NOAA Technical Memorandum NMFS-F/NEC-88

Synopsis of Principal Diseases of the Blue Crab, *Callinectes sapidus*

U. S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
National Marine Fisheries Service
Northeast Region
Northeast Fisheries Science Center
Woods Hole, Massachusetts

January 1992

Recent issues in this series

53. **Contaminants in Hudson-Raritan Estuary Water and Influence of Cold Storage upon Its Chemical Composition**. By Anthony Calabrese, Lawrence J. Buckley, and J. Christopher Powell. May 1988. vii + 37 p., 10 figs., 11 tables. NTIS Access. No. PB88-225628/AS.

54. **Epizootic Ulcerative Syndromes in Coastal/Estuarine Fish.** By Carl J. Sindermann. June 1988. v + 37 p., 8 figs., 1 table. NTIS Access. No. PB89-110803/AS.

55. **A Plan for Study: Response of the Habitat and Biota of the Inner New York Bight to Abatement of Sewage Sludge Dumping**. By Environmental Processes Division, Northeast Fisheries Center. June 1988. iii + 34 p., 5 figs., 3 tables, 4 app. NTIS Access. No. PB89-100903/AS.

56. **Characterization of the Middle Atlantic Water Management Unit of the Northeast Regional Action Plan**. By Anthony L. Pacheco, ed. July 1988. v + 322 p., 136 figs., 21 tables. NTIS Access. No. PB89-145262/AS.

57. **An Analysis and Evaluation of Ichthyoplankton Survey Data from the Northeast Continental Shelf Ecosystem.** By Wallace G. Smith, ed. August 1988. xiii + 132 p., 53 figs., 12 tables, 1 app. NTIS Access. No. PB89-122501/AS.

58. **An Indexed Bibliography of Northeast Fisheries Center Publications and Reports for 1987.** By Jon A. Gibson. August 1988. iii + 20 p. NTIS Access. No. PB89-113013/AS.

59. **Surveys of Breeding Penguins and Other Seabirds in the South Shetland Islands, Antarctica, January-February 1987.** By W. David Shuford and Larry B. Spear. September 1988. vii + 27 p., 14 figs., 1 table. NTIS Access. No. PB89-141311/AS.

60. **Survey of Antarctic Fur Seals in the South Shetland Islands, Antarctica, during the 1986-1987 Austral Summer.** By John L. Bengtson, Lisa M. Ferm, Tero J. Harkonen, Everett G. Schaner, and Brent S. Stewart. September 1988. vii + 8 p., 1 fig., 3 tables. NTIS Access. No. PB89-141303/AS.

61. **Fish as Sentinels of Environmental Health.** By Robert A. Murchelano. September 1988. iii + 16 p., 4 figs. NTIS Access. No. PB89-139737/AS.

62. **The Effects of Density Dependent Population Mechanisms on Assessment Advice for the Northwest Atlantic Mackerel Stock.** By W. J. Overholtz, S.A. Murawski, W.L. Michaels, and L.M. Dery. October 1988. v + 49 p., 7 figs., 20 tables. NTIS Access. No. PB89-151948/AS.

63. **Status of the Fishery Resources Off the Northeastern United States for 1988.** By Conservation and Utilization Division. October 1988. iii + 135 p., 51 figs., 52 tables. NTIS Access. No. PB89-130819/AS.

64. **The Shell Disease Syndrome in Marine Crustaceans.** By Carl J. Sindermann. February 1989. v + 43 p., 5 figs., 2 tables. NTIS Access. No. PB89-162523/AS.

65. **Stock Assessment Information for Pollock, *Pollachius virens* (L.), in the Scotian Shelf, Georges Bank, and Gulf of Maine Regions.** By Ralph K. Mayo, Stephen H. Clark, and M. Christina Annand. April 1989. vi + 14 p., 6 figs., 14 tables. NTIS Access. No. PB90-120676/AS.

66. **Guidelines for Estimating Lengths at Age for 18 Northwest Atlantic Finfish and Shellfish Species.** By Judith A. Pentilla, Gary A. Nelson, and John M. Burnett, III. May 1989. iii + 39 p., 18 figs., 19 tables. NTIS Access. No., PB90-120675/AS.

67. **Response of the Habitat and Biota of the Inner New York Bight to Abatement of Sewage Sludge Dumping. Second Annual Progress Report -- 1988**. By Environmental Processes Division, Northeast Fisheries Center. July 1989. vii + 47 p., 39 figs., 11 tables, 3 app. NTIS Access. No. PB90-125444/AS.

(continued on inside back cover)

NOAA Technical Memorandum NMFS-F/NEC-88

This TM series is used for documentation and timely communication of preliminary results, interim reports, or special purpose information, and has not undergone external scientific review.

Synopsis of Principal Diseases of the Blue Crab, *Callinectes sapidus*

Gretchen A. Messick and Carl J. Sindermann

Oxford Lab., National Marine Fisheries Serv., Oxford, MD 21654

U. S. DEPARTMENT OF COMMERCE
Rockwell A. Schnabel, Acting Secretary
National Oceanic and Atmospheric Administration
John A. Knauss, Administrator
National Marine Fisheries Service
William W. Fox, Jr., Assistant Administrator for Fisheries
Northeast Region
Northeast Fisheries Science Center
Woods Hole, Massachusetts

January 1992

1.a

NOTE ON SPECIES NAMES

The Northeast Fisheries Science Center's policy on the use of species names in technical publications and reports is to follow the American Fisheries Society's (AFS) lists of common and scientific names for fishes (Robins *et al.* 1991)[a], mollusks (Turgeon *et al.* 1988)[b], and decapod crustaceans (Williams *et al.* 1989)[c]. This policy applies to all issues of the *NOAA Technical Memorandum NMFS-F/NEC* series.

---

[a] Robins, C.R. (chair), R.M. Bailey, C.E. Bond, J.R. Brooker, E.A. Lachner, R.N. Lea, and W.B. Scott. 1991. Common and scientific names of fishes from the United States and Canada, 5th ed. *Amer. Fish. Soc. Spec. Publ.* 20. 183 pp.

[b] Turgeon, D.D. (chair), A.E. Bogan, E.V. Coan, W.K. Emerson, W.G. Lyons, W.L. Pratt, C.F.E. Roper, A. Scheltema, F.G. Thompson, and J.D. Williams. 1988. Common and scientific names of aquatic invertebrates from the United States and Canada: mollusks. *Amer. Fish. Soc. Spec. Publ.* 16. 277 pp.

[c] Williams, A.B. (chair), L.G. Abele, D.L. Felder, H.H. Hobbs, Jr., R.B. Manning, P.A. McLaughlin, and I. Pérez Farfante. 1989. Common and scientific names of aquatic invertebrates from the United States and Canada: decapod crustaceans. *Amer. Fish. Soc. Spec. Publ.* 17. 77 pp.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Blue crabs, *Callinectes sapidus*, are an important economic and recreational resource on the Atlantic and Gulf coasts. Accordingly, a number of pathological agents which may decrease the availability or demand for blue crabs have been studied. Investigations have emphasized diseases and parasites that may either cause mortalities or reduce market value of blue crabs. Diseases and parasites are known to cause mortalities in both wild and captive crabs. Some of the most harmful pathogens infecting wild populations of blue crabs are several microsporidans, including *Ameson michaelis*; an amoeba, *Paramoeba perniciosa*; and a dinoflagellate, *Hematodinium perezi*. Crabs held in captivity are subject to systemic bacterial infections, including *Vibrio parahaemolyticus*, and to various viral infections, including a reo-like virus, a picorna-like virus, and a bi-facies virus. Other pathogens may decrease fecundity, including the fungus *Lagenidium callinectes*, the nemertean *Carcinonemertes carcinophila*, and the parasitic barnacle *Loxothylacus texanus*. Finally, still other diseases can reduce marketability of infected crabs. Chitinoclastic bacteria including species of *Vibrio* and *Pseudomonas* produce unattractive necrotic lesions on the exoskeleton of crabs; and a trematode, *Microphallus basodactylophallus*, hyperparasitized by the haplosporidan *Urosporidium crescens*, produces undesirable black pigmented spots in the musculature of cooked crabs.

INTRODUCTION

The blue crab, *Callinectes sapidus*, is the most valuable commercial and recreational species of crab along the Atlantic and Gulf coasts. The significance of this fishery is demonstrated by the many papers and books written on the subject. As early as 1905, the life history of the blue crab was described (Hay 1905). Since then, many experimental findings and generalized data have been reported; a few of the comprehensive reports available deal with general anatomy (Pyle and Cronin 1950), form and growth (Newcombe *et al.* 1949), and histology of the blue crab (Johnson 1980). An extensive synopsis of biological data has been published (Millikin and Williams 1984) which includes most of the pertinent references to blue crab biology.

Although the blue crab has consistently been a major fishery resource in both the hard-shell and soft-shell stages, the fishery has experienced major fluctuations in landings. Outbreaks of disease combined with environmental factors have been blamed for some of the variations in crab production. Diseases have been established as the cause of mortalities, both in wild populations and in crabs contained in holding facilities (Sprague 1965; Overstreet 1977; Johnson 1983).

This synopsis provides a summary of diseases which may affect the blue crab industry by either causing mortalities, decreasing fecundity, or decreasing demand. Included are photographs, when available, for better visualization and identification of the various diseases and parasites infecting blue crabs.

Many microbial diseases have been reported from blue crabs, including those of viral, bacterial, fungal, and protozoan etiologies. A few helminths and a parasitic crustacean have also been implicated as causing either disease or reduced fecundity. The following organisms have been reported to cause mortalities or significant pathology in blue crabs. Information about each of these pathogens is summarized in Table 1, and is discussed briefly in the sections that follow.

Viruses

- Reo-like virus (RLV) + rhabdo-like virus A (RhVA)
- Chesapeake Bay virus (CBV), a picorna-like virus
- Bi-facies virus (BFV), formerly herpes-like virus (HLV)

Bacteria

- Chitinoclastic bacteria, including *Vibrio* and *Pseudomonas*
- Systemic bacteria, including *V. parahaemolyticus, Pseudomonas, Acinetobacter, Bacillus, Flavobacterium,* and a heterogenous group of coliforms, including *Escherichia coli*

Fungus

- *Lagenidium callinectes* (phycomycetous)

Protozoans

- *Ameson michaelis* (Microspora)
- *Paramoeba perniciosa* (Sarcomastigophora)
- *Hematodinium perezi* (Sarcomastigophora)
- Haplosporidan (Ascetospora)
- *Lagenophrys callinectes* (Ciliophora)

Helminths

- *Microphallus* spp. (trematode metacercariae) hyperparasitized by *Urosporidium crescens* (haplosporidan)

Table 1. Principal pathogens of blue crabs

Pathogen	Histopathology and Tissues Infected	Effect on Host	Gross Signs of Disease	Geographic Location and Prevalence	Reference
Reo-like virus (RLV); infects RNA	Rhabdolike virus A (RhVA) always found with RLV; causes cytoplasmic inclusions, increased cytoplasmic volume in hemocytes, hemopoietic tissue, and glial nerves	RLV and RhVA act synergistically, causing necrosis of hemopoietic tissue, hemocytes, and CNS; death due to nerve and hemocytic dysfunction	Sluggishness, paralysis; withdrawn blood clots incompletely	Chincoteague and Chesapeake Bays; infects crabs from high and low salinities; actual prevalence unknown	Johnson 1977a, c, 1983, 1984, 1986
Chesapeake Bay virus (CBV); infects RNA; a picorna-like virus	Causes focal infections; cytoplasmic inclusions in epithelium of gill, gut, bladder, CNS cells, and epidermis	Extensive destruction of gill and bladder epithelium and neurosecretory cells, blindness, and death	Abnormal behavior, erratic swimming, blindness	Chesapeake Bay; infects captive juveniles and probably wild populations	Johnson 1978a, b 1983, 1984, 1986
Bi-facies virus (BFV); infects DNA; formerly herpes-like virus (HLV)	Hypertrophy of nuclei of hemocytes; infected cells have refractive cytoplasmic inclusions	Nuclear hypertrophy followed by lysis of cell; death due to hemocytic dysfunction	Crabs become inactive; withdrawn blood is milky and clots improperly	Chincoteague and Assawoman Bays; infects captive and wild populations	Johnson 1976b, 1978b, 1983, 1984, 1988
Chitinoclastic bacteria, principally *Vibrio* and *Pseudomonas*, plus others	Erosion of exoskeleton, especially chelipeds and carapace overlying gills	Decreased salability; secondary infections may cause internal necrosis of tissue and death	Exoskeleton blackened and pitted; condition called "shell disease" or "box burnt disease"	Ubiquitous, particularly apparent when crabs have been injured or held in prolonged captivity	Rosen 1967, 1970; Cook & Lofton 1973; Johnson 1983; Engel & Noga 1989; Sindermann 1989
Vibrio parahaemolyticus and related facultative bacteria	Hemolymph contains bacteria; causes hemocytic aggregations in gills, heart, and other tissues	Formation of hemocytic aggregations and nodules; causes internal clotting of hemolymph	Lethargy and weakness due to systemic infections; withdrawn blood clots incompletely	Injured or stressed crabs prone to disease; mortalities reported 50% or higher in shedding tanks	Krantz *et al.* 1969; Sizemore *et al.* 1975; Tubiash *et al.* 1975; Johnson 1976c; Blake *et al.* 1980a, b; Messick & Kennedy 1990

Table 1. Continued

Pathogen	Histopathology and Tissues Infected	Effect on Host	Gross Signs of Disease	Geographic Location and Prevalence	Reference
Lagenidium callinectes (fungus)	Phycomycete zoospores settle on eggs, germinate, and extend germ tubes which develop into branched septate mycelia	Eggs fail to hatch or abnormal larvae are produced; may also kill newly hatched larvae within 48 hr	Diseased portions of egg mass appear either brownish or grey, depending on maturity of infected egg mass	Chesapeake Bay; east and Gulf coasts; 5-30% prevalence	Couch 1942; Sandoz *et al.* 1944; Newcombe & Rogers 1947; Rogers-Talbert 1948; Bland & Amerson 1973, 1974
Ameson michaelis (protozoan, microsporida	Micro-sporidans within blood cells and muscle tissue	Muscle lysis; parasite destroys and replaces musculature due to biochemical imbalance	Lethargy; muscle has chalky opaque appearance; abdomen may have white or grey color	Delaware and Chesapeake Bays southward to Gulf of Mexico	Sprague 1950, 1965, 1970, 1977; Over-street & Whatley 1975; Overstreet 1977, 1978; Couch 1983
Paramoeba perniciosa (protozoan, amoeba, Sarcomas-tigophora)	Organisms with well defined nucleus plus a secondary body in tissues; large halos may surround individual amoeba	Amoeba fills tissues, replaces hemocytes, and alters hemolymph; probably causes winter mortalities	Grey-colored abdomen and appendages; called "grey crab" disease	From Sandy Hook Bay to Chincoteague Bay; usually seen in higher salinity waters	Sprague & Beckett 1966, 1968; Sawyer 1969; Sprague *et al.* 1969; Newman & Ward 1973; Pauly *et al.* 1975; Johnson 1977b; Couch 1983
Hematodinium perezi (protozoan, parasitic dino-flagellate, Sarcomas-tigophora)	Uninucleate and binucleate plasmodial parasites in hemolymph; chromosomes condensed or diffused with no nuclear membrane	Debilitating and lethal due to ability to proliferate and replace host tissues; laboratory-infected crabs die	Lethargy and weakness; withdrawn blood milky or opaque in heavy infections, slow to clot, and contains few hemocytes	Maryland to Gulf of Mexico; found in waters with salinities greater than 11 ppt	Chatton & Poisson 1931; Newman & Johnson 1975; Newman 1977; Couch 1983
Haplosporidan (protozoan, ascetosporan)	Uninucleate and plasmodial stages of haplosporidan parasites in tissues	Causes hemocytic dysfunction	Sluggishness; hemolymph opaque white and of low viscosity	Reported in two crabs from Chinco-teague Bay, Virginia, and coastal North Carolina	Newman *et al.* 1976

Table 1. Continued

Pathogen	Histopathology and Tissues Infected	Effect on Host	Gross Signs of Disease	Geographic Location and Prevalence	Reference
Lagenophrys callinectes (protozoan, ciliate)	Ectocommensal living in lorica found on flat surfaces of gill lamellae	Secretes a protective lorica on gill lamellae; may interfere with respiratory and excretory function of gills	No gross signs; diagnosis through microscope for presence of lorica	Chincoteague and Chesapeake Bays; Gulf of Mexico; peak prevalence during summer months	Couch 1966, 1967, 1983
Microphallus basodactylophallus (digenean, fluke, hyperparasitized by *Urosporidium crescens*, haplosporidan)	Hyperparasitized *Microphallus* cysts present in thoracic muscles, hepatopancreas, and ventral ganglion	Pigmented spores debilitate and enlarge worm cyst; actual effect on crabs slight; causes reduced market value of crabs	Crab tissues contain large brownish-black metacercaria called "pepper spot" or "buckshot"	Chesapeake Bay to Texas	DeTurk 1940; Couch 1974b; Overstreet 1978, 1982
Carcinonemertes carcinophila (nemertean)	Externally infests gills and egg mass	Feeds on host's egg mass; causes reduction in reproductive potential; cements gill lamellae together	Destruction of egg mass; worms seen grossly on gills between lamellae	Gulf coast; infects crabs from high salinity habitats	Humes 1942; Hopkins 1947; Pyle & Cronin 1950; Overstreet 1978, 1982
Myzobdella lugubris (annelid, leech)	Symbiont; attaches to external carapace	Unknown; associated in some reports with mortalities, but no lesions observed	Presence of cocoons, usually near posterior margin of carapace	Atlantic and Gulf coasts; low-salinity habitats; male crab usual host	Moore 1946; Hutton & Sogandares-Bernal 1959; Sawyer *et al.* 1975; Overstreet 1978
Loxothylacus texanus (cirriped, parasitic barnacle, rhizocephalan)	Internal parasite sends root-like system throughout host's muscle; develops an external sac which serves as brood sac for nauplii larvae	Inhibits crab growth, terminates reproduction, removes individuals from fishery; may reduce up to 50% of commercial stocks in some areas	Parasite's externa protrudes under crab's apron; male crabs acquire secondary adult female sexual qualities	Gulf of Mexico; more prevalent in higher salinity waters	Reinhard 1950, 1956; Adkins 1972; Ragan & Matherne 1974; Overstreet 1978

- *Carcinonemertes carcinophila* (nemertean)
- *Myzobdella lugubris* (leech)

Parasitic Crustacean

- *Loxothylacus texanus* (rhizocephalan)

VIRAL DISEASES

Before 1966, when a virus was first reported from a European shore crab (Vago 1966), viruses from marine invertebrates were unknown. Since the 1960s, with increased availability of transmission electron microscopy, other mortality-associated viruses have been discovered in crustaceans (Johnson and Bodammer 1975; Couch 1981; Johnson 1984). Today there are seven, possibly eight, viruses reported infecting blue crabs, although not all are pathogenic or cause mortalities. Viral effects on the host may be lethal, the result of synergism with other viruses, relatively benign, or even unknown (Johnson 1978b). Of the viruses known to infect blue crabs (Table 2), three are relatively benign: baculoviruses A and B, and rhabdo-like virus B (RhVB). Two are found to be associated with other viruses and perhaps cause a synergistic effect: paramyxo-like enveloped helical virus (EHV) and RhVA. Three others are considered lethal: RLV, BFV, and CBV (Figure 1). The RhVA apparently acts synergistically with the RLV and is responsible for hemocyte and nerve tissue damage which may result in death, especially in premolt crabs about to go through ecdysis (molt), since this process is controlled by the nervous system (Johnson 1986). Multiple viral infections are frequently discovered within the same crab. Viral infections may become patent only when crabs have been exposed to stress factors such as captivity, crowding, or a degraded environment (Couch 1974a; Johnson 1977a, 1978b, 1984, 1986; Yudin and Clark 1979; Messick and Kennedy 1990).

BACTERIAL DISEASES

SHELL DISEASE

The disease syndrome known variously as shell disease, rust disease, black spot, or burnt spot was first discovered in crustaceans in 1900 in Germany by Happich (Rosen 1970), and was first reported in the Chesapeake Bay in 1967 (Rosen 1967). The syndrome, which is manifested as various kinds of exoskeletal erosions and lesions, is caused by chitinoclastic bacteria (and frequently other chitin-destroying microorganisms). Since there are numerous species of chitinoclastic bacteria found in marine environments (Benton 1935; Zobell and Rittenberg 1938; Hock 1940, 1941; Lear 1963; Hood and Meyers 1974), shell disease is one of the most common and widespread bacterial diseases of crustaceans. Chitinoclastic bacteria play an important role in the breakdown and recycling of chitin in exoskeletons of dead animals and chitinous casts from developmental stages of crustaceans in the sea (Hock 1940). Although beneficial in recycling chitin, these bacteria can be detrimental to live crustaceans (Sindermann 1989). The bacteria can cause deep, necrotic, blackened lesions on various portions of the crab's exoskeleton, thus decreasing its market value, making it unmarketable, or even allowing secondary infections to invade tissues and kill crabs.

Shell disease in blue crabs occurs naturally in all populations and has been reported from such varied geographic locations as the Louisiana and Mississippi coasts, Biscayne Bay (Florida), the South Carolina coast, Pamlico River, and the Chesapeake Bay. A large portion of blue crabs reported with shell disease has been subjected to abnormal environmental conditions (Sindermann 1989), either in the wild by being exposed to deteriorated water quality, as was thought to be the case in coastal North Carolina (Engel and Noga 1989; McKenna *et al.* 1990), or by being held in crowded conditions such as shedding or holding tanks for extended periods (Figures 2 and 3).

Most chitinoclastic bacteria isolated from lesions in blue crabs belong to several genera including *Vibrio* and *Pseudomonas* (Cook and Lofton 1973; Johnson 1983). For a detailed report on shell disease, see Sindermann (1989).

SYSTEMIC BACTERIAL INFECTIONS

Many blue crab mortalities are attributed to systemic bacterial infections, especially when the animals are subjected to crowded, confined conditions. Mortalities of 50 percent or higher have been reported in crabs held in commercial shedding facilities (Krantz *et al.* 1969). Gross signs of infection include lethargy, weakness, and possibly an enlarged chalky white area on a fifth pereiopod or the gills. Upon dissection, a premortem plasma clot can often be seen in anterodorsal and frontal blood sinuses. Hemolymph from infected crabs may have reduced clotting abilities and diminution of hemocyte numbers. The formation of hemocyte aggregations can be seen histologically in arteries and hemal spaces of various tissues, including the heart; as the infection progresses, nodules can be seen in gills, heart, antennal gland, and other organs (Johnson 1976c) (Figure 4). Apparently healthy crabs may exhibit a hemocytic response seen histologically which may be the result of recovery from a previous light bacterial infection (Messick and Kennedy 1990). Various species of bacteria have been isolated from the hemolymph of moribund and apparently healthy crabs, including *Vibrio parahaemolyticus*, a pathogen to both crabs and humans (Krantz *et al.* 1969; Davis and Sizemore 1982; Sizemore and Davis 1985), also *Pseudomonas, Acinetobacter, Bacillus, Flavobacterium,* and a

Table 2. Viral diseases of blue crabs

Virus Name and Type	Histopathology and Tissues Infected	Effect on Host	Gross Signs of Disease	Geographic Location and Prevalence	Reference
Bi-facies (BFV); formerly herpes-like (HLV); fatal; DNA virus; related to Irido-, Pox-, and Baculoviridae	Nuclei of hemocytes hypertrophied; refractile cytoplasmic inclusions	Nuclear hypertrophy followed by cell lysis; death due to hemocytic dysfunction	Just before death crabs become inactive; withdrawn blood milky and clots improperly	Assawoman and Chincoteague Bays only; captive and wild crabs; reported infections up to 13%	Johnson 1976b, 1978b, 1983, 1984, 1988
Baculo-A; benign; DNA virus (non-occluded)	Nuclei of epithelial cells of hepatopancreas hypertrophied; focal infections	Benign, since hepatopancreatic cells constantly replaced	Crabs appear healthy	Widespread along Atlantic coast; infections from 4 to 20% in adults and juveniles	Johnson 1976a, 1983, 1984; Johnson & Lightner 1988
Baculo-B; DNA virus; similar to baculovirus of *Carcinus maenas*	Nuclear hypertrophy of hemopoietic tissue and hemocytes	Lysing and dysfunction of hemocytes, otherwise effect unknown	Occurs in either normal-appearing crabs or in those with other viral infections	Tred Avon River; Chesapeake Bay	Bazin *et al.* 1974; Johnson 1983, 1984, 1986
Reo-like (RLV); fatal; RNA virus; similar to paralysis virus of *Macropipus depurator*	Cytoplasmic inclusions; increased cytoplasmic volume in hemocytes, hemopoietic tissue, and glial nerves	Rhabdo-like A (RhVA) always associated with RLV; synergistically causes paralysis and death due to nerve and hemocyte dysfunction	Slowness, paralysis; withdrawn blood does not clot completely	Actual prevalence unknown; infects crabs from various salinities from Chesapeake and Chincoteague Bays	Vago 1966; Johnson 1977a, c, 1983, 1984, 1986
Rhabdo-like A (RhVA); synergistic; RNA virus	Always seen with other viruses; infects cytoplasm of nerve ganglia, hemocytes, hemopoietic tissue	Stress related; may have synergistic effect with other virus diseases	No reported gross signs	Atlantic and Gulf coasts; may be ubiquitous	Jahromi 1977; Yudin & Clark 1978, 1979; Johnson 1978a, b, 1983, 1984, 1986

Table 2. Continued

Virus Name and Type	Histopathology and Tissues Infected	Effect on Host	Gross Signs of Disease	Geographic Location and Prevalence	Reference
Enveloped helical (EHV); RNA virus; paramyxo-like virus	Infects cytoplasm of hemocytes and hemopoietic tissue	Effect unreported; always associated with other viruses which synergisti-cally cause pathology	No reported gross signs	Chesapeake and Chinco-teague Bays; east coast of Florida; prevalence low	Johnson & Farley 1980; Johnson 1983, 1984, 1986
Rhabdo-like B (RhVB); RNA virus; formerly labeled EGV-1	Associated extra-cellularly with basal lamina of mandibular gland	"Infected" glands normal and crabs showed no sign of abnormal behavior; very similar to EHV, prob-ably same	No reported gross signs	Found only in 1 of 60 (3%) confined crabs from Galveston, Texas	Yudin & Clark 1979; Johnson 1983
Chesapeake Bay (CBV); fatal; RNA virus; picorna-like virus	Cytoplasm of epithelium of bladder, epidermis, gill, gut, and nerve cells filled with viral material; infections are usually focal	Causes destruction of epithelium of bladder, gill, and neuro-secretory cells of CNS; may produce blindness	Abnormal behavior and blindness	Juvenile crabs from Tangier Sound, probably other areas also	Johnson 1978a, b 1983, 1984, 1986

heterogenous group of coliforms including *Escherichia coli* (Colwell *et al.* 1975; Sizemore *et al.* 1975; Welsh and Sizemore 1985)[a].

Bacterial infections appear to be acquired during capture and transport, suggesting that potentially pathogenic bacteria in the water or on the exoskeleton may be introduced into tissues by wounding or other means under stressful conditions. Physical factors contributing to stress include the process of capture and handling, crowding in traps and/or holding tanks, transport out of water, exposure to elevated temperatures, and wounding by fellow captives (Johnson 1976c).

Crabs may often contain low bacterial levels in their blood due to exposure to bacteria during ecdysis, as well as their day-to-day contact with ubiquitous bacteria in the marine environment; but, their defense response would probably remove these bacteria, sometimes leaving histologic signs of the previous infections in the tissues (Messick and Kennedy 1990).

FUNGAL DISEASES

In the early 1940s, there were marked annual fluctuations in commercial catches of blue crabs noted in the Chesapeake Bay. In the same period, a parasitic fungus was discovered on egg masses of many blue crabs taken from natural spawning grounds in the lower Chesapeake Bay (Sandoz *et al.* 1944; Rogers-Talbert 1948). Samples from infected animals were examined and a new phycomycetous fungus species, *Lagenidium callinectes* Couch, was described (Couch 1942). It was believed this fungus parasite could limit the production of crab larvae and result in yearly fluctuations in crab populations. This same species of fungus was later found infecting crabs from coastal North Carolina (Bland and Amerson 1973, 1974).

Infection takes place when fungal zoospores germinate on crab egg surfaces, sending in a germ tube which develops into branched, septate mycelium. *Lagenidium callinectes* zoospores are apparently incapable of germinating without

[a] *Vibrio cholerae* and other vibrios, including *V. parahaemolyticus* and *V. vulnificus*, can cause human disease when humans ingest improperly cooked (Blake *et al.* 1980a,b) or handled crabs (Anonymous 1972).

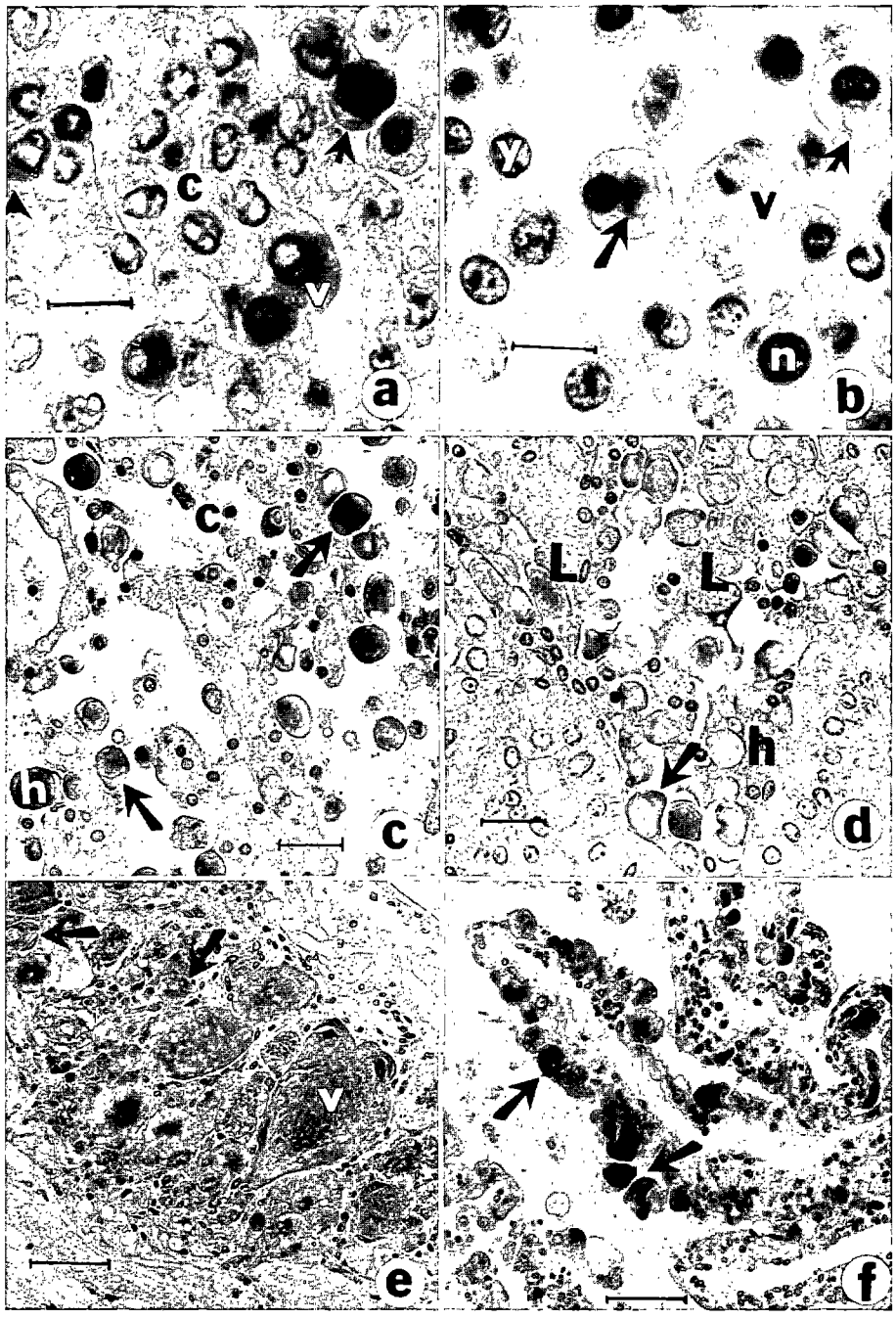


Figure 1. Viral infections causing mortalities in blue crabs.

a. Reo-like - rhabdo-like virus A (RLV-RhVA) combination in hemopoietic tissue of intermolt crab. Note increased cytoplasmic volume (v), inclusions (arrows), and normal cells (c). Bar = 10 µm.

b. RLV-RhVA infection in young hemocytes released from hemopoietic tissue. Note increased cytoplasmic volume (v), viral inclusions (arrows), nuclei of various sizes (n), and normal young hemocytes (y). Bar = 10 µm.

c. Bi-facies virus (BFV) infecting hemocytes. Note hypertrophy of nuclei (h), refractive cytoplasmic inclusions (arrows), and normal hemocytes (c). Bar = 20 µm.

d. BFV infecting hemocytes in hemal space of antennal gland. Note hypertrophy of nuclei (h), cytoplasmic inclusions (arrow), and cells about to lyse (L). Bar = 20 µm.

e. Chesapeake Bay virus (CBV) infecting thoracic ganglion of central nervous system. Note extensive destruction of neurosecretory cells (v), and normal neurosecretory cells (arrows). Bar = 50 µm.

f. CBV infecting epithelial cells of bladder. Note cytoplasmic inclusions in infected cells (arrows). Bar = 50 µm.

contact with crab eggs (Couch 1942). Infected eggs are usually below their normal size and either fail to hatch or prezoea are produced which are abnormal and rarely survive (Sandoz *et al.* 1944). Infected eggs have an atypical brown or grey color, depending on the maturity of the eggs infected. Heaviest infections occur on sponge crabs that are exposed to the fungus for the longest time, regardless of the developmental stage of the eggs (Newcombe and Rogers 1947). Infection spreads rapidly over the periphery of the egg mass, but penetration into the sponge is slow and rarely over 3-mm deep. Peripheral infections do not retard the development of eggs in the interior of the sponge; therefore, less than 25 percent of the eggs of heavily diseased sponges are infected. It is not unusual to find 80-90 percent of the crabs in a sample population to have some degree of infection. The fungus has a broad tolerance for salinity change and, experimentally, can develop in salinities from 5 to 30 ppt. *Lagenidium* can also infect larvae of blue crabs, causing extensive mortalities. Under laboratory conditions, transmission of the fungus is extremely rapid, with infections apparent within two days (Rogers-Talbert 1948).

Lagenidium callinectes has been transmitted experimentally to eggs of the oyster pea crab, *Pinnotheres ostreum*, and the mud crab, *Dyspanopeus (Neopanope) texana*, which inhabit the same areas as the blue crab (Rogers-Talbert 1948). Other strains of *Lagenidium* have been isolated in larvae from cultured shrimp in Florida, Texas, Mexico, Tahiti, and Honduras; in Dungeness crab, *Cancer magister*, larvae from Oregon; and American lobster, *Homarus americanus*, larvae from California. The fungus is considered so potentially pathogenic that attempts have been made to devise chemical control methods (Bland *et al.* 1976).

Another phycomycetous fungus, *Haliphthoros milfordensis*, was isolated initially from ova of the Atlantic oyster drill, *Urosalpinx cinerea*, but also infects penaeid shrimp, lobsters (Fisher *et al.* 1975, 1976, 1978), and, experimentally, blue crab ova. Infected ova of the blue crab can be identified by a color change; normally brown, mature ova appear dark orange-yellow and are filled with multibranched hyphae. During sporulation, long unbranched, extramatrical discharge tubes grow through the surface of the ova to the external medium. Zoospores from one infected egg are capable of infecting 20-30 other eggs in the same sponge within 24 hours. *Haliphthoros milfordensis* is morphologically similar to *L. callinectes* (Tharp and Bland 1977).

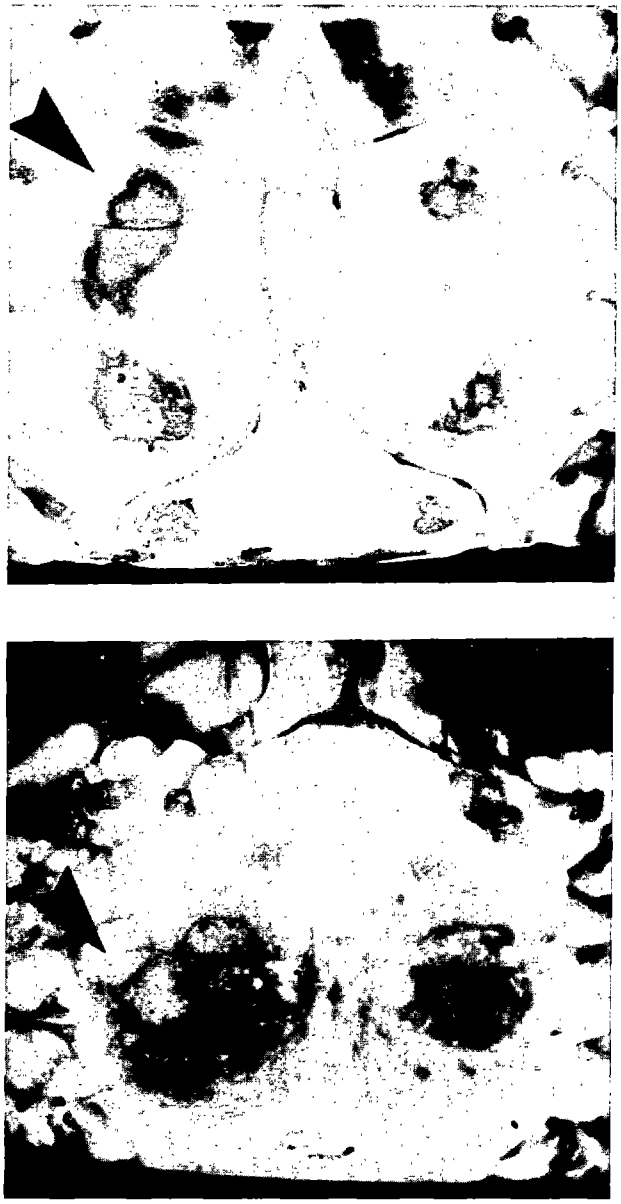


Figure 2. Shell disease in the blue crab -- early lesions (above) and advanced lesions (below). (Photographs courtesy of Dr. R.M. Overstreet, Gulf Coast Research Laboratory, Ocean Springs, Mississippi.)

PROTOZOAN DISEASES

There are many protozoans known to cause diseases in blue crabs; some occur in epizootic proportions which result in mass mortalities, but often these diseases are reported as being enzootic, killing only an occasional individual. Diseases caused by protozoans often become patent only when crabs are held in artificial environments, such as peeler shedding tanks or holding facilities for market. There are several reviews of protozoan pathogens, including those infecting blue crabs (Sindermann and Rosenfield 1967; Sindermann 1990; Sprague 1970; Sprague and Couch 1971; Overstreet 1978; Couch and Martin 1982; Couch 1983; Millikin and Williams 1984; Sparks 1985). Some of the most pathogenic protozoans found infecting blue crabs are microsporidans, including *Ameson*, *Nosema*, and *Pleistophora*; two members of the Sarcomastigophora, *Paramoeba* and *Hematodinium*; a haplosporidan-like ascetosporan; and a ciliophoran, *Lagenophrys*.

At least five different species of microsporida infect blue crabs; one that is widespread and causes considerable harm to the host is *A. michaelis* (Overstreet 1978). This

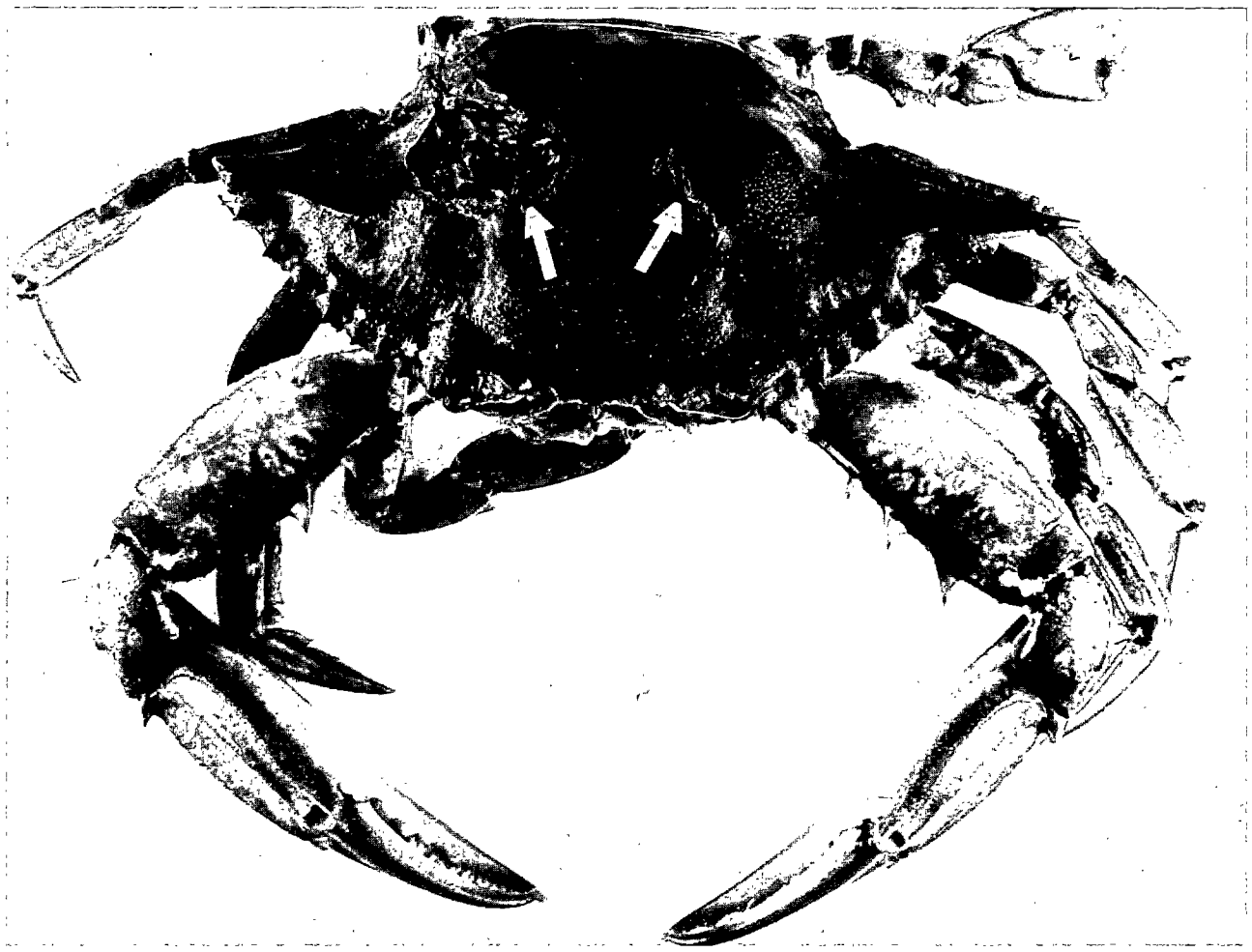


Figure 3. Blue crab from the Pamlico River, North Carolina, with extensive carapace lesions (arrows). (Photograph courtesy of Dr. D. Engel.)

microsporidan was first reported and classified as a species of *Nosema* (Sprague 1950, 1965), but was later placed in the genus *Ameson* (Sprague 1977). The disease-inducing spores are ovoid when live and measure 2.2 µm by 1.7 µm (Sprague 1965). Heavily infected crabs are called "sick crabs," since they are sluggish and have an abnormal appearance; they are often seen in shallow water and, in heavy infections, can be identified by an opaque white-to-grey ventral sternum (Sprague 1970; Overstreet 1977). Infected animals die rapidly if stressed (Overstreet and Whatley 1975). This parasite is considered highly host specific since only *Callinectes sapidus* is infected (Overstreet 1978).

Under the right conditions, spores infect host tissue by everting a long internal tube or polar filament. The single-celled spore squeezes through this extended pliable filament and invades the host's blood cells; the spore subsequently undergoes vegetative growth and produces cells which in turn produce strings of eight individual spores along muscle tissue. In heavy infections, enormous numbers of these resistant spores lyse and destroy adjacent muscle tissue and replace the normal musculature (Overstreet 1978) (Figure 5). *Ameson michaelis* may be more pathogenic than other microsporidans because it lacks pansporoblast membranes, allowing it direct contact with crab tissue. These membranes normally prevent harmful substances such as enzymes from being released to solubilize host tissue which is consequently used for nutrition by the parasite (Couch 1983). This parasite is potentially very pathogenic, especially when considering the loss of economic potential of reared crabs for the soft-shell industry. Some measures have been taken to find chemical deterrents to help reduce the spread of the parasite under controlled conditions (Overstreet and Whatley 1975).

Two other microsporida, *A. sapidus* and *Pleistophora cargoi*, can also be found parasitizing blue crabs. *Ameson sapidus* can induce extensive destruction of muscle tissue, leading to white and opaque muscles. Spores are oval and larger than *A. michaelis* at 3.5 µm by 2.13 µm (Sprague 1970; Couch and Martin 1982; Sparks 1985). Another species of microsporidan, *P. cargoi*, is characterized by its development into 16 spores; when fresh, these are ellipsoidal and measure 5.1 µm by 3.3 µm. These spores have been found in skeletal (Figure 5) and cardiac muscle of blue crabs; little is known of the effect of *P. cargoi* on blue crab

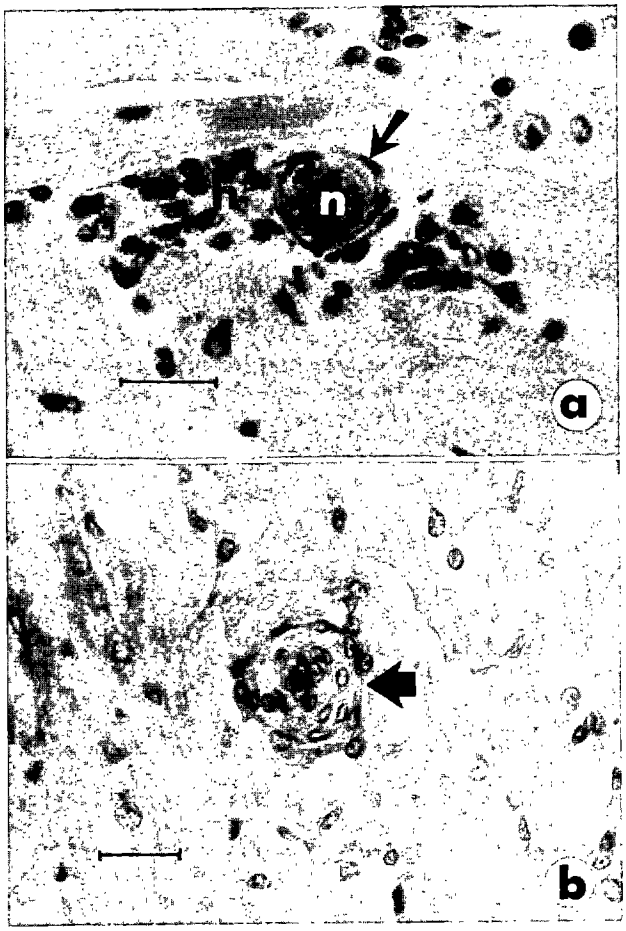


Figure 4. Bacterial infections in blue crab tissue.
a. Hemocyte aggregation (h) and nodule formation (n) in heart tissue. Note pyknotic nuclei in center of nodule and layer of flattened hyaline hemocytes encapsulating nodule (arrow). Bar = 20 μm.
b. Nodule formation in heart tissue (arrow). Bar = 20 μm.

populations (Sprague 1966, 1970; Couch and Martin 1982).

Besides the Microspora, there are other pathogenic protozoan phyla recognized as causing diseases in blue crabs. From the phylum Sarcomastigophora, there are an amoebic parasite, *Paramoeba perniciosa*, and a parasitic dinoflagellate, *Hematodinium perezi*. Both these parasites can cause lethal infections in blue crabs along the Atlantic coast of the United States.

A well documented disease of blue crabs is the "grey crab disease" caused by *P. perniciosa*. First reported in blue crabs by Sprague and Beckett (1966, 1968), the parasite was identified as the causative agent in crab shedding mortalities in Chincoteague Bay, Virginia. The condition had long been detected and reported by fishermen from Maryland and Virginia who noticed that some crabs which appeared sluggish would often have a grey ventral surface during May and June--hence, the term "grey crab disease." These crabs had reduced clotting abilities and would often die after being stressed by handling (Sawyer 1969; Sprague *et al.* 1969; Johnson 1977b; Couch 1983).

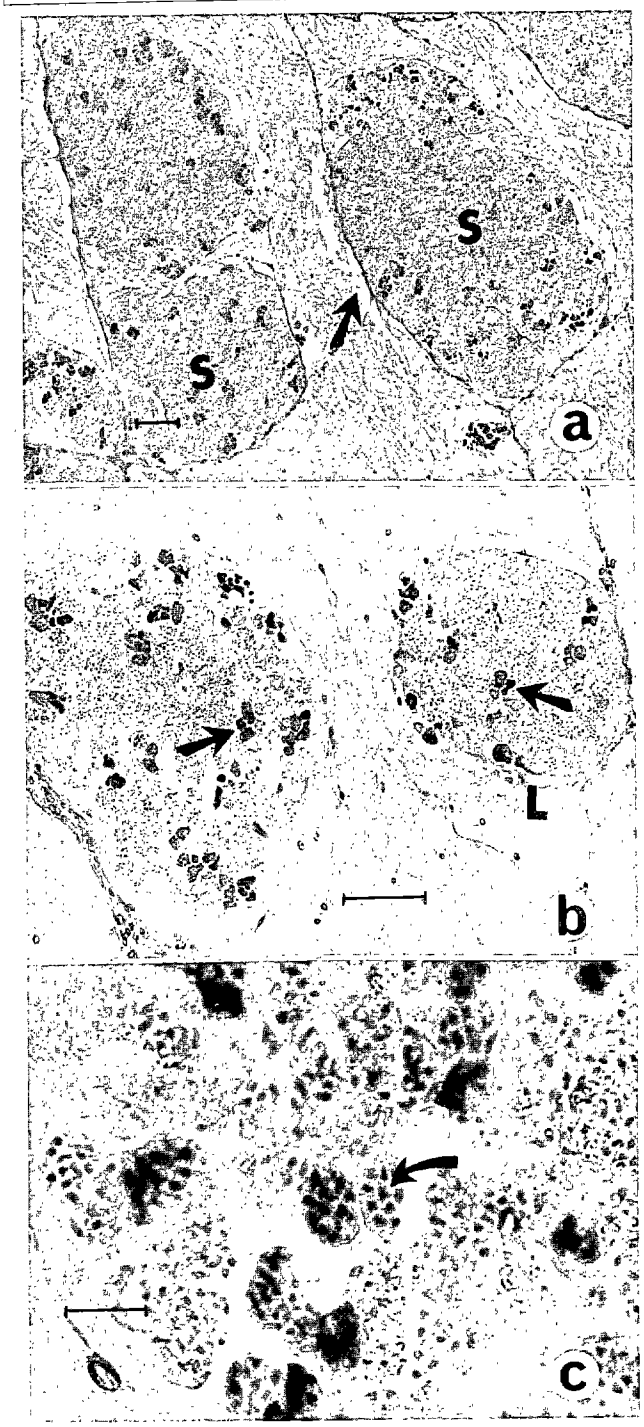


Figure 5. Microsporidan infection in blue crab skeletal muscle tissue.
a. Muscle lysis (arrow), and tissue destruction and replacement by spores (S). Bar = 50 μm.
b. Muscle lysis (L), and bundles of spores (arrows). Bar = 50 μm.
c. Note individual spores in each bundle (arrow). Bar = 10 μm.

Paramoeba is found in the hemolymph and nonepithelial tissues of blue crabs. The amoebae range in size from 3 to 35 µm, are round to elongate, contain a well defined nucleus with a large central endosome, plus a morphologically different secondary nucleus-like body in the cytoplasm. The major diagnostic characteristic of *P. perniciosa* is the presence of this secondary body in the cytoplasm (Figure 6). In heavily infected individuals, *Paramoeba* disease causes drastic changes in muscle tissues. In very heavy cases, most blood cells are replaced by the amoebae, but in light infections few amoebae may be apparent. Not all crabs infected with the parasite exhibit the grey sterna, but most of the crabs which have the grey ventral surface are lethargic and heavily infected with amoebae (Johnson 1977b; Couch 1983).

Most crabs infected by *Paramoeba* demonstrate some type of defense reaction, usually noted by the presence of degraded lysed amoebae being phagocytized by hyaline hemocytes; occasionally, there is also encapsulation of amoebae by hemocytes (Figure 6c). Hemocytes are usually seen concentrated in tissues containing amoebae, especially in the midgut and hemal spaces of the antennal gland (Johnson 1977b). The cause of death in *Paramoeba* infections is unknown. It is suggested that loss of vital tissue, such as blood cells and muscle, may lead to a state of inanition and loss of function. Blood sinuses in infected gills may become so congested with amoebae that respiratory function may fail. Glucose levels in the blood of infected crabs are reduced, perhaps due to competition between *P. perniciosa* and the infected tissue for the limited amount of available glucose. Serum protein in infected crabs is also reduced which probably causes the weakened, lethargic response (Pauley *et al.* 1975; Campbell 1984).

Peak prevalences of the parasite occur during May and June (Campbell 1984) and in dredged hibernating crabs from October to February. The disease in epizootic form could be a major factor in annual winter and late spring mortalities of blue crabs (Newman and Ward 1973; Couch 1983), and the cause of chronic low-level mortalities in late spring of nonepizootic years. Marked annual and geographic variations in the prevalences of *Paramoeba* have been noted and mortalities are most commonly reported from high-salinity coastal bays from Maryland to Georgia. The parasite appears to be opportunistic by invading the hemolymph via injuries to the exoskeleton, especially in newly molted crabs. *Paramoeba* has also been reported to infect Atlantic rock crabs, *Cancer irroratus*, American lobsters, *Homarus americanus* (Sawyer and MacLean 1978), and green crabs, *Carcinus maenas* (Campbell 1984).

Another member of the protozoan phylum Sarcomastigophora recognized as causing disease in blue crabs is the dinoflagellate *Hematodinium perezi*. This parasite was first reported in portunid crabs from France (Chatton and Poisson 1931), but was not reported as a parasite in other Crustacea until found in blue crabs sampled for *P. perniciosa* (Newman and Johnson 1975). *Hematodinium* causes a fatal disease, but infected crabs exhibit no external signs except lethargy. Drawn blood from heavily infected

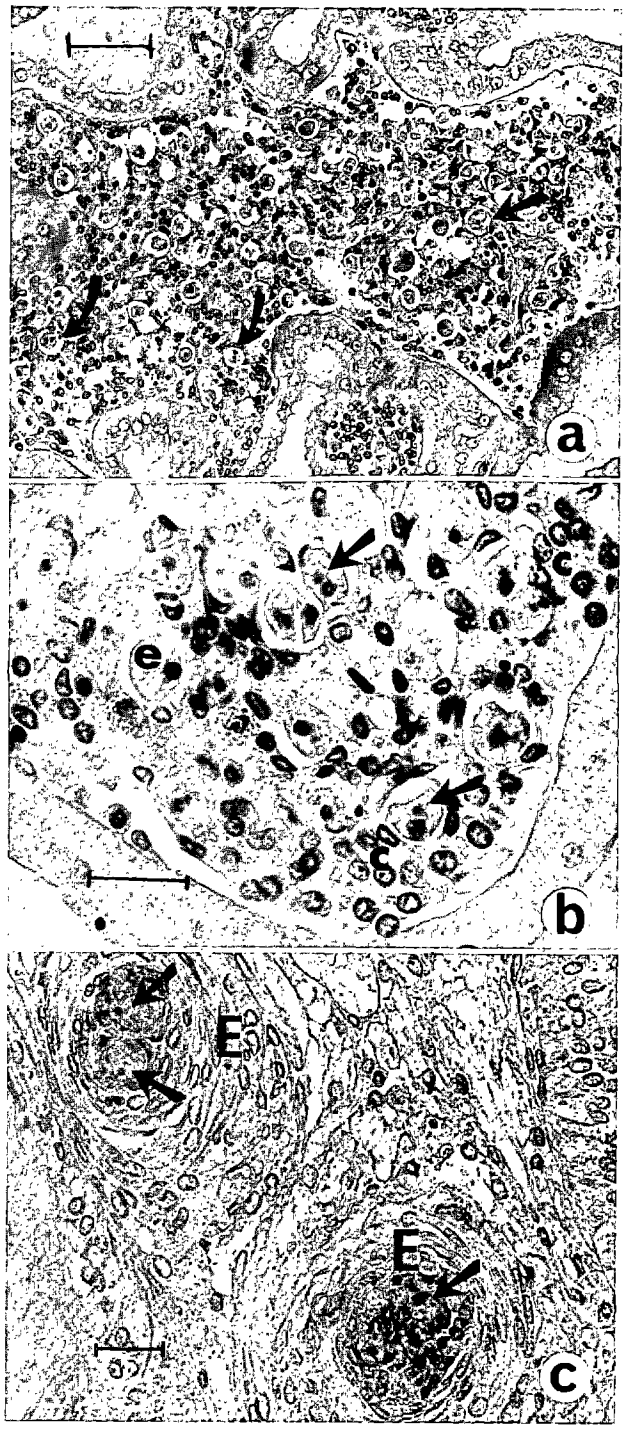


Figure 6. *Paramoeba perniciosa* infection in blue crab tissues.
a. Hemal space of antennal gland filled with parasites (arrows). Note halo surrounding individual amoebae and secondary bodies in cytoplasm of parasites. Bar = 50 µm.
b. Note nucleus with large endosome (e), secondary bodies in cytoplasm (arrow), and normal hemocytes (c). Bar = 20 µm.
c. Encapsulation (E) of degraded lysed amoebae (arrows) being phagocytized by hyaline hemocytes. Bar = 20 µm.

animals is milky, due to the presence of many uninucleate dinoflagellates; in some instances, tissues may have a pink opalescent color. The disease has been noted only in blue crabs from salinities above 11 ppt, although samples from lower salinities have been examined. Diagnosis is dependent on the presence of uninucleate or multinucleate plasmodial cells ranging in size from 5.8 to 64 µm. Nuclei are large in proportion to cytoplasmic volume and chromosomes can be either condensed or diffused. This characteristic dinokaryon nucleus is a major diagnostic indicator of the dinoflagellate (Figure 7). The parasite is found primarily in the hemolymph, but is also seen in and between muscle fibrils, gonad, and in the hepatopancreas. Reported geographic distribution in blue crabs includes the Atlantic and Gulf coasts of the United States. The parasite has been detected in crabs in all seasons except late winter and early spring (Newman and Johnson 1975; Newman 1977; Couch 1983). *Hematodinium perezi* has also been detected in other crabs, including *Carcinus maenas* and *Portunus depurator* from Europe (Chatton and Poisson 1931), the Atlantic rock crab, *Cancer irroratus*, the Jonah crab, *C. borealis*, and the lady crab, *Ovalipes ocellatus*, from the Middle Atlantic Bight of the U. S. east coast (MacLean and Ruddell 1978).

The protozoan phylum Ascetospora includes the very pathogenic haplosporidan *Haplosporidium nelsoni* which, since the 1960s, has been known to cause considerable mortalities in oysters. This phylum is poorly represented as a crustacean pathogen; only Atlantic mud crabs, *Panopeus herbstii*, from Louisiana infected with *H. louisiana* (Sprague 1963, 1978), flatback mud crabs, *Eurypanopeus depressus*, from Chincoteague Bay, Virginia, and Harris mud crabs, *Rhithropanopeus harrisii*, from Chesapeake Bay, Maryland, have been found infected with ascetosporans (Rosenfield *et al.* 1969). Two blue crabs from Virginia and North Carolina were reported infected with a haplosporidan-like organism (Newman *et al.* 1976). The prevalence of the reported disease was rare, but apparently lethal. Drawn blood from the infected crabs was opaque white, of low viscosity, and failed to clot properly. Masses of uninucleate or multinucleate plasmodial bodies filled vascular spaces and interstitial tissues (Figure 8). Uninucleate cells ranged from 3.4 to 7.3 µm, and their nuclei ranged from 1.1 to 2.2 µm in diameter. Electron microscopy revealed that these organisms were not the same as those found infecting either oysters or mud crabs, but a new species parasitizing blue crabs (Newman *et al.* 1976). Although reportedly a rare disease of blue crabs, it has the ability to proliferate in vascular spaces, thus causing fatalities in infected crabs.

Lagenophrys callinectes is an ectocommensal ciliate found attached to flat surfaces of blue crab gills (Couch 1966, 1967). The colorless, transparent, semihemispherical lorica which cements itself to the crab gill measures 48-59 µm long by 48-57 µm wide. Mortalities caused by *Lagenophrys* are most common among crabs about to molt and those being held in holding facilities, although crabs in the wild have also been reported to be heavily infested. The ciliate is most prevalent from July through November, although a decreased prevalence observed during May and June is probably due to a higher incidence of molting activity, thereby freeing infested crabs of the attached ectocommensals (Couch 1983). Although these ciliates are technically not parasites (since they do not derive nourishment directly from the host), they can debilitate crabs by preventing proper gas exchange between the water and gill tissue (Couch 1983).

Another ciliate, *Epistylis* sp., observed infesting blue crabs attaches to the margins and stems of the gill lamellae rather than the flat portions. The effect of this stalked ciliate on the health of infested crabs is unknown (Overstreet 1978) (Figure 9).

DISEASES CAUSED BY HELMINTHS

Marine decapod crustaceans are parasitized by an array of helminths. Few helminths are truly pathogenic, except in isolated instances of heavy infections in individual hosts. These usually immature worms form a spectrum of parasites, with a usually modest effect on the host. For a review of metazoan parasites in blue crabs and other crustaceans, see Overstreet (1982, 1983). Of those helminths listed, larval trematodes, nemerteans, and leeches have been reported to either reduce the market value of the captured crabs or reduce the fecundity of heavily infected populations.

TREMATODES

Metacercariae of digeneans *Microphallus basodactylophallus* and *Megalophallus* sp. often parasitize the musculature of blue crabs. These larval flukes are not easily seen unless hyperparasitized by the haplosporidan *Urosporidium crescens*. The metacercariae are easily recognized when hyperparasitized because they become enlarged (410-654 µm), darkly pigmented, and rupture easily; whereas, uninfected or lightly infected metacercariae are smaller (189-269 µm), white to cream color, and can withstand some mechanical pressure (DeTurk 1940; Couch 1974b; Overstreet 1978; Millikin and Williams 1984). Masses of dark pigmented spores in the metacercaria produce a conspicuous black spot seen easily by the unaided eye (Figure 10). Fishermen call the cysts "buckshot" or "pepper spots" and the infected crabs "pepper crabs." The presence of these "pepper spots" in cooked crab meat is objectionable and can decrease the desirability and market value of infected crabs. Very heavy infections may result in lysis of host muscle tissue surrounding the parasite. Hyperparasitized metacercariae of *Microphallus basodactylophallus* infect thoracic muscles, hepatopancreas, and ventral ganglia of blue crabs from Chesapeake Bay to Texas (Overstreet 1982). If *U. crescens* spores were released within the live crab, hemocytes or other defense mechanisms would probably kill the spores; so, as a protective measure, the spores

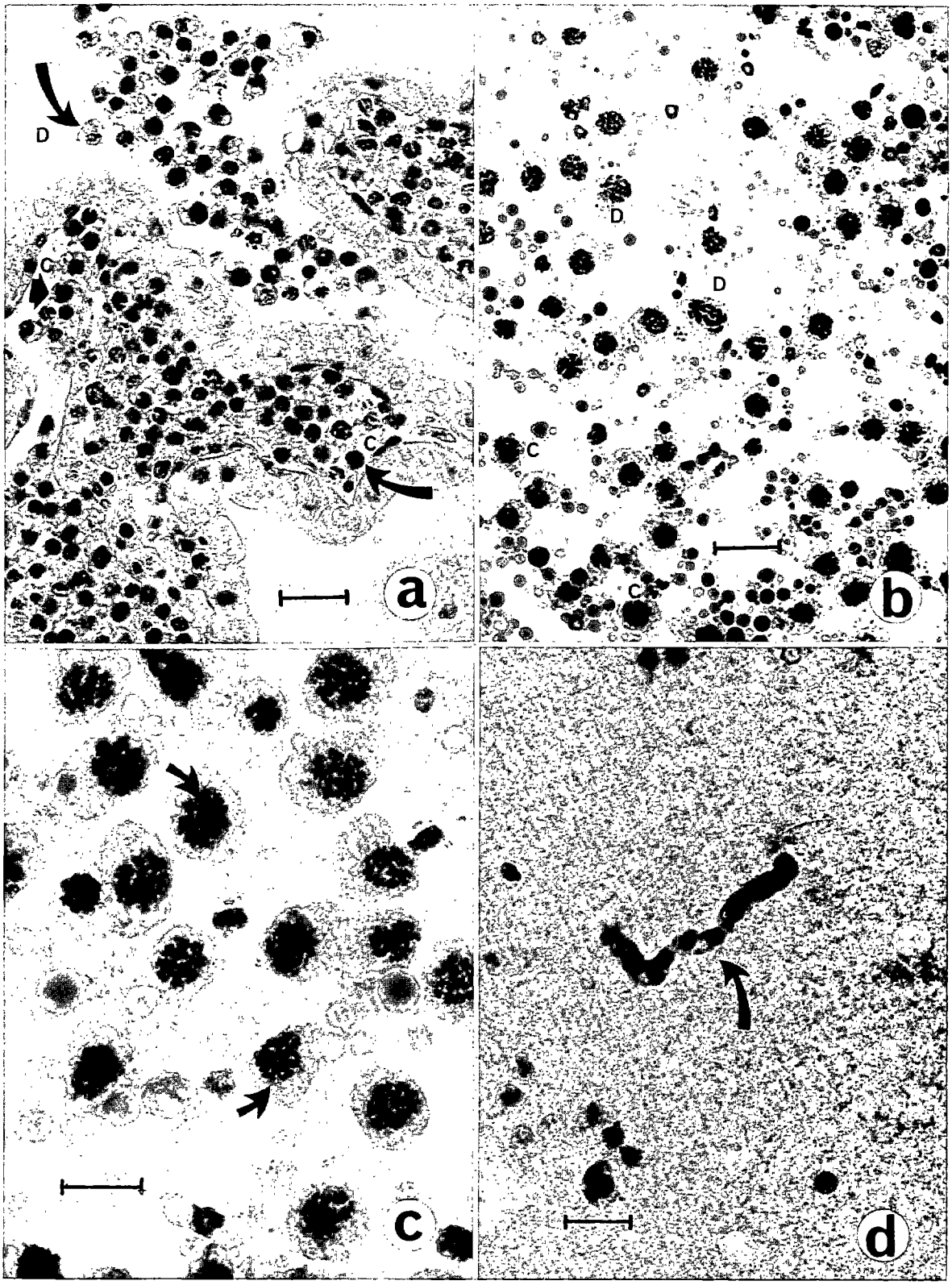


Figure 7. *Hematodinium perezi* infection in blue crab hemolymph.

a. Uninucleate plasmodial *Hematodinium* (arrows) in antennal gland. Note diffuse (D) and condensed (C) nuclei. Parasites proliferate and replace host tissue. Bar = 20 μm.

b. Uninucleate plasmodial *Hematodinium* in hemolymph smear. Note diffuse (D) and condensed (C) chromosomes. Bar = 20 μm.

c. Uninucleate plasmodial *Hematodinium* in hemolymph smear. Note lack of nuclear membranes (arrow). Bar = 10 μm.

d. Long-form multinucleate plasmodial *Hematodinium* in hemolymph smear (arrow). Bar = 20 μm.

metacercariae, although they may debilitate them. The hyperparasites are only released upon death of the crab due to rupture of the metacercarial cyst (Couch 1974b).

NEMERTEANS

The ribbon worm, *Carcinonemertes carcinophila*, is found encysted between gill lamellae of female blue crabs in high-salinity waters (Figure 11). Humes (1942) described the morphology and life cycle of this parasite. As a juvenile, the worm migrates from the gills to the host's egg mass to become sexually mature, lays eggs of its own, and returns to the gills as a mature adult (Hopkins 1947). Crab eggs provide nourishment for the worm, and a color change from cream to orange can be observed due to the ingestion of crab egg-yolk material. The parasite builds a mucus tube as a habitat in the egg mass. After mating, the female worm leaves her eggs and mucus tube and returns to the gills as an adult (Overstreet 1978). The larvae escape from the egg membranes through the action of enzymes and may remain in the tube, swim among the eggs, or leave the host and change to a free-living form, enabling them to infest other crabs (Davis 1965).

Since worms change color as a result of feeding on crab eggs, and since adults return to the gills, *C. carcinophila* can be used as a biological indicator. Nonovigerous female crabs that have already spawned once can be distinguished from nonspawning females by examining the gills of these crabs with the unaided eye. Only large bright-red adult worms occur in crabs that have spawned and only immature worms, light in color, are found in gills of females that have not spawned. This method of diagnosing spawned blue crabs is about 97-percent accurate (Hopkins 1947; Pyle and Cronin 1950; Overstreet 1978, 1982).

ANNELIDS (leeches)

The leech, *Myzobdella lugubris*, is often found attached to blue crabs from low-salinity waters, but is not considered fatal. Leeches have been implicated inconclusively in mortalities of blue crabs in Florida where 50 percent of crabs sampled had leeches attached near perforations on the carapace and articulations of the pereiopods (Figure 12) (Hutton and Sogandares-Bernal 1959). The life cycle of *M. lugubris* involves two hosts, the white catfish, *Ameiurus catus*, which provides most of the leech's nutrition, and the blue crab, on which it deposits cocoons near the rear margin of the crab's carapace. In addition to providing a surface for the cocoon to attach, the crab also acts as a means of dispersal for the young (Overstreet 1978; Millikin and Williams 1984). The parasite is common along the Atlantic and Gulf coasts of the United States and is usually not considered fatal (Moore 1946; Sawyer *et al.* 1975).

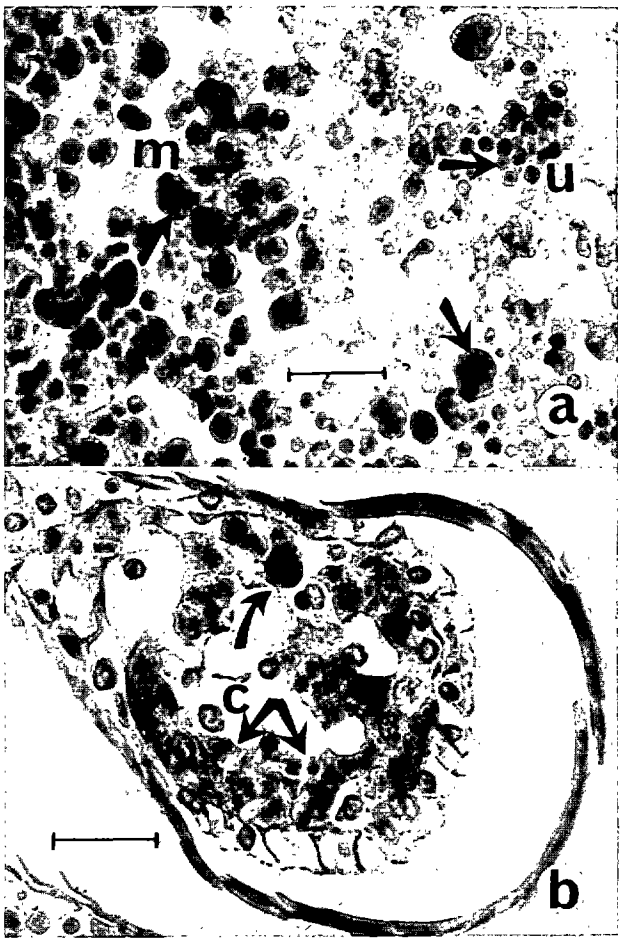


Figure 8. Haplosporidan-like organisms in hemolymph of blue crab.
a. Uninucleate (u) and multinucleate (m) plasmodial forms of haplosporidan-like parasite. Note individual spores (arrows). Bar = 20 µm.
b. Vascular space of gill filled with haplosporidan-like plasmodia (arrows); (c) is a normal hemocyte. Bar = 20 µm.

ECTOCOMMENSAL AND PARASITIC BARNACLES

Ectocommensal barnacles are observed frequently on blue crabs. The gooseneck or pedunculate barnacle, *Octolasmis mulleri*, is found on gills of blue crabs in high-salinity waters. These may interfere with crab respiration in heavy infestations due to inhibited scaphognathite movement, but usually they cause no harm to the host (Walker 1974; Overstreet 1978; Millikin and Williams 1984). Another ectocommensal, the acorn barnacle *Chelonibia patula*, is found attached to the carapace of blue crabs, especially older crabs, after their terminal molt. The only hardship these barnacles may present to the host is excess weight on the carapace which in heavy infestations may inhibit crab mobility (Overstreet 1978).

A truly parasitic sacculinid barnacle infecting blue crabs is the rhizocephalan *Loxothylacus texanus*. Postmolt juvenile crabs are infected by swimming *L. texanus* cypris larvae. The interna or "root system" of the barnacle penetrates the soft abdominal joint of the crab and is extended into internal tissues when the crab molts. The externa of the barnacle, consisting of a brood pouch for gonads and larvae, protrudes from under the crab's abdomen, appearing as a bulge which can be seen grossly. The barnacle causes a restriction in growth; infected individuals are only between 3 and 8 cm wide, thereby making them unavailable to the blue crab fishery. Infected crabs are often termed "button crabs" and appear as miniature adult females, with secondary sexual characteristics transformed to those of a female (Figure 13). The abdominal segments of infected males become wider; six distinct segments develop instead of the normal four; and abdominal musculature appears similar to that of a mature female. The aprons of infected immature females are transformed to broader, rounded aprons similar to mature females (Reinhard 1950, 1956; Adkins 1972).

Castration may result from a malfunction of the androgenic gland in males; in experiments in which the organ was removed from a young male, female characteristics developed, including production of ovarian tissue in the testes. It is conceivable that *L. texanus* causes malfunction to this gland, or possibly hormones produced by the barnacle's ovaries cause over-feminization of infected crabs (Overstreet 1978). Infections have been reported from the Gulf coast only and are usually seen in high-salinity waters (Ragan and Matherne 1974). Epizootics were reported in Mississippi in 1965 and 1977 (Overstreet 1978). Since diseased crabs are below market size, actual prevalence of the parasite is hard to estimate, and because the parasite removes crabs from the fishery, it causes significant economic losses during epizootics.

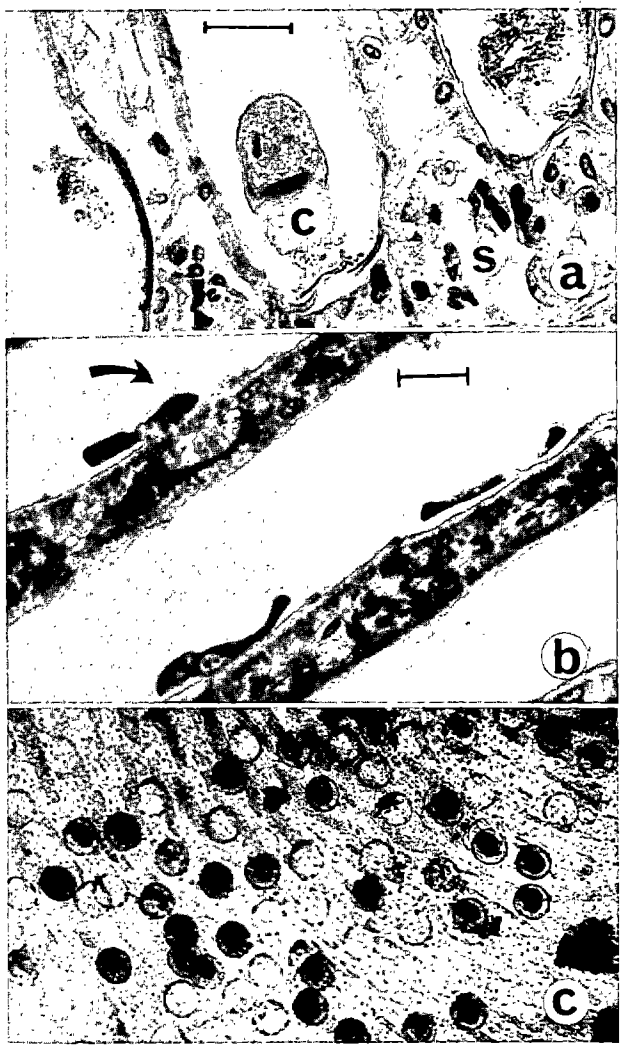


Figure 9. Ciliates infesting blue crab gills.
a. Stalked ciliate, *Epistylis*, attached to stem (S) of blue crab gill. Bar = 20 μm.
b. *Lagenophrys* ciliates cemented to flat surfaces of gill. Note semitransparent lorica. Bar = 40 μm.
c. Infestation of the gills of a blue crab by the ciliate *Lagenophrys*. (Photograph courtesy of Dr. J.A. Couch.)

SUMMARY

From the abundant literature available on diseases of blue crabs, it becomes obvious that there are several severe pathogens that can cause mortalities in both wild and captive blue crabs. Because of the economic importance of the blue crab fishery, it is apparent that diseases should be controlled whenever possible. Through increased research, better methods of control can be devised to help reduce the damage diseases can cause, as was the case with "sick crab" disease caused by microsporidans. After finding that this disease is transmitted by crabs ingesting infected tissues from dead diseased crabs, watermen were told to stop crushing diseased crabs and throwing them back into the water. Instead, crabbers were told to dispose of the crabs properly on shore, thus reducing some of the potential spread of the parasite.

Diseases commonly found in crabs held in captivity such as bacterial and viral infections can be aggravated by stress. The viruses considered pathogenic in blue crabs are: (1) the reo-like virus (RLV) acting synergistically with rhabdo-like virus A (RhVA), (2) the Chesapeake Bay virus (CBV), and (3) the bi-facies virus (BFV). The RLV-RhVA combination is known to cause mortalities in shedding systems. Perhaps by devising better methods of reducing stress in these animals, mortalities could be lowered, thus allowing for increased productivity and profits for the soft-shell crab producer.

Shell disease, caused by chitinoclastic bacteria, occurs frequently in crabs held in captivity for prolonged periods. This disease could be limited by reducing captivity time; when this is impossible, some kind of prophylactic treatment to exclude chitinoclastic bacteria would help reduce the incidence of this disfiguring disease which may make crabs unmarketable.

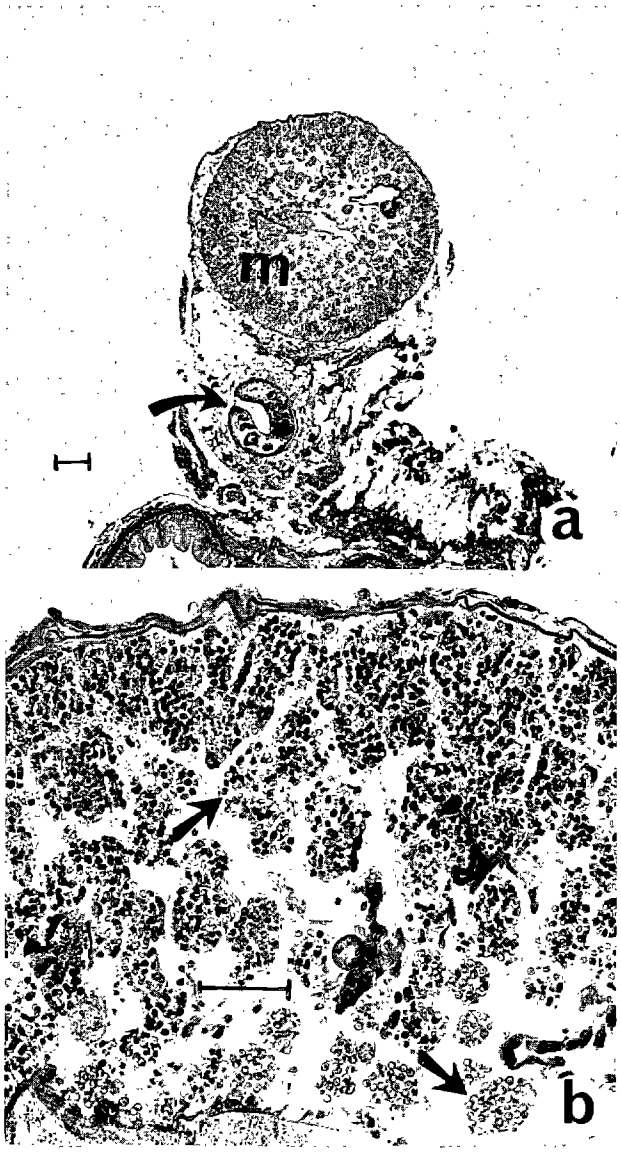


Figure 10. *Microphallus basodactylophallus* hyperparasitized by *Urosporidium crescens*.
a. Hyperparasitized metacercariae (m) and unparasitized metacercariae (arrow). Bar = 100 µm.
b. Pockets of haplosporidans *U. crescens* (arrows) parasitizing *M. basodactylophallus* metacercariae. Bar = 50 µm.

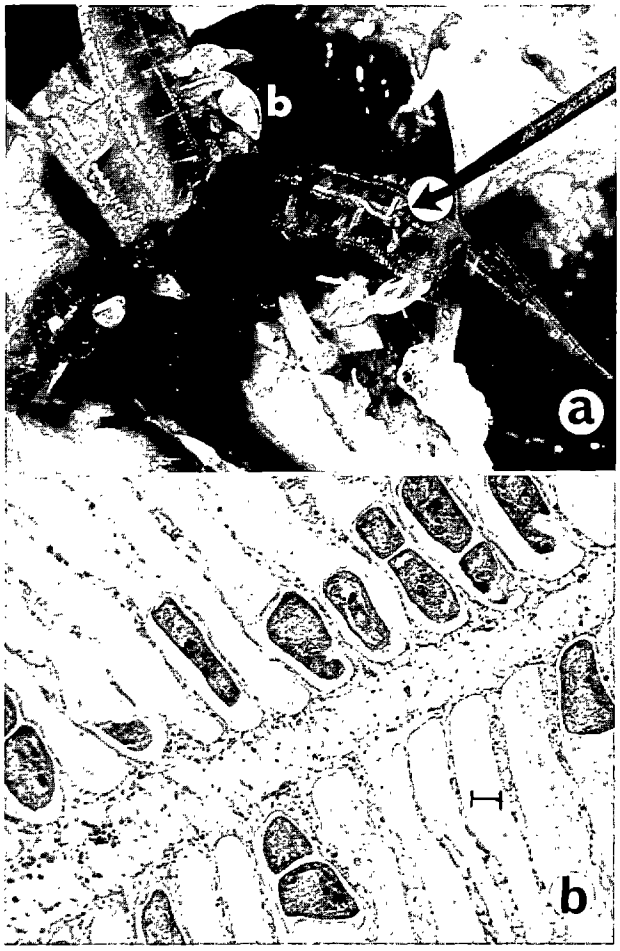


Figure 11. Ribbon worms between blue crab gill lamellae.
a. Long orange ribbon worm *(Carcinonemertes carcinophila)* pulled out from between gill lamellae. Other light-colored regions on gills are additional specimens. Also note barnacle on gills. Both barnacle and ribbon worm indicate that the crab had inhabited high-salinity water. (Photograph courtesy of Dr. R.M. Overstreet.)
b. Histologic section showing nemerteans encysted between gill lamellae. Bar = 100 µm.

Systemic bacterial infections are usually manifested in crabs that have been stressed by one or more factors. By reducing stress, both in the shedding system and during capture, these infections could be lowered. Crabbers should be advised to treat peeler crabs with better care by providing shade, reducing handling stress, and trying to reduce crowded conditions, if possible. Crab shedders could reduce stress to captive crabs by providing them with good water flow and well oxygenated water, and by keeping their systems clear of sediments and high levels of metabolic byproducts.

Although diseases which occur frequently in captive crabs have the potential for being treated, diseases which occur most often in wild populations cannot usually be treated or controlled. Unfortunately, these are diseases that may cause the most harm to crab populations. One of these, the fungus *Lagenidium callinectes*, attacks egg masses, thus causing reduced fecundity. Protozoan diseases can induce mass mortalities in wild populations of blue crabs. The most detrimental protozoans found infecting blue crabs are microsporidans, including *Nosema* and *Pleistophora*. It is known that these pathogens are transmitted directly when healthy crabs ingest dead sick crabs, so crab fishermen have been told not to return sick crabs to the waters as they had done previously. Other protozoans of significance are two

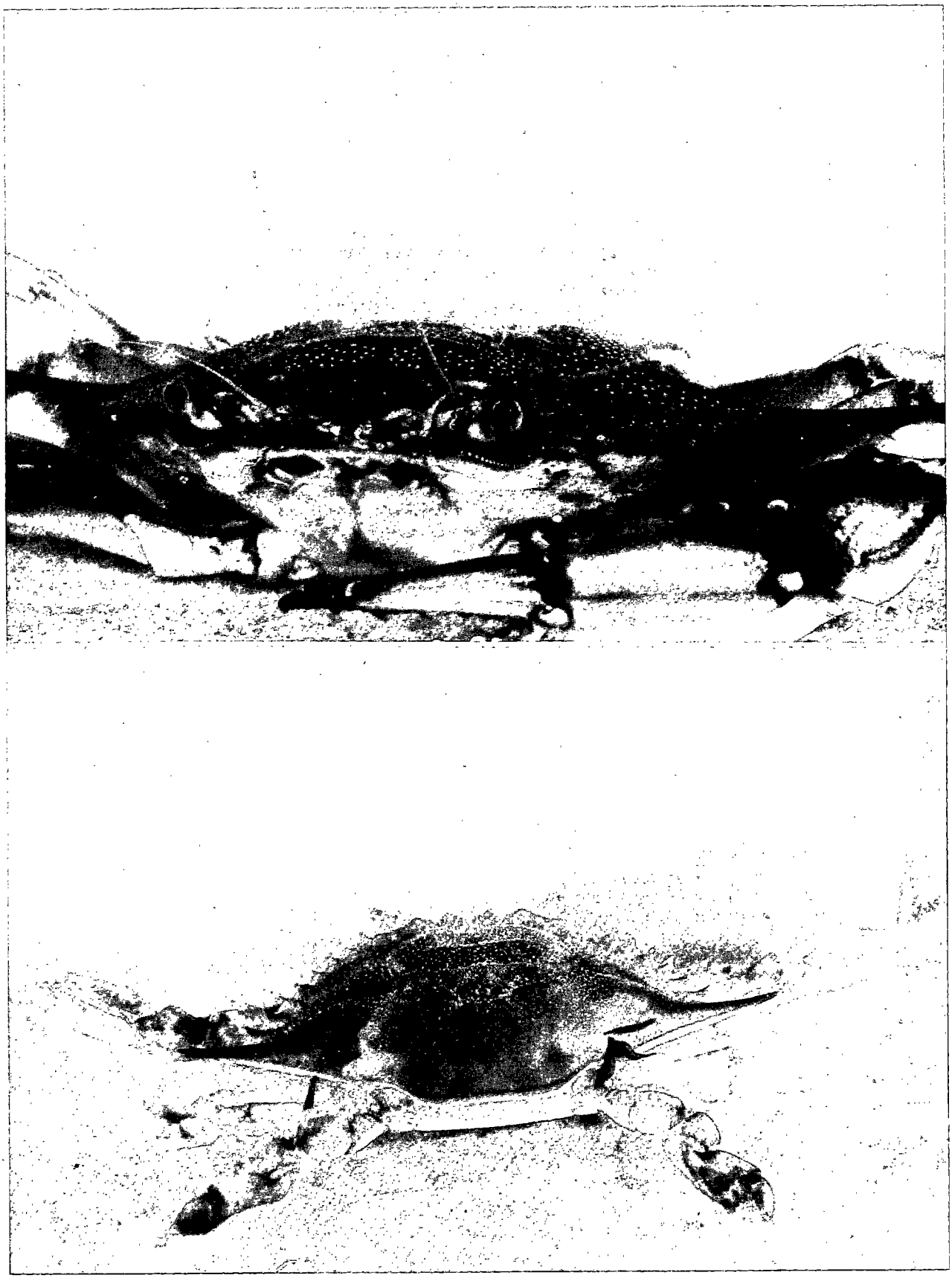


Figure 12. Leech *(Myzobdella lugubris)* on blue crab. Top, several specimens of the leech on the crab's anterior. Bottom, several specimens on crab's posterior; most individuals deposit cocoons near the rear margin. (Photographs courtesy of Dr. R.M. Overstreet.)

members of the phylum Sarcomastigophora, *Paramoeba perniciosa* and *Hematodinium*; a member of the phylum Ciliophora, *Lagenophrys callinectes*; and a member of the phylum Ascetospora, a haplosporidan.

Some diseases do not cause mortalities, but reduce the demand for crabs because of the unappetizing features they produce in the crabs. Besides shell disease, mentioned earlier, a digenean trematode, *Microphallus basodactylophallus*, hyperparasitized by the haplosporidan *Urosporidium crescens* can cause reduced demand for infected crabs. These cause enlarged, darkly pigmented "pepper spots" in muscle tissue of infected crabs. Cooked crab meat with these foreign bodies appears unappetizing. Another parasite which removes crabs from the fishery and causes an interesting morphological change in young crabs is caused by a parasitic barnacle, *Loxothylacus texanus*. This parasite attacks young crabs and causes a marked change in secondary sexual characteristics, making infected individuals appear as miniature adult females. The parasite castrates infected males, thus reducing potential reproductive abilities of the population.

In conclusion, blue crabs are subject to a number of diseases, some with a high potential to cause mass mortalities and others which seem to be more a nuisance than a threat to the crab's health. Methods have been devised to reduce the potential risk of some of these diseases and further research should provide additional information, leading to better control--especially in captive populations.

ACKNOWLEDGMENTS

We wish to thank Fred Kern, Dr. Eric May, and Dr. Aaron Rosenfield for manuscript review and helpful comments, and Martin Newman and Dr. Phyllis Johnson for use of archived specimens. We are also indebted to Jane Keller for diligent typing, editing, and counsel, to Susie Hines for assistance in obtaining library materials, and to Jay Lewis for photography advice.

REFERENCES CITED

Adkins, G. 1972. Notes on the occurrence and distribution of the rhizocephalan parasite *(Loxothylacus texanus* Boschma*)* of blue crabs *(Callinectes sapidus)* in Louisiana estuaries. [La. Wildl. Fish. Comm.,] *Tech. Bull.* No. 12. 13 pp.

Anonymous. 1972. *Vibrio parahaemolyticus* gastroenteritis--United Kingdom. [U.S. Public Health Serv.,] *Morbid. Mortal. Week.* Rep., Mar. 25: 99, 104.

Bazin, F., P. Monsarrat, J.R. Bonami, G. Croizier, G. Meynadier, J.M. Quiot, and C. Vago. 1974. Particules virales de type Baculovirus observees chez le Crabe *Carcinus maenas*. *Rev. Trav. Inst. Peches. Marit.* 38: 205-208.

Benton, A.G. 1935. Chitinovorous bacteria. A preliminary survey. *J. Bacteriol.* 29: 449-463.

Blake, P.A., D.T. Allegra, J.D. Snyder, T.J. Barrett, L. McFarland, C.T. Caraway, J.C. Feeley, J.P. Craig, J.V. Lee, N.D. Puhr, and R.A. Feldman. 1980a. A possible endemic focus in the United States. *New. Engl. J. Med.* 302(6): 305-309.

Blake, P.A., R.E. Weaver, and D.G. Hollis. 1980b. Diseases of humans (other than cholera) caused by vibrios. *Annu. Rev. Microbiol.* 34: 341-367.

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

Bland, C.E., and H.V. Amerson. 1974. Occurrence and distribution in North Carolina waters of *Lagenidium callinectes* Couch, a fungal parasite of blue crab ova. *Chesapeake Sci.* 15: 232-235.

Bland, C.E., D.G. Ruch, B.R. Salser, and D.V. Lightner. 1976. Chemical control of *Lagenidium*, a fungal pathogen of marine Crustacea. [Univ. N.C. Sea Grant Prog.,] *Sea Grant Publ.* UNC-SG-76-02. North Carolina State University, Raleigh, N.C. 38 pp.

Campbell, D.G. 1984. The abundance and distribution of *Paramoeba perniciosa*. Ph.D. Thesis. Johns Hopkins University, Baltimore, Md. 188 pp.

Chatton, E., and R. Poisson. 1931. Sur l'existence, dans le sang des Crabes, de Peridiniens parasites: *Hematodinium perezi* n. g., n. sp. (Syndinidae). *C.R. Soc. Biol.* 105: 553-557.

Colwell, R.R., T.C. Wicks, and H.S. Tubiash. 1975. A comparative study of the bacterial flora of the hemolymph of *Callinectes sapidus*. *Mar. Fish. Rev.* 37(5-6): 29-33.

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

Couch, J.A. 1966. Two peritrichous ciliates from the gills of the blue crab. *Chesapeake Sci.* 7: 171-176.

Couch, J.A. 1967. A new species of *Lagenophrys* (Ciliata: Peritrichida: Lagenophryidae) from a marine crab, *Callinectes sapidus*. *Trans. Amer. Microsc. Soc.* 86: 204-211.

Couch, J.A. 1974a. An enzootic nuclear polyhedrosis virus of pink shrimp: ultrastructure, prevalence, and enhancement. *J. Invertebr. Pathol.* 24: 311-331.

Couch, J.A. 1974b. Pathological effects of *Urosporidium* (Haplosporida) infection in microphallid metacercariae. *J. Invertebr. Pathol.* 23: 389-396.

Couch, J.A. 1981. Viral diseases of invertebrates other than insects. Pages 127-160 *in* E.W. Davidson, ed. Pathogenesis of invertebrate microbial diseases. Allanheld Osmun & Co. Publishers, Totowa, N.J.

Couch, J.A. 1983. Diseases caused by Protozoa. Pages 79-111 *in* A.J. Provenzano, ed. The biology of Crustacea. Vol. 6. Pathobiology. Academic Press, New York.

Couch, J.A., and S. Martin. 1982. Protozoan symbionts and related diseases of the blue crab, *Callinectes sapidus* Rathbun from the Atlantic and Gulf coasts of the United

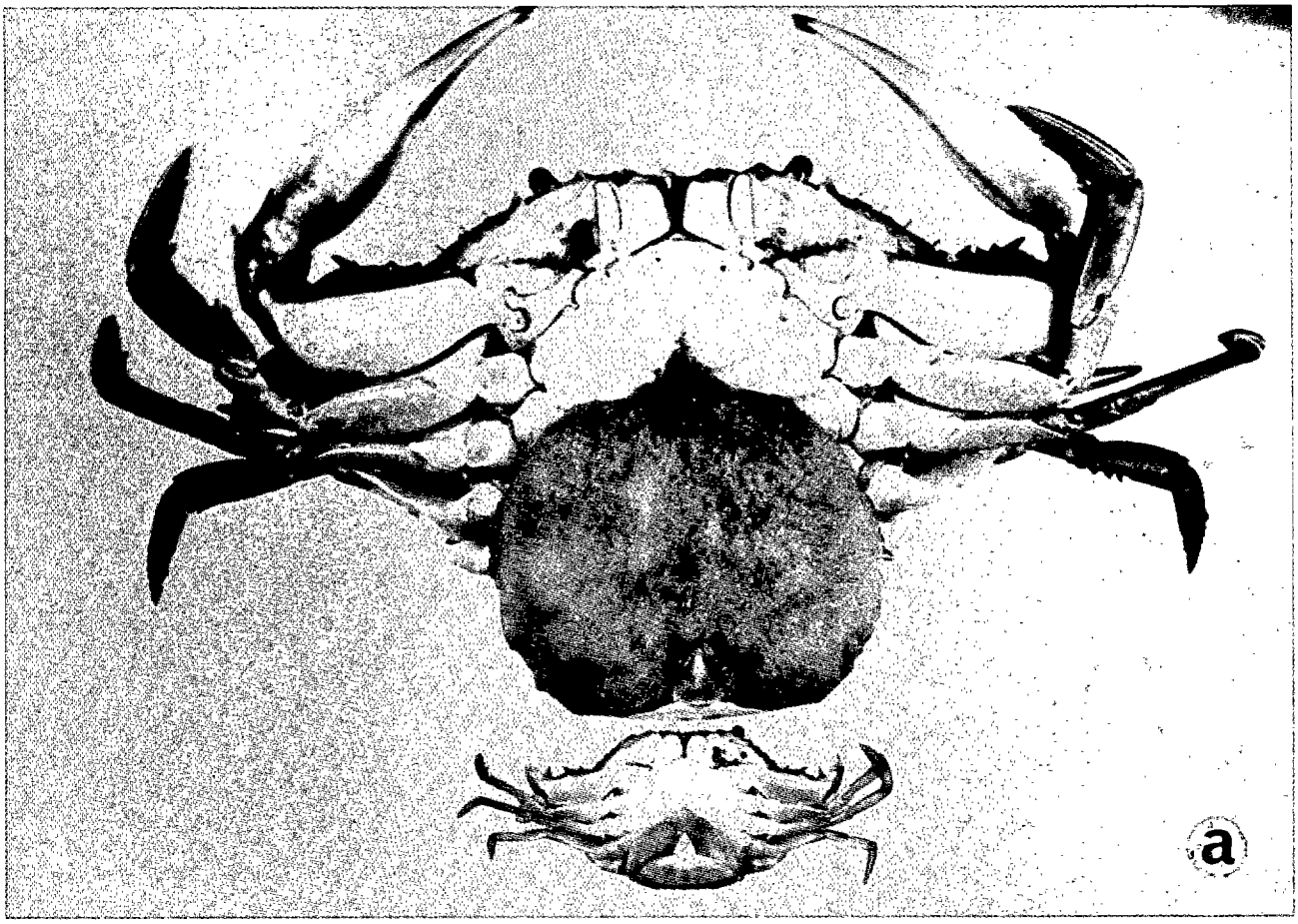
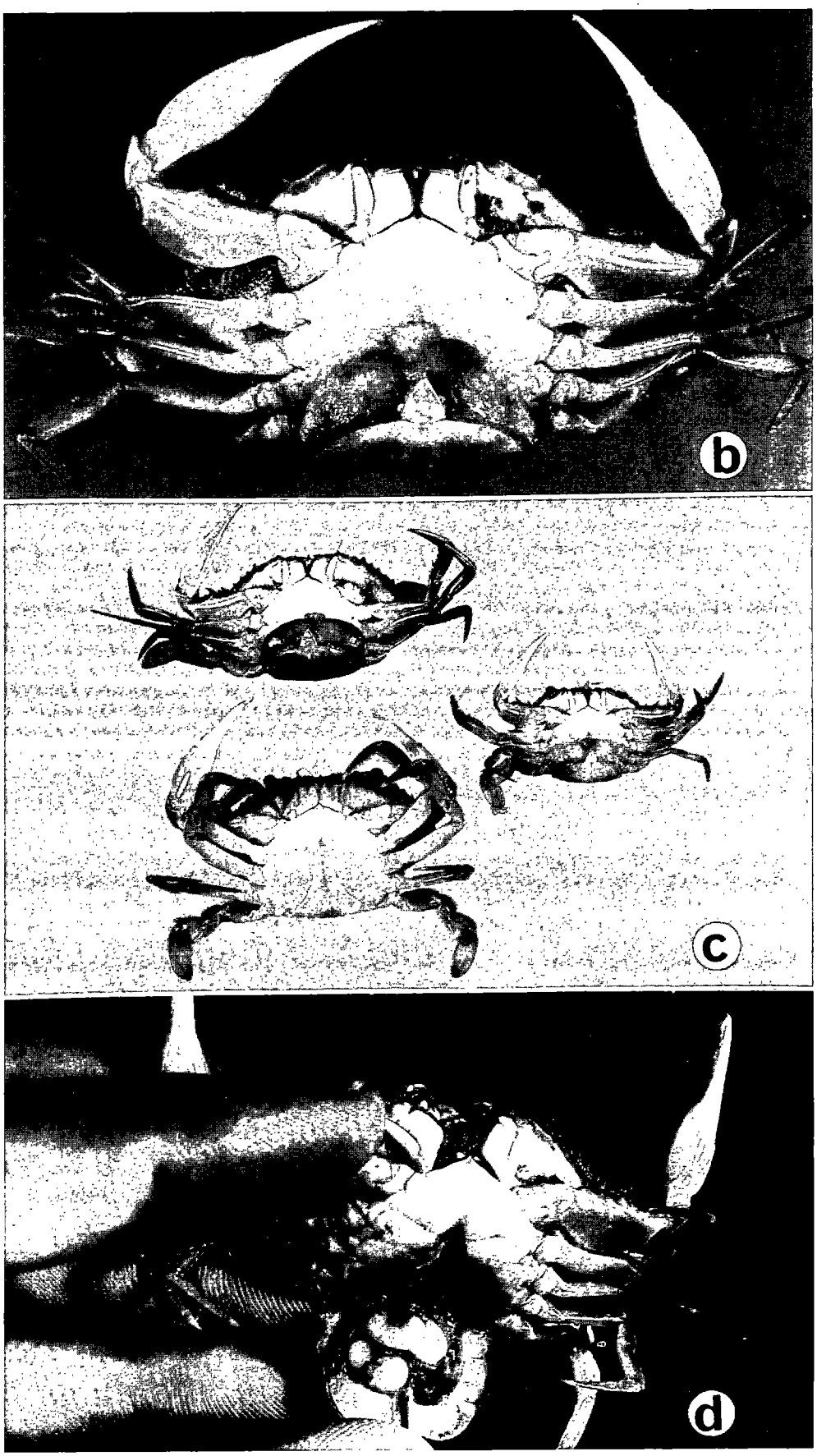


Figure 13. Sacculinid barnacle, *Loxothylacus texanus*, infecting blue crab. (Photographs courtesy of Dr. R.M. Overstreet.)

a. A blue crab infected with the rhizocephalan *L. texanus* (bottom), compared to a normal egg-bearing female (top).
b. The protruding pouch (externa) under the crab's abdomen contains larvae and gonads (ovary and testes). Long, narrow extensions invade most other tissues.
c. Top crab has dark externa, indicating an older infection than that of the yellowish externa of the middle crab. The larger lower crab reveals the small size of infected crabs. It is an immature female, whereas the aprons on the infected crabs reveal the apron of a mature crab. Infections also modify the shape of the male's abdomen into that of a female.
d. Crab with three developing externae. Usually only one externa occurs per crab, but occasionally over five may be present.


b
c
d

States. Pages 71-81 *in* H.M. Perry and W.A. Van Engel, eds. Proceedings of the Blue Crab Colloquium, Biloxi, Miss. Gulf States Marine Fisheries Commission, Ocean Springs, Miss.

Couch, J.N. 1942. A new fungus on crab eggs. *J. Elisha Mitchell Sci. Soc.* 58: 158-162.

Davis, C.C. 1965. A study of the hatching process in aquatic invertebrates: XX. The blue crab, *Callinectes sapidus*, Rathbun, XXI. The nemertean, *Carcinonemertes carcinophila* (Kolliker). *Chesapeake Sci.* 6(4) 201-208.

Davis, J.W., and R.K. Sizemore. 1982. Incidence of *Vibrio* species associated with blue crabs *(Callinectes sapidus)* collected from Galveston Bay, Texas. *Appl. Environ. Microbiol.* 43(5): 1092-1097.

DeTurk, W.E. 1940. The occurrence and development of a hyperparasite *Urosporidium crescens* sp. nov. (Sporozoa, Haplosporidia), which infests the metacercariae of *Spelotrema nicolli*, parasitic in *Callinectes sapidus*. *J. Elisha Mitchell Sci. Soc.* 56: 231-232.

Engel, D.W., and E.J. Noga. 1989. Shell disease in the blue crabs of the Pamlico River. *Environs*, Feb. 1989: 3-5.

Fisher, W.S., E.H. Nilson, and R.A. Shleser. 1975. Effect of the fungus *Haliphthoros milfordensis* on the juvenile stages of the American lobster, *Homarus americanus*. *J. Invertebr. Pathol.* 26: 41-45.

Fisher, W.S., E.H. Nilson, L.F. Follett, and R.A. Shleser. 1976. Hatching and rearing lobster larvae *(Homarus americanus)* in a disease situation. *Aquaculture* 7: 75-80.

Fisher, W.S., E.H. Nilson, J.F. Steenbergen, and D.V. Lightner. 1978. Microbial diseases of cultured lobsters: a review. *Aquaculture* 14: 115-140.

Hay, W.P. 1905. The life history of the blue crab *(Callinectes sapidus)*. *Rep. U.S. Bur. Fish.* 1905: 395-413.

Hock, C.W. 1940. Decomposition of chitin by marine bacteria. *Biol. Bull. (Woods Hole)* 79: 199-206.

Hock, C.W. 1941. Marine chitin-decomposing bacteria. *J. Mar. Res.* 4: 99-106.

Hood, M.A., and S.P. Meyers. 1974. Distribution of chitinoclastic bacteria in natural estuarine waters and aquarial systems. Pages 115-121 *in* R.S. Amborski, M.A. Hood, and R.R. Miller, eds. Proceedings of Gulf Coast Regional Symposium on Diseases of Aquatic Animals. *La. State Univ. Publ.* No. LSU-SG-74-05.

Hopkins, S.H. 1947. The nemertean *Carcinonemertes* as an indicator of the spawning history of the host, *Callinectes sapidus*. *J. Parasitol.* 33: 146-150.

Humes, A.G. 1942. The morphology, taxonomy and bionomics of the nemertean genus *Carcinonemertes*. *Ill. Biol. Monogr.* 18(4): 1-105.

Hutton, R.F., and F. Sogandares-Bernal. 1959. Notes on the distribution of the leech, *Myzobdella lugubris* Leidy, and its association with mortality of the blue crab, *Callinectes sapidus* Rathbun. *J. Parasitol.* 45: 384, 404, 430.

Jahromi, S.S. 1977. Occurrence of rhabdovirus-like particles in the blue crab, *Callinectes sapidus*. *J. Gen. Virol.* 36: 485-493.

Johnson, P.T. 1976a. A baculovirus from the blue crab, *Callinectes sapidus*. Page 24 *in* Proceedings of the First International Colloquium on Invertebrate Pathology and IXth Annual Meeting of the Society for Invertebrate Pathology, Kingston, Ont. Queen's University, Kingston, Ont.

Johnson, P.T. 1976b. A herpeslike virus from the blue crab, *Callinectes sapidus*. *J. Invertebr. Pathol.* 27: 419-420.

Johnson, P.T. 1976c. Bacterial infection in the blue crab, *Callinectes sapidus*: course of infection and histopathology. *J. Invertebr. Pathol.* 28: 25-36.

Johnson, P.T. 1977a. A viral disease of the blue crab, *Callinectes sapidus*: histopathology and differential diagnosis. *J. Invertebr. Pathol.* 29: 201-209.

Johnson, P.T. 1977b. Paramoebiasis in the blue crab, *Callinectes sapidus*. *J. Invertebr. Pathol.* 29: 308-320.

Johnson, P.T. 1977c. Reovirus-like virus of blue crabs. Pages 103-105 *in* C.J. Sindermann, ed. Disease diagnosis and control in North American marine aquaculture. Elsevier, New York.

Johnson, P.T. 1978a. New information on viral diseases of the blue crab, *Callinectes sapidus*. Page 55 *in* S.E. Milligan, ed. International Colloquium on Invertebrate Pathology and XIth Annual Meeting of the Society for Invertebrate Pathology, Prague, Czechoslovakia.

Johnson, P.T. 1978b. Viral diseases of the blue crab, *Callinectes sapidus*. *Mar. Fish. Rev.* 40(10): 13-15.

Johnson, P.T. 1980. Histology of the blue crab, *Callinectes sapidus*: a model for the Decapoda. Praeger, New York. 440 pp.

Johnson, P.T. 1983. Diseases caused by viruses, rickettsiae, bacteria, and fungi. Pages 1-78 *in* A.J. Provenzano, ed. The biology of Crustacea. Vol. 6. Pathobiology. Academic Press, New York.

Johnson, P.T. 1984. Viral diseases of marine invertebrates. Pages 65-98 *in* O. Kinne and H.P. Bulnheim, eds. Diseases of marine organisms. *Helgol. Meeresunters.* 37.

Johnson, P.T. 1986. Blue crab (*Callinectes sapidus* Rathbun) viruses and the diseases they cause. Pages 13-19 *in* H.M. Perry and R.F. Malone, eds. Proceedings of the National Symposium on the Soft-Shelled Blue Crab Fishery. Gulf Coast Research Laboratory, Biloxi, Miss.

Johnson, P.T. 1988. Development and morphology of an unusual nuclear virus of the blue crab *Callinectes sapidus*. *Dis. Aquat. Org.* 4: 67-75.

Johnson, P.T., and J.E. Bodammer. 1975. A disease of the blue crab, *Callinectes sapidus*, of possible viral etiology. *J. Invertebr. Pathol.* 26: 141-143.

Johnson, P.T., and C.A. Farley. 1980. A new enveloped helical virus from the blue crab, *Callinectes sapidus*. *J. Invertebr. Pathol.* 35: 90-92.

Johnson, P.T., and D.V. Lightner. 1988. Rod-shaped nuclear viruses of crustaceans: gut-infecting species. *Dis. Aquat. Org.* 5: 123-141.

Krantz, G.E., R.R. Colwell, and E. Lovelace. 1969. *Vibrio parahaemolyticus* from the blue crab *Callinectes sapidus*

in Chesapeake Bay. *Science* 164: 1286-1287.

Lear, D.W., Jr. 1963. Occurrence and significance of chitinoclastic bacteria in pelagic waters and zooplankton. Pages 594-610 *in* C. Oppenheimer, ed. Symposium on Marine Microbiology. Thomas, Springfield, Ill.

MacLean, S.A., and C.L. Ruddell. 1978. Three new crustacean hosts for the parasitic dinoflagellate *Hematodinium perezi* (Dinoflagellata: Syndinidae). *J. Parasitol.* 64: 158-160.

McKenna, S., M. Jansen, and M.G. Pulley. 1990. Shell disease of blue crabs, *Callinectes sapidus* in the Pamlico River, North Carolina. [N.C. Div. Mar. Fish.,] *Spec. Sci. Rep.* No. 51. 30 pp.

Messick, G.A., and V.S. Kennedy. 1990. Putative bacterial and viral infections in blue crabs, *Callinectes sapidus* Rathbun, 1896 held in a flow-through or a recirculation system. *J. Shellfish Res.* 9: 33-40.

Millikin, M.R., and A.B. Williams. 1984. Synopsis of biological data on the blue crab, *Callinectes sapidus* Rathbun. [U.N.,] *FAO* [Food Agr. Org.] *Fish. Synop.* No. 138, NMFS-S-138. 39 pp.

Moore, J.P. 1946. The anatomy and systematic position of *Myzobdella lugubris* Leidy (Hirudinea). *Not. Nat. (Phila.)* 184: 1-12.

Newcombe, C.L., and M.R. Rogers. 1947. Studies of a fungus parasite that infects blue crab eggs. *Turtox News* 25(9): 1-7.

Newcombe, C.L., F. Campbell, and A.M. Eckstine. 1949. A study of the form and growth of the blue crab *Callinectes sapidus* Rathbun. *Growth* 13: 71-96.

Newman, M.W. 1977. *Hematodinium* disease of blue crabs. Pages 211-213 *in* C.J. Sindermann and D.V. Lightner, eds. Disease diagnosis and control in North American marine aquaculture. Elsevier, New York.

Newman, M.W., and C.A. Johnson, III. 1975. A disease of blue crabs *(Callinectes sapidus)* caused by parasitic dinoflagellates, *Hematodinium* sp. *J. Parasitol.* 61: 554-555.

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

Newman, M.W., C.A. Johnson, III, and G.B. Pauley. 1976. A *Minchinia*-like haplosporidan parasitizing blue crabs, *Callinectes sapidus*. *J. Invertebr. Pathol.* 27: 311-315.

Overstreet, R.M. 1977. Microsporosis of blue crabs. Pages 200-203 *in* C.J. Sindermann and D.V. Lightner, eds. Disease diagnosis and control in North American marine aquaculture. Elsevier, New York.

Overstreet, R.M. 1978. Marine maladies? Worms, germs, and other symbionts from the northern Gulf of Mexico. Blossman Printing, Ocean Springs, Miss. 140 pp.

Overstreet, R.M. 1982. Metazoan symbionts of the blue crab. Pages 81-87 *in* H.M. Perry and W.A. Van Engel, eds. Proceedings of the Blue Crab Colloquium, Biloxi, Miss. Gulf States Marine Fisheries Commission, Ocean Springs, Miss.

Overstreet, R.M. 1983. Metazoan symbionts of crustaceans. Pages 155-250 *in* A.J. Provenzano, ed. The biology of Crustacea. Vol. 6. Pathobiology. Academic Press, New York.

Overstreet, R.M., and E.C. Whatley. 1975. Prevention of microsporidosis in the blue crab, with notes on natural infections. *Proc. World Maricult. Soc.* 6: 335-345.

Pauley, G.B., M.W. Newman, and E. Gould. 1975. Serum changes in the blue crab, *Callinectes sapidus*, associated with *Paramoeba perniciosa*, the causative agent of grey crab disease. *Mar. Fish. Rev.* 37(5-6): 34-38.

Pyle, R., and E. Cronin. 1950. The general anatomy of the blue crab *Callinectes sapidus* Rathbun. [Univ. Md.,] *Chesapeake Biol. Lab. Publ.* 87. 40 pp.

Ragan, J.G., and B.A. Matherne. 1974. Studies of *Loxothylacus texanus*. Pages 185-203 *in* R.L. Amborski, M.A. Hood, and R.R. Miller, eds. Proceedings of Gulf Coast Regional Symposium on Diseases of Aquatic Animals. *La. State Univ. Publ.* No. LSU-SG-74-05.

Reinhard, E.G. 1950. An analysis of the effects of a sacculinid parasite on the external morphology of *Callinectes sapidus* Rathbun. *Biol. Bull. (Woods Hole)* 98: 277-288.

Reinhard, E.G. 1956. Parasitic castration of Crustacea. *Exp. Parasitol.* 5: 79-107.

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

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

Rosen, B. 1970. Shell disease of aquatic crustaceans. Pages 409-415 *in* S.F. Snieszko, ed. A Symposium on Diseases of Fishes and Shellfishes. *Amer. Fish. Soc. Spec. Publ.* 5.

Rosenfield, A., L. Buchanan, and G.B. Chapman. 1969. Comparison of the fine structure of spores of three species of *Minchinia* (Haplosporida, Haplosporidiidae). *J. Parasitol.* 55: 921-941.

Sandoz, M.D., M.R. Rogers, and C.L. Newcombe. 1944. Fungus infection of eggs of the blue crab, *Callinectes sapidus* Rathbun. *Science* 99: 124-125.

Sawyer, R.T., A.R. Lawler, and R.M. Overstreet. 1975. Marine leeches of the eastern United States and the Gulf of Mexico with a key to the species. *J. Nat. Hist.* 9: 633-667.

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

Sawyer, T.K., and S.A. MacLean. 1978. Some protozoan diseases of decapod crustaceans. *Mar. Fish. Rev.* 40(10): 32-35.

Sindermann, C.J. 1989. The shell disease syndrome in marine crustaceans. *NOAA* [Nat. Ocean. Atmos. Admin.] *Tech. Mem. NMFS* [Nat. Mar. Fish. Serv.]-*F/NEC*-64.

Sindermann, C.J. 1990. Principal diseases of marine fish and shellfish. Academic Press, New York. 1037 pp.

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

Sizemore, R.K., and J.W. Davis. 1985. Source of *Vibrio* spp. found in the hemolymph of the blue crab, *Callinectes sapidus. J. Invertebr. Pathol.* 46: 109-110.

Sizemore, R.K., R.R. Colwell, H.S. Tubiash, and T.E. Lovelace. 1975. Bacterial flora of the hemolymph of the blue crab, *Callinectes sapidus:* numerical taxonomy. *Appl. Microbiol.* 29(3): 393-399.

Sparks, A.K. 1985. Synopsis of invertebrate pathology exclusive of insects. Elsevier Scientific Publishers, Amsterdam, The Netherlands. 423 pp.

Sprague, V. 1950. Notes on three microsporidian parasites of decapod Crustacea of Louisiana coastal waters. *Occas. Pap. Mar. Lab., La. State Univ.* No. 5: 1-8.

Sprague, V. 1963. *Minchinia louisiana* n. sp. (Haplosporidia, Haplosporididae), a parasite of *Panopeus herbstii. J. Protozool.* 10: 267-274.

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 *Pleistophora* (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. Pages 416-430 *in* S.F. Snieszko, ed. A Symposium on Diseases of Fishes and Shellfishes. *Amer. Fish. Soc. Spec. Publ.* No. 5.

Sprague, V. 1977. Systematics of Microsporidia. Pages 1-30 *in* L.A. Bulla and T.C. Cheng, eds. Comparative pathobiology, Vol. 2. Plenum, New York.

Sprague, V. 1978. Comments on trends in research on parasitic diseases of shellfish and fish. *Mar. Fish. Rev.* 40: 26-30.

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

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

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

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

Tharp, T.P., and C.E. Bland. 1977. Biology and host range of *Haliphthoros milfordensis. Can. J. Bot.* 55: 2936-2944.

Tubiash, H.S., R.K. Sizemore, and R.R. Colwell. 1975. Bacterial flora of the hemolymph of the blue crab, *Callinectes sapidus*: most probable numbers. *Appl. Microbiol.* 29(3): 388-392.

Vago, C. 1966. A virus disease in Crustacea. *Nature* 209: 1290.

Walker, G. 1974. The occurrence, distribution and attachment of the pedunculate barnacle *Octolasmis mulleri* (Coker) on the gills of crabs, particularly the blue crab, *Callinectes sapidus* Rathbun. *Biol. Bull. (Woods Hole)* 147: 678-689.

Welsh, P.C., and R.K. Sizemore. 1985. Incidence of bacteremia in stressed and unstressed populations of the blue crab, *Callinectes sapidus. Appl. Environ. Microbiol.* 50(2): 420-425.

Yudin, A.I., and W.H. Clark, Jr. 1978. Two viruslike particles found in the ecdysial gland of the blue crab, *Callinectes sapidus. J. Invertebr. Pathol.* 32: 219-221.

Yudin, A.I., and W.H. Clark, Jr. 1979. A description of rhabdovirus-like particles in the mandibular gland of the blue crab, *Callinectes sapidus. J. Invertebr. Pathol.* 33: 133-147.

Zobell, C.E., and S.C. Rittenberg. 1938. The occurrence and characteristics of chitinoclastic bacteria in the sea. *J. Bacteriol.* 35: 275-287.

☆ U. S. GOVERNMENT PRINTING OFFICE: 1992--500-031--40007

(continued from inside front cover)

68. **MARMAP Surveys of the Continental Shelf from Cape Hatteras, North Carolina, to Cape Sable, Nova Scotia (1984-87). Atlas No. 3. Summary of Operations.** By John D. Sibunka and Myron J. Silverman. July 1989. iv + 197 p., 36 figs., 2 tables. NTIS Access. No. PB90-160656/AS.

69. **The 1988 Experimental Whiting Fishery: A NMFS/Industry Cooperative Program.** By Frank P. Almeida, Thurston S. Burns, and Sukwoo Chang. August 1989. v + 16 p., 9 figs., 11 tables, 1 app. NTIS Access. No. PB90-160664/AS.

70. **Summer Distribution of Regulated Species on Georges Bank with Reference to the 1988 Experimental Whiting Fishery.** By Frank P. Almeida, Sukwoo Chang, and Thurston S. Burns. September 1989. v+ 25 p., 74 figs., 1 table. NTIS Access. No. PB90-206525/AS.

71. **Allocation of Statewide-Reported MRFSS Catch and Landings Statistics between Areas: Application to Winter Flounder.** By Frank P. Almeida. September 1989. v + 18 p., 5 figs., 6 tables, 2 app. NTIS Access. No. PB90-246745.

72. **Status of the Fishery Resources off the Northeastern United States for 1989.** By Conservation and Utilization Division, Northeast Fisheries Center. December 1989. iv + 110 p., 50 figs., 93 tables. NTIS Access. No. PB90-170622/AS.

73. **Surface and Bottom Temperature Distribution for the Northeast Continental Shelf.** By David G. Mountain and Tamara J. Holzwarth. December 1989. iii + 32 p., 31 figs. NTIS Access. No. PB90-252172/AS.

74. **Shell Disease of Crustaceans in the New York Bight.** By C.J. Sindermann (chair.), F. Csulak (EPA coord.), T.K. Sawyer (rapp.), R.A. Bullis, D.W. Engel, B.T. Estrella, E.J. Noga, J.B. Pearce, J.C. Rugg, R. Runyon, J.A. Tiedemann, and R.R. Young. December 1989. vii + 47 p., 7 figs., 4 tables, 4 app. NTIS Access. No. PB90-195058/AS.

75. **An Indexed Bibliography of Northeast Fisheries Center Publications and Reports for 1988.** By Jon A. Gibson. December 1989. iii + 32 p. NTIS Access. No. PB91-112912.

76. **Finfish Maturity Sampling and Classification Schemes Used during Northeast Fisheries Center Bottom Trawl Surveys, 1963-89.** By Jay Burnett, Loretta O'Brien, Ralph K. Mayo, Jeffrey A. Darde, and Margot L. Bohan. December 1989. iii+14 p., 11 tables, 1 app. NTIS Access. No. PB91-128389/AS.

77. **Shell Disease among Red Crabs Inhabiting Submarine Canyons of the New York Bight.** By Randall R. Young. December 1989. iii+9 p., 18 figs., 5 tables. NTIS Access. No. PB90-194762/AS.

78. **Seasonal Distribution Patterns of Commercial Landings of 45 Species off the Northeastern United States during 1977-88.** By Sukwoo Chang. October 1990. v+130 p., 246 figs. NTIS Access. No. PB91-160846.

79. **Contaminants in Sediment and Fish Tissue from Estuarine and Coastal Sites of the Northeastern United States: Data Summary for the Baseline Phase of the National Status and Trends Program Benthic Surveillance Project, 1984-1986.** By Vincent S. Zdanowicz and Donald F. Gadbois. December 1990. x + 138p., 67 figs., 21 tables. NTIS Access. No. PB92-147453/AS.

80. **Distribution of Sexually Immature Components of 10 Northwest Atlantic Groundfish Species Based on Northeast Fisheries Center Bottom Trawl Surveys, 1968-86.** By Susan E. Wigley and Wendy L. Gabriel. January 1991. iv + 17 p., 23 figs., 3 tables. NTIS Access. No. PB92-101617.

81. **Status of the Fishery Resources Off the Northeastern United States for 1990.** By Conservation and Utilization Division, Northeast Fisheries Center. January 1991. iv + 130 p., 50 figs., 82 tables. NTIS Access. No. PB91-213785.

82. **Response of the Habitat and Biota of the Inner New York Bight to Abatement of Sewage Sludge Dumping: Third Annual Progress Report–1989.** By Anne L. Studholme, Merton C. Ingham, and Anthony Pacheco, eds. February 1991. vi + 57 p., 74 figs., 20 tables, 1 app. NTIS Access. No. PB91-208199.