e Furunculosis;e

(3)e Kidney disease; ande

(4)e Sporocytophaga disease.e

We can anticipate future problems with virus diseases -- several of which are current threats to fresh-water salmonid culture.

GENERAL REFERENCES:

Novotny, A. J. 1972. The marine culture of vertebrates. In. Aquaculture--Potential in the Maritimes Region. Proc. Sympos. Fed. Dept. Fish. and Canad. Soc. Envir. Biol. Halifax N.S. April 1972, pp. 38-53.

Rucker, R. R., B. J. Earp and E. J. Ordal. 1954. Infectious diseases of Pacific salmon. Trans. Amer. Fish. Soc. 83: 297-312.

(1) VIBRIOSIS OF SALMON

COMMON NAME: Vibriosis

SPECIES AFFECTED: Chinook salmon, Oncorhynchus tshawytscha
Chum salmon, Oncorhynchus keta
Sockeye salmon, Oncorhynchus nerka
Pink salmon, Oncorhynchus gorbuscha
Coho salmon, Oncorhynchus kisutch
Atlantic salmon, Salmo salar
Cherry salmon, Oncorhynchus masu
Rainbow trout, Salmo gairdneri
Cuthroat trout, Salmo clarki

GROSS SIGNS: Red necrotic lesions of the abdominal musculature and erythema at the bases of fins and within the mouth. Evidence of hemorrhaging in the gills, skin and intestines. Inactivity, cessation of feeding, reddened vent, exophthalmia, and extensive mortalities. Acute form of the disease may produce little externally-apparent pathology, especially in young pink and chum salmon.

CAUSE: Infection by marine bacterium, Vibrio anguillarum (and possibly other closely-related vibrios).

METHOD OF DIAGNOSIS: Gross observations, then bacterial culture with selective media. Vibrios may frequently be observed in large numbers in wet (saline) mounts from spleen tissue.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Stress from transfer shock, overcrowding, low oxygen, or rough handling can precipitate disease particularly when water temperatures are above 10°C. Disease can be transferred ino salt water.

EFFECT ON HOST: The most serious pathogen of salt-water reared salmon at present. Causes major mortalities in rearing pens, ponds, and tanks. At least two strains of Vibrio anguillarum seem important as pathogens of salmon at present in the Pacific Northwest -- one which grows rapidly in culture and affects fish early in the salt water phase, and one which grows slowly in culture and kills fish later in their salt water growth.

TREATMENT:

Sulfonamides: sulfamerazine, sulfadiazine, sulfasoxazole.
Sulfamerazine -- 5 gm/100 lb fish/day for 10 days or 15 gm/100 lb fish in dry starter diet.
Terramycin -- 4 gm/day/100 lb fish for 10 days.
Chloramphenicol -- 2.5 to 3.5 gm/100 lb fish/day for 7-10 days.
Nitrofurazone -- 56 mg/kg fish/day.
Intramuscular injections of chloromycetin and streptomycin (large fish).

Furanace, a chemotherapeutic developed in Japan, may have potential in treating vibrio infections in marine fish. Inhibition of cultured strains of vibrios occurs; therapeutic levels of the chemical can be produced by short-duration baths; and rapid elimination from the blood occurs following exposure.

PREVENTIVE MEASURES:

Avoid excessive crowding and handling.
Immunization -- orally or by inoculation.
2 gm sulfamerazine/100 lb fish/day in food.
Furazolidone -- .02% in diet over 2 weeks.
(Sulfamerazine and furazolidone not recommended as routine measures because of possible selection for resistant strains).

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

Ubiquitous in marine and estuarine environment, reaching infectious and epizootic levels under favorable environmental conditions and host stresses. Known in salmon throughout the Pacific rim, from Oregon to Japan, including Alaska and British Columbia. Reported in the British Isles, the Baltic, and the Northwest Atlantic from New Hampshire to New Brunswick. A pathogen of many marine and estuarine fishes -- cod, eels, mullet, ayu, etc.

NOTE:

An oral vaccine composed of moist whole cells of V. anguillarum killed with 0.3% formalin was incorporated in Oregon moist pellet diet at a rate of 2 mg per gram and fed to salt water-held chinook and coho, with encouraging results, although further long-term studies are needed. Atlantic salmon are also susceptible to vibrios, and some protection has been demonstrated by similar oral immunization.a

KEY REFERENCES:

Anderson, D. P. 1974. Diseases of Fishes. Book 4. Fish Immunology. Snieszko, S. F. and Axelrod, H. R. (eds.). TFH Pubs., Jersey City, N. J.

Anderson, J. I. W., and D. A. Conroy. 1970. Vibrio disease in marine fishes. In. Snieszko, S. F. (ed.). A sympsium on diseases of fishes and shellfishes. pp. 266-272. Amer. Fish. Soc. Spec. Pub. No. 5, Wash., D. C.

Anonymous. 1973. Vibriosis in Atlantic salmon. F.A.O. Aquacult. Bull. 6(1): 17.

Anonymous. 1973. Control of vibrios. F.A.O. Aquacult. Bull. 5(2): 17-18.

Cisar, J. O., and J. L. Fryer. 1969. An epizootic of vibriosis in chinook salmon. Bull. Wildl. Dis. Assoc. 5: 73-76.

Evelyn, T. P. T. 1971. First records of vibriosis in Pacific salmon cultured in Canada, and taxonomic status of the responsible bacterium, Vibrio anguillarum. J. Fish. Res. Bd. Canada 28: 517-525.

Fryer, J. L., J. S. Nelson and R. L. Garrison. 1972. Vibriosis in fish. pp. 129-133. In. Moore, R. W. (ed.). Progress in Fishery and Food Science, Vol. 5, Univ. Wash.. Pubs. Fish., New Series.

Pearse, L., R. S. V. Pullin, D. A. Conroy and D. McGregor. 1974. Observations on the use of Furanace for the control of vibrio disease in marine flatfish. Aquaculture 3: 295-302.

Rucker, R. R., B. J. Earp and E. J. Crdal. 1954. Infectious diseases of Pacific salmon. Trans. Amer. Fish. Soc. 83: 297-312.

(2) FURUNCULOSIS OF SALMON

FURUNCULOSIS OF SALMON

COMMON NAME: Furunculosis

SPECIES AFFECTED: Sockeye salmon, Oncorhynchus nerka
Chum salmon, Oncorhynchus keta
Chinook salmon, Oncorhynchus tschawytscha
Pink salmon, Oncorhynchus gorbuscha
Coho salmon, Oncorhynchus kisutch
Atlantic salmon, Salmo salar

GROSS SIGNS: Acute: sudden increase of mortality, few or no external lesions.
Subacute: more gradual increase of mortality with the formation of furuncles (external lesions, soft blisterlike necrotic areas filled with blood).
Chronic: low mortality, intestinal inflammation, variable hemorrhages.
Latent: No mortality or diseases signs, but bacterium can be isolated.

CAUSE: Infection by the bacterium Aeromonas salmonicida.

METHOD OF DIAGNOSIS: Isolation on standard furunculosis agar medium of bacteria from kidney tissues and external lesions of infected fish.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Transmission direct, and possibly transovarian also. Highly infectious in fresh water in dormant state; active disease triggered by stresses of transfer to salt water. Pathogen is sufficiently salt tolerant that infections can be transferred in sea water. Pink and chum salmon more susceptible than chinook. The pathogen has been isolated from surf smelt, Thallicthys pacificus, residing in salt water pens with infected chinook salmon.

EFFECT ON HOST: May cause mortality. Outbreaks begin in June and continue into autumn.

TREATMENT:

Nitrofurans: especially Furazolidone (Furoxone)	1.2 gm/100 lb fish/day in feed, a 20 days
Sulfonamides (particularly sulfamerazine)	10 gm/100 lb fish/day in feed, for 14 days

Oxytetracycline, chloramphenicol. (Note: recent publications suggest that sulfas and oxytetracycline may not prevent mortalities in chinook salmon).

PREVENTIVE MEASURES: Lowered water temperatures, development of resistant fish when possible. Oral immunization being tested.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:

Found in salmon held in fresh and salt water, in sablefish, also in trout and cyprinids from United States west coast. Isolated from all species of Pacific salmon in salt water.

KEY REFERENCES:

Anonymous. 1973. Furunculosis of chinook salmon in salt water. FAO Aquacult. Bull. 5(2): 18.

Anonymous. 1973. Diseases of pen-reared salmon. FAO Aquacult. Bull. 5(3-4): 15.

Bullock, G. L. 1971. Identification of fish pathogenic bacteria. In. Diseases of Fishes (S. F. Snieszko and H. R. Axelrod, eds.). TFH Pubs. Jersey City, N. J. Book 2a: 151 pp.

Bullock, G. L., D. A. Conroy and S. F. Snieszko. 1971. Bacterial diseases of fishes. In. Diseases of Fishes (S. F. Snieszko and H. R. Axelrod, eds.). TFH Pubs. Jersey City, N.J., Book 2a: 151 pp.

Evelyn, T. P. T. 1971. An aberrant strain of the bacterial fish pathogen Aeromonas salmonicida isolated from a marine host, the sablefish (Anoplopoma fimbria) and from two species of cultured Pacific salmon. J. Fish. Res. Bd. Canada 28: 1629-1634.

Evelyn, T. P. T. 1971. First records of vibriosis in Pacific salmon cultured in Canada, and taxonomic status of the responsible bacterium, Vibrio anguillarum. J. Fish. Res. Bd. Canada 28: 517-525.

Herman, R. L. 1968. Fish furunculosis 1952-1966. Trans. Amer. Fish. Soc. 97: 221-230.

Herman, R. L. 1969. Oxytetracycline in fish culture -- a review. Tech. Pap. Bur. Sport Fish. U. S. Fish Wildl. Serv. No. 31, 9 pp.

Herman, R. L. 1973. A review of the prevention and treatment of furunculosis. In. Symposium on the major communicable fish diseases in Europe and their control. (W. A. Dill, ed.). EIFAC Tech. Pap. No. 17, Suppl. 2, pp. 170-174.

Scott, M. 1968. The pathogenicity of Aeromonas salmonicida (Griffin) in sea and brackish waters. J. Gen. Microbiol. 50: 321-327.

(3) KIDNEY DISEASE OF SALMON

KIDNEY DISEASE OF SALMON

COMMON NAME: Kidney disease

SPECIES AFFECTED: Coho salmon, Oncorhynchus kisutch, held in salt-water pens. (Also identified from chinook, pink and chum salmon).

GROSS SIGNS: External signs variable. Infected fish may cease feeding; fail to move with school; spend much time near surface; appear dark when viewed from above; exhibit exophthalmia; become edematous; exhibit furuncles laterally and ventrally. In other instances there may be no outward signs. During second year in salt water, large vigorously feeding fish may die suddenly.

CAUSE: Bacterium, Cornybacterium sp.

METHOD OF DIAGNOSIS: Dissection of dying fish discloses severely diseased kidneys. Gram stain reveals dense concentrations of gram positive rods.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Carried over from fresh water. Transmission in salt water not yet demonstrated.

EFFECT ON HOST: May contribute to mortalities in salt-water rearing facilities. Generally appears during first winter in salt water.

TREATMENT: None known for salt-water rearing; temporary arrest by sulfonamides. Erythromycin (100 mg/kg fish/day has been used in fresh water.

PREVENTIVE MEASURES: Careful selection of stock from disease-free hatcheries. Diet has been found to influence occurrence and severity of the disease in fresh water.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
West coast of North America.

KEY REFERENCES:

Anomymous. 1972. Effect of diet on occurrence of kidney disease in salmonids. FAO Aquaculture Bull. 5(1): 17.

Earp, B. J., C. H. Ellis and E. J. Ordal. 1953. Kidney disease in young salmon. Wash. Dept. Fish. Spec. Rept. Ser. No. 1, 74 pp.

Ordal, E. J. and B. J. Earp. 1956. Cultivation and transmission of etiological agent of kidney disease in salmonid fishes. Exp. Biol. Med. 92: 85-88.

(4) SPOROCYTOPHAGA DISEASE OF SALMON

SPOROCYTOPHAGA DISEASE OF SALMON

COMMON NAME: Sporocytophaga disease (salt-water myxobacteriosis)

SPECIES AFFECTED: Sockeye salmon, Oncorhynchus nerka

GROSS SIGNS: Lesions, sometimes quite large, on body surfaces but not on gills; skin in area of infection has abraded appearance.

CAUSE: Myxobacterium Sporocytophaga sp. (often with mixed infection with Vibrio anguillarum.

METHOD OF DIAGNOSIS: Isolation of myxobacteria from lesions and formation of microcysts in culture.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Form infective microcysts capable of resisting adverse environmental conditions. Occurs in epizootics in salt-water rearing pens. Sporadic during early summer.

EFFECT ON HOST: Can cause severe mortalities in salt-water rearing pens, especially when V. anguillarum is also present.

TREATMENT: Oxytetracycline and chlorotetracycline baths at 1 ppm for 1 hour.
PMA (pyridylmercuric acetate) or Timsan (ethylmercuric phosphate) baths at 1 ppm for 1 hour.
Note: Accumulation of mercury in fish tissues makes use of these chemicals questionable. Furanace baths have been suggested.

PREVENTIVE MEASURES: Not reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Pacific Northwest (possibly in Atlantic Salmon from Maine).

KEY REFERENCES:

Amend, D. V. 1970. Myxobacterial infections of salmonids: prevention and treatment. In. Snieszko, S. F. (ed.). A symposium on diseases of fishes and shellfishes. pp. 258-265. Spec. Pub. No. 5, Amer. Fish. Soc., Wash., D. C. 526 pp.

Grace, J. B. 1951. The life cycle of Sporocytophaga. J. Gen. Microbiol. 5: 519-524.

Pacha, R. E., and E. J. Ordal. 1970. Myxobacterial diseases of salmonids. In. Snieszko, S. F. (ed.). A symposium on diseases of fishes and shellfishes. pp. 243-257. Spec. Pub. No. 5, Amer. Fish. Soc., Wash., D. C. 526 pp.

Stanier, R. Y. 1942. The Cytophaga group; a contribution to the biology of Myxobacteria. Bact. Rev. 6: 143-160.

Wood, J. W. 1968. Diseases of Pacific salmon: their prevention and treatment. State of Wash. Dept. Fish Hatcheries.

Pompano Diseases

Pompano, because of its relative scarcity, high unit value, and delicious taste, has been the subject of numerous (mostly unsuccessful) mariculture ventures during the past decade. Research and development efforts have encountered problems with diets, water quality, and inability to produce larvae consistently from artificial spawning. Culture has been attempted in tanks, floating pens, and silos. Some disease problems have appeared -- but thus far those reported are mostly ones which can be controlled readily. It seems that the real culprits are still not visible, or at least not yet reported in the published literature. Only four diseases are summarized here:

(1) White spot disease;

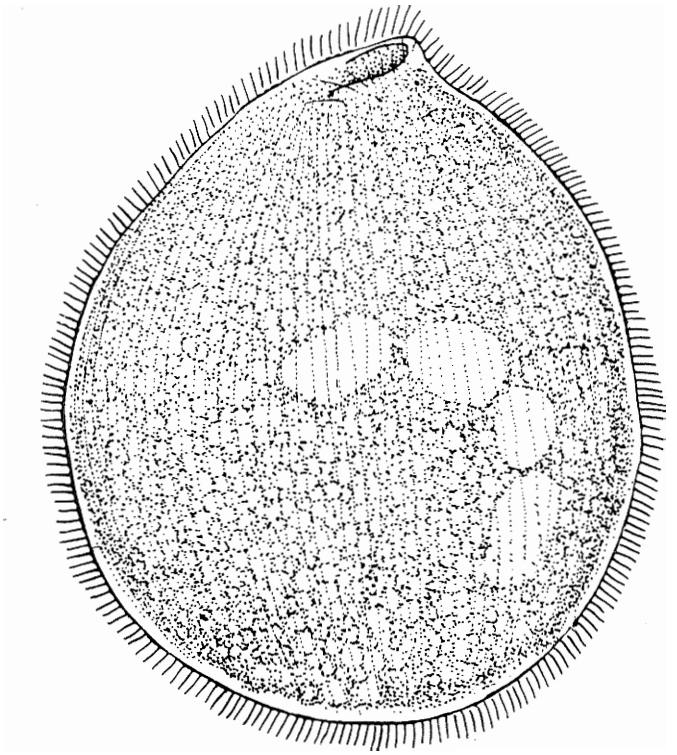
(2) Cardiac myxosporidiosis;

(3) Monogenetic trematode infestation; and

(4) Fatty liver degeneration.

Other parasites have been identified from pompano, but they do not seem to constitute present threats to mariculture. Two rather indefinite reports suggest bacterial infections as a cause of observed mortalities, but the organisms were not identified. Another recent report indicates that the protozoans Trichodina and Scyphidium can be sporadic problems.

(1) WHITE SPOT DISEASE OF POMPANO



Trophozoite of Cryptocaryon. (From Wilkie and Gordon, 1969).

WHITE SPOT DISEASE
OF POMPANO

COMMON NAME: White spot disease

SPECIES AFFECTED: Pompano, Trachinotus carolinus

GROSS SIGNS: Pinhead size white cysts on gills and body surfaces, including eyes. Infestations produce small lesions, erosion of gills, and excessive mucous production.

CAUSE: Ciliate protozoan, Cryptocaryon irritans

METHOD OF DIAGNOSIS: Dissect fresh unpreserved cysts, examine under low magnification for typical ciliated organism.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Parasite numbers can increase rapidly in sea water tanks, when mature parasites drop off the host, encyst on the bottom, and undergo multiple divisions to produce large numbers of motile stages.

EFFECT ON HOST: Heavy infestations, particularly of gills, may be fatal; heavy infestations may blind the fish.

TREATMENT: Formalin, tris, and cupric acetate in sea water can be effective in all but heavy infections (see Nigrelli and Ruggieri, 1966).

PREVENTIVE MEASURES: Cleaning and sterilization of tanks in which juveniles are held, to eliminate encysted stages.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Thus far reported from pompano in Florida, but known as a parasite of marine aquarium fishes in many parts of the world.

KEY REFERENCES:

Nigrelli, R. F. and G. D. Ruggieri. 1966. Enzootics in the New York Aquarium caused by Cryptocaryon irritans Brown 1951 (=Ichthyophthirius marinus Sikama 1961), a histophagous ciliate in the skin, eyes, and gills of marine fishes. Zoologica 51: 47-102.

Wilke, D. W. and H. Gordon. 1969. Outbreak of cryptocaryoniasis in marine aquaria at Scripps Institution of Oceanography. Drum and Croaker. 69(1): 23-31.

(2) CARDIAC MYXOSPORIDIOSIS OF POMPANO

CARDIAC MYXOSPORIDIOSE
OF POMPANO

COMMON NAME: Cardiac Myxosporidiosis

SPECIES AFFECTED: Pompano, Trachinotus carolinus

GROSS SIGNS: Sporadic mortalities of n holding tanks; poor growth. Di scloses small white cysts on surface the heart.

CAUSE: Myxosporidan protoan, _____ sp.

METHOD OF DIAGNOSIS: Characteristic elongate myxosporidan spores in fresh smears from cysts removed from heart.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Unknown.

EFFECT ON HOST: Can weaken and kill individual hosts, particularly in presence of other environmental stresses.

TREATMENT: Not reported.

PREVENTIVE MEASURES: Clean and sterilize holding tanks; remove and destroy abnormal individuals.

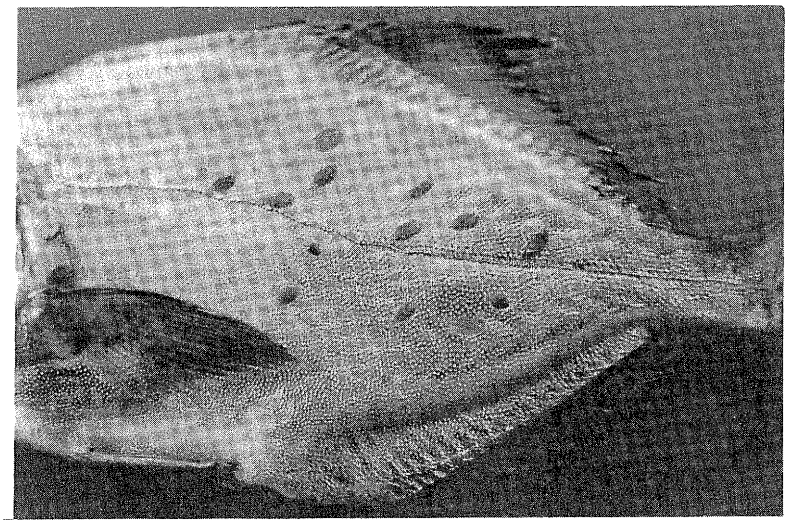
KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Known thus far from one experimental rearing facility in Florida.

KEY REFERENCES:

Kudo, R. R. 1966. Protozoology, 5th Ed. C. C. Thomas Co., Springfield, Ohio. 1174 pp.

(3) MONOGENETIC TREMATODE INFESTATION OF POMPANO

MONOGENETIC TREMATODE INFESTATION OF POMPANO



Juvenile tank-reared pompano with monogenetic trematodes on body surface. Photograph supplied by H. Kumpf, Southeast Fisheries Center.

MONOGENETIC TREMATODE INFESTATION OF POMPANO

COMMON NAME: Monogenetic trematode infestation

SPECIES AFFECTED: Pompano, Trachinotus carolinus

GROSS SIGNS: Small white worms observed on gills; pigmented leaf-like worms occur on body surfaces of tank-held juveniles.

CAUSE: Bicotylophora trachinoti (on gills) and Benedenia sp. on body surfaces.

METHOD OF DIAGNOSIS: Gross observation; low power microscopic examination of living worms removed from gills or body surfaces.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Direct life cycle and reinfection can cause rapid buildup of parasite numbers.

EFFECT ON HOST: Rarely causes mortality, but heavy infestations may be contributory to death of host in presence of other environmental stresses. Gill epithelium damaged in heavy Bicotylophora infestations.

TREATMENT: Fish placed in tank of 250 ppm formalin for 35 minutes (gill trematodes). Reduced salinities also constitute an effective treatment.

PREVENTIVE MEASURE: Avoid overcrowding; use formalin dips if numbers of worms become apparent.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: Known thus far from experimental rearing facilities in Florida.

KEY REFERENCES:

Finucane, J. H. 1970. Pompano mariculture in Florida. Amer. Fish Farmer 1(4): 5-10.

Kumpf, H. E. 1972. Temperature-salinity tolerance of the Florida pompano, Trachinotus carolinus (Linnaeus). Univ. Miami Ph. D. Thesis. 102 pp. (unpub).

Smith, T. I. J. 1973. The commercial feasibility of rearing pompano, Trachinotus carolinus (Linnaeus), in cages. Univ. Miami Sea Grant Tech. Bull. No. 26, 62 pp.

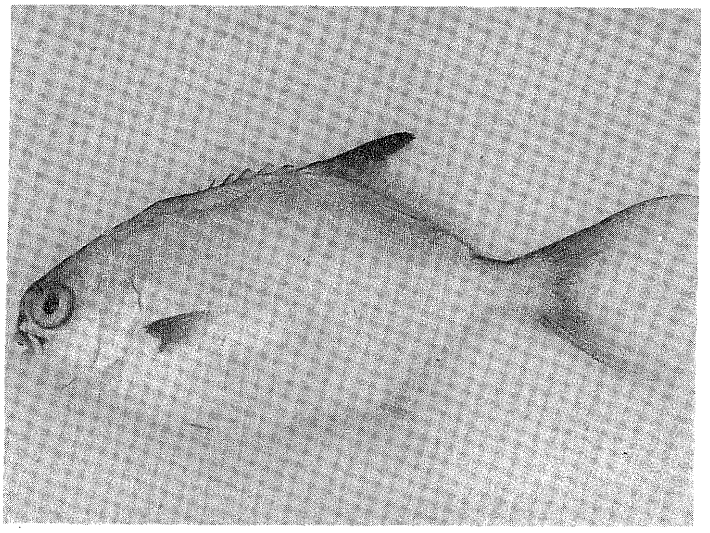
Swingle, W. E. and W. M. Tatum. 1971. Experiments in the culture of marine species in floating baskets. Ala. Dept. Conser. Nat. Res. (Seafoods) Div., Fed. Aid Ann. Prog. Rep. 2-86-R-2, 26 pp.

Williams, E. H. Jr. 1972. Parasitic infestation of some marine fishes before and after confinement in feeding cages. Ala. Marine Resources Bull. No. 8: 25-31.

Williams, E. H. Jr. (in press). Parasites of some mariculture fishes before and after cage culture. Proc. Fourth Food-Drugs from the Sea Conf., Mayaguez, P.R.

(4) FATTY LIVER DEGENERATION IN POMPANO

FATTY LIVER DEGENERATION IN POMPANO



Pompano with "dropsy-like" distention of abdomen, found in dietary deficiency.

FATTY LIVER DEGENERATION IN POMPANO

COMMON NAME: Fatty degeneration of liver

SPECIES AFFECTED: Pompano, Trachinotus carolinus

GROSS SIGNS: Poor growth, emaciation, listlessness, cessation of feeding, and sporadic mortalities of juvenile pompano held in tanks and floating pens; fluid buildup in body cavity, producing a dropsy-like condition.

CAUSE: Dietary deficiency, probably of protein.

METHOD OF DIAGNOSIS: Dissection of abnormal fish discloses liver of light tan color; blood indicates severe anemia.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Appears in juveniles fed on artificial diet deficient in protein.

EFFECT ON HOST: Poor growth; sporadic mortalities if diet unchanged.

TREATMENT: Augment diet with natural protein.

PREVENTIVE MEASURES: Provide complete diets (at present, artificial pellet food augmented with liver, ground squid, etc.).

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Reported from several experimental rearing facilities in Florida.

KEY REFERENCES:

Finucane, J. H. 1970. Pompano mariculture in Florida. Amer. Fish Farmer 1(4): 5-10.

Smith, T. I. J. 1973. The commercial feasibility of rearinga pompano, Trachinotus carolinus (Linnaeus), in cages.a U.aMiami Sea Grant Tech. Bull. No. 26.a

Striped Bass Diseases

Salt-water rearing of striped bass is being attempted on a limited scale. Interest derives from its market acceptability, and its importance as a sport fish. One east coast hatchery has been successful in routine hatching and rearing of this species to juvenile stages. Suggestions have been made to utilize heated power plant effluents to rear this and other mariculture species -- by providing year-round growth in temperate areas where ambient water temperatures are low in winter.

Several diseases and abnormalities of striped bass have been observed -- some in these same heated effluents. Those included in this summary include:

(1)e Lymphocystis;e

(2)e Fin rot;e

(3)e Pasteurella disease;e

(4)e Myxosporidian disease; ande

(5)e Deformities.e

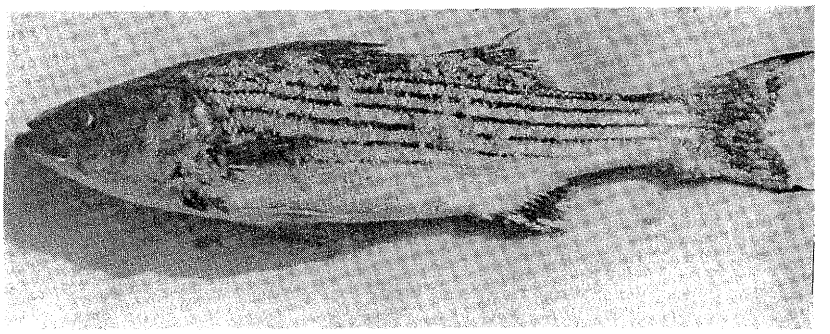
This list is short, but it will undoubtedly grow in proportion to mariculture attempts with the species. Other parasites, such as the nematode Philometra rubra in the viscera, and the copepod Ergasilus labracis on the gills, are known from natural populations, and may become important in culture.

GENERAL REFERENCES:

Paperna, I. and D. E. Zwerner. 1974. Diseases and parasites of striped bass: a pilot study. Res. Rept. (unnumbered), Va. Inst. Mar. Sci., Gloucester Pt., Va., 28 pp.

(1) LYMPHOCYSTIS IN STRIPED BASS

LYMPHOCYSTIS DISEASE OF STRIPED BASS



Lymphocystis disease of striped bass.

LYMPHOCYSTIS DISEASE
OF STRIPED BASS

COMMON NAME: Lymphocystis

SPECIES AFFECTED: Striped bass, Roccus saxatilis

GROSS SIGNS: Raised grayish nodules on fins and body surfaces of the fish, often becoming confluent in heavy infections. Often associated with fin erosion.

CAUSE: Virus.

METHOD OF DIAGNOSIS: Gross examination, followed by finding of enormously hypertrophied cells in histological sections.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Has been reported in epizootic proportions in American plaice from the Grand Banks, in striped bass from heated power plant effluents, and in marine aquarium fish.

EFFECT ON HOST: Rarely fatal, but reduces acceptance and marketability of affected fish.

TREATMENT: None reported -- fish usually recover spontaneously.

PREVENTIVE MEASURES: None reported.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Known in striped bass from North American east coast. Also common in many other species of marine and fresh-water fishes.

KEY REFERENCES:

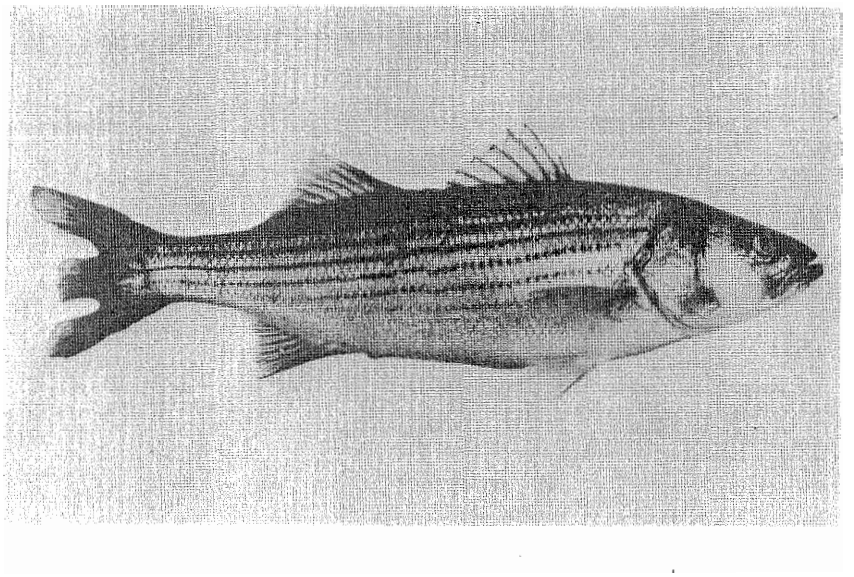
Anonymous. 1973. Fish disease alert. Amer. Littoral Soc. Newsletter, Aug. 1973. p. 2.

Krantz, G. E. 1970. Lymphocystis in striped bass, Roccus saxatilis, in Chesapeake Bay. Chesapeake Sci. 11: 137-139.

Nigrelli, R. F. and G. D. Ruggieri. 1965. Studies on virus diseases of fishes. Spontaneous and experimentally-induced cellular hypertrophy (Lymphocystis disease) in fishes of the New York Aquarium, with a report of new cases and an annotated bibliography. (1874-1965). Zoologica 50: 83-96.

(2) FIN ROT OF STRIPED BASS

FIN ROT DISEASE OF STRIPED BASS



Striped bass with early signs of fin rot.

FIN ROT DISEASE OF STRIPED BASS

COMMON NAME: Fin rot, Tail rot

SPECIES AFFECTED: Striped bass, Roccus saxatilis (and many other species of fish).

GROSS SIGNS: Fin necrosis; caudal fin initially in some species, also dorsal and anal fins. Skin hemorrhages and ulcers.

CAUSE: Bacterial invasion by vibrios, pseudomonads, and aeromonads, probably induced by environmental stress.

METHOD OF DIAGNOSIS: Gross examination.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY: Probably a complex synergistic group of pathogens enhanced by presence of pollutants or other environmental stresses. May be enhanced by high bacterial populations in water.

EFFECT ON HOST: Severe erosion of fins interferes with locomotion; bacterial infections may become systemic; ulcerations provide entry for other secondary invaders.

TREATMENT: Terramycin may be partially effective.

PREVENTIVE MEASURES: Adequate diet and water quality; avoid overcrowding and drastic temperature changes; use care in handling.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM: Wide spectrum of marine and euryhaline food fishes in many parts of the world, including flounders, bluefish (Pomatomus saltatrix), sea trout (Cynascion regalis) and sea herring (Clupea harengus). Probably one of the most common non-specific diseases of marine and fresh-water fishes in captive environments.

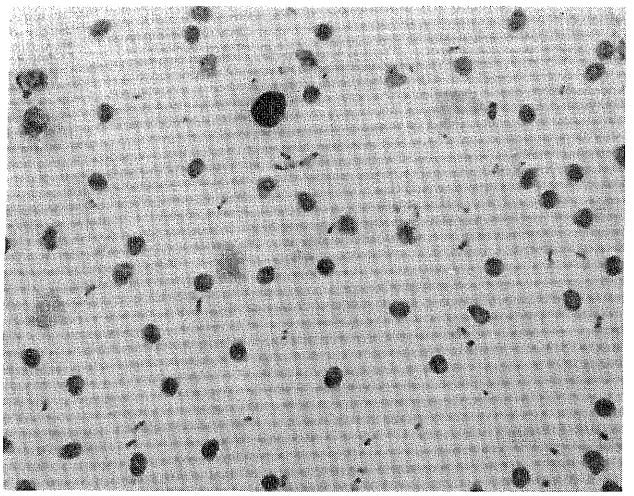
KEY REFERENCES:

American Littoral Society. 1972. Special Newsletter Alert -- Fin rot. Amer. Littoral Soc. Newsletter, August, 1972, 4 pp.

Mahoney, J. B., F. H. Midlige and D. G. Deuel. 1973. A fin rot disease of marine and euryhaline fishes in the New York Bight. Trans. Amer. Fish. Soc. 102: 596-605.

(3) *PASTEURELLA* DISEASE OF STRIPED BASS

PASTEURELLA DISEASE OF STRIPED BASS



Pasteurella disease of striped bass. Stained blood smear with polar staining bacteria. Modified from Allen et al., 1966.

PASTEURELLA DISEASE OF STRIPED BASS

COMMON NAME: Pasteurella disease

SPECIES AFFECTED: Striped bass, Roccus saxatilis
White perch, Roccus americanus

GROSS SIGNS: No pathological external indication of infection. Internally, Pasteurella infections produce extensive bacteremia, often with white spots in viscera of striped bass but not in white perch -- possibly because of greater resistance and increased likelihood of chronic infections in striped bass.

CAUSE: Bacterium Pasteurella piscicida.

METHOD OF DIAGNOSIS: Isolation of P. piscicida from blood and associated tissues, using selective culture media. Organisms are gram-negative, bipolarly staining, cytochrome oxidase-positive pleomorphic non-motile rods.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:

Pasteurella piscicida, isolated from white perch dying during the 1963 extensive mortalities in Chesapeake Bay, was not found in extensive bacterial isolations made by Allen et al. (1966) from normal white perch in 1964 and 1965. That the organism was of low virulence was suggested by the fact that an inoculum of 10^7 cultured cells was required to produce LD50's in white perch. It seems likely that striped bass, which are closely related to white perch, are even less susceptible to P. piscicida, but that the epizootic in white perch resulted in sufficient infection pressure on the striped bass population to produce infections and mortalities, when combined with predisposing environmental conditions. Antigenically, P. piscicida does not indicate close similarity to the plague bacillus.

PASTEURELLA DISEASE OF STRIPED BASS

An epizootic and associated mortalities in striped bass was reported to have occurred in 1972. Progressive necrosis of spleen, liver, kidney and intestine was characteristic of moribund fish.

EFFECT ON HOST: Mortalities of striped bass reported in 1963 epizootic were mostly larger size groups; few reports of juvenile fish were noted. Experimentally, deaths can be produced within 6 days following inoculation.

TREATMENT: Not described for striped bass, but the Japanese have found sulfonamides, antibiotics and nitrofurazones effective against P. piscicida infections in yellowtail. Sulfonamides are used prophylactically by incorporation in Oregon pellets at 200-400 mg/kg body weight for 6 days. Chloramphenicol is effective mixed with food at 20-40 mg/kg body weight for 5 or more days. Resistance to chemotherapeutics has been found.

PREVENTIVE MEASURES: Minimize other environmental stresses where possible.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
First reported from Chesapeake Bay and tributaries. A similar or identical form identified in Japan. Possibly of widespread occurrence in estuarine and marine waters.

NOTE: A bacterial pseudotuberculosis in cultured yellowtail, Seriola quinqueradiata, was determined by Kusuda (1972) to be caused by Pasteurella piscicida. The disease was first reported by Kubota, Kimura, Kusuda, and Egusa (1970) and new information has been supplied by Matsusato (1974). Serious mortalities have occurred in yellowtail farms due to the disease since 1968.

KEY REFERENCES

Allen, N. E. and M. J. Pelczar Jr. 1967. Bacteriological studies on white perch (Roccus americanus). Chesapeake Sci. 8: 135-154.

Allen, N. E., F. C. Szoka Jr. and M. J. Pelczar Jr. 1966. Microbiological studies on the white perch (Roccus americanus) and its estuarine environment. Univ. Md., Dept. Microbiol., Res. Rept. No. 4, 117 pp.

Janssen, W. A. and M. J. Surgalla. 1968. Morphology, physiology and serology of a Pasteurella species pathogenic for white perch (Roccus americanus). J. Bact. 96: 1606-1610.

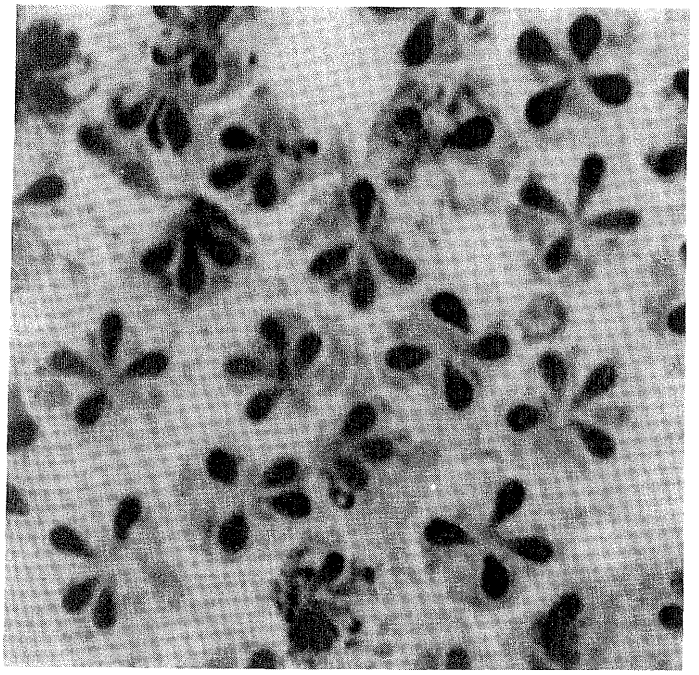
Matsusato, T. (in press). Bacterial pseudotuberculosis of cultured yellowtail. Proc. UJNR Symposium on Aquaculture Diseases. Tokyo, 1974.

Paperna, I. and D. E. Zwerner. 1974. Diseases and parasites of striped bass: a pilot study. Research Report (unnumbered), Virginia Institute of Marine Science, 28 pp.

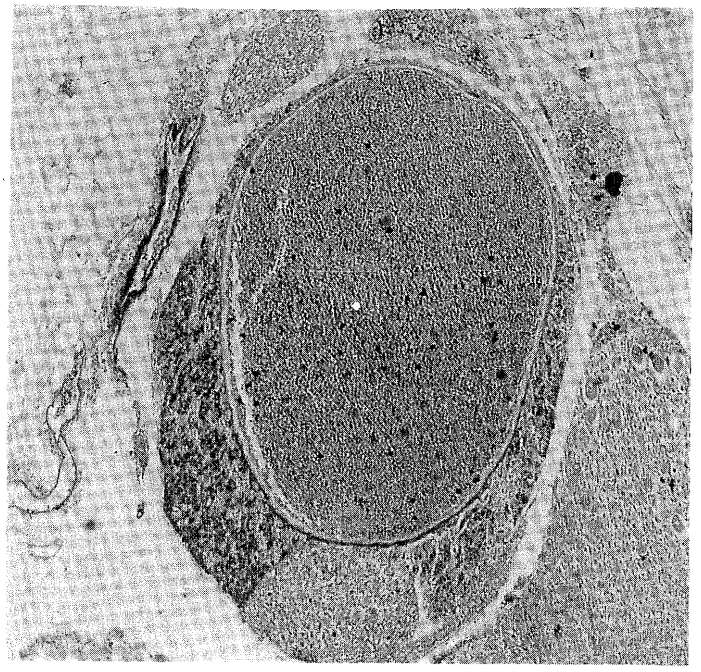
Snieszko, S. F., G. L. Bullock, E. Hollis and J. G. Boone. 1964. Pasteurella sp. from an epizootic of white perch (Roccus americanus) in Chesapeake Bay tidewater areas. J. Bact. 88: 1814-1815.

(4) MYXOSPORIDIAN DISEASE

OF STRIPED BASS



Myxosporidian disease of striped bass.
Stained spores of Kudoa cerebralis.
Photograph supplied by David E. Zwerner,
Virginia Institute of Marine Science.



Myxosporidian cyst in connective tissue adjacent
to cranial nerve trunk of striped bass. Photograph
supplied by David E. Zwerner, Virginia Institute
of Marine Science.

MYXOSPORIDIAN DISEASE
OF STRIPED BASS

COMMON NAME: Myxosporidian disease

SPECIES AFFECTED: Striped bass, Roccus saxatilis

GROSS SIGNS: No external signs. Parasite cyst grossly visible in dissected fish in cranial cavity around brain.

CAUSE: Myxosporidian protozoan, Kudoa cerebralis

METHOD OF DIAGNOSIS: Parasite cysts in cranial cavity, with very typical quadrate Kudoa spores disclosed by microscopic examination of fresh smears.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
This is the first Kudoa to be associated with the nervous system of fish. Cysts located in connective tissue associated with nervous system. Higher prevalence in estuarine samples than in oceanic samples suggests that transmission occurs in estuaries. Juveniles not found to be infected in very limited sampling.

EFFECT ON HOST: Cysts causes distortion and displacement of neural elements at infection site and in adjacent ganglia and nerves. Behavioral abnormalities not reported.

TREATMENT: Not reported, but would be difficult if not unlikely.

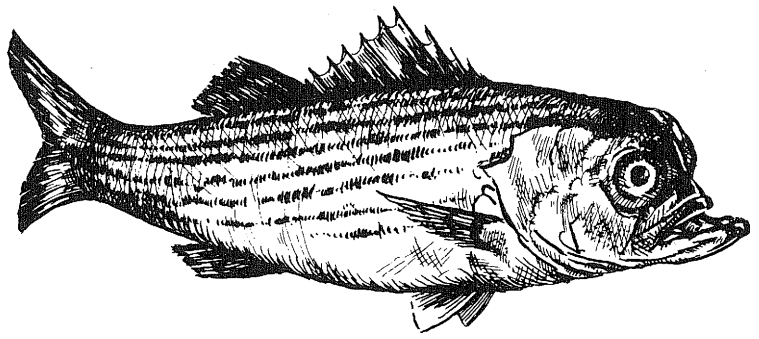
PREVENTIVE MEASURES: Not reported, but from the history of a similar disease (whirling disease of salmonids caused by Myxosoma cerebralis), drastic steps, such as complete sterilization of culture areas and restrictions on transfer of infected stock, must be taken to prevent dissemination of the disease.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Lower Chesapeake Bay and Atlantic coast of Virginia.

KEY REFERENCES:

Paperna, I. and D. E. Zwerner. 1974. Kudoa cerebralis sp.n. (Myxosporidea, Chloromyxidae) from the striped bass, Morone saxatilis (Walbaum). J. Protozool. 21: 15-19.

(5) DEFORMITIES IN STRIPED BASS



"Pug-headed" striped bass.

DEFORMITIES IN
STRIPED BASS

COMMON NAME: Deformities (particularly pug-headedness)

SPECIES AFFECTED: Striped bass, Roccus saxatilis

GROSS SIGNS: Abnormal body structure, particularly pug-headedness, spinal curvature, spinal compression, fin anomalies.

CAUSE: Not fully understood, but probably of at least three origins; genetic anomalies, abnormal embryonic development, and environmentally-induced larval abnormalities.

METHOD OF DIAGNOSIS: Gross observation.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Embryonic and larval stages of fish are particularly susceptible to environmental changes (such as low oxygen, high temperatures, presence of chemical contaminants, presence of toxins or metabolites, etc. Effects may be expressed as structural anomalies, as well as physiological ones.

EFFECT ON HOST: Reduced mobility, rendering affected individual more susceptible to predators and less successful in feeding.

TREATMENT: None.

PREVENTIVE MEASURES: Stable and optimum environmental factors during spawning, hatching and early rearing.

KNOWN GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Anomalies such as these can be found in most fish species, but in very low numbers in natural populations. Culture environment may allow survival of many more abnormal individuals than would be the case in natural waters.

NOTE: A study of deformities in striped bass is being conducted by C. R. Hickey Jr. (New York Ocean Science Laboratory) and B. H. Young (New York State Dept. Envir. Conservation). Many types and degrees of deformities, especially pug-headedness and other skeletal anomalies, have been noted.

KEY REFERENCES

Dawson, C. E. 1964. A bibliography of anomalies of fishes. Gulf Res. Rept. 1: 308-399.

Hickey, C. R. 1972. Common abnormalities in fishes, their causes and effects. Tech. Rept. No. 13, N. Y. Ocean Sci. Lab., 20 pp.

Mansueti, R. 1958. Eggs and young of the striped bass, Roccus saxatilis. Maryland Dept. Res. and Ed. Contrib. No. 112, 35 pp.

Mansueti, R. 1960. An unusually large pugheaded striped bass, Roccus saxatilis, from Chesapeake Bay, Maryland, Chesapeake Sci. 1: 111-113.

Turner, J. L. and T. C. Farley. 1971. Effects of temperature, salinity, and dissolved oxygen on the survival of striped bass eggs and larvae. Calif. Fish and Game 57: 268-273.

MARINE TURTLE DISEASES

Interest in sea turtle farming, concentrating on the green sea turtle, Chelonia mydas, has been revived within the past five years, following a long hiatus after the heyday of marine fish hatcheries in the first few decades of this century. One large operation on Grand Cayman Island, for example, now has a stock of some 100,000 turtles of varying ages, including those captured in various parts of the world and transferred to ponds as well as those raised at the facility.

The crowding inherent in this or any mariculture operation produces additional stresses and provides a means of rapid dissemination of infectious agents. Thus far two diseases of green sea turtles have been recognized: a viral skin disease named "gray patch disease", and a coccidian disease of the digestive tract.

Additionally, rearing experiments with loggerhead sea turtles, Caretta caretta, have encountered problems with Aeromonas infections, which have so far been described very briefly.

(1) GRAY PATCH DISEASE

OF GREEN TURTLES

GRAY PATCH DISEASE
OF TURTLES

COMMON NAME: Gray patch disease

SPECIES AFFECTED: Green turtle (Chelonia mydas)

GROSS SIGNS: Grayish spreading lesions with slightly raised edges that eventually become macerated, on skin of neck and flippers. Carapace sometimes affected. Pustular lesions which do not spread may also occur.

CAUSE: Herpes-type virus.

METHOD OF DIAGNOSIS: Histopathology of lesions showing enlarged nuclei with intranuclear inclusions; electron microscopic demonstration of viral particles of herpes type -- dense inner core with two outer membranes.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Epizootics reported in five groups of hatchlings, with 70-98% of individuals affected. Mortality peaks 9 weeks after onset of signs.

EFFECT ON HOST: Maceration and erosion of skin and carapace. Resolution of lesions in some animals, death in others. Heavily infected hatchlings die; individuals over one year old that have survived an infection will be immune.

TREATMENT: Only experimental treatment with metabolic inhibitors is now available, and some response seen in severely infected turtles.

CONTROL: Probably a stress-induced disease, since crowding drastically increases occurrence of the infections.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Unknown, but reported from hatchlings in Grand Cayman Island and in those imported from Australia.

KEY REFERENCES:

Haines, H. G., A. Rywlin and G. Rebell. (in press). A herpesvirus disease of farmed green turtles (Chelonia mydas). Proc. Fifth Ann. Workshop, World Mariculture Soc., Charleston, S. C. (1974).

(2) AEROMONAS DISEASE OF LOGGERHEAD TURTLES

AEROMONAS DISEASE OF LOGGERHEAD TURTLES

COMMON NAME: Aeromonas disease

SPECIES AFFECTED: Loggerhead sea turtle, Caretta caretta

GROSS SIGNS: Superficial lesions (not further described).

CAUSE: Bacterium Aeromonas sp.

METHOD OF DIAGNOSIS: Isolations of Aeromonas from lesions.

LIFE HISTORY, BIOLOGY, EPIZOOTIOLOGY:
Turtles hatched in captivity and fed on various combinations of fish, crabs, and trout pellets exhibited lesions due to Aeromonas infections when three months old.

EFFECT ON HOST: Superficial lesions (mortalities not reported).

TREATMENT: Not reported.

PREVENTIVE MEASURES: Not reported, but diet was considered inadequate, as evidenced by poor growth.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Aeromonas spp. are common inhabitants of aquatic environments, and may be facultatively pathogenic to animals living under conditions of environmental stress. Infections of loggerhead sea turtles by Aeromonas have been reported thus far only from Georgia.

KEY REFERENCES:

Ingle, R. M. and F. G. Walton Smith. 1949. Sea turtles and the turtle industry. Spec. Pub. Univ. Miami Marine Lab., Univ. Miami Press. 107 pp.

Stickney, R. R., D. B. White and D. Perlmutter. 1973. Growth of green and loggerhead sea turtles in Georgia on natural and artificial diets. Bull. Georgia Acad. Sci. 31: 37-44.

(3) COCCIDIAN DISEASE

OF GREEN TURTLES

COCCIDIAN DISEASE OF GREEN TURTLES

COMMON NAME: Coccidian disease

SPECIES AFFECTED: Green turtle, Chelonia mydas

GROSS SIGNS: Early hatchlings most seriously affected. Emaciation; lethargy; no external lesions; intestinal casts may occur in heavy infections.

CAUSE: Coccidian protozoan, probably a member of the genus Caryospora.

METHOD OF DIAGNOSIS: Intestinal mucosa heavily invaded; oocysts in feces may be induced to sporulate in 2% dichromate solution.

LIFE HISTORY, BIOLOGY, EPIZOOTICLOGY:
Reported as epizootic in tank-held hatchling populations, with a peak at 30 days after hatching, followed by declining mortalities to 60-70 days. Course of disease in individual hatchling seems brief -- less than one week.

EFFECT ON HOST: Coccidian infections involve invasion and destruction of intestinal mucosa; oocysts shed in feces and mature externally. Oocyst masses may produce congestion or occlusion of intestine.

TREATMENT: None yet reported as successful. Tetracycline and sulfamethazine administered orally, in water, and by injection not effective. Avian anticoccidial agents should be tested.

CONTROL: Sanitation an important factor, since oocysts occur in fecal sludge at bottom of tanks. Tanks should be cleaned and disinfected regularly. Contact with adult carriers should be avoided.

GEOGRAPHIC RANGE OF CAUSATIVE ORGANISM:
Reported as epizootic in hatchlings from Grand Cayman Island.

KEY REFERENCES:

Rebell, G. (in press). Proc. Fifth Annual Workshop, World Mariculture Society, Charleston, S. C. (1974).

NON-SPECIFIC TOXICANT-INDUCED PATHOLOGY

Occasionally in aquaculture situations non-specific mortalities or abnormalities can occur which may be traced to toxicants of various kinds -- heavy metals, pesticides, petroleum, industrial chemicals -- in the environment. Such contamination may result from land runoff, aerial dissemination, accidental spills, or deliberate discharges.

Effects are twofold -- on the exposed marine population, and potentially on the human consumer. Effects on the marine animal may take the form of reduced growth, poor spawning, poor larval survival, mortalities, anomalies in body structure, and greater susceptibility to pathogens. It may well be, for example, that effects of contaminants and toxins could be expressed at time of molting in Crustacea. Effects on human consumers, except for a few extreme incidents such as Minamata Disease in Japan, remain as potential problems.

Documentation of contaminant effects on aquaculture species is voluminous and increasing. Some of the information has been summarized recently (Sindermann, in press; Snieszko, 1974) and a few samples of the original literature are appended to this section. As a few examples of the kind of information available, Couch and Nimmo (in press) have suggested interactions between chlorinated hydrocarbon

contamination and prevalence of virus disease in shrimps, and between pesticide pollution and prevalence of fin rot in fish. Other studies at the Gulf Breeze (Florida) Environmental Research Laboratory have demonstrated reduced growth and tissue pathology in oysters following exposure to pesticides (Lowe et al., 1972). Couch (in press) has reviewed the extensive literature on pesticide effects on fish. Duke et al., 1970, demonstrated harmful effects of polychlorinated biphenyls (PCB's) on a spectrum of estuarine species, and Butler (1962, 1966, 1973) has reviewed effects of DDT on oysters, shrimps, and other species. Documentation of effects of petroleum components on fish and shellfish is equally voluminous.

Aquaculture areas must be protected from contamination by a system of laws combined with vigorous enforcement. Such a system does not now exist in the United States or elsewhere -- in fact the one country (Japan) most heavily committed to mariculture at present, is confronted and threatened today with a rising tide of coastal pollution of frightening proportions -- such that some aquaculture efforts have had to be abandoned. Mortalities and signs of stress in aquaculture populations must therefore be investigated from the viewpoint of possible toxicants as well as infectious disease.

REFERENCES:

Butler, P. A., 1966. Pesticides in the marine environment. J.eApp. Ecol. 3 (suppl.): 253.e

Butler, P. A. 1973. Organochlorine residues in estuarine mollusks, 1965-72 -- National Pesticide Monitoring Program. Pestic. Monit. J. 6: 238-362.

Butler, P. A. 1962. Reaction of some estuarine mollusks to environmental factors. In: Biological problems in water pollution. Third Seminar. U. S. Dept. Health, Educ., Welfare, Public Health Serv. Publ. No. 999-WP-25, 1965: 92-104.e

Couch, J. A. and D.eR. Nimmo. 1974. Ultrastructural studies of shrimp exposed to the pollutant chemical polychlorinated biphenyl (Aroclor 1254). Bull. Soc. Pharm. Envir. Pathologists 2(2): 17-20.

Couch, J. A. and D. R. Nimmo. (in press). Detection of interactions between natural pathogens and pollutants in aquatic animals. Proc. Reg. Sympos. Disease in Aquatic Animals. LSU, 1974.

Couch, J. A. (in press). Histopathologic effects of pesticides and related chemicals on the livers of fishes. In. "Pathology of Fishes". Proc. Sympos. on Fish Pathology, Univ. Wisconsin Press.

Davis, H. C. 1961. Effects of some pesticides on eggs and larvae of oysters (Crassostrea virginica) and clams (Venus mercenaria). Comm. Fish. Rev. 23(12): 8-23.

Davis, H. C. and H. Hidu. 1969. Effects of pesticides on embryonic development of clams and oysters and on survival and growth of larvae. Fish. Bull. U. S. Fish Wildl. Serv. 67: 393-404.

Duke, T. W., J. I. Lowe and A. J. Wilson Jr. 1970. A polychlorinated biphenyl (Aroclor 1254) in the water, sediment, and biota of Escambia Bay, Florida. Bull. Envir. Contam. Toxicol. 5: 171-180.

Ghittino, P. 1972. Aquaculture and associated diseases of fish of public health importance. J. Amer. Vet. Med. Assoc. 161: 1476-1485.

Hansen, David J. and A. J. Wilson Jr. 1970. Significance of DDT residues from the estuary near Pensacola, Fla. Pestic. Monit. J. 4: 51-56.

Hansen, D. J., P. R. Parrish, J. I. Lowe, A. J. Wilson Jr. and P. D. Wilson. 1971. Chronic toxicity, uptake, and retention of Aroclor(R) 1254 in two estuarine fishes. Bull. Environ. Contam. Toxicol. 6: 113-119.

Harrington, R. W. and W. L. Bidlingmayer. 1958. Effects of dieldrin on fishes and invertebrates of a salt marsh. J.eWildl. Manage. 22: 76-82.e

Jensen, S., A. G. Johnels, M. Olsson and G. Otterlind. 1969. DDT and PCB in marine animals from Swedish waters. Nature. Lond. 224, 247-250.

Koeman, J. H., M. C. Ten Noever deeBrauweand R. H. de Vos. 1969. Chlorinated biphenyls in fish, mussels and birds from the River Rhine and the Netherlands coastal area. Nature. Lond. 221, 1126-1128.

Lowe, J. I., P. R. Parrish, J. M. Patrick Jr. and F. Forester. 1972. Effects of the polychlorinated biphenyl Aroclor 1254 on the American oyster Crassostrea virginica. Mar. Biol. 17: 209-214.

Lowe, J. I., P. D. Wilson, A. J. Rick and A. J. Wilson Jr. 1971. Chronic exposure of oysters to DDT, toxaphene and panthion. Proc. natn. Shellfish. Ass. 61, 71-79.

Mihursky, J. A., A. J. McErlean and V. S. Kennedy. 1970. Thermal pollution, aquaculture and pathobiology in aquatic systems. J. Wildl. Dis. 6: 347-355.

Nimmo, D. R., D. J. Hansen, J. A. Couch, N. R. Cooley, P. R. Parrish and J. I. Lowe. (in press). Toxicity of Aroclor 1254 and its physiological activity in several estuarine organisms. Arch. Envir. Contam. Toxicol.

Nimmo, D. R., Blackman, R. R., Wilson, A. J. Jr. and Forester, J. 1971. Toxicity and distribution of Aroclor(R) 1254 in the pink shrimp Penaeus duorarum. Mar. Biol. 11: 191-194.

Sindermann, C. J. (in press). The role of environmental contaminants in open system mariculture. Proc. First Caribbean Oceaneering Conf. San Juan, P.R. (1973).

Snieszko, S. F. 1974. The effects of environmental stress on out-breaks of infectious diseases of fishes. J. Fish. Biol. 6: 197-208.e

Wisely, B. and R. A. P. Blick. 1967. Mortality of some marine invertebrate larvae in mercury, copper, and zinc solutions. Aust. J. Mar. Freshw. Res. 18: 63-72.

Zitko, V. 1971. Polychlorinated biphenyls and organochlorin pesticides in some freshwater and marine fishes. Bull. envir. Contam. Toxicol. 6, 464-470.

DISEASES AND ABNORMALITIES CAUSED BY INADEQUATE NUTRITION

This handbook has been concerned almost exclusively with infectious diseases of marine species -- those diseases for which a specific pathogen may be identified. In addition to the toxicant effects just described, inadequate nutrition may also exert severe impact on cultured populations -- again in a variety of ways. Growth may be slow, survival may be poor, spawning may be poor or inhibited completely, and mortalities may ensue. Effects of nutritional deficiencies may be direct, or they may be expressed in increased disease susceptibility, followed by mortalities from infectious diseases -- often produced by facultative organisms which are not serious problems in unstressed populations.

Chemical definition of nutritional requirements of most mariculture species is at present unavailable. Adequacy of diets is often deduced from growth rates and survival of cultured animals, and much experimentation with diets is currently underway. Thus, opportunity for less-than-optimum food is great, and the consequences may be apparent in form of slow growth or poor survival, or they may be masked by outbreaks of infectious disease.

In essence, then, mariculture operations in considering the impact of disease must contend with a closely interlocking triumvirate of water quality, nutrition, and pathogens. An outbreak of infectious disease may often have its impetus in poor water quality or inadequate diet -- implying that mere treatment of the disease (assuming that a treatment exists) is not always enough, but that chemical imbalances in the environment or the food must also be corrected if long-term success is to be achieved.

Much nutritional information has been accumulated in fresh-water culture which has application to mariculture. Prepared pelleted diets developed for salmonid culture are being used and modified for marine species. The need for research in nutrition of fresh-water species was recognized by the establishment of Eastern and Western Fish Nutrition Laboratories by the Bureau of Sport Fisheries and Wildlife. Those laboratories have confronted problems of deficiency diseases such as that of thiamine and vitamin E, and with hepatomas associated with aflatoxin from storage fungi. Specific nutritional requirements have been determined for a few fresh-water species, and additional deficiency diseases recognized, but much remains to be done with marine species -- both fish and shellfish.

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CHEMOTHERAPY

Chemicals added to culture systems may serve two functions: (1)ethey may reduce or eliminate pathogens, and (2) they may reducee or control populations of heterotrophic microorganisms which may act as facultative pathogens of animals under stress.

Principal problems encountered with use of chemicals or antibiotics include the following:

(1)e They may have negative effects on biological filters ine controlled recirculated systems -- particularly one nitrifying bacteria;e

(2)e They may have negative effects on algal food, or on algaee present in fish larval rearing tanks;e

(3)e They may leave undesirable or harmful residues in culturede animals.e

A wide range of chemicals have been used to control fish diseases -- mostly in fresh water, but lately in salt water as well. The sulfas had earlier and continuing utility, with the various antibiotics being applied soon after. At present there is much interest in the nitrofurans -- a class of chemotherapeutics first developed by the Japanese against bacterial fish diseases. The development of drug resistance has been and continues to be a significant problem.

Chemotherapy and chemical prophylaxis have been listed as last resort methods in disease control (Wolf and Snieszko, 1964; Herman, 1970), with more favored methods being sanitation, development of resistant stocks, immunization, and environmental manipulation. This principle should be an important one in mariculture disease control as well.

One very important general point which must be kept clearly in mind when considering chemical methods of disease control is the extreme restriction on use of chemicals to treat animals being raised for food. The Eastern Fish Disease Laboratory (Leetown, West Virginia) in its excellent leaflet series on fish diseases includes the following standard statement, which should be heeded well by mariculturists:

> "If fish or shellfish are to be used for human or animal food, they can be treated with drugs or chemicals only in accordance with current laws and regulations. Federal agencies having such regulations are Food and Drug Administration (DHEW) and the Department of Agriculture. State and local agencies may also have regulations."

At present, for example, only salt, glacial acetic acid and sulfamerazine are approved by FDA for use on all food fish, while terramycin is restricted to use with trout, salmon, and catfish. This means that such common and useful substances as formalin, furanace, copper sulfate, acriflavin, and potassium permanganate may not be used legally in treatment of species destined for human consumption.

It should also be pointed out that some chemicals (such as malachite green) may be carcinogenic, or may cause other damage to humans who handle the compounds. Some potentially harmful chemicals may actually be used, even though they are not cleared, and such chemicals may have persistent residues in the harvested product destined for human consumption. Other chemicals may affect food chain organisms in the natural environment; their widespread use should be discouraged. Accelerated clearance of chemicals, or publication of definitive data on their harmful effects should thus have high priority.

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DISEASE PROBLEMS CREATED BY INTRODUCTION OF SPECIES FROM OTHER GEOGRAPHIC AREAS

The history and probable future development of mariculture in the United States has included and will undoubtedly continue to include introduction of species from other countries. Such introductions pose additional disease problems, especially when they take place in open system culture in bays and estuaries. Introduced species may be susceptible to endemic pathogens, parasites, commensals, and predators in new grow-out areas, and the native species may be susceptible to pathogens, parasites, commensals, and predators imported with the introduced species. Probably the best example of the realities of disease problems caused by introductions is that of the Japanese seed oysters brought to the United States west coast. A copepod parasite, Mytilicola, was introduced with the Japanese oysters, and affected stocks of native oysters (Ostrea lurida). Oyster drills were also introduced. A bacterial disease, resulting in "focal necrosis" of oyster tissues was also introduced, as far as can be determined. Disease problems in oysters on the coast of France may have been exacerbated in recent years by massive introductions of Crassostrea anguillata from other European countries and Crassostrea gigas from Japan. Mortalities--many of them unexplained but at least some probably caused by pathogens--have occurred and are occurring in native Ostrea edulis as well as in the introduced species.

Since transfers and introductions will certainly continue, a plan of action should be developed to limit risks of disease. Such a plan should include the following:

(1)e Before any attempt at introduction, a detailed diseasee study, concentrating on possible microbial diseases, should be carried out where the species is native. This must be more than a quick parasite survey.

(2)e The species to be introduced should be examined ine closed or controlled systems for an extended period -- up to a year -- to see if any unique disease problems emerge.

(3)e Brood stocks should be maintained in closed or controllede systems; larvae should be removed from any contact with brood stock; and only the offspring should be permitted to be placed in open waters. Brood stocks should then be developed from these offspring, and the original stocks destroyed. (It might be noted here that disease can become a major problem in maintenance of brood stock -- whether introduced or not -- and an important consideration should be preventing transmission to eggs or larvae).

(4)e Initial introductions of offspring of foreign species ine open waters should be small ones, to make manageable any problems which emerge -- but should be done in several areas, since environmental conditions may not be suitable in all areas.

At present, much international attention is directed toward limiting the spread of certain viral diseases of salmonids and carp by controlling transfers of eggs and fish. Diseases for which specific testing and certification are proposed to be required before export include Infectious Pancreatic Necrosis (IPN), Infectious Hematopoetic Necrosis (IHN), and Viral Hemorrhagic Septicemia (VHS) of salmonids and Spring Viremia (SV) of carp. These diseases are characteristic of fresh-water culture; no diseases of marine fish or shellfish are included in the proposal, which has been prepared by FAO, but international mechanism is being developed which could address any future problems created by transfer and introductions of marine species.

APPENDIX I. REFERENCES TO DISEASES IN MARICULTURE--BOOKS AND REVIEWS

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NOTE: Even though this handbook is confined to mariculture diseases in the United States, it is relevant to point out the leadership of Japan, both in culture of marine animals and in developing control methods for mariculture diseases. The Japanese have, for example, developed drugs such as Furanace, specific for aquatic diseases; they have developed a number of research groups actively concerned with aquaculture diseases; they have published a journal, "Fish Pathology" dealing with diseases in aquaculture; and they have published several books on aquaculture disease. Two recent books containing information on aquaculture diseases (fresh- and salt-water) are "Methods of Curing Fish Diseases", Japan Aquaculture Newspaper Co., Tokyo (1970), 206 pp., "Fish Diseases and Treatment", Midori Shobo Pub. Co., Tokyo (1970), 225 pp. Additionally, Egusa and Nakajima (1973) have published a bibliography of fish diseases in Japan (Fish Pathol. 7: 137-229).

APPENDIX II
RESEARCH GROUPS CONCERNED WITH DISEASES OF MARINE ANIMALS

Name of Group	Address	Approximate Size Including Support Staff	Unit Leader(s) or Principal Investigator(s)	Areas of Particular Interest or Competence
Private or Industry Supported				
Aquacultural Res. Corp.	Dennis, Mass.	12	E. J. Petrovits R. R. Seshadri F. S. Stevens R. J. Howes	Mariculture diseases, vertebrate and invertebrate
Ralston Purina Co.	St. Louis, Mo.	4	J.a Barkatea	Microbiological studies of shrimp
Shelter Island Oyster Co.	Greenport, N. Y.	3	P.a Chanleya	Hatchery diseases of bivalve mollusks
Private Universities				
The Johns Hopkins Univ., School of Hygiene and Public Health	Baltimore, Md.	3	F.a Banga	Microbiology, virology, immunology, and hematology of invertebrates
Lehigh University	Bethlehem, Pa.	10	T.a C. Chenga	Invertebrate pathology, nematode pathogens in fish
University of Miami Rosenstiel School of Marine Sciences	Miami, Fla.	1	E.a Iversena	Parasitology, microbiology, pollution effects, shrimp mariculture

Name of Group	Address	Approximate Size Including Support Staff	Unit Leader(s) or Principal Investigator(s)	Areas of Particular Interest or Competence
University of Miami School of Medicine	Coral Gables, Fla.	4	M. Sigele	Virology, tissue culture searches for antibiotic and anti-tumor substances in marine invertebrates
"	"	5	H. G. Haines	Diseases of marine turtles
College and University Consortium				
New York Ocean Science Laboratory	Montauk, N. Y.	2	C. R. Hickey, Jr.	Abnormalities in marine fish
State Universities				
University of California	Bodega Bay, Calif.	3	R.eShlesere E.eNilsone W.eFishere	Crustacean diseases
University of California at Davis, Dept. of Pathology	Davis, Calif.	2	S.eWellingse	Neoplasms in fish
Cornell University N. Y. State Veterinary College	Ithaca, N. Y.	3	J.eGillespiee L.eLeibovitze J.eTimoneye	Microbial diseases of shellfish
University of Delaware College of Marine Studies	Newark and Lewes, Del.	4	M.eR. Trippe	Molluscan diseases

Name of Group	Address	Approximate Size Including Support Staff	Unit Leader(s) or Principal Investigator(s)	Areas of Particular Interest or Competence
East Carolina University Dept. of Biology	Greenville, N.C.	1	C.oE. Blando	Fungus diseases of Crustacea
Louisiana State University Dept. of Microbiology	Baton Rouge, La.	2	R.oAmborskio	Diseases of aquatic animals
Louisiana State University Dept. of Zool. Physiology	Baton Rouge, La.	1	E.oWeidnero	Protozoan diseases
University of Maryland Dept. of Microbiology	College Park, Md.	17	R. R. Colwell	Effects of pollutants on bacterial invasiveness; bacterial flora of healthy and diseased fish; effect of human pathogens on marine animals; bacterial taxonomy
University of Maryland Chesapeake Biol. Lab.	Solomons, Md.	2	V.oSpragueo	Pathogenesis, distribution and taxonomy of marine protozoan
Northwestern State College Biological Science Dept.	Natchitoches, La.	2	D.oM. Kruseo	Parasitology of shrimps
Oregon State University Dept. of Microbiology	Corvallis and Newport, Ore.	13	J.oFryero	Vibriosis of salmonids; oral immunization of salmonids
Oregon State University Dept. General Science	Corvallis, Ore.	1	M.oMixo	Invertebrate pathology

Name of Group	Address	Approximate Size Including Support Staff	Unit Leader(s) or Principal Investigator(s)	Areas of Particular Interest or Competence
University of Rhode Island Lab. for Animal Pathology	Kingston, R. I.	3	R.e Wolkee	Histopathology, microbiology, vibriosis in flounders & salmon
San Diego State University	San Diego, Calif.	8	J.e Mathewsone H.e Schapiro F.e Steenbergere	Gaffkya infection of lobsters in aquaculture microbiology, pathology, immunology
Texas A&M University College of Veterinary Medicine	College Station, Tex.	10	R.e Nickelsone C.e Vanderzante	Microbial diseases of fish & shellfish, histopathology, parasitology, seafood microbiology
Texas A&M University Dept. Wildlife and Fisheries Science	College Station, Tex.	1	S. K. Johnson	Diseases of shrimps
Texas A&M University Dept. of Oceanography	Galveston, Tex.	2	S.e Ray J.e Mackine	Molluscan shellfish diseases

Name of Group	Address	Approximate Size Including Support Staff	Unit Leader(s) or Principal Investigator(s)	Areas of Particular Interest or Competence
Government Laboratories				
California Dept. Fish & Game Marine Resources Lab.	Menlo Park, Ca.	2	S.e Katkanskye	Invertebrate diseases
Florida Dept. of Natural Resources	St. Petersburg, Fla.	1	J.e Quicke	Marine shellfish diseases
Gulf Breeze Environmental Research Lab., EPA	Gulf Breeze, Fla.	1	J. A. Couch	Diseases of shrimps, particularly those related to environmental contaminants
Gulf Coast Research Laboratory	Ocean Springs, Miss.	12	D.e Cooke R.e Overstreete A. Lawlere	Parasitology & microbiology of shrimp and fish
Gulf Coastal Fisheries Center Galveston Laboratory, Natl. Marine Fisheries Service, NOAA (Commerce)	Galveston, Tex.	4	D.e Lightnere C.e Fontainee	Diagnostic studies on finfish and shellfish; wound repair and mycotic infections of shrimp
Halifax Laboratory, Fisheries Research Board of Canada	Halifax, N.S.	5	J.e Stewarte	Lobster diseases
Middle Atlantic Coastal Fisheries Center, Oxford Laboratory, Natl. Marine Fisheries Service, NOAA (Commerce)	Oxford, Md.	15	A. Rosenfielde	Comparative and experimental pathology of fish and shellfish

Name of Group	Address	Approximate Size Including Support Staff	Unit Leader(s) or Principal Investigator(s)	Areas of Particular Interest or Competence
New York Dept. of Envir. Conservation	Stony Brook, N.Y.	1	B. H. Young	Abnormalities in marine fish
Northwest Fisheries Center, Natl. Marine Fisheries Service, NOAA (Commerce)	Seattle, Wash. and Manchester, Wash.	6	A. Novotnyo H.oHodginso L.oHarrello W.oGronlundo	Diseases of salmon including vibriosis, kidney disease and furunculosis
Pacific Biological Station Fisheries Research Board of Canada	Nanaimo, B. C.	3	L.oMargoliso Z.oKabatao T.oEvelyno	Parasitology and microbiology of fish and crustacea
Virginia Institute of Marine Science	Gloucester Point, Va.	4	J.oAndrewso F.oPerkinso	Molluscan diseases -- protozoa and fungi in particular
Western Fish Diseases Lab., Bur. of Sport Fisheries and Wildlife (Interior)	Seattle, Wash.	12	R.oRuckero G.oWedemeyero	Diseases of salmonids; oral immunization against vibriosis