PRINCIPAL DISEASES OF COMMERCIALLY IMPORTANT MARINE BIVALVE MOLLUSCA AND CRUSTACEA

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ABSTRACT

Diseases of commercially important marine bivalve mollusks and crustaceans can cause mortalities in natural and captive populations. Oysters have suffered frequent and extensive mass mortalities. Epizootic disease has sometimes been indicated, as for example in "dermocystidium disease," caused by a fungus, in the Gulf of Mexico, and "Delaware Bay disease," caused by a protozoan, in the Middle Atlantic States. Fungus organisms have also been implicated in "shell disease" of European oysters and a fatal disease of bivalve larvae in hatcheries. Several species of haplosporidan Protozoa cause serious mortalities of oysters and mussels. Larger animal parasites, such as larval trematodes and parasitic copepods, can affect reproduction, growth, and survival of bivalve mollusks.

Diseases of crustaceans are usually less well known. Effects of pathogens are most apparent in captive populations, where mortalities may result from outbreaks of microbial agents. Lobsters have two known bacterial diseases: the often very lethal "gaffkaemia" which occurs in wild populations, but severely affects impounded stocks; and "shell disease," caused by chitin-destroying bacteria, which is important in captive populations, probably by interfering with respiration of infected individuals. Mortalities of captive blue crabs have also been attributed to possible respiratory impairment caused by ciliate gill associates. Microsporidan Protozoa can be important parasites of crabs and shrimps; depending on their habitat within the host, they may destroy muscle tissue or gonads and have been postulated as causes of mortality. Among the larger parasites of crustaceans, rhizocephalan barnacles have long been known to cause degeneration of gonads in crabs of many species.

The described pathogens of commercial invertebrate species probably constitute only a small percentage of the many disease agents that affect marine populations. Many mass mortalities have been described in the literature; disease has been implicated in some of them, but the etiologic agent rarely has been determined precisely, and the relative importance of other environmental factors has been assessed infrequently. Diseases and parasites can also exert significant "background" effects such as continued low-level mortality, depressed reproductive capacity, and increased susceptibility to predation.

Disease control measures are possible and have been applied to a few populations of sedentary inshore marine invertebrates. Possible methods of control include quarantine, selective breeding of disease-resistant strains of shellfish, environmental manipulation (dredging of growing areas, chemical treatments, control of density of planting, and scheduled harvest), and more extensive use of artificial environments such as hatcheries and artificial ponds.

As understanding of factors that influence the numbers of animals in the sea has expanded, it has become evident that disease, among other environmental variables, can drastically affect abundance. This fact has been clearly demonstrated in populations of sedentary inshore invertebrates. Many marine invertebrate species, harvested in great numbers, constitute marine crops of high value. Some, such as mussels, oysters, clams, crabs, lobsters, and shrimps, occur in inshore or estuarine waters, and have been cultivated in varying degrees in different parts of the world. Under natural conditions or under cultivation, mass mortalities occasionally occur; here disease can be an important contributing factor.

The word "disease," as used in this paper, includes abnormalities resulting from microbial pathogens or parasite invasion, and tumors. Not included are genetically or environmentally induced abnormalities, or physiological disturbances not related to an infectious agent or parasite. For each host group considered in this review a sequence of diseases, beginning

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with those caused by bacteria (virus diseases, with but a single exception (Vago, 1966), have not been identified in marine invertebrates) and progressing to fungi, protozoans, and larger parasites, has been followed. Often a host species may be infected by several well-defined pathogens, as well as assorted parasites that have variable impact on the host population. The common and the scientific names of parasites and hosts are usually both given when the organisms are first mentioned in the text, after that either one may be used.

No general review has been made of the literature on diseases of marine invertebrates (Steinhaus, 1965), but particular groups—especially those of commercial importance—have received some attention. Dollfus (1921a), Pelseneer (1928), Ranson (1936), and Fischer (1951) have summarized information about the parasites and diseases of mollusks—particularly oysters—and Hutton, Sogandares-Bernal, Eldred, Ingle, and Woodburn (1959) reported on parasites and diseases of some of the commercial shrimps. Certain general aspects of invertebrate diseases, such as immune mechanisms, have been considered (Cantacuzène, 1923, 1928; Huff, 1940; Baer, 1944; Steinhaus, 1949; Stauber, 1961), and a few research groups, such as Frederik B. Bang and his associates at The Johns Hopkins University, and Albert K. Sparks and his co-workers at the University of Washington, have been concerned with comparative pathology of invertebrates (Bang, 1956, 1961, 1962; Bang and Bang, 1962; Bang and Lemma, 1962; Levin and Bang, 1964; Rabin and Bang, 1964; Sparks and Pauley, 1964; Pauley and Sparks, 1965).

Much of our knowledge about diseases of marine invertebrates concerns species of economic importance, particularly the bivalve molluscan and crustacean shellfish. This paper is concerned only with the important diseases of these two groups. We have attempted to encompass as much literature as possible from widely separated areas. Many diseases of marine invertebrates are inadequately characterized, and it is probable that others have not even been recognized. Microbial pathogens that have been implicated in mass mortalities include bacteria, fungi, and protozoans. Several of the larger parasites have been found to be pathogenic under specific conditions. Not included here are most of the parasites and diseases of noncommercial species—those species that may be of great significance in the cycles of life in the sea, but which are not of significant direct importance as food for humans. Among the groups thus excluded are gastropods and cephalopods, barnacles, copepods, and most of the smaller crabs. Also excluded are many diseases that have been incompletely described in the scientific literature.

A summarization of knowledge in any area of research, however specialized in its scope, is subjective and in some ways frustrating to the reviewer; yet a consolidation of research results can be useful, particularly to the nonspecialist. Much published information about diseases of marine bivalve mollusks and crustaceans has accumulated, and at least a representative fraction of the available literature has been considered in this paper. Preparation of a bibliography of molluscan shellfish diseases has been a continuing project of the BCF (Bureau of Commercial Fisheries) Biological Laboratory, Oxford, Md., for 6 years; this bibliography, as well as standard bibliographic and abstracting sources, has been used in preparing the manuscript. There is little representation of the Russian literature—this may be in part a reflection of the relative lack of emphasis placed on shellfish in Russian fishery research, as well as the limited availability of translations of Russian literature. Some of the older European literature, particularly that on specific parasites of invertebrates, has not been considered in this paper but is accessible through references cited in more recent publications. Although necessarily limited in content, this review attempts to assess the state of knowledge about the role of disease in two major groups of commercial marine invertebrates.

DISEASES OF BIVALVE MOLLUSCA

Most of the commercial bivalve mollusks occur in shallow inshore waters, often intertidally, where they are accessible to quantitative evaluation and observation. Unusual mortalities are more apparent here than in offshore populations. As a result, literature on

mass mortalities of species such as oysters and mussels is voluminous. Disease has sometimes been demonstrated to be the cause of deaths; in other situations, disease has been strongly suspected, or the cause has not been determined. Within the past decade knowledge about molluscan shellfish diseases has increased at a greatly accelerated pace, largely because of concern about mortalities which have occurred in widely scattered populations. Literature on oyster diseases is most abundant; that on mussel and clam diseases is less voluminous.

OYSTERS

The 20th century has been a difficult and troublesome period for oysters (family Ostreidae) in many parts of the world (Orton, 1924a; Roughley, 1926; Gross and Smyth, 1946; Logie, 1956; Mackin, 1961; Sindermann, 1966c). Decline in abundance of oysters actually started late in the 19th century, probably caused in large part by indiscriminate harvesting and destruction of beds. Extensive mortalities from unknown causes also contributed to decreased oyster production. The rate of decline on the North American east coast and in other geographic areas has recently increased because of large-scale mortalities, several of which have been caused by disease. Largely because of their worldwide economic importance, oysters are among the most thoroughly studied of marine animals—especially their diseases and parasites. Interest in oyster diseases has logically arisen from catastrophic mortalities in many parts of the world. Many unsolved problems remain but the body of literature is large, and is growing rapidly.

Among the important diseases of oysters are microbial diseases and those caused by helminths and parasitic crustaceans.

Microbial Diseases

Bacteria, fungi, and protozoans are the principal causes of microbial diseases in oysters.

Bacteria.—Reports of mass mortalities of Pacific oysters, *Crassostrea gigas* (Thunberg), have been published recently in Japan (Fujita, Matsubara, Hirokawa, and Araki, 1953, 1955; Takeuchi, Takemoto, and Matsubara, 1960; Ogasawara, Kobayashi, Okamoto, Furukawa, Hisaoka, and Nogami, 1962; Imai, Numachi, Oizumi, and Sato, 1965; Kan-no, Sasaki, Sakurai, Watanabe, and Suzuki, 1965; Mori, Imai, Toyoshima, and Usuki, 1965; Mori, Tamate, Imai, and Itikawa, 1965; Numachi, Oizumi, Sato, and Imai, 1965; Tamate, Numachi, Mori, Itikawa, and Imai, 1965). Takeuchi et al. (1960) implicated a gram-negative, motile, 1– to 3–μ bacillus, probably an *Achromobacter*, in large-scale mortalities in Pacific oyster culture areas of Hiroshima Bay since 1946. Experimental infections were achieved with cultured bacteria, but the organisms could be isolated from healthy as well as sick oysters, and from sea water. Moribund oysters had diffuse cell infiltration, massive increase in bacterial numbers, and tissue necrosis.

Numachi et al. (1965) found up to 20 percent infection with gram-positive bacteria (not further identified) in oysters during mass mortalities in Matsushima Bay, Japan, in the early 1960's. The disease was called "multiple abscesses," but the authors did not think that a causal relation existed between bacteria and mortalities. A similar disease was found in 1965 by staff members of the BCF Biological Laboratory, Oxford, Md., in seed oysters (less than 1 year old) imported to the U.S. west coast from Matsushima Bay, and in adult oysters from Willapa Bay, Wash. The disease has been labeled "focal necrosis" (fig. 1). Studies of the etiologic agent and its pathogenicity

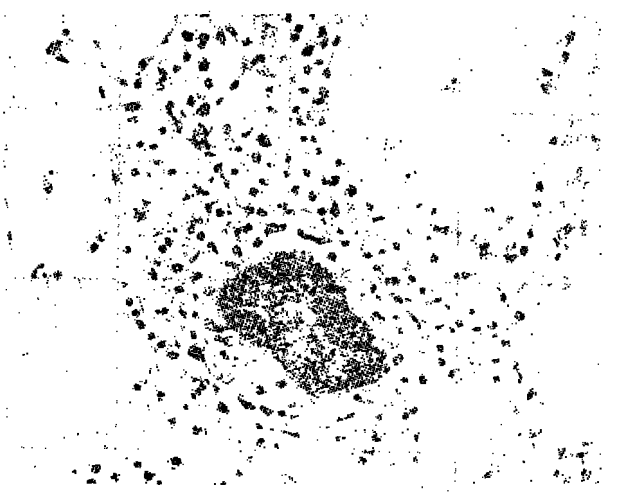

FIGURE 1.—"Focal necrosis" in connective tissue of Pacific oyster from Willapa Bay, Wash., surrounded by extensive leucocytic infiltration (× 700).

are in progress. The disease affects seed oysters as young as 6 months, as well as adults. The necrotic foci or multiple abscesses may represent the resistant or arrested disease state, and the active, fulminating phase may have already killed susceptible members of the population.

Several bacterial pathogens of bivalve larvae have been isolated (Guillard, 1959; Tubiash, Chanley, and Leifson, 1965). Identified only as *Aeromonas* sp. or *Vibrio* sp., the organisms killed larvae and juveniles of five bivalve species tested, including American oysters, *Crassostrea virginica* (Gmelin), and European oysters, *Ostrea edulis* L., but did not affect adults.

Fungi.—Oysters have several fungus diseases, some of serious consequence. Identification of fungus pathogens, especially in the early literature, has often been tentative, and only a few adequate characterizations of etiologic agents have been made.

A relatively well-known fungus infecting oysters from the Atlantic and Gulf coasts of the United States is *Dermocystidium marinum*. First described by Mackin, Owen, and Collier (1950), the pathogen has been the subject of much research. A useful diagnostic technique, based on antibiotic fortified fluid thioglycollate medium, was devised by Ray (1952, 1966b) during attempts to culture the organism. A number of authors have shown that *D. marinum* causes oyster mortalities (Mackin et al., 1950; Mackin, 1953; Andrews and Hewatt, 1954; Hewatt and Andrews, 1954; Ray, 1954a, 1954b, 1954c). Pathological changes in infected oysters were described by Mackin (1951). Invasion takes place through the gut epithelium and possibly through the mantle. The epithelium is destroyed; the parasite lyses the basement membrane and is distributed by the blood to all parts of the body (fig. 2). All tissues are invaded and damaged, and multiple abscesses are formed. Normal gonad development is inhibited, infected oysters become severely emaciated (Ray, Mackin, and Boswell, 1953; Ray, 1954b), and growth is retarded (Menzel and Hopkins, 1955b).

Temperature is important in the epizootiology of dermocystidium disease (Hewatt and Andrews, 1956). Infections and associated mortalities rise during the warmer months and decline during colder periods. Mortalities decline in winter, probably because of reduced parasite metabolism, rather than elimination of the organism. Failure to find *D. marinum* consistently north of Chesapeake Bay suggests that prolonged low temperature may be a significant limiting factor. Andrews (1965) found that *D. marinum* proliferates readily only at temperatures above 25° C., and overwinters as subpatent infections. His further observation, that the partial destruction of oyster populations by a protozoan disease resulted in decreased prevalence of the fungus organism, suggests that *Dermocystidium* is dependent upon direct transmission from one oyster to another. Other evidence for direct transmission was provided by Ray and Mackin (1955).

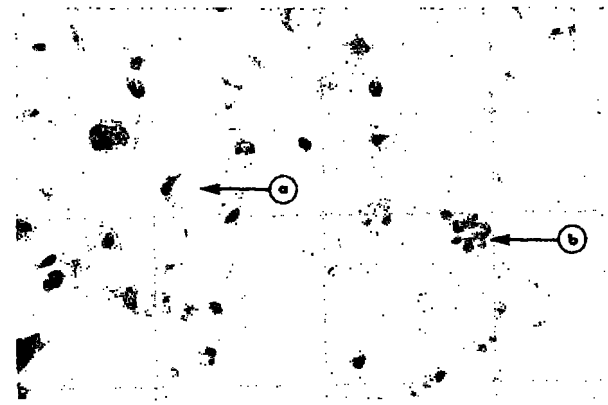

FIGURE 2.—Dermocystidium disease of American oysters. Histologic section of infected oyster showing diagnostic "hypnospore" (a) and "rosette" (b) (× 1,000).

Infections and resulting mortalities are reduced in salinities below 15 o/oo. Ray (1954b) found evidence that low salinity retarded development of terminal infections in laboratory populations. Ray and Chandler (1955) suggested that excessively high salinities may also be unfavorable for *Dermocystidium*. Mackin (1956) found a positive correlation between high salinity and high incidence of the fungus, but he observed that the salinity tolerance range was wide in experimental studies. Dilution of infective elements by inflow of fresh water was suggested as an important limiting factor and a possible control measure.

D. marinum is abundant in waters of the southern United States. Ray (1966a) who surveyed the occurrence of the fungus in the Gulf of Mexico in 1961 and 1962, found infections in 35 of 39 oyster samples, and prevalences as high as 100 percent. Hoese (1964) was able to find *D. marinum* in the digestive tracts and feces of fish, oyster drills, and crabs that had fed on dying and dead infected oysters. He speculated that transmission of the fungus might be furthered by scavengers and that scavengers may release the parasite from host tissue.

D. marinum is common in oysters from most high-salinity areas of the South Atlantic and Gulf of Mexico coasts of the United States, but is absent in a few such areas. Hoese (1963) attempted assays of water samples from different coastal locations in one section of the Texas coast to determine their inhibitory effects on development of fungus hypnospores. Although results were not conclusive, water samples from certain localities apparently stopped hypnospore development. Hoese speculated that the absence of *D. marinum* may be related to those *Spartina* salt marshes where consistently high salinities occur.

In addition to its common occurrence in *C. virginica*, *Dermocystidium* has been found in other species. Ray (1954b) reported it in the leafy oyster, *Ostrea frons* L., from Florida and in horse oysters, *O. equestris* Say, from Texas. The organism was not found, however, in the mangrove oysters, *C. rhizophorae* (Guilding), from Puerto Rico, in *O. edulis* from Holland, or in the rock oyster, *C. commercialis* (Iredale and Roughley), from Australia. *O. lurida* (Carpenter), the Olympia oyster, was experimentally infected by exposure to infected *C. virginica*. *Dermocystidium*-like organisms have also been seen in other mollusks and annelids. Andrews (1955) found what he termed "*Dermocystidium*-like" organisms in 12 of 16 mollusk species from the Chesapeake Bay area.

This important fungus pathogen of oysters continues to be the subject of much research. Knowledge of its biology has been summarized by Ray (1954b), Ray and Chandler (1955), Andrews and Hewatt (1957), and Mackin (1962). Mackin and Boswell (1956) proposed a life cycle for *D. marinum* that included a saprophytic stage leading to production of an infective spore. Recently, Perkins and Menzel (1966) described motile biflagellate stages that were also postulated to be infective to oysters. Mackin and Ray (1966) grew the organism on beef-serum agar plates and suggested that it belongs in the genus *Labyrinthomyxa*, a member of the Labyrinthulales. Culture of a *Dermocystidium* similar to *D. marinum* in chemically defined medium (Goldstein, Belsky, and Chasak, 1965) should make possible the study of isolates from many areas to determine whether one species or a species complex exists, and should permit more precise determination of the taxonomic affinities of the *Dermocystidium* group of protistan parasites.

Korringa (1947, 1951a, 1951c) reported that mortalities of the European oyster in Holland, beginning in 1930, were caused by a fungus disease characterized by formation of green or brown pustules on the inner shell surfaces. Activity of the fungus varied directly with temperature, and the outbreak was said to be intensified by widespread use of cockle shells as spat collectors. Thin parts of oyster shells were perforated by the disease agent, which proliferated after reaching the interior surfaces. The fungus had been identified earlier as a species of *Monilia* by Voisin (1931), who found the infection, called "shell disease," in 40 percent of oysters imported into France from Holland in 1931. Cole (1950) and Cole and Waugh (1956) found infections in the European oyster from Brittany and in Portuguese oysters, *C. angulata* (Lamarck), grown in England. Infections were common in beds where old shells were abundant. Cole and Hancock (1956) found the disease in almost all beds of native European oysters, and described two distinct forms: the typical one characterized by greenish rubbery warts and knobs on the inside of the shell, particularly in the region of the muscle attachment; and an atypical form in which young oysters had thickened shells with numerous white patches but had no deformation of the muscle attachment area.

Another disease of the European oyster, which may be identical to shell disease, has been misnamed "foot disease" or "maladie du pied" (Dollfus, 1921a). It has long been known on the coast of France; Giard (1894) described

its etiologic agent as a bacterium, *Myotomus ostrearum*, but further definitive studies of the causative organism are needed. The disease is localized in the shell under the attachment of the adductor muscle, where it causes roughening and blistering of the shell and degeneration of adjacent muscle tissue. The muscle may become detached as irregular cysts are formed. Major mortalities occurred on oyster beds at Arcachon, France, in 1877 (Hornell, 1910; Orton, 1937). The cause was not determined, but some evidence of "foot disease" was found. Galtsoff (1964) reported the rare occurrence of the disease in the American oyster from the southern United States, but did not consider it a serious threat to oyster populations. Durve and Bal (1960) reported the rare occurrence of a shell disease which they considered to be "maladie du pied" in the backwater oyster, *C. gryphoides* (Schlotheim) from India.

Davis, Loosanoff, Weston, and Martin (1954) isolated a fungus, later described as *Sirolpidium zoophthorum* (Vishniac, 1955), from hatchery-produced oyster and clam larvae. The infections were rare, but they produced occasional epizootics that killed most of the cultured larval population in 2 to 4 days. Juvenile as well as larval bivalves were infected; growth ceased and death followed soon after infection. Infected cultures of bivalve larvae contained large numbers of motile biflagellate zoospores of the fungus. The authors speculated that an epizootic of the fungus could occur among lamellibranch larvae in nature.

There are several inconclusive reports of organisms resembling actinomycetes in oysters. Eyre (1924) reported *Cladothrix dichotoma* from oysters examined during the great mortalities of 1919–23 in western Europe. The isolate was not pathogenic in experimental studies. Dollfus (1921a) stated that Eyre's isolate was a species of *Nocardia*. Pettit (1921) also identified a *Nocardia* from *O. edulis*, but Dollfus (1921b) considered this to be merely normal cell reticulum of the oyster. Mackin (1962) described a "mycelial disease" of *Crassostrea virginica*, which he thought might be caused by an actinomycete. We have recently seen an organism similar to that described by Mackin in *C. angulata* from France.

Protozoa.—A variety of Protozoa parasitize oysters (Rosenfield, 1964; Sindermann, 1966b), and certain Sporozoa [1] are serious pathogens. Two haplosporidans, *Minchinia costalis* (Wood and Andrews) and *M. nelsoni* Haskin, Stauber, and Mackin, have caused oyster mortalities on the North American east coast within the past decade (fig. 3).

M. costalis is found in seaside bays of Maryland and Virginia, along the lower eastern shore of Virginia, and in Delaware Bay (Andrews, Wood, and Hoese, 1962; Wood and Andrews, 1962; Sprague, 1963; Haskin, Stauber, and Mackin, 1966; Couch, 1967b). First recognized in moribund and dead oysters from Hog Island Bay, Va., in 1959 by Wood and Andrews, *M. costalis* was held responsible, on the basis of epizootiological evidence, for sharp peaks of mortality in early summer. The pathogen and mortalities caused by it continue to characterize Maryland and Virginia seaside oyster populations.

The second haplosporidan species, *M. nelsoni*, has a wider distribution—from Connecticut to North Carolina. It has caused extensive mortalities and drastic decline of the oyster fishery in Delaware Bay beginning about 1957 and in lower Chesapeake Bay beginning in 1959 (Mackin, 1960; Engle and Rosenfield, 1963; Andrews, 1964). In each affected area, mortalities have often exceeded 95 percent for several years. Because of the severe impact of the *M. nelsoni* epizootic on oyster stocks of the Middle Atlantic States, a number of research groups—university, State, and Federal—have participated in scientific studies since the late 1950's, and significant papers have been published recently. Haskin et al. (1966) named the plasmodial stage of the parasite as *Minchinia nelsoni:* Couch, Farley, and Rosenfield (1966) associated the plasmodium with spore and prespore stages; and Barrow and Taylor (1966) confirmed, with immunological techniques, the association of plasmodium and spore. Andrews (1964, 1966) described aspects of the epizootiology of the disease in Virginia waters, and Haskin, Canzonier, and Myhre (1965) briefly summarized the epizootiology in Delaware Bay.

[1] Throughout this paper an attempt has been made to conform to the revised classification of the Protozoa as proposed by Honigberg, Balamuth, Bovee, Corliss, Gojdics, Hall, Kudo, Levine, Loeblich, Weiser, and Wenrich (1964), and modified by Sprague (1966a).

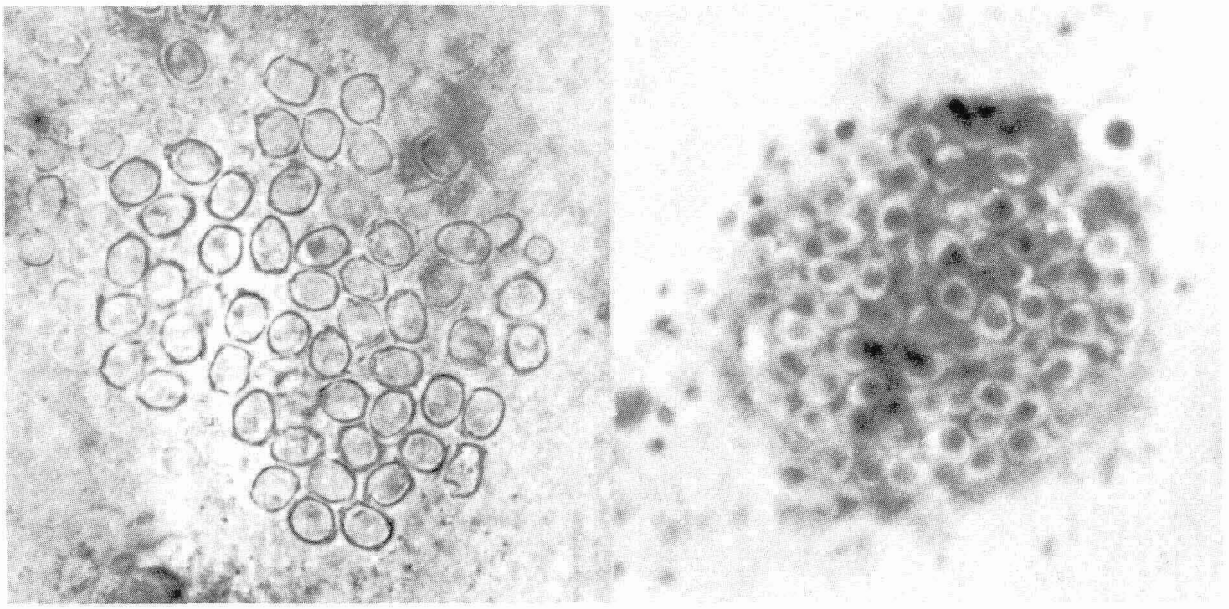

FIGURE 3.—Sporocysts of *Minchinia nelsoni* (formerly known as "MSX"), the etiolic agent of "Delaware Bay disease" in an American oyster—fresh preparation at left, and stained section at right (× 1,000).

Farley (1965), after completing a 5-year histopathological study of Chesapeake Bay oysters, categorized natural infections according to extent of invasion and nature of host response.

As an example of the effects of *M. nelsoni* on the U.S. oyster fishery, landings in New Jersey waters of Delaware Bay in the late 1940's and early 1950's had fluctuated around 6 million pounds of shucked meats until the mid-1950's, when disease decimated the stocks. Landings fell precipitously to a low of 167,000 pounds in 1960, and no significant recovery has occurred (fig. 4). Comparable effects have been felt in the high-salinity waters of lower Chesapeake Bay, another major oyster producing area.

Thus far, one alleviating influence seems to be salinity. *M. nelsoni* occurs in waters whose salinity consistently exceeds 15 o/oo; during 3 years (1963–65) of drought along the Atlantic coast, the pathogen invaded areas of middle Chesapeake Bay formerly free of the disease (Rosenfield and Sindermann, 1966). Temperature may also be important, since the pathogen appears to be quiescent during the winter.

Although several life history stages have been recognized for both species of *Minchinia*, and concurrent infections have been found (Couch, 1967b), routes of infection and methods of transmission are still unknown. Because several research groups are actively concerned with oyster diseases on the U.S. east coast, particularly with *M. nelsoni*, increased understanding of this and other pathogens should develop rapidly. Recently, we have seen in Pacific oysters from Taiwan plasmodial parasites morphologically very similar to those found in American oysters.

Léger and Hollande (1917) described another haplosporidan, *Chytridiopsis ovicola*, infecting the eggs of European oysters taken at Marennes, France. The parasite was relatively rare and occurred only in certain ovarian follicles of parasitized oysters.

Nematopsis ostrearum Prytherch, a gregarine parasite of the American oyster, was held (Prytherch, 1938, 1940) to be the cause of extensive mortalities in Virginia and Louisiana. Later studies (Sprague, 1949; Sprague and Orr, 1955) indicated, however, that *Nematopsis* did not cause deaths of oysters and suggested that *Dermocystidium* infections may have complicated earlier results. Owen, Walters, and Bregan (1952) found no correlation between *Nematopsis* infections and oyster mor-

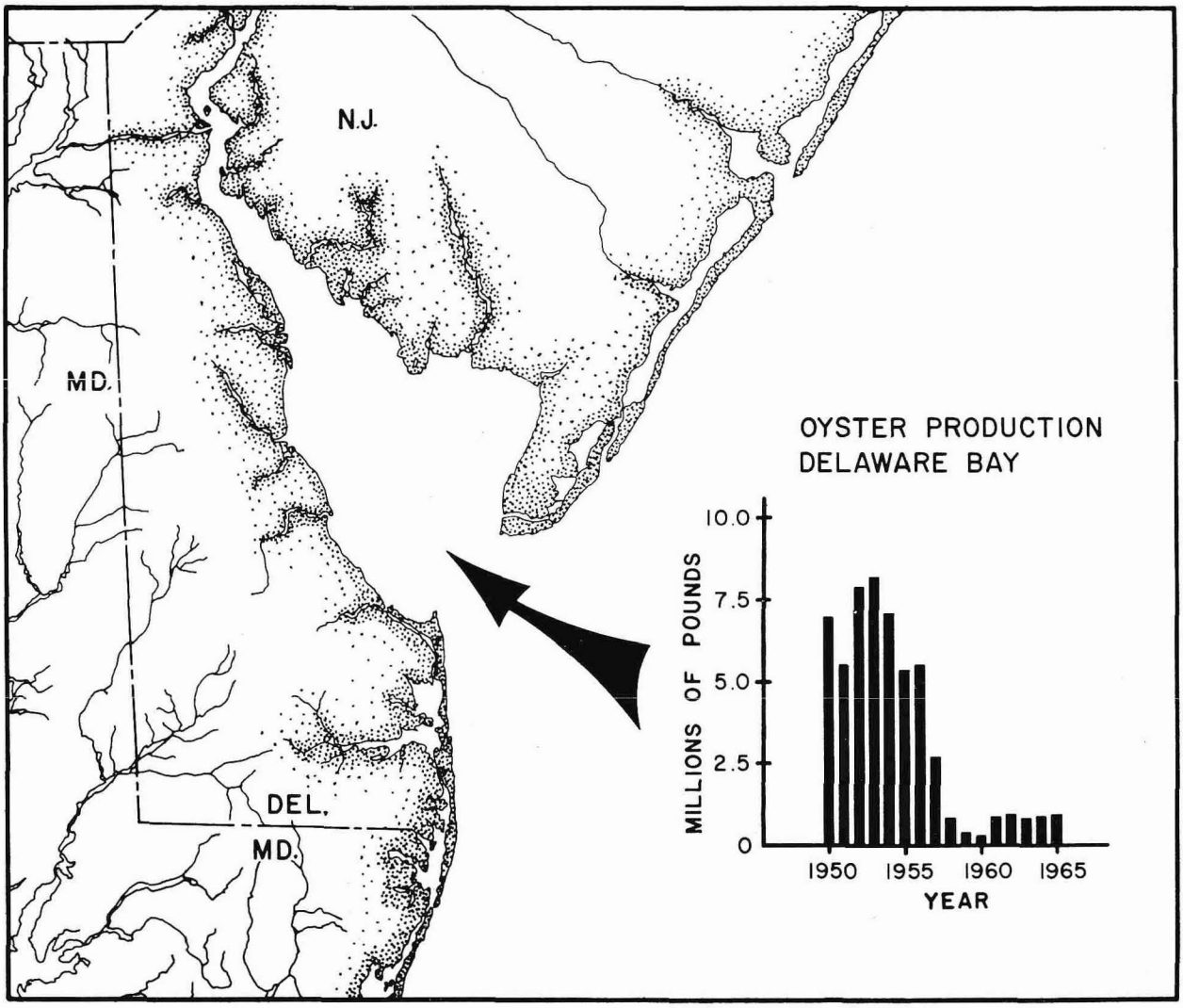

FIGURE 4.—Oyster production in New Jersey waters of Delaware Bay, 1950–65.

talities in Louisiana. Sprague and Orr (1953, 1955) demonstrated that *N. ostrearum* as described by Prytherch, was actually two species, which they designated *N. ostrearum* (emended) and *N. prytherchi*. Spores of *N. prytherchi* are larger and more elongate, localize in the gills rather than in the mantle, and have vegetative stages only in the crab *Menippe mercenaria* (Say). The life cycle of *N. ostrearum* includes a mud crab host—*Panopeus herbstii* Milne-Edwards, *Eurypanopeus depressus* (Smith), or *Eurytium limosum* (Say). In many oyster-growing areas most oysters are infected, although infections are rarely heavy. Mackin (1962) has pointed out the lack of tissue reaction to the parasite and the lack of evidence for existence of lysins or toxins. Feng (1958) found that a dynamic equilibrium existed between acquisition by the oyster of new parasites and elimination of spores.

Nematopsis was found to be widely distributed on the Atlantic and Gulf coasts of the United States by Landau and Galtsoff (1951). They found heavy infections in oysters from certain localities, such as the mouth of Chesapeake Bay, but relations with other ecological factors such as abundance of crabs were not apparent. The intensity of infection was cumulative and increased with age of the host. No evidence was obtained to indicate that *Nematopsis* infection caused poor meats or mortality of oysters.

Hexamita sp., a flagellate protozoan, occurs frequently in the digestive tract of oysters, but its parasitic or saprozoic role has not been adequately determined. First described by Certes (1882) as a commensal in European oysters, the flagellate was later held responsible for oyster mortalities from "pit disease" in Holland (Mackin, Korringa, and Hopkins, 1952). Clear evidence of pathological effects was not obtained, and heavy bacterial infections further complicated the study. *Hexamita* was also blamed for mortalities of Olympia oysters in the State of Washington (Stein, Denison, and Mackin, 1961), but again clear evidence of pathogenicity was not presented. Scheltema (1962), who examined the relation between *Hexamita* and American oysters from Delaware Bay, concluded that the organism did not contribute significantly to deaths of oysters. He suggested, as did Stein et al. (1961), that *Hexamita* may act as a pathogen during periods of low environmental temperatures and low host metabolism but that prevalence declines at higher temperatures because the processes in oysters which act to remove the trophozoites exceed the reproductive rate of the flagellate. We have recently seen *Hexamita* in Pacific oysters from Korea and Taiwan.

Several ciliate parasites have been described from American oysters. A member of the genus *Sphenophrya* was reported by the BCF Biological Laboratory, Oxford, Md. (Anonymous, 1965), as the cause of an oyster disease characterized by formation of large cysts on the gills. Richardson [2] and Laird (1961) identified ciliates in the gut of *C. virginica* from Prince Edward Island, Canada, as *Orchitophrya stellarum* Cépède. Prevalence was low, but infections were heavy and the intestinal epithelium had been invaded. *O. stellarum* is known as a serious pathogen of starfish, in which it causes gonad destruction (Cépède, 1911; Smith, 1936; Vevers, 1951). Laird speculated that the organism may be a regular and possibly harmful parasite of oysters and that starfish may become infected from them.

Mackin (1962) mentioned a ciliate parasite of oysters from the Atlantic and Gulf coasts of the United States, which he considered to be *Ancistrocoma pelseneeri*, a well-known parasite of mussels. The ciliates were abundant in the digestive tracts of oysters infected with *Dermocystidium marinum*, but Mackin did not believe that they were pathogenic to the oyster host.

Two amebae are known from American oysters. Hogue (1914, 1921) described *Vahlkampfia calkensi* and *V. patuxent*, which are parasitic in the digestive tract. She distinguished the two species on the basis of differences in the cyst wall. No evidence of pathogenicity was found, nor were these forms demonstrated to be other than saprozoic. Additional ameboid organisms isolated from American oysters were reported briefly by Sawyer (1966).

Diseases caused by Helminths

Both trematodes and cestodes parasitize oysters.

Trematodes.—European and American oysters are parasitized by larval trematodes of the genus *Bucephalus*. *B. haimeanus* was first reported by Lacaze-Duthiers (1854) from European oysters in the Mediterranean Sea, and *B. cuculus* was described by McCrady (1874) in American oysters from South Carolina. Sporocysts occur in the gonad and digestive gland of the oyster (fig. 5), and sterilize the host. The tentative life cycle of the parasite (Tennent, 1906) includes minnows (Cyprinidae) or mullets (Mugilidae) as second intermediate hosts, and gars (Lepisosteidae) as definitive hosts. Hopkins (1954, 1957b) reported parasitization of more than one-third of the oyster population in localized areas of the United States, although prevalence generally was much lower, particularly in open waters. Menzel and Hopkins (1955a, 1955b) suggested that early infections temporarily stimulate growth of the oyster, but that older infections retard growth. Hopkins (1957b) made the interesting, if somewhat facetious, observation that *Bucephalus* might be considered a gastronomically beneficial parasite in southern waters, since infected oysters have an excellent flavor and are fat-looking and glycogen-rich throughout the year, whereas normal oysters are spawned out, thin, and relatively tasteless during part of the year.

[2] Data provided in Fisheries Research Board of Canada, Manuscript Report Series (Biology), mimeographed, unnumbered, 1939, "Report on the studies of eastern coast oysters during the season of 1939," by L. R. Richardson.

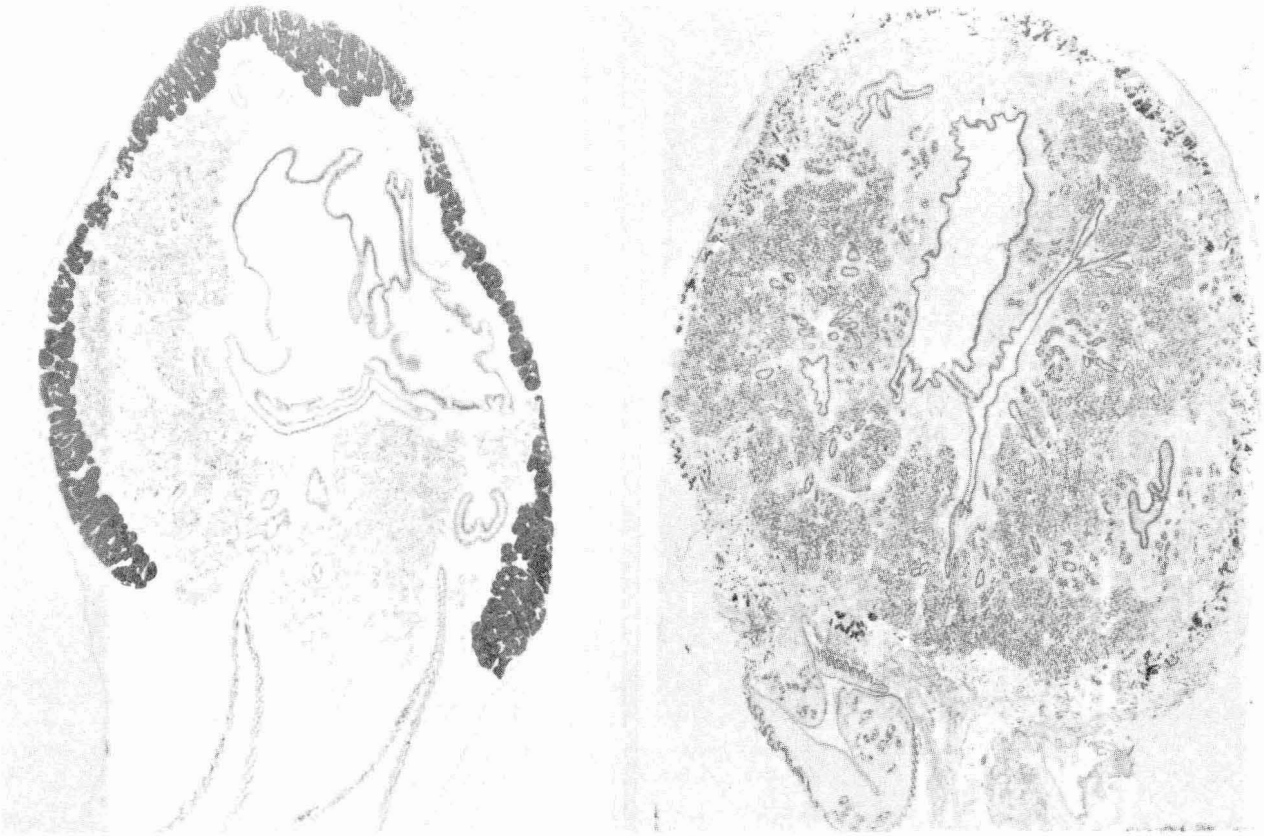

FIGURE 5.—Histological sections of oysters: (a) normal, and (b) parasitized by *Bucephalus* (× 3.5). Note in (b) the almost complete destruction of gonad tissue, which stains darkly in (a).

Recently, Cheng (1965) and Cheng and Burton (1965) used histochemical methods to examine the host-parasite relation of the American oyster and the larval trematode, *Bucephalus* sp. Parasitization caused marked changes in the distribution of fats.

Larval trematodes of the family Bucephalidae also occur in New Zealand oysters, *Ostrea lutaria* Hutton. Millar (1963) reported that oysters imported from New Zealand and maintained for breeding studies in Scotland were frequently parasitized and that the percentage mortality was much higher among parasitized individuals than among normal ones. We have recently found Pacific oysters from Taiwan to be infected with larval *Bucephalus*.

Hyperparasitization of sporocysts of *Bucephalus* in American oysters from the Gulf of Mexico was reported by Mackin and Loesch (1955). This hyperparasitization produced blackish discoloration of oyster mantle and viscera, and destruction of sporocysts, followed by release of the haplosporidan spores into host oyster tissue. Pronounced cellular reaction was elicited in the localized areas where spores were found in oyster tissue; the authors described some abnormal development of the hyperparasite in such tissue. The hyperparasite was not named, but it was considered on the basis of spore morphology to be a haplosporidan distinct from the parasite *Urosporidium pelseneeri* (Caullery and Chapellier) found in clams of the genus *Donax*.

Sprague (1964) described a microsporidan hyperparasite, *Nosema dollfusi*, of *Bucephalus* and speculated that escape of the protozoan into the tissues of the oyster could contribute to the death of the molluscan host. Shuster and

Hillman (1963) and Cheng (1964) made a similar speculation about haplosporidan hyperparasites of oysters.

Other larval trematodes occur on and in oysters. Fujita (1925, 1943) described *Gymnophalloides tokiensis*, a metacercaria which encysts, often in great numbers, on the mantle and gills. The host's physiology is disturbed, growth is halted, and reproduction is inhibited. Marine birds are definitive hosts for the parasite. Metacercariae of *Proctoeces ostrea* Fujita are also found in Japanese oysters. About 10 percent of the oysters in Hiroshima Bay were infected by the larval trematode, which localizes in gonad tissue. European oysters harbor the related *Proctoeces maculatus* Looss. Definitive hosts are labrid fishes in Europe, and snappers and red groupers, *Pagrosomus major* and *Epinephetus akaara,* in Japan.

Massive invasion by metacercariae in American oysters from the Texas coast was reported recently by Little, Hopkins, and Schlicht (1966). Feeding experiments showed that the trematodes were *Acanthoparyphium spinulosum* (Johnston), which matures in the intestine of shore birds. Most of the oysters examined had metacercariae in the mantle; the number averaged 45 worms per oyster.

Cestodes.—Oysters in several regions of the world are parasitized by larval cestodes of the genus *Tylocephalum*. These parasites are Lecanicephaloidea that occur as adults in the digestive tracts of elasmobranchs. Sparks (1963) reported heavy infections of *Tylocephalum* in American oysters introduced in Hawaii. In an addendum, Sparks noted that oysters from Florida had been reported by the BCF Biological Laboratory, Oxford, Md., to harbor similar larval cestodes (fig. 6). We have found similar larvae in oysters from Georgia and North Carolina. The coracidium of *Tylocephalum* was recently reported in the stomach and gills of American oysters collected at Pearl Harbor, Hawaii (Cheng, 1966). Penetration of gill or digestive epithelium was postulated from study of histologic sections. The pronounced cellular reaction in the subepithelial tissues—including encapsulation of the larvae—was described. Larval cestodes, probably *Tylocephalum,* have been found in Pacific oysters from Japan and Taiwan by staff members of the BCF Biological Laboratory, Oxford, Md. Members of this tapeworm group are known as parasites of pearl oysters in the Far East and were held to be the cause of pearl formation (Herdman, 1904; Herdman and Hornell, 1906; Shipley and Hornell, 1904, 1906; Southwell, 1924). Jameson (1912) presented convincing evidence, however, that invasion by larval trematodes, rather than larval cestodes, was more important in pearl development. Pearl formation around trematode and cestode larvae which invade the mantle and die appears to follow a similar pattern of host responses in bivalve mollusks (Wright, 1966).

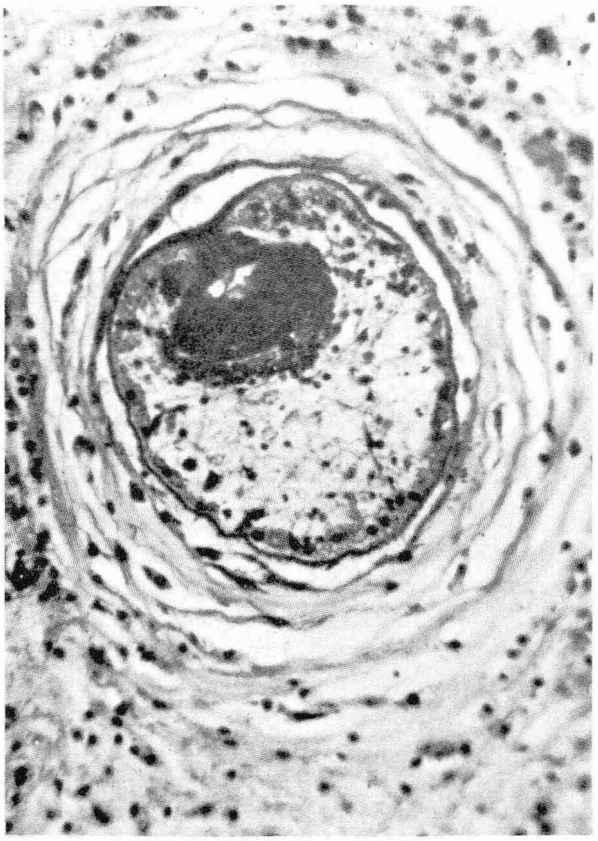

FIGURE 6.—*Tylocephalum* in American oyster from Apalachicola Bay, Fla. (× 70).

Diseases Caused by Parasitic Crustaceans

A parasitic copepod, *Mytilicola orientalis,* was described from the digestive tract of the Pacific oyster from Japan by Mori (1935). The parasite was transferred to the United States west coast with imports of seed oysters from Japan, and was described as *M. ostrea* by Wil-

son (1938), who was apparently unaware of Mori's report. Odlaug (1946) stated that Olympia oysters infected with even small numbers of *M. orientalis* had a lower condition index than uninfected oysters. Chew, Sparks, and Katkansky (1965) found a similar relation in Pacific oysters, and Sparks (1962) demonstrated pathological changes in gut epithelium and underlying tissues of these oysters infected with the copepod. Mori and Odlaug reported *M. orientalis* as a parasite of mussels (*Mytilus crassitesta* Lischke and *M. edulis* L.) as well as oysters, both in Japanese and United States waters. Another species, *Mytilicola intestinalis*, first described by Steuer (1902, 1903), has been blamed by Korringa (1950, 1959) and others for widespread mortalities of sea mussels, *Mytilus edulis*, in Europe but is found only rarely in European oysters (Baird, Bolster, and Cole, 1950; Hepper, 1953, 1956) and has not been reported to cause significant mortalities. A small specimen, identified as *Mytilicola intestinalis*, was found by Pearse and Wharton (1938) in an American oyster from the Florida coast. Humes (1954) believed that this copepod was probably *M. porrecta*, which he described from mussels and clams from Louisiana.

Crabs of the genus *Pinnotheres* occasionally inhabit the shell cavity of oysters, where their activities and effects suggest that they are parasites rather than commensals (Christensen and McDermott, 1958; Haven, 1959). Stauber (1945) studied a sudden increase in abundance of the oyster crab, *P. ostreum* (Say), in American oysters of Delaware Bay in 1941. He observed that the crabs robbed the oyster of food and injured the gills, resulting in a weakened condition of the host. Stauber found in 1941 that 90 percent of the oysters of Delaware Bay harbored four to six crabs in the first parasitic stage. He ascribed unusual mortalities in certain oyster populations to debilitation caused by high abundance of crabs. In 1942 abundance of crabs dropped to 25 to 30 percent, and continued to decline in following years.

Another *Pinnotheres* was described from Madagascar oysters, *Ostrea vitrifacta* Sowerby, by Poisson (1946); infestation was accompanied by development of a characteristic irritating flavor in the parasitized oyster. He speculated that this flavor might be traced in some way to the coelenterate *Sertularia*, which often grows on shells of oysters that contain *Pinnotheres*. Korringa (1952a) pointed out the striking similarity of this observation with the popular belief in Holland that eating mussels parasitized by *P. pisum* causes "nettle rash."

Tumors

Tumors in oysters have been reported. Benign mesenchymal tumors of pericardial origin have been described from oysters by Ryder (1887) and Smith (1934). The nodular, polypoid, pediculated growth found by Smith was over 3 cm. in greatest dimension. Sparks, Pauley, Bates, and Sayce (1964a) described a stalked mesenchymal tumor from the Pacific oyster that appeared to be histologically similar to that found by Smith. Sparks, Pauley, Bates, and Sayce (1964b) also reported a Pacific oyster with a tumorlike fecal impaction of unknown origin, accompanied by inflammation and encapsulation. The literature on tumors of other species of mollusks, as well as those of other invertebrates, has been reviewed by Scharrer and Lochhead (1950).

MUSSELS

Marine mussels, particularly *Mytilus edulis*, are abundant and palatable. They are grown by highly developed culture methods, especially in Western Europe. Culture of a single species, often under crowded conditions, however, increases vulnerability to disease. As with cultivated oysters, extensive and repeated mortalities, presumably caused by disease, have occurred in mussel beds. The parasitic copepod, *Mytilicola*, is the only specific pathogen that has been shown to cause mortalities in European mussels, although other parasites, particularly haplosporidan Protozoa and larval trematodes, have been reported.

Microbial Diseases

Eggs of the sea mussel from the western North Atlantic are occasionally infected with a haplosporidan, *Chytridiopsis mytilovum*. Field (1923) first described the parasite and several of its life history stages. Recently Sprague (1965b) has redescribed life history stages in more detail. The parasite has a high prevalence in some samples, although the proportion of infected eggs to normal eggs in any

individual is low. Vincentiis and Renzoni (1963) recognized what seems to be the same organism in eggs of the Mediterranean edible mussel, *Mytilus galloprovincialis* L., from the Gulf of Naples. A similar parasite, *Chytridiopsis ovicola*, has been reported from eggs of the European oyster (Léger and Hollande, 1917).

Taylor (1966) described a disease of California mussels, *M. californianus* Conrad, caused by another haplosporidan, *Haplosporidium tumefacientis*. The disease, characterized by tumefactions of the digestive gland, was found in 23 of 1,114 individuals examined. The gross enlargement of the gland was apparently due to plasmodia of the parasite; no necrosis was reported. Samples of sea mussels collected simultaneously from the California coast by Taylor were not parasitized by the haplosporidan.

Sea mussels from the Baltic were found by Raabe (1934, 1936, 1938, 1949) to harbor a number of ciliates, including *Ancistrocoma pelseneeri* Chatton and Lwoff, *Kidderia mytili* (DeMorgan), *Ancistruma mytili* (Quennerstedt), and *Hypocomides mytili* (Chatton and Lwoff). The same species of ciliates have also been identified from *Mytilus* sampled in other geographic areas (Kidder, 1933; Chatton and Lwoff, 1934; Kozloff, 1946). Although many ciliates occur in bivalve Mollusca (Fenchel, 1965), a parasitic role has not been adequately determined for most of them.

Diseases Caused by Helminths

Invasion of mussels by larval trematodes is thought to be responsible in part for pearl formation. Jameson (1902) believed that most mussel pearls resulted from encystment of metacercariae and encapsulation by the host. Herdman (1904), who studied sea mussels in England, found pearls very common near Piel and attributed them to invasion by larvae of *Distomum (Gymnophallus) somateriae*.

The literature on trematode-induced pearl formation in mussels has been reviewed by Stunkard and Uzmann (1958). The relation between pearls in European sea mussels and trematode parasites was first described by Garner (1872), and later by Dubois (1901, 1903, 1907, 1909) who proposed the name *Distomum margaritarum (Gymnophallus margaritarum)* for parasites found in reddish-brown spots which served as foci for pearl formation in mussels from the French coast. Jameson (1902) stated that the larval trematodes resembled *D. somateriae* which had been described as an adult from the intestine of the eider duck, *Somateria mollissima*, by Levinsen (1881). Jameson referred the parasite to the genus *Lecithodendrium* Looss and described the process of pearl formation by the mantle of the mussel around the metacercariae. Giard (1903, 1907) confirmed these observations on pearl formation. Odhner (1905) designated the larvae causing pearl formation in mussels as *Gymnophallus bursicola*. Similar metacercariae were found by Stafford (1912) in mussels from the Gulf of Saint Lawrence. Jameson and Nicoll (1913) reviewed pearl formation in mussels and concluded that several gymnophallid larvae were involved. Since then other gymnophallid cercariae have been associated with metacercariae in mussels (Palombi, 1924; Cole, 1935; Rees, 1939). Stunkard and Uzmann (1958) fed mussels from Long Island to newly hatched eider ducks and recovered adult gymnophallids, probably *G. bursicola*.

Other larval trematodes have been described from mussels. Cole (1935) reported "orange sickness" of sea mussels at Conway in Wales. The color was due to masses of orange pigmented trematode sporocysts in the mantle and throughout the body; the tailless cercariae they contained were described as *Cercaria tenuans*. A similar condition had been noted previously by Atkins (1931a). Cole also described a second larval trematode infestation, caused by *Bucephalus mytili*. Uzmann (1953) found microcercous trematode larvae in sea mussels from Long Island and Connecticut that had a similar orange coloration. Described as *Cercaria milfordensis*, the larvae were primarily parasites of the blood vascular system of the host and had foci in the blood vessels of the mantle. Sporocyst development precluded normal gametogenesis in mussels; infected mussels held in aquaria had unusually high mortalities. Uzmann suggested that *C. milfordensis* infections are probably lethal to the host under unfavorable environmental conditions.

Diseases Caused by Parasitic Crustaceans

A well-documented example of the effects of

disease on mussel populations is that of the invasion of the north European sea mussel stocks by the copepod *Mytilicola intestinalis*. A fascinating body of literature has accumulated about this parasite and its effects on mussels; only a sampling of the many published papers is cited here. Steuer (1902) first described the parasite from the intestines of Mediterranean edible mussels, and Pesta (1907) outlined the life history. Korringa (1950, 1959) described the relatively sudden appearance of *Mytilicola* in sea mussel stocks of the Netherlands in 1949 and its subsequent spread to many mussel beds during the following decade. The organism was known to occur in Mediterranean mussels since the beginning of the 20th century (Monod and Dollfus, 1932), and in 1938 was found near Cuxhaven, Germany, from whence it was assumed to have spread westward to the Netherlands. Spread was thought by Korringa and others to be aided by mussel encrusted ships, by movement of planktonic larvae, and by transfer of seed mussels from infested areas. *Mytilicola* was also very abundant in localized areas of the English coast in 1946. Korringa stated that the condition of mussels was generally correlated with intensity of parasitization; mussels with less than 5 copepods were still healthy, those with 5 to 10 were visibly thinner, and more heavily infested lots suffered serious mortalities. According to Meyer-Waarden and Mann (1954) and Mann (1956), gonad weights of infested individuals were 10 to 30 percent less than those of nonparasitized mussels.

There is some indication, however, that *M. intestinalis* exerts a less severe effect on populations of *Mytilus galloprovincialis* than on those of *M. edulis*, possibly because of its longer association with *M. galloprovincialis* and the consequent better adaptation of host and parasite—as was pointed out by Fleury, Lubet, and Le Dantec (1951). Hrs-Brenko (1964), for example, found no difference in condition index of parasitized and unparasitized mussels *(M. galloprovincialis)* on the Yugoslav Adriatic coast, and Genovese (1959) made similar findings on the Italian coast.

Infestation of sea mussels led to poor growth, thin meats (Cole and Savage, 1951; Mann, 1951), cream-colored rather than dark brown liver, failure of byssal development, and a dirty red-brown color. Reproduction of the parasite was accelerated by warm water, and the many young parasites present in the summer invaded and killed mussels. Deaths occurred among mussels of all sizes, including "seed." Mussels fell from culture racks and died during transport to markets (Brienne, 1964). Density of mussel beds was believed to directly influence survival and multiplication of the parasite. Infestations were light in areas where the mussels were thinly scattered and near the surface of the water. Because of the continued spread of *Mytilicola* in the Netherlands, an extensive scheme of repeated dredging of natural beds, transfer of lightly infested stocks, and destruction of heavily infested stocks was outlined by Korringa (1959) to create a barrier to further invasion.

Mytilicola in mussel populations grown on floats in Spain was studied by Andreu (1963). He found the infestation to be greater near shore where tidal currents were weak. Vertical distribution of the parasite in cultured mussels grown on 6–m. ropes was uniform in areas of strong currents but increased with depth where currents were weak. Such findings agree well with those of Hepper (1955), who concluded from field observations that mussels raised from the bottom, or in fast-moving water at either end of an estuary, were less heavily infested with *Mytilicola* than those on the bottom, in slow-moving water, or in the mid-regions of estuaries. Hepper felt that control of the copepod was possible by using off-bottom culture or by locating culture beds in fast-moving water or at the brackish-water ends of estuaries.

M. intestinalis, except for one doubtful North American record (Pearse and Wharton, 1938), is known only from Europe. It has been reported from Germany (Caspers, 1939; Meyer and Mann, 1950, 1952a, 1952b; Meyer-Waarden and Mann, 1956), the Netherlands (Korringa, 1951b, 1952b, 1953, 1957a), Belgium (Leloup, 1951, 1960), Scotland, England, and Ireland (Ellenby, 1947; Grainger, 1951; Hockley, 1952; Thomas, 1953; Bolster, 1954; Waugh, 1954), the north coast of France (Dollfus, 1914, 1927; Monod and Dollfus, 1932; Brienne, 1964), the northwest coast of Spain (Andreu, 1960, 1961,

1963), the Mediterranean Sea (Bassedas, 1950; Meyer-Waarden and Mann, 1953), and the Adriatic Sea (Steuer, 1902; Pesta, 1907; Meyer-Waarden and Mann, 1953). Waugh (1966) has mapped the recent distribution of the parasite in northern Europe. A conference to review and discuss problems of parasitization by *Mytilicola* was held in Paris, and the proceedings were published in 1951 (Cole, 1951a; Dollfus, 1951; Havinga, 1951; Heldt, 1951; Korringa, 1951b; Korringa and Lambert, 1951; Lambert, 1951a, 1951b; Leloup, 1951; Meyer and Mann, 1951). It was agreed that *Mytilicola* constituted a severe threat to the mussel industry of Europe, but whether the copepod was a direct or indirect cause of death was left undecided. Continuing mortalities associated with the presence of *Mytilicola* (Brienne, 1964), however, indicate a causal relationship, possibly influenced by stresses of spawning, high temperatures, and inadequate food supply.

Another species, *Mytilicola porrecta* Humes, occurs in ribbed and recurved mussels (*Modiolus demissus* Sowerby and *Mytilus recurvus* Rafinesque) in the Gulf of Mexico. Humes (1954) found as many as 15 individuals per mussel, but no pathology or mortality was indicated. A third species, *Mytilicola orientalis*, known to occur in *Mytilus edulis* and *M. crassitesta*, was recently reported from the California mussel by Chew, Sparks, and Katkansky (1964).

Pinnotherid crabs of several species, best known as parasites of oysters, also occur in mussels. McDermott (1962) found that *Pinnotheres ostreum* and *P. maculatus* cause gill damage and palp erosion in *M. edulis*. Earlier, Atkins (1931b) described similar palp abnormalities in mussels from England.

CLAMS

Many species of bivalves called by the general term "clam" are harvested throughout the world. Some species constitute a significant commercial crop in many coastal areas; other species are fished for sport or are ignored. Changes in clam abundance have been documented, although mass mortalities comparable to those in oysters and mussels have not been reported. Mass deaths may pass unnoticed in sediment-hidden clams; it may be for this reason that information on diseases of clams is scarce. Among the diseases and parasites that are known in clams are: several protistan organisms, larval trematodes, larval cestodes, parasitic copepods, and tumors.

Microbial Diseases

Coe's (1955) study of population fluctuations of the California bean clam, *Donax gouldi* Dall, included a description of a possible fungus parasite "apparently similar to *Dermocystidium marinum*" as a cause of mass mortalities during the summer. Moribund clams of all ages were heavily infected with "irregularly spherical or ovoid cells, 2 to 6 microns in diameter." The identity of the pathogen was not further determined, however, and the information presented is insufficient to identify it as a *Dermocystidium*.

Much earlier, Léger (1897) found a coccidian, *Hyaloklossia pelseneeri*, in kidneys of *Donax* sp. and *Tellina* sp. in Europe, and Léger and Duboscq (1917) described another coccidian, *Pseudoklossia glomerata*, parasitic in *Tapes floridus* L. and *T. virgineus* L. from the Mediterranean Sea.

Ciliate parasites have been described from soft-shell clams, *Mya arenaria* L., by Uzmann and Stickney (1954). The peritrich *Trichodina myicola* Uzmann and Stickney was found, often in large numbers, on the palps. These infections were often accompanied by the nonpathogenic thigmotrich *Ancistrocoma myae* (Kofoid and Busch). *A. myae* had been described earlier from *M. arenaria* sampled in California (Kofoid and Busch, 1936; Kozloff, 1946) and in Massachusetts (Chatton and Lwoff, 1950). Kozloff considered the ciliate identical to *A. pelseneeri*, a common parasite of sea mussels. Fenchel (1965) also found *Ancistrocoma myae* in nearly 100 percent of *M. arenaria* sampled from two locations in Denmark.

Diseases Caused by Helminths

Several life history stages of diverse trematodes occur in the soft-shell clam. Uzmann (1952) reported sporocysts and cercariae *(Cercaria myae)* from gonads and digestive gland of this clam from Massachusetts, and held that parasitization resulted in a condition known as "water belly." (Subsequent observations, sum-

marized by Dow and Wallace (1961), suggest that this condition may be a general sign of physiological disturbance.) Uzmann considered *C. myae* to be the same species as that reported by Stafford (1912) from the soft-shell clam in the Gulf of Saint Lawrence. Hutton (1953) believed that the larvae were members of the genus *Gymnophallus*. Stunkard and Uzmann (1958) discussed gymnophallid sporocysts and cercariae from the soft-shell clam, and Stunkard (1960) found echinostome metacercariae of the genus *Himasthla* in palps and gills of clams from the Maine coast. Three species were recognized. Earlier, Stunkard (1938) had demonstrated experimentally that cercariae of *Himasthla* would penetrate and encyst in the gills of *Mya arenaria* and a number of other bivalves, and Uzmann (1951) had reported natural occurrence of *Himasthla quissetensis* (Miller and Northrup) in *M. arenaria*. Susceptibility and response of a number of marine pelecypods, including four species of clams, to cercariae of *H. quissetensis* was tested experimentally by Cheng, Shuster, and Anderson (1966). Metacercariae were found in all clams and mussels but not in oysters used in the study.

Several larval trematodes have been reported from clams of the genus *Donax*. Giard (1897, 1907) identified bucephalid and gymnophallid cercariae. Rees (1939) found gymnophallid metacercariae, and Young (1953) described the life cycle of a monorchid, *Postmonorchis donacis*, whose larvae occur in the California bean clam. Hopkins (1958) identified sporocysts and cercariae of three species, and metacercariae of two species, in coquina clams, *Donax variabilis* Say, from the Texas coast. Infections by larval trematodes were considered by Pelseneer (1896, 1906, 1928) to be responsible for reduced abundance of *Donax vittatus* in France, and Coe (1946) held that trematode parasites (probably *Postmonorchis donacis*) were important in controlling population size in California *Donax gouldi*.

Fujita (1906, 1907, 1943) has described two larval trematode parasites of asari clams, *Tapes philippinarum* Adams and Reeve, from Japan. Parasitic castration of the hosts was observed.

Hopkins' (1957a) brief but excellent exposition of the role of parasitism in marine communities referred to an interesting interrelationship of host, parasite, and hyperparasite in the case of *Donax trunculus* parasitized by trematodes, which in turn were parasitized by the haplosporidan *Urosporidium pelseneeri* (Caullery and Chappellier). The often severe fluctuations in abundance of this clam have been attributed to shifts of balance in this tripartite relationship (Caullery and Chappellier, 1906; Cépède, 1911). Other haplosporidan and microsporidan hyperparasites of *Donax* have also been described (Guyénot, Naville, and Ponse, 1925; Dollfus, 1946; Mackin and Loesch, 1955).

MacGinitie and MacGinitie (1949) surveyed a number of species of clams from the Pacific coast of the United States for parasitization by larval tapeworms. Encysted larvae of the cestode genus *Anabothrium* were found, occasionally in large numbers, in the foot muscles of the gaper clam, *Schizothaerus nuttallii* Conrad. The definitive host of the *Anabothrium* sp. found in clams was identified by MacGinitie and MacGinitie as the bat stingray, *Myliobatis californicus*.

Sparks and Chew (1966) described remarkable levels of parasitization of littleneck clams, *Venerupis staminea* (Conrad), from Humboldt Bay, Calif., by larval tetraphyllidean cestodes of the genus *Echeneibothrium*. Cysts of the worm were closely packed throughout the tissues of the clams, which were abnormally exposed on the surface of gravel beds. Adult *Echeneibothrium*, with bothridia similar to those of larvae in clams, were found in bat stingrays caught in the same area.

Diseases Caused by Parasitic Crustaceans

Clams, like certain other bivalves, harbor parasitic copepods. Hoshina and Kuwabara (1959) described *Mytilicola mactrae* from Japanese *Mactra veneriformis* Reeve. About half the clams in a sample of 69 were infested. Yamaguti (1939) described another species from *Brachidontes senhausi* (Reeve), and Humes (1954) found *M. porrecta* in a single hard clam, *Mercenaria mercenaria* (L.), from the Gulf of Mexico.

Tumors

Hueper (1963) reported cauliflowerlike papillary tumors at the anterior end of soft-shell

clams collected from Chesapeake Bay. He termed the condition "endemic" and reported it in about 2 percent of clams collected from certain bay areas.

OTHER BIVALVE MOLLUSKS OF COMMERCIAL IMPORTANCE

There are two other groups of commercially important bivalve mollusks—scallops and pearl oysters—for which some information on diseases and parasites is available. Mass mortalities caused by disease have not been reported in either group. Scallops are infected by protozoan and trematode parasites, and occasionally are affected by a shell disease. Pearl oysters harbor a number of larval trematode parasites.

Scallops

Although major mortalities have occurred in scallop populations (Dickie and Medcof, 1963; Medcof and Bourne, 1964; Merrill and Posgay, 1964; Sanders, 1966), none has been definitely associated with disease. In fact, only a few diseases and parasites are known, and their effects on the hosts are slight.

Two parasites of scallops have been recognized. A coccidian, *Pseudoklossia pectinis*, was described as a rare parasite in the kidney tubules of the great scallop, *Pecten maximus*, at Roscoff, France, by Léger and Duboscq (1915b), who found usually light infections with no extensive pathology. Sporocysts and fork-tailed cercariae of a trematode (not further identified) were found by Linton (1915) in large bay scallops, *Aequipecten irradians*, from Woods Hole, Mass. Infections were rare.

An abnormal brown discoloration of meats was studied by Medcof (1949) in sea scallops, *Placopecten magellanicus* (Gmelin), off the south coast of Nova Scotia, Canada. He considered the condition to result from extensive invasion of the shell by a boring sponge. In advanced stages, the shell was completely honeycombed, causing excessive inner shell deposition and producing weak shrunken individuals which, Medcof assumed, died eventually from effects of the shell disease. Meat yields from heavily infected scallops were less than half those of normal individuals, but only older scallops (8 or 9 years old) were infected.

Pearl Oysters

Pearl-producing bivalves of the family Pteriidae, called "pearl oysters" but actually taxonomically remote from edible oysters of the family Ostreidae, occur in many parts of the world (Sivalingam, 1962). Interest in parasites of pearl oysters has naturally centered on those larval worms considered responsible for pearl formation (Jameson, 1902, 1912; Wright, 1966), but a few other parasites and diseases have been recognized.

Parasites of the pearl oysters of Ceylon, *Margaritifera (Pinctada) vulgaris* Schum., were studied by Shipley and Hornell (1904) and Southwell (1911, 1912) particularly with regard to the role of parasitic worms in pearl formation. These authors described several stages of cestode larvae, some clearly trypanorhynchid, from the digestive gland and gills of the pearl oyster. The worms occurred, often in great numbers, in fibrous capsules. Several larval trematodes were also found, but only one, described as *Muttua margaritiferae*, occurred in abundance. Metacercariae localized in the gills. Other metacercariae, described as *Musalia herdmani*, were found in the muscles, mantle, and foot. An aspidobothrid trematode, *Aspidogaster margaritiferae*, occurred in the pericardial cavity, and several species of encysted larval nematodes were seen in the gonads, stomach walls, and adductor muscles.

Pearl oysters of Japan, *Pinctada martensii*, are commonly infested with sporocysts and cercariae of a bucephalid trematode described as *Bucephalus margaritae* by Ozaki and Ishibashi (1934). Experimental infections of several species of small fishes with cercariae from pearl oysters (Sakaguchi, 1962, 1966a) produced metacercariae morphologically the same as those identified as *B. varicus* by Manter (1940). Adult trematodes were found in the digestive tracts of carangid fishes, *Caranx sexfasciatus* and *C. ignobilis*, which were abundant in the waters near oyster farms where pearl oysters were heavily infested (Sakaguchi, 1966b). Marked decline in condition of pearl oysters resulted from invasion by larval trematodes. Sporocysts were found to overwinter in the host, and cercarial production began again when water temperatures rose in spring (Sakaguchi, 1965). Pearl oysters infected in the pre-

ceding year suffered high mortalities after insertion of pearl cores, and high percentages of pearls produced by infected individuals were of low quality (Sakaguchi, 1964).

DISEASES OF CRUSTACEA

Crustacea such as crabs, lobsters, and shrimps are among the most valuable of marine crops in many parts of the world. Large populations of crustaceans occur on the continental shelves, and often part or all of the life cycle is spent in estuarine or inshore waters. Here individuals may be observed and studied in natural habitats as well as in the landed catches. These studies have disclosed certain parasites and diseased conditions. Disease may have severe effects on survival, particularly when crabs and lobsters are impounded before sale. Diseases exist in natural populations of Crustacea as well, although effects are less apparent than in captives or in more sedentary marine animals. No widespread epizootic is known for marine Crustacea that would be comparable to "krebspest," a fungus disease that swept through populations of freshwater crayfishes of Europe (Schikora, 1906, 1926; Schäperclaus, 1935; Nybelin, 1935; Mannsfield, 1942).

CRABS

Many species of crabs have great commercial value in various parts of the world. Consequently, diseases and parasites have been included in studies of factors which affect abundance. Microbial diseases, helminths, and parasitic crustaceans occur in crabs.

Microbial Diseases

Among the microbial diseases of crabs are those caused by a virus, several fungi, bacteria, and a variety of protozoans.

Viruses.—Virus diseases have not been reported from marine invertebrates, with the exception of one described recently, and only very briefly, by Vago (1966), in swimming crabs, *Portunus depurator* (L.), from the French Mediterranean coast. Gross disease signs included the slow development of paralysis, and sometimes a slight darkening (presumably of the exoskeleton) in later phases of the disease. Virus particles were seen with the electron microscope; inoculation of blood from infected animals produced disease signs in healthy crabs; and infections were obtained with ultrafiltrates and ultracentrifugates of homogenized tissues from sick crabs. No indication of disease prevalence was given by Vago.

Recently, Sprague and Beckett (1966) have published a preliminary note on a disease of soft-shell and molting blue crabs, *Callinectes sapidus* Rathbun. The disease, of undetermined but possibly viral etiology, was called "gray crab disease." It occurred in crabs from seaside bays of Virginia, where it apparently caused some deaths among captive crabs.

Bacteria.—King crabs, *Paralithodes camtschatica* (Tilesius) and *P. platypus* Brandt, from the eastern North Pacific are occasionally affected by "rust disease," which seems to result from action of chitin-destroying bacteria on the exoskeleton. Microorganisms of this type are common in the sea (ZoBell and Rittenberg, 1938; Hock, 1940, 1941) but usually degrade the exoskeletons of dead animals and do not affect living individuals. Over thirty species of chitin-destroying bacteria are known, of which half have been isolated from shells of crustaceans.

Bright, Durham, and Knudsen[3] described observations of rust disease in landed catches of king crabs from Kachemak Bay, Cook Inlet, Alaska, as well as experimental studies of the bacteria involved. The disease was characterized by progressive darkening and softening of the exoskeleton, particularly on the ventral surfaces. Underlying living tissues were unaffected. Natural infections reached 11 percent in larger older crabs in 1957 but were much lower in 1958 and 1959. Shell abrasions and injuries served as foci of the disease, which developed experimentally within 2 weeks. The disease was not carried over to the new exoskeleton after molting, but recently shed crabs were highly susceptible because the new shell was easily punctured or abraded. Chitin-destroying bacteria were isolated from infected

[3] Data furnished from unpublished contract report, "King crab investigations of Cook Inlet, Alaska," by Donald B. Bright, Floyd E. Durham, and Jens W. Knudsen of the Allan Hancock Foundation, University of Southern California, Los Angeles, to BCF Biological Laboratory, Auke Bay, Alaska, June 1960. (Cited with permission of Laboratory Director, BCF Biological Laboratory, Auke Bay, Alaska.)

crabs, and cultured organisms produced the disease experimentally in normal crabs. Similar bacteria were also isolated from sea water in Kachemak Bay. The authors concluded that the disease would not affect the commercial fishery seriously unless the catch of crabs was substantially less than annual recruitment, since larger individuals, which do not molt annually, were more frequently infected.

Fungi.—Eggs of blue crabs from lower Chesapeake Bay were found to be parasitized by a fungus *Lagenidium callinectes* Couch (Couch, 1942; Sandoz, Rogers, and Newcombe, 1944; Sandoz and Rogers, 1944; Newcombe and Rogers, 1947; Rogers-Talbert, 1948). Infected eggs either failed to hatch, or gave rise to abnormal zoea larvae. Infection levels were as high as 90 percent of a sample of ovigerous female crabs, and up to 25 percent of the eggs in a "sponge" (egg mass). Penetration of the egg mass was slow and did not exceed 3 mm. This fact, combined with the short (2-week) incubation time, permitted normal development of much of the egg mass internal to the infection. Experimentally, the fungus developed normally in salinities from 5 to 30 o/oo. The fungus was transmitted experimentally to the eggs of two other species of crabs (the oyster crab and the mud crab, *Neopanope texiana* Rathbun) inhabiting the same Bay area.

Pea crabs *(Pinnotheres)* taken from the sea mussel at Plymouth, England, were parasitized by the fungus *Leptolegnia marina* (Atkins, 1929, 1954a). The mycelium was usually found in the gills but penetrated other body organs and appendages as well. Zoosporangia developed in the appendages, and large numbers of zoospores were released upon the death of the host. No further growth of the fungus took place in dead crabs, and no external development, beyond papillae of zoospore exit tubes, was seen. Atkins (1954b, 1955) described two other fungi, *Plectospira dubia* and *Pythium thalassium*, which infect eggs of pea crabs and other Crustacea.

Protozoa.—Gregarines are common parasites of many Crustacea, and an extensive and at times confusing literature has accumulated. Many members of the group have been reported from crabs. For example, species of the genus *Cephaloidophora* occur in spider and fiddler crabs of the United States east coast (Watson, 1915, 1916a, 1916b; Kamm, 1922), in the striped shore crab, *Pachygrapsus crassipes*, of the Pacific coast, and in the Mediterranean "flat crab," *Pachygrapsus marmoratus* (Ball, 1938; Théodoridès, 1961, 1962). As mentioned in the discussion of oyster diseases, several representatives of the genus *Nematopsis* occur in Atlantic species of mud crabs (Prytherch, 1940; Ball, 1951; Sprague and Orr, 1955). Although the gregarines are not usually considered serious pathogens of Crustacea, Ball pointed out that masses of the parasites may occlude the lumen of the intestinal caeca and may cause sloughing or thinning of the epithelium.

Several microsporidans are parasites of crabs. Sprague (1965a) described a species of *Nosema* parasitic in muscles of the blue crab. Infected muscles became opaque with a coarse fibrous texture, and heavy infections caused lysis of myofibrils. He considered the parasite to be common and widespread in Chesapeake Bay, and believed it might be a significant factor in crab mortality. Sprague (1966b) also described *Plistophora cargoi* from the skeletal and cardiac muscles of a single blue crab from the Patuxent River, Md. Earlier, Perez (1905a, 1905b) reported *Nosema pulvis* in the muscles of the green crab, *Carcinus maenas* (L.), and Perez (1907) also described ovarian infections with a microsporidan, *Thelohania maenadis*, in green crabs from Arcachon, France. The parasite normally occurred in the body muscles.

Ciliates are also significant parasites of crabs. Serious mortalities of molting blue crabs from Chesapeake Bay occurred in the summers of 1965 and 1966. Their gills had a massive infestation of peritrichous ciliates (fig. 7) of the genera *Lagenophrys* and *Epistylus* (Couch, 1966, 1967a). Mortalities were most severe among crabs in holding tanks just before or after molting, but wild crabs were also heavily infested, and fishermen reported mortalities. Infestations of gills frequently seemed heavy enough to interfere with respiration.

Another ciliate, *Anophrys sarcophaga* Cohn, is found in the blood of green crabs. Originally described as a free-living form (Cohn, 1866), it was first seen in the blood of crabs by Cattaneo (1888). Poisson (1930) described the

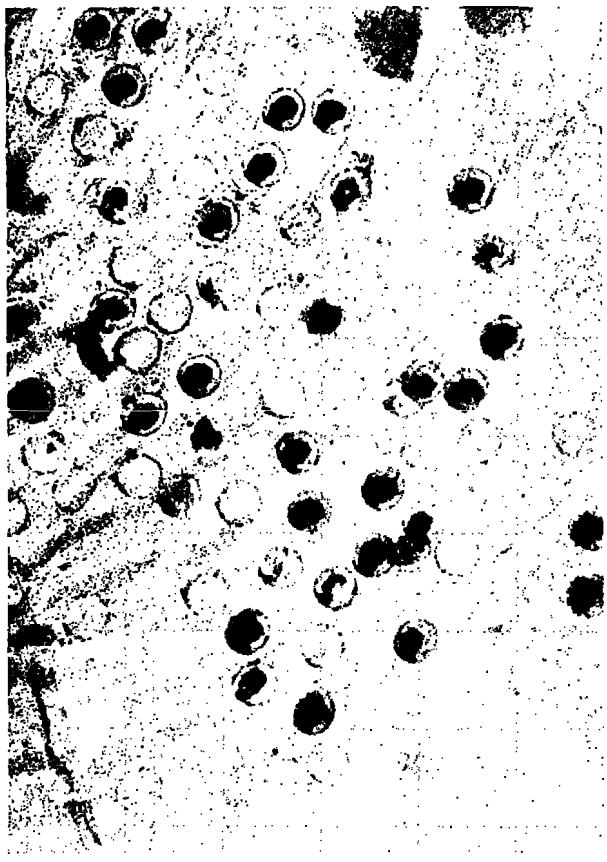

FIGURE 7.—*Lagenophrys* encysted on gills of blue crab (× 70).

active and encysted forms of the parasite in great detail. He also cultured the organism. *Anophrys* apparently ingests large numbers of host amebocytes, and multiplies until the blood becomes a dense "soup" of motile parasites. The disease, though often fatal once infection occurred, was relatively rare on the French coast. Experimental infections, achieved by injecting the ciliates, killed crabs in 2 to 7 days. Certain individual crabs seemed resistant to infection, but no antibody response was detected. Effects on the parasites included inhibition of reproduction, immobilization, and eventual death.

Diseases Caused by Helminths

Helminth infestations include larval trematodes and cestodes, as well as nemerteans and leeches.

Trematode metacercariae are common in several species of crabs. Larvae of *Microphallus* (*Spelotrema*) *nicolli*, encysted in body muscles of blue crabs, were described by Cable and Hunninen (1940). Adult trematodes occur in young herring gulls, *Larus argentatus*, and sporocyst generations in the snail *Bittium alternatum* (Say). A haplosporidan hyperparasite, *Urosporidium crescens* [De Turk], has been found in metacercariae of *Microphallus nicolli* from blue crabs caught in North Carolina (Anonymous, 1940). One third of the crabs examined contained metacercariae, but the extent of hyperparasitization was not determined. The host metacercarial tissue was often completely destroyed, leaving little more than a sack of protozoan spores. Invasion by *Urosporidium* was thought to occur before encystment of cercariae. Masses of dark pigmented spores in the destroyed metacercaria produce a black spot, which has led to the application of the descriptive colloquial term "pepper crab" to hyperparasitized individuals. "Pepper spots" occur most commonly in fat bodies, digestive gland, and muscles of the crab.

Metacercariae of *Microphallus* (*Spelotrema*) *carcini* Lebour were reported from the hepatopancreas of green crabs from the English Channel (Guyénot et al. 1925), the Mediterranean (Timon-David, 1949), and elsewhere. Hyperparasitization of metacercariae by the microsporidan *Nosema (Plistophora) spelotremae* was reported by Guyénot et al. Metacercariae of *Microphallus similis* (Jägerskiöld) occur in the hepatopancreas of the green crab of the western North Atlantic (Stunkard, 1957). Young green crabs were killed in 10 to 20 days by massive experimental exposure to cercariae of *M. similis*.

Larval diphyllidean cestodes (*Echinobothrium affine* Diesing) have been reported by Dollfus (1964a, 1964b) from green crabs sampled at Roscoff on the coast of France.

Juvenile nemerteans, *Carcinonemertes carcinophila* (Kölliker), encyst on the gills of the blue crab. After the female crab spawns, the worms excyst and migrate to the egg mass, where they mature, lay eggs, then return to the gills (Davis, 1965). Hargis (1959) has cited this parasite as an indicator of host physiology, since Hopkins (1947) pointed out that increased size and more noticeable color were characteristic of reencysted worms after the

host has spawned. This and other nemerteans have been described from a number of crabs, including the green crab, of Europe (Coe, 1902; Humes, 1942).

A localized mortality of blue crabs, thought to be caused by parasitization by leeches, *Myzobdella lugubris* Leidy, was reported from a Florida river by Hutton and Sogandares-Bernal (1959). A sample of 7 crabs had 32 leeches attached near the base of the legs and near perforations in the exoskeleton. The parasite is known from the Atlantic and Gulf coasts of the United States but had not previously been considered to cause mortalities (Moore, 1946).

Other leeches occur on crabs. Oka (1927) described *Carcinobdella kanibir* from Japanese edible crabs, *Chionoecetes opilio*. Egg cases and adults of the leech *Notostomobdella cyclostoma* (Johansson) are common on Alaska king crabs, particularly during summer (Moore and Meyer, 1951; Bright et al., see footnote 3). Undescribed worms, probably leeches, were seen by MacKay (1942) on the abdomens of female Dungeness crabs, *Cancer magister* Dana, from British Columbia. The worms were much larger on egg-bearing crabs and were found chiefly among the eggs.

Diseases Caused by Parasitic Crustaceans

Parasitic crustaceans—rhizocephalans, isopods, and copepods—also infest crabs.

Many species of crabs, in many parts of the world, are parasitized by rhizocephalan Cirripedia. These parasites invade the host's body and cause degeneration of the gonads (Reinhard, 1956). The tumorlike body of the rhizocephalan ramifies throughout much of the crab and causes extensive morphological changes. The crab is usually sterilized, secondary sex characters are modified, and molting is often inhibited (Giard, 1888; Potts, 1906; Smith, 1906; Cantacuzène, 1925; Reinhard, 1950; Ichikawa and Yanagunachi, 1957).

Brachyuran or true crabs are parasitized by members of the rhizocephalan family Sacculinidae. In U.S. waters, crabs most frequently parasitized are the green crab of the Atlantic coast, and the masking crab, *Loxorhynchus grandis*, the kelp crab, *Pugettia producta* (Randall), and the black-clawed crab, *Lophopanoepeus bellus* (Stimpson) of the Pacific coast. Green crabs and swimming crabs, *Macropipus* (*Portunus*) *holsatus* (Fabricius), from the English coast are parasitized by *Sacculina carcini* Thompson (Delage, 1884; Day, 1935; Foxon, 1940). Blue crabs from the Gulf of Mexico are parasitized by the rhizocephalan *Loxothylacus texanus* (Hopkins, 1957a). Mud crabs, *Eurypanopeus depressus* (Smith), from lower Chesapeake Bay (Virginia) were discovered by Van Engel, Dillon, Zwerner, and Eldridge (1966) to have high incidences of the sacculinid *Loxothylacus panopaei* (Gissler). The localized nature of the infestations suggested that the parasite had been introduced with its hosts in shipments of oysters from the Gulf of Mexico.

Many species of anomuran crabs may be parasitized by rhizocephalans. King crabs, *Paralithodes platypus*, from Alaskan waters are occasionally invaded, probably by a species of *Peltogaster* (J. B. Kirkwood, written communication, April 14, 1967). Hermit crabs are also frequently invaded by members of the family Peltogastridae. Reinhard (1942), who examined 3,092 *Pagurus pubescens* Krøyer from the Maine coast of the United States, found 13.7 percent parasitized by *Peltogaster paguri* Rathke. The same rhizocephalan occurs on the coast of France, where its host is *Pagurus bernhardus* (L.) (Perez, 1927, 1928, 1931a, 1931b, 1931c). Infestation can have significant effects on crab populations, since parasitization usually causes degeneration of host gonads. Perez (1929, 1931a), however, found interesting evidence for sterilization and mortalities of *Peltogaster paguri* because of hyperparasitization by the epicaridean isopod *Liriopsis pygmaea* (Rathke); in some samples from northern France, most of the rhizocephalans were parasitized. Perez believed that this parasitization was an important control for *Peltogaster* populations.

Epicaridean isopods can also be significant parasites of crabs. Two families are of importance: the Bopyridae, which live principally in the gill chambers, and the Entoniscidae, which invade the haemocoele. In some species morphological modification for parasitic existence parallels that in rhizocephalans (Veillet, 1945). Effects on the crab host often include sterilization and changes in secondary sexual characteristics (Tucker, 1930; Reverberi, 1943, 1952;

Reinhard and Buckeridge, 1950). Parasitization of crabs by female entoniscids causes internal deformities, including reduction in size of organs (Atkins, 1933; Reinhard, 1945) and changes in the nervous system (Matsumoto, 1953).

Copepods are known as parasites of crab eggs. Connolly (1929) described *Choniosphaera cancrorum* from the egg masses of the American rock crabs, *Cancer borealis* and *C. irroratus*. Johnson (1957) found the same species on green crabs from the Maine coast. Gnanamuthu (1954) described *Choniosphaera indica* from gills and egg masses of an Indian edible crab, *Neptunus sanguinolentus*. Copepod larvae were found between the crab's gill lamellae, probably feeding on tissue fluids; adults apparently suck fluids from the crab eggs. Many other species of copepods, particularly of the family Choniostomatidae, are parasitic on Crustacea (Hansen, 1897, 1904, 1923).

An extensive and fascinating body of literature on rhizocephalan, epicaridean, and other crustacean parasites and hyperparasites of Crustacea has accumulated (Giard and Bonnier, 1887, 1895; Smith, 1906; Shiino, 1942; Veillet, 1945; Reinhard, 1944, 1956; Baer, 1951; Nicol, 1960).

LOBSTERS

Lobsters, because of their great economic importance in North America and Europe, have been subjects of many scientific studies, including some concerned with diseases and parasites. Because of the practice of holding lobsters in pounds and live cars, occasionally for extended periods and frequently under crowded conditions, mortalities have been observed and causes examined. Two bacterial diseases have significant effects on impounded lobsters. Among the known larger parasites are trematodes, nematodes, acanthocephalans, and annelid worms.

Microbial Diseases

A bacterial disease, caused by gram-positive tetrad-forming encapsulated cocci, described as *Gaffkya homari* Hitchner and Snieszko, is known from wild and impounded populations of American lobsters, *Homarus americanus* Milne-Edwards. The disease (gaffkaemia) was first noted on the Maine coast in 1946 (Hitchner and Snieszko, 1947; Snieszko and Taylor, 1947; Getchell, 1949). "Red-tail" disease, as it was originally called, is characterized by a variable pink coloration of the ventral abdomen, pink blood, prolonged clotting time, and drastic reduction in blood phagocytes. Infected lobsters become progressively weaker, and mortalities may reach 50 percent after short periods of storage. Mortalities increase sharply if water temperature exceeds 15° C. Moribund lobsters move to shoal water and die in a "spread-eagle" position.

Goggins and Hurst (1960)[4] have provided information about two epizootics of gaffkaemia along the entire Maine coast, one in 1946–47 and another in 1959–60, with losses as great as 58 percent of impounded populations. They found that the pathogen could live and multiply outside the lobster, in the slime on lobster cars, crates, tanks, and live wells. *Gaffkya* was also isolated from mud of tidal pounds and from sea water several miles from infected pounds. The disease was transmitted directly by allowing presumably healthy lobsters to feed on infected individuals or by holding healthy lobsters in sea water containing the pathogen. Incubation time was 14 to 21 days, although the animals possibly were already gaffkaemic before the start of the experiments. Treatment of tidal pounds with calcium hypochlorite reduced populations of the pathogen in bottom mud and reduced subsequent losses of impounded lobsters.

The disease organism is often present in wild populations. Stewart and MacDonald (1962) and Stewart, Cornick, Spears, and McLeese (1966) isolated *Gaffkya* from 96 of 2,035 recently caught lobsters in Canada and found the disease to be widespread in the Canadian Atlantic region. Cornick and Stewart (1966) recovered, from presumptive tests for *Gaffkya* in Canadian lobsters, several other kinds of bacteria, including *Micrococcus*, *Pseudomonas*, *Achromobacter*, and *Brevibacterium*—none of which was considered to be pathogenic. Rabin (1965) found a *Gaffkya*-like organism in lob-

[4] Data provided in unpublished mimeographed report of Department of Sea and Shore Fisheries, Augusta, Maine, "Progress report on lobster gaffkyaremia (Red Tail)," by P. L. Goggins and J. W. Hurst, 1960.

sters from Woods Hole, Mass., and Wood (1965a, 1965b) isolated *Gaffkya*-like organisms from two European lobsters (*Homarus vulgaris* Milne-Edwards) from the North Sea. Wood observed lobster mortalities in storage tanks in southern England in 1962 and recovered *Gaffkya* with cultural and biochemical characteristics similar to Canadian and United States isolates. Epizootics of gaffkaemia have been reported from European lobsters in Ireland (Gibson, 1961), Norway, and the Netherlands (Roskam, 1957). Gibson noted that diseased lobsters were also infested with the "gill maggot," *Nicothoë astaci* Audouin and Milne-Edwards, which was absent from uninfected individuals. Cross sections of the parasitic copepod were used by Gibson to determine the presence of the disease in the host.

Experimental studies of host-parasite relationships showed that lobsters became infected and died a few days after inoculation with *G. homari* (Rabin, 1965). Prior inoculation with *Vibrio* endotoxin did not enhance the infection and prior inoculation of heat-killed *Gaffkya* did not alter the course of infection. Lobster serum stimulated in vitro growth of *G. homari* in almost every test, but growth of *Vibrio* was sometimes inhibited. Studies of possible defense mechanisms of lobsters against *G. homari* have also been carried out at the Halifax (Nova Scotia) and Saint Andrews (New Brunswick) stations of the Fisheries Research Board of Canada (Fisheries Research Board of Canada, 1966). Lobster serum, as indicated by Rabin's work, had no bactericidal activity against the pathogen but instead promoted its growth.

A preliminary note by Bell and Hoskins (1966) described experimental transmission of *G. homari* to Dungeness crabs and spot shrimps (*Pandalus platyceros*). Infection was achieved by intramuscular inoculation, but not by ingestion or contact. Mortalities were produced in both species.

A second bacterial disease of lobsters, known as "shell disease" (Hess, 1937), is caused by chitin-destroying gram-negative bacilli. Hess isolated chitin-degrading bacteria from live lobsters impounded at Yarmouth, Nova Scotia, but collected from various parts of the Canadian Maritime provinces. This was the first report of attacks by such microorganisms on living Crustacea. The disease was characterized by a pitting and sculpturing of the exoskeleton (fig. 8); although it was first seen in impounded

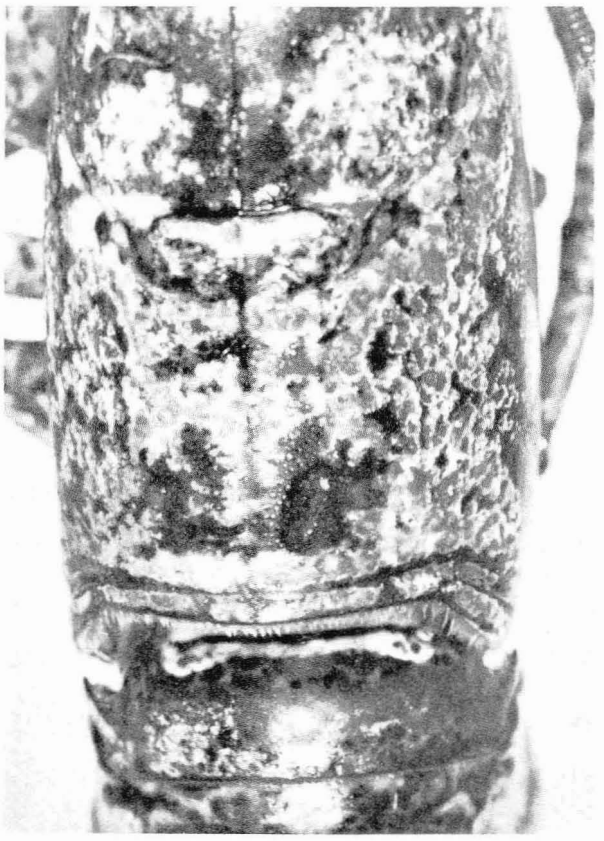

FIGURE 8.—"Shell disease" of American lobster.

lobsters, similar conditions were later observed in freshly caught lobsters from several widely separated Canadian fishing grounds. Initial lesions occurred on the walking legs, and were distinguished by white outer margins, from which the bacteria were most readily isolated. Hess found the disease relatively rare in natural populations but noted severe shell erosion and weakening of lobsters stored in pounds over the winter. Microorganisms isolated were biochemically and physiologically similar to *Bacillus chitinovorous* Type II and Type XIV of Benton (1935). All isolates were able to decompose pure chitin in saline solution containing no other nitrogen or carbon source. None of Hess' isolates—nor, for that matter, isolates

prepared in subsequent work—was reported to reproduce the disease experimentally.

Significant mortalities of lobsters accompanied the shell disease; Taylor (1948) found that 71 percent of infected captive lobsters died from the disease, but observed no correlation between mortality and intensity of external shell erosion. Contraction of the disease by healthy lobsters placed in sea-water tanks with infected individuals indicated direct transmission. The disease developed slowly, requiring at least 3 months before the advanced stages were reached. Progress of chitin destruction was directly temperature-dependent, and new shell laid down after molting was not affected, except by reinfection.

Sawyer and Taylor (1949) observed that shell disease produced thickening or complete destruction of the chitinous layer of the gill filaments. No living gill tissue was attacked, but the authors postulated respiratory impairment as an important consequence of the disease. The infection appeared to be entirely external, confined to the exoskeleton, and not invading living tissue nor transmitted internally. Sawyer and Taylor also reported the disease to be present on the Maine coast as well as in Canada, and considered it a potential threat to the lobster industry, in view of the ease of transmission and the observed mortalities of captive individuals. The method of infection of lobsters is unknown; lodging of bacteria in pores and ducts of the shell was proposed by Sawyer and Taylor as a route of invasion.

Another recently recognized disease of lobsters from the Maine coast, probably of fungus etiology, is called "mottling disease." Characterized by yellowish splotches in an otherwise dark green exoskeleton (fig. 9), the condition has been known for many years as a color variation. Affected individuals are called "leopard lobsters" (Herrick, 1895, 1911). The shell condition and color result from progressive growth of areas of necrosis in underlying tissues and, in advanced cases, even blisters of the shell. The areas of necrosis expand slowly in lobsters held in sea water tanks. Our histological examination of diseased tissues disclosed numerous Schiff-positive, subspherical, heavy-walled bodies 30 to 60μ in diameter. Tentatively, the

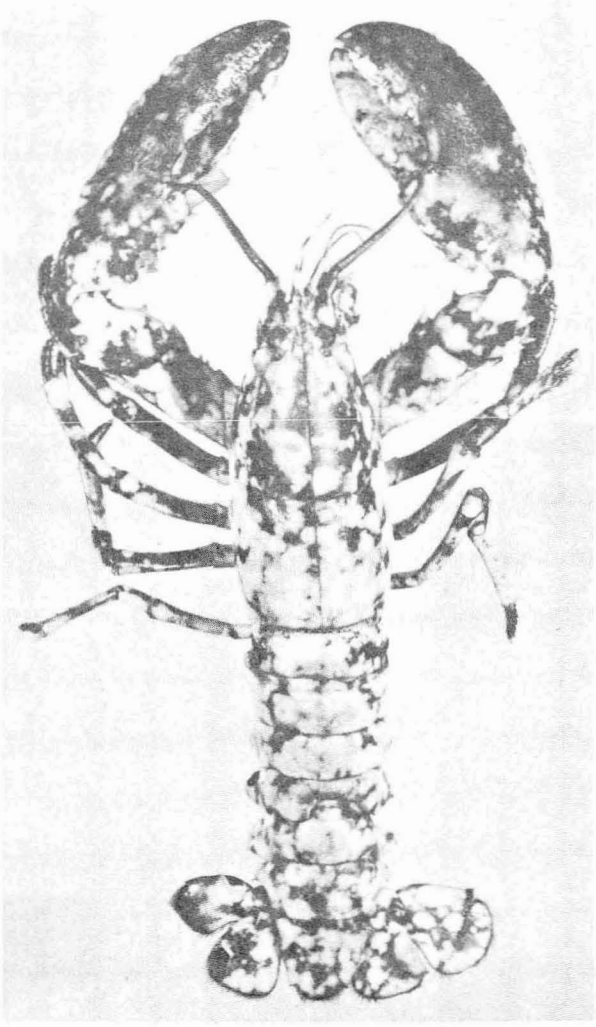

FIGURE 9.—"Leopard lobster" with external signs of mottling disease.

organism is considered a chytrid fungus. Preliminary attempts at culture and transmission have been unsuccessful. The disease occurs infrequently in Gulf of Maine lobster populations and has not been reported from other areas.

Dannevig (1928, 1939) in a report on Norwegian lobster hatcheries, described infection and destruction of eggs on the female by the suctorian *Ephelota gemmipara* Hertwig. The protozoan was found on newly caught individuals, and increased tremendously on lobsters in hatching boxes. Dannevig attributed substantial decreases (90 percent) in production of larvae to the effects of the parasite. The organism was abundant only in certain years.

The gregarine protozoan, *Porospora gigantea* (Van Beneden), has been reported as parasitic in the digestive tract of the European lobster (Hatt, 1928, 1931), and was found in all of 202 American lobsters examined from the Magdalen Islands, Gulf of Saint Lawrence, Canada, by Montreuil (1954). *Porospora nephropis* has been described from the Norway lobster, *Nephrops norvegicus* L., by Léger and Duboscq (1915a) and Tuzet and Ormières (1962).

"Spiny lobsters" (family Palinuridae) are of economic importance in many parts of the world, but little is known about their diseases (Sims, 1966). One fatal and apparently infectious disease of *Panulirus argus* (Latreille) from Florida waters was observed by H. W. Sims (written communication, April 8, 1967). Affected individuals became disoriented and their abdomens were "milky"—a condition reminiscent of microsporidan infections of shrimps (Sprague, 1950a) and fresh-water crayfishes (Sprague, 1950b; Sogandares-Bernal, 1962), as was pointed out by Sims. Two fungus parasites, *Ramularia branchialis* and *Didymaria palinuri*, have been described recently from the gills of *Panulirus vulgaris* in Italy by Sordi (1958). *R. branchialis* was also found on the gills of *Homarus vulgaris*.

Diseases Caused by Helminths

Immature aspidobothrid trematodes, *Stichocotyle nephropis* Cunningham, encyst in the stomach and intestinal walls of lobsters—*N. norvegicus* and *H. americanus*—from Europe and North America (Cunningham, 1887; Nickerson, 1894; Herrick, 1895; Odhner, 1910; Montreuil, 1954; MacKenzie, 1963). Montreuil found the parasite in lobsters taken near the mouth of the Bay of Fundy in the Gulf of Maine but not in more than 500 lobsters examined from the Gulf of Saint Lawrence. Adult *S. nephropis* parasitize several species of skates and rays (Odhner, 1898; Linton, 1940).

A larval nematode, tentatively assigned to the genus *Ascarophis* Van Beneden, has been recognized from lobsters taken off northeastern United States (Anonymous [Uzmann], 1966; Uzmann, 1967a). Adults of the genus occur in fishes, particularly gadoids (Uspenskaya, 1953). Larvae occurred commonly in lobsters from Georges Bank and several canyons along the edge of the Continental Shelf south of Cape Cod, Mass., but were absent in lobsters from near the coast. The larvae were encysted in the rectal wall of 25 percent of the offshore lobsters examined by Uzmann, who speculated that larvae from lobsters reach maturity in abundant cod (*Gadus morhua* L.) and haddock (*Melanogrammus aeglefinus* (L.)) populations of Georges Bank.

A larval acanthocephalan, probably of the genus *Corynosoma*, was identified in American lobsters from the Gulf of Saint Lawrence and elsewhere in the Canadian Maritime Provinces by Montreuil (1954). The worms were usually encysted in the thin wall of the intestine, although some had apparently perforated the gut and encysted in the heart and body muscles. Montreuil believed that accidental gut perforation may provide a route of entry for secondary invaders, and account for appreciable mortality. Feeding experiments with cats and seals suggested that the stage of *Corynosoma* in the lobster is not infective to mammals.

Havinga (1921), in a discussion of artificial lobster rearing in the Netherlands, described the attachment of a small green annelid worm, *Histriobdella homari* Van Beneden, to the eggs and to all parts of the bodies of larval and adult lobsters in Norway. He attributed poor success in production of larvae to effects of the worm. The same parasite had been observed earlier (Sund, 1914, 1915) in massive numbers on eggs of lobsters held in floating boxes at Korshavn, Norway, where it was held responsible for destruction of the brood. Every female lobster was infested with thousands of worms, and they also occurred on larvae. Although *H. homari* had not been reported previously from American lobsters, Uzmann (1967b) has recently found it to be widely distributed on the gills of lobsters in New England coastal waters from Maine to Connecticut, and on those from Georges Bank as well.

Diseases Caused by Parasitic Crustaceans

A blood-sucking parasitic copepod, *Nicothoë astaci* Audouin and Milne-Edwards, has been found on the gills of European lobsters. The parasite was prevalent in Scottish waters (Thomas, 1954; Mason, 1958, 1959) and was seen by Korringa (1957b) in lobsters being

held in the Netherlands. Mason found infestations to be occasionally heavy, with a maximum of 1,700 copepods on a single lobster. He concluded that such heavy infestations could harm the host through loss of blood and reduction in gill surface available for gas exchange, but that the usual level of infestation (100 or less per lobster) caused little or no harm to the host

Tumors

One of the earliest observations of neoplasms in invertebrates, according to Scharrer and Lochhead (1950), was made by McIntosh and reported by Prince (1897). A lobster tumor originated in the stomach wall and pushed through the carapace behind the eyes, enlarged, and finally killed the lobster.

SHRIMPS

Many shrimps of the families Penaeidae and Pandalidae are of worldwide commercial significance. Shrimps are the most valuable fishery resource in the United States (Lyles, 1966). Parasites and diseases, which may have adverse effects on shrimp stocks, have been studied, particularly in the Gulf of Mexico. Several diseases caused by Microsporida are known, and larval helminths—trematodes, cestodes, and nematodes—have been reported. Isopods and rhizocephalans have also been observed.

Microbial Diseases

Protozoan parasites have been shown to be of significance to commercial shrimp populations of the Gulf of Mexico. Several microsporidan Protozoa cause a condition known as "cottony" or milky" shrimps. *Nosema nelsoni* was described by Sprague (1950a) from the brown shrimp, *Penaeus aztecus* Ives. Affected body muscles had an opaque white discoloration, and black pigment spots occurred externally. The disease was common in bait shrimps as well as in those processed as food. Infected individuals did not survive well in bait tanks and were also discarded in processing plants, thus representing significant losses to the industry (Woodburn, Eldred, Clark, Hutton, and Ingle, 1957; Hutton, Sogandares-Bernal, and Eldred, 1959). Sprague (1950a) found microsporidan spores in the gonads of the white shrimp, *P. setiferus* (L.), some of which he described as *Thelohania penaei*. Earlier, Viosca (1945) reported that "about 90 percent" of white shrimp in Louisiana waters were infected in 1919 by a protozoan disease (not further identified) which destroyed the reproductive organs. If the disease was caused by *T. penaei*, the microsporidan may play a role in fluctuations of the host species when present at epizootic levels. Iversen and Manning (1959) described still another microsporidan, *Thelohania duorara*, from the musculature of pink shrimp, *P. duorarum* Burkenroad, of the Gulf of Mexico. Infected individuals were relatively rare, however, in landed catches. Brazilian brown shrimp, *P. brasiliensis*, also harbor the same parasite (Iversen and Van Meter, 1964).

Several gregarine Protozoa also occur in shrimps. Sprague (1954) tentatively identified as *Nematopsis penaeus* a gregarine from the digestive tract of brown shrimp from Louisiana. The same parasite was observed by Kruse (1959a, 1959b) and Hutton, Sogandares-Bernal, Eldred, Ingle, and Woodburn (1959) in several species of shrimps from Florida. Sprague mentioned the possibility of extensive damage to the intestinal epithelium of individuals heavily infected by *Nematopsis*. Kruse described a second gregarine from the digestive tracts of brown and pink shrimps in Florida, and Hutton, Sogandares-Bernal, Eldred, Ingle, and Woodburn (1959) observed the same parasite with high frequency in certain samples of pink shrimp from Florida.

Diseases Caused by Helminths

Shrimps occasionally harbor larval helminths. Metacercariae of the trematode *Opecoeloides fimbriatus* (Linton) were found by Hutton, Sogandares-Bernal, Eldred, Ingle, and Woodburn (1959) and Sogandares-Bernal and Hutton (1959) in several Florida species. Larval *Microphallus* sp. have been reported from the body muscles and hepatopancreas of pink shrimp (Hutton, Sogandares-Bernal, and Eldred, 1959; Hutton, Sogandares-Bernal, Eldred, Ingle, and Woodburn, 1959).

Several larval cestodes of the order Trypanorhyncha have been found in the digestive gland and other organs of shrimps. Larval *Prochristianella penaei* Kruse were identified from four species of shrimps—brown, pink, white, and humpback (*Trachypeneus constrictus* (Stimpson))—from the Florida coast (Sparks and

Mackin, 1957; Woodburn et al., 1957; Hutton, Sogandares-Bernal, Eldred, Ingle, and Woodburn, 1959; and Kruse, 1959a, 1959b). Aldrich (1965) found the same cestode larvae in *Penaeus aztecus* and *P. setiferus* from the Texas coast. The adult worm was identified from the Atlantic stingray *Dasyotis sabina* LeSueur. Another trypanorhynch larva, unidentified, was seen by Ward (1962) in great numbers in the abdominal muscles, gills, and pericardium of the white shrimp from the Gulf of Mexico.

Trypanorhynch larvae have been found in commercial shrimps from other parts of the world. Yamaguti (1934) reported larvae, probably *Tetrarhynchus rubromaculatus* Diesing, in *Penaeopsis* sp. from Japan. Heldt (1949) took a larval cestode resembling *Eutetrarhynchus ruficollis* (Eysenhardt) from *Penaeus trisulcatus* Leach from the North African coast.

Larval nematodes of the genus *Contracaecum* were found in Florida shrimps by Woodburn et al. (1957), Hutton, Sogandares-Bernal, Eldred, Ingle, and Woodburn (1959), and Kruse (1959b). Margolis and Butler (1954) observed adult nematodes, *C. aduncum*, in a single specimen of northern pink shrimp *Pandalus borealis* Krøyer from British Columbia, Canada.

Diseases Caused by Parasitic Crustaceans

Epicaridean isopods are well-known parasites of Crustacea, and several genera occur on shrimps. Baer (1951), for example, stated that the epicaridean *Hemiarthrus abdominalis* (Krøyer) had been recovered from 20 species of shrimps belonging to the genera *Pandalus* and *Spirontocaris*. Joseph Uzmann (personal communication, Jan. 31, 1967) has found *H. abdominalis* on northern pink shrimp from the Gulf of Maine. The parasite has also been reported on *P. borealis* from Greenland (Horsted and Smidt, 1956) but not from Norway or England (Dahl, 1949; Allen, 1966).

Ricketts and Calvin (1962) described the occurrence of the bopyrid isopod *Argeia pugettensis*, which caused unilateral protuberances of the carapace of the black-tailed shrimp, *Crago nigricauda* (Stimpson), from the Pacific coast of the United States. Infestation was estimated at 3 to 5 percent. Japanese "red prawns," *Penaeopsis akayebi* Rathbun, are frequently (up to 70 percent) infested with another bopyrid *Epipenaeon japonicus* Thielemann. Hiraiwa and Sato (1939) found the gonads of parasitized individuals reduced, or in some males, completely atrophied. Presence of the branchial parasite *Bopyrus squillarum* on the shrimp *Leander serrifer* causes suppression of the ovaries and the breeding characters of the pleopods (Yoshida, 1952).

Several rhizocephalans have been reported as parasites of shrimps. Potts (1912) described *Mycetomorpha vancouverensis* from *Crago communis* Rathbun, and Calman (1898) described *Sylon hippolytes* from the dock shrimp, *Pandalus danae* Stimpson, both from Puget Sound, Wash.

DISEASES AS A POSSIBLE CAUSE OF MASS MORTALITIES

Many physical, chemical, and biological variables contribute directly or indirectly to mortalities of commercially valuable marine invertebrates. Various environmental factors and some of their effects have been discussed by Dexter (1944), Brongersma-Sanders (1957), Coe (1957), Mackin (1961), Dickie and Medcof (1963), Medcof and Bourne (1964), and Merrill and Posgay (1964). It seems clear that the actual cause of death in many mass mortalities is often undetermined, even after exhaustive studies such as those of Orton (1924a, 1924b), who studied oyster mortalities in England in 1920–21, and Roughley (1926), who examined oyster mortalities in Australia in 1924–25.

Disease has often been suspected as a cause of mortalities, but the actual disease agent often has proved to be elusive. Hirsch (1921), Dollfus (1923), and Korringa (1952a) reported major mortalities of sea mussels, probably due to a contagious disease, in the period 1900–19. The mortalities reached a peak in 1914–16. Sick mussels lost their byssal attachment, mantles were retracted, meats were thin, and adductor muscles were weak. Histological and bacteriological examinations were inconclusive. Soon thereafter—1919–23—catastrophic mortalities of European oysters occurred in western Europe. Deaths began in 1919 in Mar Piccolo, near Taranto, Italy (Cerruti, 1941), and quickly spread to England and other European countries. Orton (1924a) suspected, but was

unable to demonstrate, a bacterial pathogen. Although no infectious agent was directly associated with the mortalities, disease signs such as mantle retraction, pale digestive gland, muscle degeneration, and pustules on the shell and mantle were seen. Ulcerations and pustules on the body and mantle, and shell pustules containing dead or moribund leucocytes were observed in oysters from England and the Netherlands during periods of mortality (Orton, 1937). These signs often result from disease. The exhaustive studies of Orton were supplemented by those of Eyre (1923, 1924), who isolated a number of species of bacteria from sick and healthy oysters, but doubted that any were true pathogens.

Korringa (1952a) gave an excellent historical account of these mortalities of oysters in Europe. Cultured-oyster beds in France, England, Denmark, Germany, and the Netherlands were affected almost simultaneously. Many natural beds were also destroyed. A few isolated populations—Helgoland and Brittany—were not affected until several years later. Mortalities did not occur in Portuguese oysters during this time. Although environmental factors such as poor food supply and low temperatures were held by some to be causes of the catastrophic mortalities (Gaarder and Alvsaker, 1941; Spärck, 1950), the available evidence strongly indicates an infectious disease (Cole, 1951b; Fischer, 1951; Korringa, 1952a).

A mortality, with characteristics very similar to those seen in the European oyster, was described by Roughley (1926) in populations of rock oysters, *Crassostrea commercialis* (Iredale and Roughly), from Australia. Oysters died in 1924 and 1925 in Georges River, New South Wales. Disease signs, such as abscesses and ulcerations, were observed, and a bacterial pathogen was suspected—possibly combined with winter environmental stresses.

Disease-associated mortalities, with a history of long and frustrating scientific study, were first observed in 1915 in American oysters of Prince Edward Island, Canada (Needler and Logie, 1947). In the period 1915–33, the disease (commonly known as "malpeque disease") spread around the Island and destroyed most of the oyster stocks—some of which required 20 years to return to previous levels of abundance (Logie, 1956). During the outbreak, oysters apparently developed resistance to the causative organism, whose identity remains undetermined. Beginning in 1955, mortalities, probably due to the same disease, began in waters of the adjacent mainland of New Brunswick across Northumberland Strait. Oyster populations along the entire northern coast of New Brunswick and Nova Scotia were decimated, but mass transfer of disease-resistant oysters from Prince Edward Island waters, beginning in 1957, has hastened the recovery of the fishery (Logie, Drinnan, and Henderson, 1960; Drinnan and England, 1965).

Pacific oysters imported as seed from Japan and planted in waters of the States of Washington and California began to die in significant numbers in the late 1950's. Oysters in their second year after introduction were most commonly killed; peaks of mortalities occurred in late summer; and deaths were most often observed at the heads of bays. In the absence of other obvious environmental changes, and because of the selective nature of the mortalities, it seems logical to suspect disease. A pathological condition described by us as "focal necrosis," has been found in seed from Japan and in several samples of larger oysters from beds in Washington. As many as 30 percent of the individuals in a sample were affected. In addition, a haplosporidan parasite, morphologically similar to the pathogen *Minchinia nelsoni* associated with recent mortalities on the U.S. east coast, has been seen by staff members of the BCF Biological Laboratory, Oxford, Md., in Pacific oysters from the State of Washington; a similar organism was recognized recently in a sample of seed oysters from Taiwan. Pereyra (1964) mentioned a "multinucleated MSX-like organism, possibly pathogenic" in a dying oyster from Oyster Bay, Wash. No clear association has yet been made, however, of specific pathogens with mortalities of *C. gigas* on the Pacific coast of the United States, and it is quite possible that other environmental factors are operative in the mortality areas.

The Japanese literature contains numerous historical accounts of mass mortalities of oysters dating back to 1915. Although disease was often suspected, specific pathogens were usually not identified. Takeuchi et al. (1960) mentioned

large-scale deaths of oysters in Kanasawa Bay, beginning in 1915 and continuing for a number of years. Over 80 percent of the oysters in that bay died annually. Ogasawara et al. (1962) reported similar mass mortalities on the Miura peninsula, beginning in 1927 and continuing for 10 years. Oyster farms along the coast of the peninsula lost 50 to 80 percent of their crop annually. More recent mortalities of 2-year-old oysters have occurred in Hiroshima Bay and adjacent localities, beginning in 1945 (Fujita et al., 1953). A 10-year study (Takeuchi, Matsubara, Hirokawa, and Tsukiyama, 1955, 1956; Takeuchi, Matsubara, Hirokawa, and Matsuo, 1957; and Takeuchi et al., 1960) provided somewhat inconclusive evidence that a bacterial pathogen was responsible for the mortalities.

A series of papers by Tohoku Regional Fisheries Research Laboratory (Imai et al., 1965; Kan-no et al., 1965; Mori, Imai, Toyoshima, and Usuki, 1965; Mori, Tamate, Imai, and Itikawa, 1965; Numachi et al., 1965; Tamate et al., 1965) described mass mortalities of oysters in Matsushima Bay, Miyagi Prefecture, Japan, that have occurred annually in late summer since 1961. Environmental, physiological, and pathological factors were examined. Pathological changes were observed, and mortalities were considered to be related to metabolic changes during fattening and spawning. Mortalities exceeded 60 per cent per year in certain areas of the Bay during 1961–64. A gram-positive bacterium was found in multiple abscesses in as many as 20 percent of oysters in certain samples (Numachi et al., 1965), but a causal relation with mortalities was not established. Our later studies suggest that the disease condition is the same as that called "focal necrosis" in adult Pacific oysters from Washington. The pathogen warrants further observation, since the abscesses may represent only the chronic stage of infection in resistant hosts, whereas the acute disease may have a significant effect on mortality. An ameboid organism, often present in large numbers and accompanied by pronounced host response, has also been found in oysters from the Matsushima Bay mortality area.

Blue crab populations on the coasts of North and South Carolina have been affected by extensive mortalities beginning in 1965 (Lunz, 1967). Significant impact on population size was indicated by a marked drop in catch per unit effort in 1966, as compared with the previous 5 years. Newspapers stated that great numbers of crabs were washed up on beaches or littered the bottoms of creeks, and that catches were drastically reduced. Disease has been suspected (Lunz, 1967), but no clear evidence of a pathogen has been obtained. Such mortalities in wild crabs are apparently distinct from the long-recognized high levels of deaths in crab shedding floats (Beaven and Truitt, 1939), which may in part be associated with parasites or diseases (Couch, 1966; Sprague and Beckett, 1966).

A few mass mortalities of marine invertebrates have been definitely ascribed to epizootics caused by specific pathogens (Sindermann, 1963). Recurring mortalities of American oysters in the Gulf of Mexico were found to be caused by the fungus *Dermocystidium marinum*. Exerting its effects in higher salinities and temperatures among dense aggregations of hosts, the pathogen can cause annual mortalities in excess of 50 percent. Development and use of a presumptive test, with thioglycollate medium (Ray, 1952), has established the presence of the organism in oysters throughout the Gulf of Mexico and northward along the Atlantic coast as far as Connecticut. Although the fungus may at times reach epizootic levels in particular areas, its most significant effect is probably that of continuing attrition, year after year, during periods of high sea-water temperature. Effects of the disease on commercial beds are now controlled to some extent by planting and harvesting at prescribed times of the year and by spreading oysters thinly on the beds.

Major mortalities, with consequent severe depression of the oyster fishery, occurred in Delaware and Chesapeake Bays on the U.S. east coast, beginning in the late 1950's. A haplosporidan parasite with distinctive characteristics, *Minchinia nelsoni*, has been associated with the mortalities. Epizootic areas have had oyster losses in excess of 90 percent, and some indications are appearing of increased resistance among survivors. The disease, like that caused by *Dermocystidium*, exists and exerts severe effects in salinities above 15 o/oo. Seed beds and oyster stocks in low-salinity areas have not

been destroyed. Recently the organism has been found in oyster populations on the coasts of New York and North Carolina—well outside previous areas of high mortality.

Studies of these serious pathogens of oysters —*D. marinum and M. nelsoni*—have revealed the very important role of a "salinity barrier" to certain diseases. The fungus *D. marinum* exerts severe effects on oyster populations in high-salinity waters of the Gulf of Mexico but does not flourish in low-salinity areas. *M. nelsoni*, which has seriously affected oyster stocks of the Middle Atlantic States, also occurs in higher salinities. Both pathogens seem confined to salinities above 15 o/oo; this fact has made possible the continuation of production in parts of coastal areas affected by these epizootics.

Inhibitory effects of low temperature have been well illustrated for these serious oyster pathogens. *D. marinum* causes warm-weather mortalities in American oysters; in fact, the plantings of seed oysters are timed to take advantage of the relative quiescence of the disease in cooler seasons. Surveys of *Dermocystidium* have indicated marked decline in winter. *M. nelsoni*, of Chesapeake Bay and Delaware Bay, is similarly quiescent in winter. New infections are not apparent, prevalence of disease declines, existing infections seem less active, and mortalities are reduced.

Mackin (1961) attempted, from a review of the literature, to itemize characteristics of mortalities of oysters due to various causes. As one who has published extensively on the role of disease in oyster populations, he naturally turned his attention toward mortalities caused by infectious agents. Among many interesting comments in his paper, Mackin stated that "all oyster producing bays are endemic areas for one or more diseases" and that "not only are bivalve mollusks frequent hosts for pathogens, but they are regularly parasitized by a unique group of low fungi." Mackin further stated his belief that "of all causes of mortality, disease ranks first." Disease, then, can cause significant, if temporary, reductions in population abundance of marine invertebrates. Such reductions may exceed 95 percent of existing stocks. Additionally, there is every indication that serious but undescribed diseases exist among marine invertebrates. Mackin (1962), for example, mentioned a number of pathological conditions in oysters that were not associated with known pathogens. Rust disease of Pacific king crabs, which we have described on the basis of an unpublished report, is a commonly recognized condition in the fishery, but has not been described in the published scientific literature.

Destruction of most of a population by epizootics and mass mortalities, of course, also reduces pathogen numbers, because the possibility of finding a new susceptible host at a critical point in the life cycle is reduced.

Less spectacular mortalities, which also have severe continuing depressive effects on host population size, are probably more common than large-scale or mass mortalities. Minor fluctuations in abundance may be attributable to such "background" mortalities. Also, those parasites and diseases that do not kill the host may act as indirect agents of mortality. Abnormal individuals are rendered more vulnerable to predation in many ways: their body muscles may be partially destroyed, covering or erosion of their gills may interfere with respiration, or their normal protective coloration may be modified or obscured. For example, Hopkins (1957a) has observed that blue crabs prey more frequently on oysters which cannot close their shells as quickly or as tightly as normal oysters. Any increase in parasite burden must reduce the probability of survival in an environment where death, early and sudden, is the rule rather than the exception. For parasites with complex life cycles involving two or more hosts, consumption of an earlier host in the cycle—one weakened by the parasite—by the right predator may be critical to the completion of the cycle.

Another prominent effect of parasitization of marine mollusks and crustaceans is sterilization of the host. Larval trematodes are notable for destroying the gonads of gastropods and bivalves, and parasitic barnacles and certain isopods produce similar effects in crustaceans. In areas where levels of parasitization are high, the reproductive capacity of the host population may be seriously impaired. In a study of the ecological relation of the marine snail, *Littorina littorea*, and its trematode parasite, *Cryptoctyle lingua*, Sindermann and Farrin

(1962), and Sindermann (1965, 1966a) found prevalences of the parasite of over 50 percent in certain coastal areas, indicating that the reproductive potential of snail populations was suppressed by that amount. An excellent review of parasitic castration of Crustacea has been provided by Reinhard (1956).

Effects of disease can be generally categorized as catastrophic, resulting in mass mortalities, or continuing, producing a constant drain on population numbers. Although disease is always with us, and mortalities have undoubtedly occurred in the past, new factors have been introduced by man to set the stage for the spread of epizootic disease. For example, oysters are transferred promiscuously from one geographic area to another; populations are often crowded in dense beds, sometimes in areas where natural populations did not exist previously; drastic physical and chemical changes have been made in oyster habitats; and new predators have been introduced. A dominant mortality factor—disease—has been aided by human activities; it must be controlled, if we are to achieve maximum production of cultivated inshore mollusks and crustaceans.

Many different environmental factors—physical, chemical, and biological—can kill oysters, crabs, or other animals of commercial value (Brongersma-Sanders, 1957). Any single factor may become overriding, however, at a particular time in the life of a species—and in this paper we have described several examples of how the factor of disease can reduce the abundance of marine species.

It is easy, of course, to overextend any point of view; we do not imply here that every decrease in abundance can be blamed on disease. An excellent case could also be made from the published literature for the significant role of predation—particularly during population peaks of particular predator species—as a major cause of fluctuations in abundance of commercially valuable species. Man-made changes in environment can also affect abundance. Because of industrial pollution, shellfish populations have been eliminated from certain localized areas within estuaries and along the coast. In addition, other types of pollution have made extensive areas in rivers and bays unavailable for the harvesting of shellfish. It is likely that mass mortalities are, and have always been, natural methods of population regulation—but, until recently, these mortalities would have been accepted with the same dazed bewilderment and inaction that must have characterized the behavior of our ancestors during the plagues of the dark ages. We can now look to methods of environmental control and stock manipulation, particularly for sedentary shallow water species such as oysters, clams, and even certain Crustacea, as part of the methodology of an increasingly complex system of cultivation of our inshore waters.

CONTROL OF DISEASES OF MARINE INVERTEBRATES

The original, persistent, and largely erroneous feeling about disease in marine populations is that little can be done about it. This pessimistic attitude is definitely unwarranted for species that live inshore—particularly the sedentary invertebrates—where practical measures of disease control are possible and have already been applied in some situations. Possible methods include the following:

1. *The transfer of susceptible animals into epizootic areas, or of individuals from such areas, should be prevented.* Because each disease is discrete in terms of transmission and infectivity, risks of transfer will vary as well. When intermediate or alternate hosts play a significant role in maintaining the disease in a given geographic area, transfer of infected individuals to other areas where these hosts are absent may be a reasonable management procedure. Diseases that have been demonstrated experimentally to be transmitted directly, however, such as *Dermocystidium* infections of oysters, may be maintained at epizootic levels by repeated introduction of susceptible animals.

2. *Disease-resistant stocks should be developed by selective breeding of survivors.* Epizootics of several oyster diseases have apparently produced increased resistance among survivors. During the outbreak of "malpeque disease" in Prince Edward Island (Canada) oysters, resistance developed to an unidentified pathogen, and stocks returned to previous levels of abundance after several decades. That the pathogen is still present is indicated by deaths of suscep-

tible oysters from other geographic areas introduced into Prince Edward Island waters. Resistant stocks were drawn upon to repopulate other oyster growing areas of the Gulf of Saint Lawrence that had been subsequently decimated by the same disease.

Evidence is accumulating that increased resistance to the haplosporidan pathogen *Minchinia nelsoni* is developing among oysters that have survived the disease in the Middle Atlantic States. The disease has been at epizootic levels in some Chesapeake Bay populations for several years. Perhaps resistant strains can be developed with presently available hatchery techniques. Aggregation of survivors on natural beds to which adequate cultch has been added could also do much to improve reproduction, spatfall, and return to full production.

3. *Basic information about the life history and ecology of the disease agent must be accumulated, to define vulnerable stages or restrictive environmental requirements.* As an example, several oyster pathogens, such as *M. nelsoni*, are limited to salinities in excess of 15 o/oo. Plantings during epizootics can be restricted to low-salinity areas, and temporary transfer of infected stocks to low salinities may retard or eliminate infections. Mechanical and chemical treatments can also reduce disease prevalence. Effects of gaffkaemia on impounded lobster populations have been reduced by treating bottom muds of pounds with calcium hypochlorite. Damages of dermocystidium disease to oysters have been lessened by planting oysters thinly on the beds, by harvesting within 2 years, and by planting and harvesting at prescribed seasons to take advantage of the decrease in pathogen activity during the colder months.

Korringa (1959) outlined an extensive program to control the spread of the parasitic copepod *Mytilicola* in cultivated mussel stocks of the Netherlands. Included were extensive dredging of adjacent natural beds, transfer of lightly infested stocks, and destruction of heavily infested beds.

Korringa (1951a) found "shell disease" of oysters to be caused by a fungus that perforated the shell—a fungus that thrived on old shells. He attributed the outbreak of shell disease in 1930 in the Netherlands to the practice of spreading enormous quantities of cockle shells on the beds. The disease declined when the spat collectors were placed in areas free of the disease, when old shells were cleared from beds, and when infected young oysters were dipped in mercuric disinfectant (Korringa, 1948, 1949, 1951c).

Biological control of other hosts in the life cycle of parasites, or biological control of the parasite itself, are also possible approaches.

4. *Production could be maintained in artificial environments where disease can be controlled.* Some progress has been made in this direction with the development of hatchery methods of producing seed oysters and clams (Loosanoff and Davis, 1963). Bacterial and fungal epizootics in larval culture tanks can be prevented, or their effects reduced, by ultraviolet treatment of filtered sea water, antibiotic treatment of sea water in standing water cultures, maintenance of general cleanliness of all utensils used in handling larvae, and ultraviolet treatment of phytoplankton food derived from impure mass cultures.

Shellfish production in artificial ponds (Shaw, 1965) offers distinct possibilities of disease and predator control, beginning with disease-free and disease-resistant brood stock and progressing to filtration and ultraviolet treatment of recirculated water; important also are careful control of contaminants in mass phytoplankton cultures and elimination of shellfish associates that act as alternate or intermediate hosts of disease agents.

CONCLUSIONS

Many of the great fisheries of the world have undergone large fluctuations in supply. The causes of these fluctuations, although subjects of much discussion, have rarely been precisely determined. Reduction in abundance of commercially valuable marine species has been attributed to overfishing, failure of spawning, sudden and drastic changes in temperature and salinity, and many other factors. One biological factor that has received too little attention is disease. The fact that marine animals become ill and die, often in vast numbers, has been largely ignored. Events in commercial shellfish

populations in this century, however, have forced us to look closely at disease as a cause of mass mortalities of epic proportions, and of subsequent major declines in abundance of commercial species.

Molluscan and crustacean species of economic value as food have been affected by diseases, some of which have produced epizootics with resultant mass mortalities. Much attention has been directed toward oyster diseases, possibly because oysters have been cultivated more intensively than most other inshore or estuarine species. Microbial diseases—including those of bacterial, fungal, and protozoal etiology—have affected oyster stocks in many parts of the world. Bacterial diseases have had serious effects on lobsters, and a number of Protozoa, particularly Microsporida, affect crab and shrimp populations.

It is often difficult to establish the precise cause of death of marine invertebrates—to determine whether a suspected pathogen is a primary or secondary invader. Environmental and physiological factors can be inextricably associated with apparent disease; their relative effects are often not easy to assess. Thus, the literature on mass mortalities contains measurements of many environmental variables, descriptions of physiological conditions of host animals, and reports of suspected disease agents —but too frequently the studies have been unable to point to a single cause of death. The search for a single cause may have been an oversimplified approach to a complex problem. In other situations, epizootics of specific pathogens, possibly influenced to some extent by environmental factors, can be directly related to the state of resistance of the host population, the virulence and infectivity of the pathogen, and infection pressure.

Mass mortalities, many of undetermined causes but some definitely the result of disease, have occurred in commercial invertebrate populations. These mortalities are a natural method of regulating population size; they have received increasing scrutiny in recent years. The development of methods of cultivation and of limited manipulation of the inshore environment should make it possible to reduce or eliminate the serious threat of disease to populations of commercial shellfish.

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