HELGOLÄNDER MEERESUNTERSUCHUNGEN
Helgoländer Meeresunters. 37, 149–166 (1984)

Epizootiology of diseases of oysters *(Crassostrea virginica)*, and parasites of associated organisms in eastern North America*

J. D. Andrews

Virginia Institute of Marine Science,
School of Marine Science;
Gloucester Point, Virginia 23062, USA

ABSTRACT: Haplosporidan parasites of oysters have been reported from four continents. Those of the genera *Minchinia, Haplosporidium,* and *Marteilia,* which cause serious diseases of oysters, have been intensively studied. Epizootiology of these highly pathogenic species is well known. Life cycles are obscure for all haplosporidans because artificial infections have not been achieved. The high pathogenicity of newly-discovered haplosporidan diseases to native oysters in eastern North America and western Europe may indicate that these are exotic pathogens parasitizing susceptible oysters not previously exposed to these disease agents. Epizootiology of two haplosporidan pathogens along the middle Atlantic Coast of North America during 25 years of disease activity is discussed. *Haplosporidium nelsoni* sporulates only rarely and its life cycle remains unconfirmed. Resistant oysters were developed in nature and from laboratory breeding. *Haplosporidium costale* which causes "Seaside Disease" in high-salinity waters appears to be a more acclimated disease with regular patterns of infection and mortality. Several minor parasites whose life cycles and host species need more study are mentioned.

STATUS OF OYSTER FISHERIES ALONG MIDDLE ATLANTIC COAST OF NORTH AMERICA IN THE 1980'S

The oyster fisheries of Delaware Bay and Chesapeake Bay were severely depressed after 1957 and 1959, respectively, when a new disease appeared caused by *Haplosporidium nelsoni* (MSX), a haplosporidan parasite (Haskin et al., 1966). Production declined greatly (Sindermann, 1976) and recruitment of seed stocks fell sharply after mortalities caused by the disease. Oystermen were forced to alter cultural practices to avoid or minimize the effects of the disease. Mortalities were severe and growth was inhibited. Adjustments of cultural practices because of the disease were quite different in the two major growing areas of Delaware Bay and Chesapeake Bay. This was because of differences in prevailing salinity regimes. Fortunately, the seed areas for both of these bays were located in low-salinity waters (< 18 ‰) where in most years the disease did not kill oysters (Fig. 1).

Delaware Bay oystermen resumed the practice of transplanting seed oysters from relatively low to high salinities where the disease was prevalent. Growing areas were

* Contribution No. 1147. Virginia Institute of Marine Science; Gloucester Point, Virginia 23062

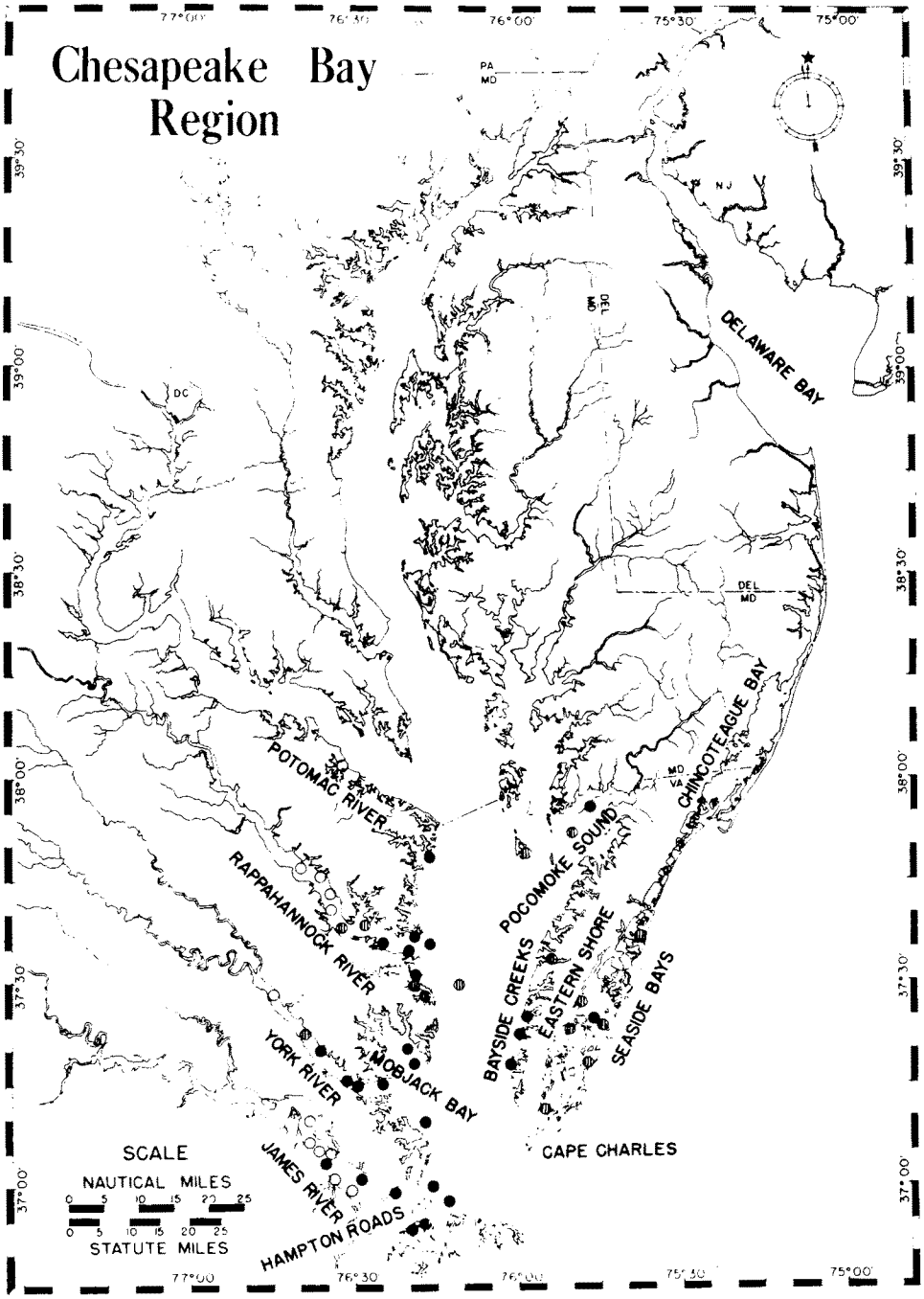

Fig. 1. Map of Chesapeake Bay showing major estuaries and sampling areas where beds and trays of oysters were monitored for diseases, 1959 to 1982

located in high salinity waters where MSX was enzootic (Haskin & Ford, 1982). This was possible only because strains of Delaware Bay oysters with resistance to the disease had been selected after 95 % of each year class was killed over a period of about 10 years. Infections and deaths occurred in oysters on nearly all the seed beds as well as in growing areas during the early years of 1957 to 1960 and again in a drought period from

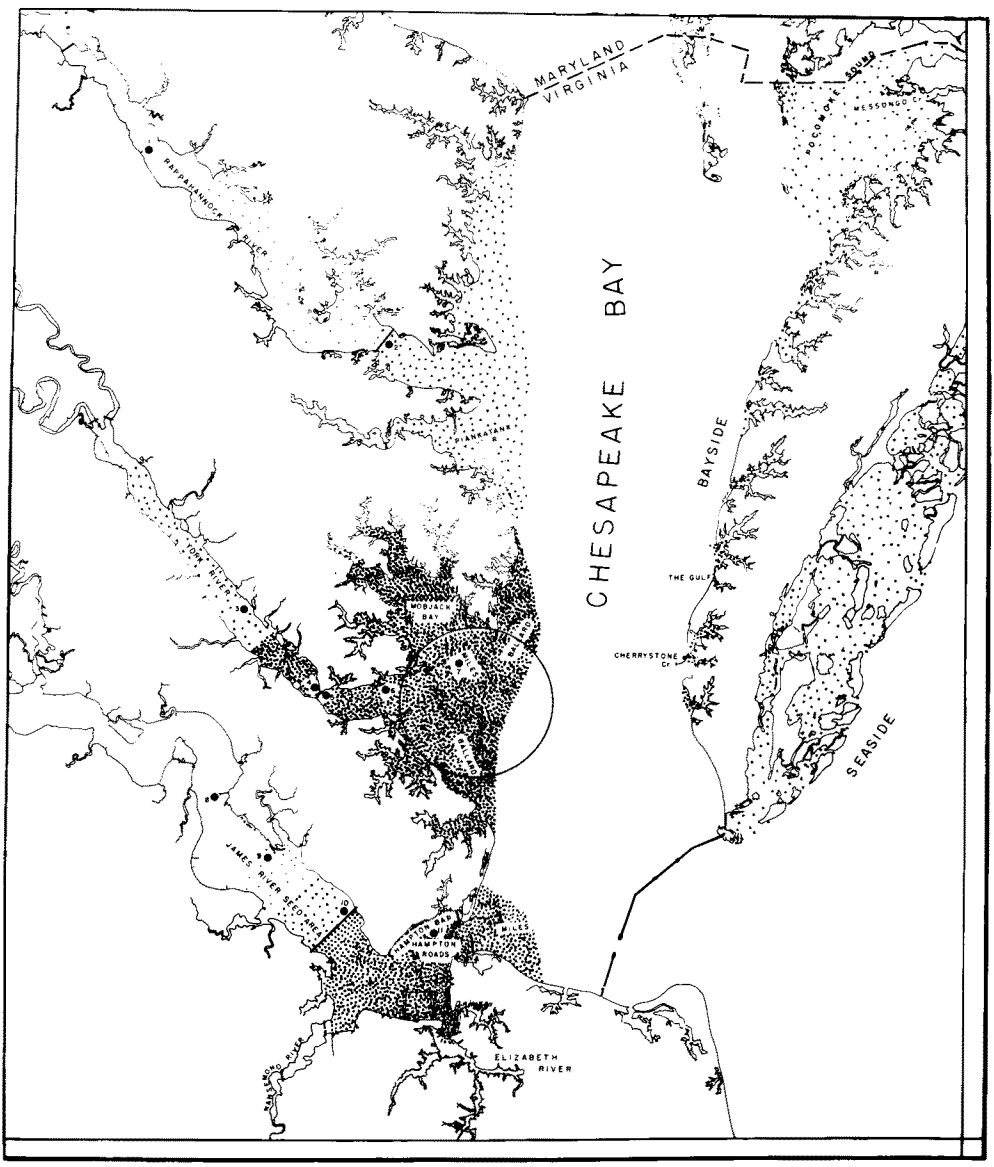

Fig. 2. Distribution of *Haplosporidium nelsoni* in Virginia by intensity of activity based on mortalities and prevalences of the disease. MSX is enzootic in heavily stippled areas but it spread through lightly stippled areas during periods of drought and high salinities

1963 to 1967. In areas where the disease was enzootic, mortalities of oysters were reduced to one-half the original rates by selection for innate resistance (Ford & Haskin, 1982). Persistent culling of susceptible oysters by the disease over 25 years maintained a resistance level in broodstocks; mortalities caused by the disease were low in the seed area during most years.

In Chesapeake Bay, the area enzootic for *Haplosporidium nelsoni* is confined to the lower Bay below the Rappahannock River mouth in Virginia (Fig. 2) (Andrews & Wood, 1967). Prior to 1960, most market oysters in Virginia were produced on private beds leased from the state, whereas public beds, except low-salinity seed beds, were relatively unproductive. All privately planted beds of oysters in the enzootic area were destroyed by the disease and they have not been replanted since 1960. Broodstocks in rivers and in the Bay above the enzootic area were lightly selected by the disease only during the high-salinity period of the mid-1960's. Oysters in Maryland including the Potomac River were therefore highly susceptible to *H. nelsoni* as were seed oysters from the James River which supplies Virginia planters. Naturally occurring wild oysters which are resistant to the disease are not available in Chesapeake Bay; therefore, the enzootic area cannot be used to grow market oysters. Annual mortality of susceptible James River seed oysters planted in enzootic areas averaged 50 to 60 % the first year and 40 to 50 % the second year over a period of 20 years (Andrews, unpubl.). This resulted in 70 to 80 % deaths from the disease alone during the two years of growth required before marketing of oysters. Oystermen were compelled to move upriver and upbay into low-salinity waters to culture oysters (Andrews & Wood, 1967).

EFFECTS OF SALINITY ON DISTRIBUTION OF MSX DISEASE

Salinity is the major physical factor controlling distribution of *Haplosporidium nelsoni* along the middle Atlantic Coast. Lowest annual salinities occur in late winter and spring when freshwater discharge is greatest. Salinities increase gradually in summer and fall as widespread rainfalls are replaced by sporadic thunderstorms of local distribution which cause little freshwater discharge. The mouth of the Rappahannock River, which marks the upbay limit of the enzootic area, exhibits late-summer salinities of about 20 ‰. Salinities > 20 ‰ provide a favorable environment for the disease to cause high mortalities. A similar isohaline value was reported by Haskin & Ford (1982) for the upper enzootic limit of *H. nelsoni* in Delaware Bay, on a line which is located near the lower edge of the seed area. In the James River seed area, occasional summer infections of MSX are expulsed by oysters the following April and May when salinities are < 10 ‰ (Andrews, 1983). An invasion of non-enzootic areas into Virginia and Maryland occurred in 1981 and 1982 during three years of drought (1980–1982), which was accentuated by a dry winter and spring (1980–1981) with < 50 % of normal river discharge into the Bay (Andrews, 1983). These 1982 infections were mostly eliminated from non-enzootic areas during a wet winter and spring in 1983. Some disease mortalities occurred in Virginia and Maryland estuaries above the enzootic area in 1981 and 1982.

Haplosporidium nelsoni is distributed along the coast in high-salinity areas from South Carolina to Massachusetts, but it causes serious infections and mortalities only from Virginia to New Jersey. This geographic restriction of serious disease activities and

mortalities is unexplained. Temperatures in Delaware Bay and Chesapeake Bay range widely from 0 °C in winter to 30 °C in summer, but the disease agent survives low temperatures and kills oysters during warm seasons.

SEASONALITY OF INFECTION AND MORTALITY FROM *HAPLOSPORIDIUM NELSONI* (MSX)

The life cycle of *Haplosporidium nelsoni* is unknown. Farley (1967, 1975) described the multiplication stages in oysters which were recognized much earlier by Haskin et al. (1966). Spores were produced so rarely that not until a slight increase in occurrence of sporulation in 1965 and 1966 did Couch et al. (1966) link them to plasmodial stages. Artificial infections have not been induced and the source of infection is unknown. The period of infectivity extends from mid-May to 1 November (Andrews & Frierman, 1974). Oysters transplanted from disease-free areas after 1 November do not develop clinical infections until the following July. Oysters begin dying about 1 August each year (Figs 3, 4 and 5), and deaths may occur in as little as six weeks after first exposure. A few oysters with early-summer infections may die in late winter from failure to withstand 2 to 3 months of winter dormancy (Andrews, 1966, 1968).

Oysters exposed in enzootic areas after 1 August develop subclinical infections which may become patent either as early as December in Delaware Bay (Ford & Haskin, 1982) or as late as May of the following year in Virginia (Andrews & Frierman, 1974).

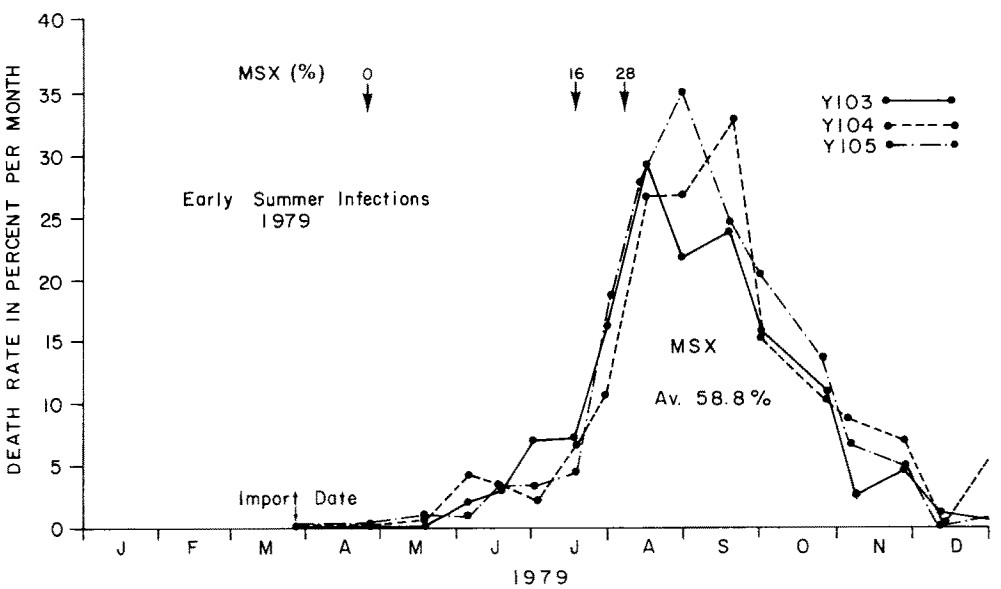

Fig. 3. Typical mortality pattern caused by *Haplosporidium nelsoni* in susceptible James River oysters during their first year of exposure. Data from three replicate groups (Y103, Y104 and Y105) of oysters in trays at Gloucester Point in the York River are shown for 1979. Prevalences of infections in percentages were obtained only for beginning of mortality period which occurs typically about 1 August each year

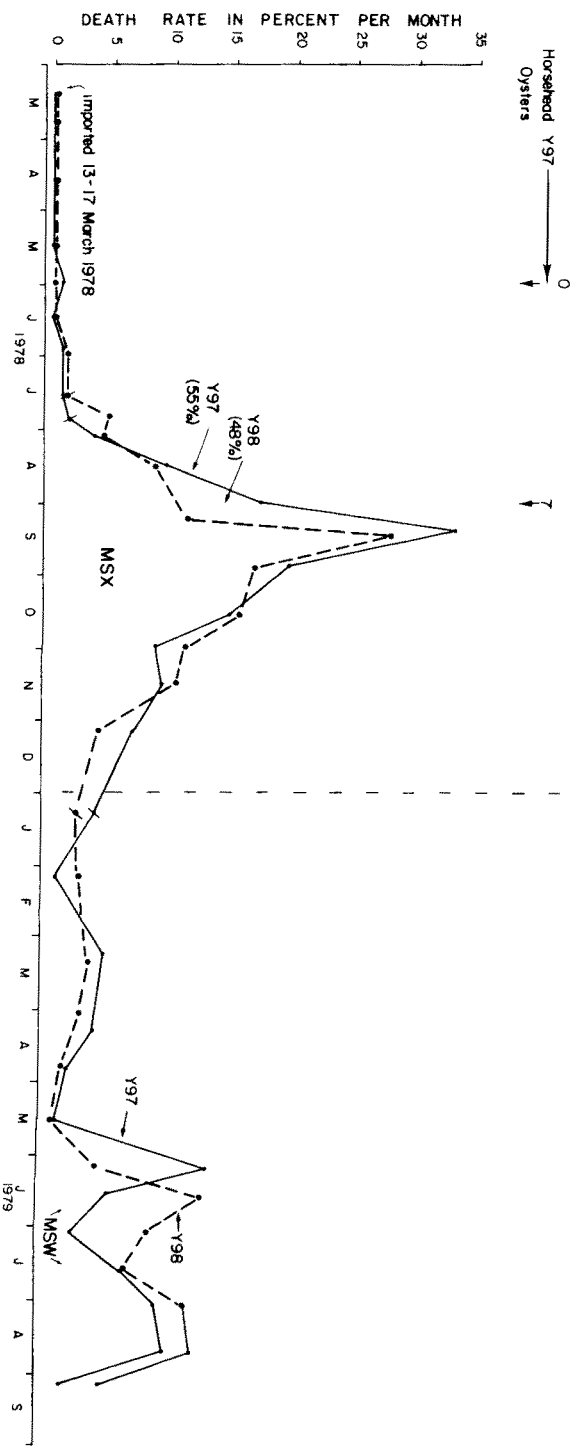

Fig. 4. Mortality in susceptible oysters (trays Y97 and Y98) caused by *Haplosporidium nelsoni* at Gloucester Point, 1978 and 1979. Late-summer infections almost failed in 1978 as shown by low mortality in June–July 1979. Survivors had 20 % infection of MSX on 20 November 1980. Prevalences shown as number of infections per 25 live oysters

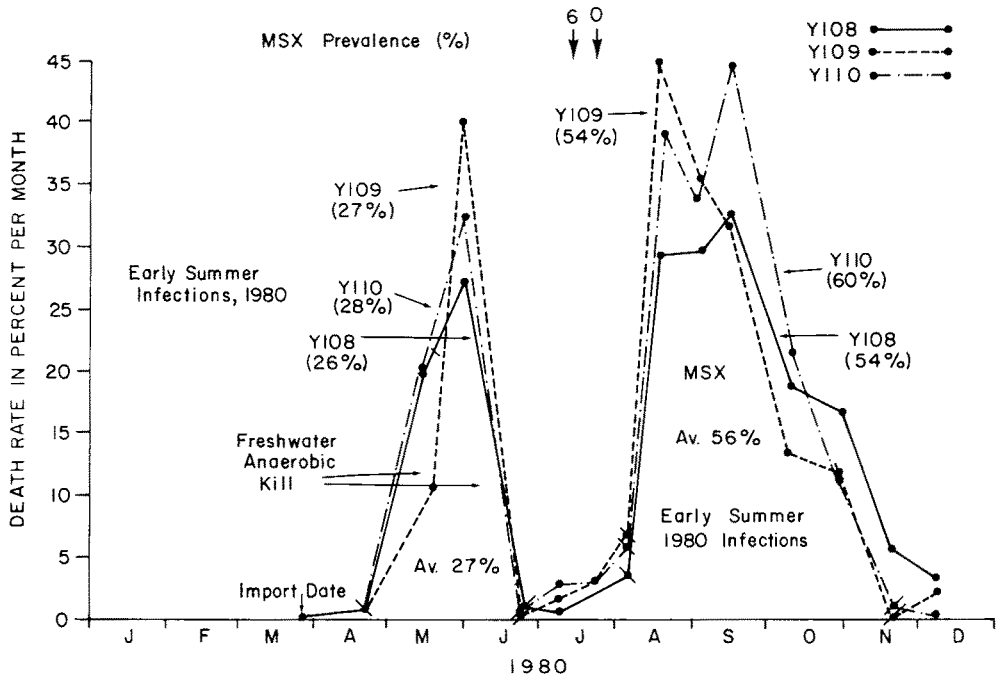

Fig. 5. Pattern of MSX mortality in susceptible oysters in 1980 (trays Y108, Y109 and Y110). The May mortality was caused by heavy fall rains and low salinities in winter and spring of 1979–1980 during a dormant period. Oysters died anaerobically with closed shells but dead oysters (gapers) did not become evident until May when water temperatures rose. Dead oysters provided evidence that cause of late-summer kill was MSX

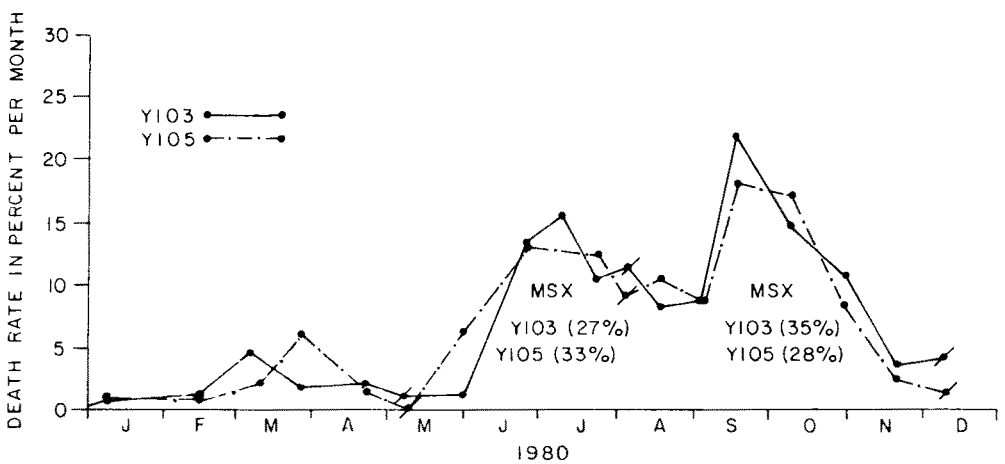

Fig. 6. Pattern of MSX mortality during second year of exposure of oysters in trays Y103 and Y105. June–July deaths resulted from infections in late-summer of 1979, and August to November deaths were caused by infections in early-summer of 1980. These deaths occurred after 59 % of oysters were killed by MSX in 1979 (see Fig. 3)

Deaths from late-summer infections are delayed until June and July of the following year (Fig. 6) when summer temperatures prevail again (Andrews, 1982).

Only plasmodial stages are commonly seen in live oysters and gapers (dead oysters). Initial site of infection is usually in the gills where large nucleated plasmodia are often localized in epithelial tissues. Infections become systemic through distribution within blood sinuses. Oysters that die early often exhibit light infections (< 1 plasmodium/430× field). Sporulation is rare in oysters most years (< 1 case/2000 infections per year), and oysters do not die promptly from it. Before sporulation occurs, plasmodia must migrate to epithelia of digestive tubules. During sporulation, multiplication of nuclei and enlargement of sporocysts to 30 to 50 μm occurs in tubule epithelia causing their distension into the lumen. In *Crassostrea virginica*, spores are too rare to be a probable source of infection.

DISEASE CONTROL WITH RESISTANT OYSTERS

Absence of resistant broodstocks in Chesapeake Bay forced oystermen to discontinue planting oysters in high-salinity areas. They now use only low-salinity areas (< 20 ‰) where susceptible oysters can be cultured successfully. Absence of planted oyster beds in the area enzootic for *Haplosporidium nelsoni* forced the use of tray oysters to monitor the disease in Virginia (Andrews, 1979). Biologists realized early that the only feasible method for control of the disease was to select and breed resistant oysters. This was achieved naturally in Delaware Bay because all broodstocks were exposed to the disease (Haskin & Ford, 1979), but it required laboratory breeding in Chesapeake Bay

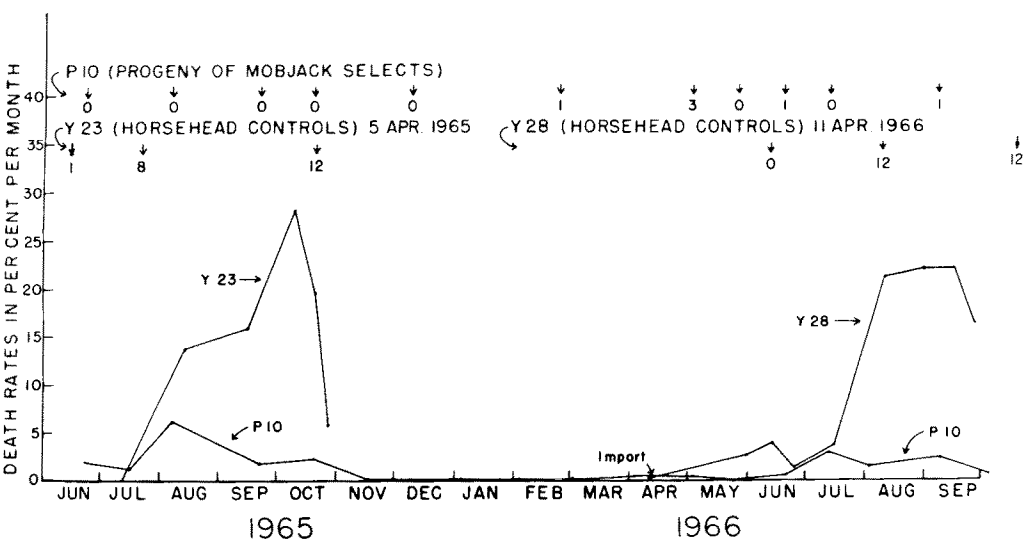

Fig. 7. Monthly mortality rates and prevalence of *Haplosporidium nelsoni* in resistant oysters (P10) are compared to those for susceptible James River seed oysters (trays Y23 and Y28). All lots were exposed to MSX disease in trays in the York River at Gloucester Point, Va., an enzootic area. Prevalences are shown as number of infections in samples of 25 oysters. April dates of importation of susceptible, disease-free oysters are given on graph

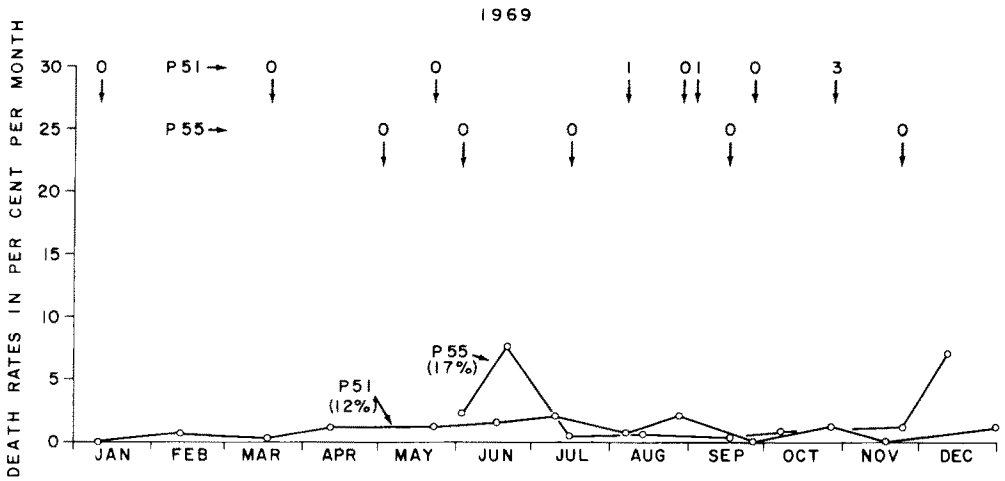

Fig. 8. Mortality and prevalence of *Haplosporidium nelsoni* in two lots of resistant progeny bred from P10 oysters after 3 and 4 years of exposure to the disease. Trays P51 and P55 contained second generation progeny of James River oysters which were exposed to the disease on beds in Mobjack Bay from 1959 to 1964 and suffered 95 % mortality. Prevalences are number of infections in 25 live oysters

where large stocks of susceptible brood oysters survived in low-salinity waters (Andrews, 1968).

In 1964, oysters surviving from 1959 and 1960 private plantings of James River seed in Mobjack Bay and other beds in high-salinity waters where the disease was endemic were dredged for broodstocks. Reduction of oyster populations on these beds from deaths caused by *H. nelsoni* exceeded 95 % during 4 or 5 years of exposure to the disease. Progeny of survivors bred in the laboratory exhibited strong resistance to MSX when exposed in trays within the enzootic area (Figs 7 and 8). Susceptible oysters were exposed to MSX as controls to insure that high levels of infection and deaths occurred each year (Andrews & Frierman, 1974). After 15 years of selection and breeding, brood oysters with strong resistance exhibited few *H. nelsoni* infections and mortalities of < 10 % per year in trays. Over 100 separate lots from various parents were field tested for 2 or 3 years. Unfortunately, it was not economically feasible in hatcheries to produce resistant seed oysters in commercial quantities. Because planted acreage decreased to about one-half of the 1950's level, natural seed oysters were adequate in quantity to meet market demand and continued to sell for the low price of $ 2 per bushel. A bushel contains about 1000 2- and 3-year-old seed oysters.

OCCURRENCE OF *HAPLOSPORIDIUM COSTALE* (SSO) CAUSING SEASIDE DISEASE

A new disease of oysters was discovered by Wood & Andrews (1962) in coastal bays of Virginia; it is enzootic in high-salinity waters along the ocean coast from Chesapeake Bay to Maine. Sharp increases in mortality from mid-May to mid-June (Fig. 9), regular sporulation throughout connective tissues, and all plasmodia developing into sporonts

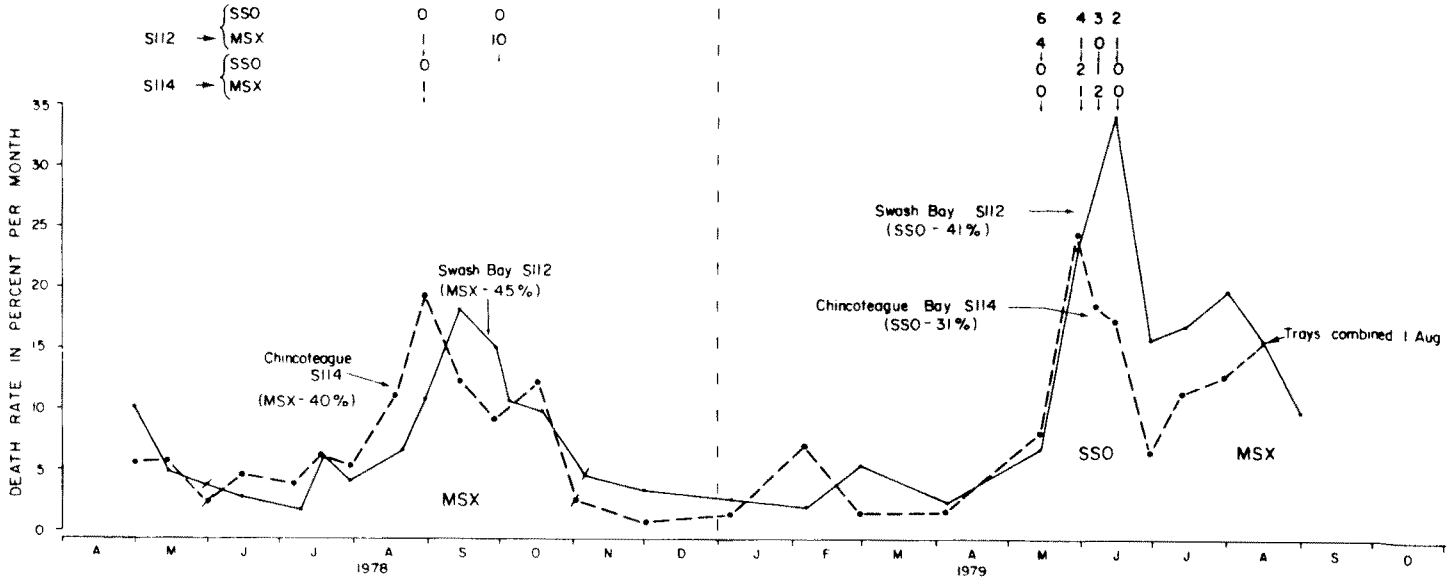

Fig. 9. Susceptible James River oysters were transplanted to Seaside of Eastern Shore (trays S112 and S114) where they acquired MSX infections in early summer and many died in late summer of 1978. Survivors showed a mixture of *Haplosporidium nelsoni* and *H. costale* infections in May and June 1979. Despite some overlapping of periods of mortality, a typical *H. costale* peak can be seen in May–June

were characteristics distinctive from those observed for *H. nelsoni* (Andrews et al., 1962). Subclinical infections were initiated in May and June, but they did not become patent until the following March. By rapid multiplication in spring, the agent produced abundant plasmodia, all of which developed into small sporocysts (10–20 μm). Oysters died promptly after sporulation although many spores did not reach maturity before gaper meats were scavenged by crabs and small fishes (Andrews & Castagna, 1978). All oysters in which sporulation occurred died because infections were systemic, but some oysters with light plasmodial infections recovered. No clinical evidence of infections was found during 8 to 10 months of incubation before tiny plasmodia appeared beneath intestinal epithelia in spring. The site of infection appeared to be the epithelia of the digestive tract.

This pathogen occurred only in high-salinity waters (> 25 ‰) from Virginia to Maine, but it caused mortalities mostly between Chesapeake and Delaware Bays on the Middle-Atlantic Coast. It was presumed to have been enzootic for many years. *H. costale* appeared to have a well-defined annual life cycle with close regularity in timing of maturing stages which is probably normal for a haplosporidan. New infections occurred during the period of mortality in May and June as shown by a series of trial importations of disease-free oysters. Artificial infections could not be induced by injecting or feeding spores.

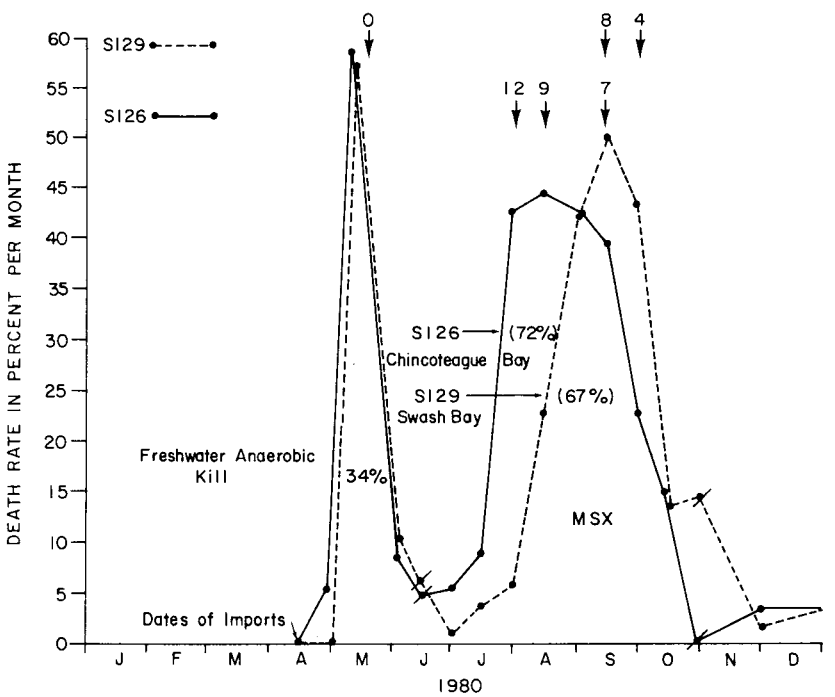

Fig. 10. Mortality in two seaside bays caused by *Haplosporidium nelsoni* in susceptible James River oysters on Eastern Shore of Virginia (S126 and S129). After 15 years of low MSX mortalities on Seaside, death rates increased to very high levels after 1975. The May mortality was caused by excessive rainfall over the James River drainage area in the fall and winter of 1979 and 1980. Oysters exposed to freshwater for several months were dead or dying in April when transplanted to Seaside

Haplosporidium costale disease was depressed after 1976 by a sharp increase in *H. nelsoni* infections and mortalities in seaside bays (Fig. 10). The latter pathogen killed oysters much more quickly than *H. costale* thereby removing oysters with early infections of *H. costale.* Experiments to test the response of oysters resistant to *H. nelsoni* to Seaside Disease (SSO) were inconclusive because of interference by the pathogen of Delaware Bay disease (MSX). It seems to have developed a more virulent strain in Seaside bays in recent years. Continuous exposure to and selection by haplosporidan parasites is probably necessary to sustain innate resistance, particularly if susceptible broodstocks in low-salinity sanctuaries continue to contribute larvae that alter oyster genomes.

LIFE CYCLES OF HAPLOSPORIDANS

The list of haplosporidans parasitic for invertebrates is growing rapidly. Sprague (1967) listed 45 species of haplosporidans in this group including those with insects as hosts. Some of these were microsporidans for which polar filaments had not been demonstrated. Caullery (1953) removed others considered to be fungi earlier. Haplosporida may still be a "wastebasket" for incorrectly identified parasites. There remains some question about the haplosporidan classification of two new oyster parasites of the genus *Marteilia* in *Ostrea edulis* in Europe (Grizel et al., 1974) and in *Crassostrea commercialis* in Australia (Wolf, 1972) which Perkins (1979) considers to be haplosporidans on the basis of EM structures called haplosporosomes.

It is significant that *Crassostrea gigas* was introduced into western North America, Australasia and Western Europe from Japan, yet different new diseases appeared on each continent. It is difficult to establish the origin of new disease agents except by circumstantial evidence such as timing and location of importations. *C. gigas* appears to be little affected by any of these new diseases, but native oysters are quite susceptible. All growing areas in temperate zones of the northern hemisphere, except eastern North America, are now dependent upon this vigorous species, but until artificial infections can be induced, and life cycles of haplosporidans documented, it is difficult to assess the risk of additional importations in spreading diseases and parasites (Andrews, 1980). Several diseases with unnamed agents were reported along the western Atlantic Coast (Sprague, 1971).

There is disagreement over characters to be used in separating the two common genera of haplosporidans in oysters. Sprague (1970, 1978) believes that tails on spores are a suitable generic character for separating the genera *Minchinia* and *Haplosporidium.* Perkins & van Banning (1981) found surface ultrastructure of spores to be useful in separating species in three genera of Balanosporida. Andrews (1983) and A. Farley (Oxford, MD; pers. comm.) contended that site of sporulation in the epithelia of digestive tubules *(M. nelsoni)* versus sporulation throughout all connective tissues in *Minchinia costale* and *M. armoricana* are more important generic traits than tails or surface ornamentation on spores. The latter species has tails associated with spore wrappings whereas *M. costale* does not. Therefore, *M. armoricana* would be placed in a separate genera by Sprague's criteria despite many similarities to *M. costale* in physiology and morphology. Lauckner (1983) reports the genus *Minchinia* to be occupied, and places its species back in genus *Haplosporidium.* This solves the problem of classification for the present.

The variety of invertebrates parasitized by haplosporidans is extensive even by phyla, although as a group haplosporidans are less economically important than microsporidans (Sprague, 1979). Molluscs, annelids, crustaceans and trematodes are common hosts for these pathogens. Often haplosporidans are parasites of the digestive tract which they tend to discolor when sporulation occurs (van Banning, 1977). Those species that produce systemic infections, such as are described herein for oysters, cause serious mortalities. In contrast, the hyperparasites of the genus *Urosporidium* in parasitic trematodes are apparently insignificant factors in the health of crabs and bivalves. The trematode *Bucephalus cuculis* is widespread geographically in oysters in Chesapeake Bay, but it is only rarely parasitized by an unnamed species of haplosporidan (Andrews, unpublished data). Cercaria of a trematode in blue crabs are frequently infected with *Urosporidium crescens* (DeTurk, 1940). A xanthid crab *Panopeus herbstii* is commonly parasitized in Virginia probably by the species *Minchinia louisiana* (Sprague, 1963; Perkins, 1975). Another xanthid crab *Rhithropanopeus harrisi,* common in Chesapeake Bay, was recently reported by Marchand (1974) to host a *Haplosporidan* species in France. It is not known whether the parasite was introduced with crabs, or if it is a new host for a parasite of a European crab species. The host specificity of haplosporidans is not well known. The severe disease epizootics in the oysters *O. edulis* and *C. angulata* in France (Marteil, 1969) in the late 1960's and 1970's were probably examples of native hosts being exposed to introduced pathogens.

A new haplosporidan (*Haplosporidium* sp.) parasitic in several species of shipworms *(Teredo)* in New Jersey may be linked to introductions of subtropical species of shipworms that depend on warm power plant effluents for winter survival (Hillman et al., 1982). Prevalences of the parasite are temporally variable suggesting that complex interactions of hosts and pathogen occur. This *Haplosporidium* parasite of *Teredo* has spores about the same size as those of *H. nelsoni,* but it has not been found in *Bankia* which is the most common shipworm genus in Chesapeake Bay and New Jersey waters. Because of its limited distribution, the shipworm pathogen seems to be an unlikely alternate or reservoir host for *H. nelsoni* which causes the severe disease in oysters. *Haplosporidium* sp. in *Teredo* may be an introduced pathogen of shipworms.

After 25 years of studies of the two *Haplosporidium* diseases of oysters along the Atlantic Coast, some clues as to their life cycles should become evident. Life cycles have not been demonstrated for any haplosporidans. Artificial infections have not been induced in the few species studied intensively (*H. nelsoni, H. costale, H. chitonis,* and *Marteilia refringens*). Spores for *H. nelsoni* are rarely found in oysters despite 25 years of intensive study, and it is unlikely that they are the infective source for the disease. Most investigators believe, therefore, that MSX must have an alternate or reservoir host. Andrews (1983, in press) listed epizootiological evidence for and against existence of other hosts.

Evidence for another host includes: (1) Proximity of infected oysters to disease-free groups does not alter timing or level of infections and mortalities. The disease appears not to be directly transmissable. (2) Scarcity of oysters within enzootic areas does not affect disease level. Oysters are no longer planted in the enzootic areas of Chesapeake Bay. (3) Failure to sporulate and prolonged periods of infectivity, mortality, and erratic occurrence of sporulation indicate that there is poor adaptation of parasite and host to each other. This suggests that *H. nelsoni* is a pathogen introduced with exotic oysters or

other invertebrates. (4) The wide variety of invertebrates parasitized by haplosporidans may provide an alternate host. (5) Uniformity and wide distribution of infections may require a mobile carrier or ubiquitous host to distribute infective particles throughout the area enzootic for the disease. (6) Failure of MSX to cause morbidity and mortality south of Chesapeake Bay and north of New Jersey may be due to absence or scarcity of a normal host producing infective spores. The pathogen occurs in these waters with low prevalence of disease.

Evidence against other hosts includes: (1) Infection periods match mortality periods in *H. nelsoni* and *H. costale*. Periods are long (5 months) for MSX and short (2 months) for SSO. (2) After an initial year of localized epizootics, dispersal of MSX occurred rapidly to full geographic distribution throughout subsequent enzootic areas both in Chesapeake and Delaware Bays. An alternate host must be highly mobile or planktonic to explain wide geographic fluctuations in range during dry periods. (3) If MSX were introduced, a new alternate host among native species seems unlikely to exist and to have spread the disease so rapidly. Introduced marine species are rare along the Atlantic Coast of North America where the climate is a rigorous continental-type. (4) Initial infections are acquired on food-collecting organs, particularly the gills, apparently from low dosage of infective particles that are waterborne for long distances. A reservoir host would not fulfill an ecological role under these conditions. (5) Infection and mortality levels are too consistent in intensity by stations and years to be induced by intermittent visits of reservoir host organisms. Only small planktonic organisms could be cosmopolitan so long and be so widely distributed each year. (6) Haplosporidans that are hyperparasites of trematodes and nematodes have complex life cycles in terms of invasion and infection of their parasitic hosts without the complications of additional alternate hosts. (7) Many haplosporidans sporulate in molluscan hosts regularly; therefore, oysters appear to be suitable hosts that have all the necessary stages for successful life cycles of these parasites. (8) Barrow (1965) claimed to have transmitted *H. pickfordae,* in freshwater snails from Lake Erie in the laboratory. His experimental specimens may have been infected in natural waters earlier. (9) Occurrence in *C. gigas* from Asia of haplosporidans with spores similar in shape and size to those of *H. nelsoni* suggests that this pathogen was introduced in oysters, probably without an alternate host. The same argument applies to *Marteilia* pathogens found in *C. gigas* introduced into France and Australia from Japan.

MINOR PARASITES OF MOLLUSCS AND DECAPODS IN CHESAPEAKE BAY

Cephaline gregarines (Porosporidae) of the genus *Nematopsis* are common in molluscs and decapod crustaceans in Chesapeake Bay. Except for the species found in mud crabs which alternate with oysters as hosts for *N. ostrearum,* the crustacean hosts are unknown. South of Virginia, the species *N. prytherchi* is found in gills of oysters with *Menippe mercenaria* as the decapod host (Sprague & Orr, 1955). Gregarines are common in decapods, however. Encysted spores are found in most species of bivalve molluscs including *Brachidontes recurvus* (hooked mussels), and *Anomia simplex* (jingles) which are often attached to oysters. Gymnospores from crustaceans, collected by molluscs while feeding, become encysted as thick-walled spores without multiplication; there-

fore, the pathologic effect on molluscan hosts is minimal. Nearly all oysters in Chesapeake Bay are infected with porosporid spores except in very low salinity waters where crabs are absent.

The larval trematode *Bucephalus cuculus* occurs regularly in < 5 % of oysters *(Crassostrea virginica)* throughout Chesapeake Bay. Little or no hemocytic response to the parasite occurs. Oysters are castrated and slowly starved to death as host tissues are replaced by multiplying sporocysts. The alternate hosts of *B. cuculus,* as described by Tennent (1909), have not been confirmed by careful infection experiments (Hopkins, 1957). How embryos discharged in faeces of freshwater gars, *Lepisosteus osseus,* can infect oysters in saline waters is not clear. The other fish host, presumed to be *Menidia* in which encysted immature stages are found, is also unconfirmed. Probably these are stages of several bucephalid species.

A haplosporidan hyperparasite of *Bucephalus cuculus* was reported by Mackin & Loesch (1955), but only plasmodial infections have been found in Maryland (Sprague, 1970). Over 300 infections of *B. cuculus* were found in 167 000 stained oyster sections in Virginia. These yielded only 4 cases of plasmodial infections by the hyperparasite. Thousands of bushels of Gulf of Mexico oysters have been shucked at waterside packing houses around Chesapeake Bay in recent years. Perhaps the hyperparasite is being casually introduced, for it is challenging to contemplate the biology of the host-parasite relationship and the tenacity of such a rare pathogen.

A sacculinid parasite of mud crabs was introduced into Chesapeake Bay from the Gulf of Mexico in the early 1960's. Live xanthid crabs were found in trucks hauling oysters to Virginia. The two most abundant species of xanthid crabs in Chesapeake Bay (Ryan, 1956) were infected by *Loxothylacus panopaei,* the sacculinid (Van Engel et al., 1966). Susceptible xanthid crabs of the species *Eurypanopeus depressus* and *Rhithropanopeus harrisi* became scarce; a relatively rare crab *Neopanope texana sayi* became the most abundant crab in Virginia waters (Andrews, unpublished data). Sacculinids are easily diagnosed by reproductive sacs or externae on aprons of crabs from which planktonic larvae are released.

Haplosporidans of the genus *Urosporidium* are found as hyperparasites in two commercial shellfish species, *Spisula solidissima* the surf clam, and *Callinectes sapidus* the blue crab. *U. spisuli* (Perkins, 1971) parasitizes a nematode in the surf clam; *U. crescens* (DeTurk, 1940) is a pathogen of metacercariae of a trematode and it causes "pepper" crabs. Sporulation of haplosporidans often discolors tissues of primary hosts which makes meats of parasitized seafood species unacceptable.

Microsporidan parasites of the genus *Chytridiopsis,* which occur in eggs of oysters, are not important pathogens along the Atlantic Coast of North America (Sprague, 1965). No infections were found in *C. virginica.* This egg parasite occurs in *Mytilus edulis* on the east coast. Becker & Pauley (1968) reported an unnamed ovarian parasite in *Crassostrea gigas* on the west coast of North America which is probably a *Chytridiopsis* sp. A rare microsporidan occurs in *Bucephalus cuculus* (Sprague, 1964). Microsporidans are not important as disease agents in oysters, but some agents are pathogenic in decapods.

The European shell disease of *Ostrea edulis* (Alderman & Jones, 1971) was found in flat oysters on Prince Edward Island, Canada (R. E. Drinnan, Halifax; pers. comm.). This disease was reported not to attack oysters of the genus *Crassostrea,* but it could become a

serious problem in the warm waters of eastern North America if it should become established in oysters or other shellfish. *Ostrea edulis* does not withstand warm seasons of the Atlantic Coast of North America, and it will not be grown commercially south of New England.

The Apicomplexan *Perkinsus marinus* cannot be called a minor disease agent along the southeastern coast of North America (Ray, 1954; Mackin, 1962; Andrews & Hewatt, 1957). It requires temperatures > 20 °C for several months each year to cause enzootic mortalities in oysters. If introduced into Western Europe, this species is unlikely to persist. It requires dying oysters in close proximity to other oysters and large dosages to transmit infections. *P. marinus* requires an abundance of oysters and warm saline waters to proliferate and spread. It thrives best at temperatures > 25 °C and salinities > 15 ‰ in Virginia. Regular harvesting of crops, fallowing of beds, and avoidance of infected seed oysters provide management controls for the disease; however, it persists on pilings along shores wherever oysters are recruited regularly. It occurs primarily from Chesapeake Bay south to South America.

LITERATURE CITED

Alderman, D. J. & Jones, E. B. G., 1971. Shell disease of oysters. – Fishery Invest., Lond. (Ser. 2) *26,* 1–19.

Andrews, J. D., 1966. Oyster mortality studies in Virginia. V. Epizootiology of MSX, a protistan pathogen of oysters. – Ecology *47,* 19–31.

Andrews, J. D., 1968. Oyster mortality studies in Virginia. VII. Review of epizootiology and origin of *Minchinia nelsoni.* – Proc. natn. Shellfish Ass. *58,* 23–36.

Andrews, J. D., 1979. Oyster diseases in Chesapeake Bay. – Mar. Fish. Rev. *41* (1–2), 45–53.

Andrews, J. D., 1980. A review of introductions of exotic oysters and biological planning for new importations. – Mar. Fish. Rev. *42* (12), 1–11.

Andrews, J. D., 1982. Epizootiology of late summer and fall infections of oysters by *Haplosporidium nelsoni,* and comparison to annual life cycle of *Haplosporidium costalis,* a typical haplosporidan. – J. Shellfish Res. *2,* 15–23.

Andrews, J. D., 1983. *Minchinia nelsoni* (MSX) infections in the James River seed-oyster area and their expulsion in spring. – Estuar. coast. Shelf Sci. *16,* 255–269.

Andrews, J. D. Epizootiology of haplosporidan diseases of oysters. – J. Invertebr. Pathol. (In press.)

Andrews, J. D. & Castagna, M., 1978. Epizootiology of *Minchinia costalis* in susceptible oysters in Seaside bays of Virginia Eastern Shore, 1959–1976. – J. Invertebr. Pathol. *32,* 124–138.

Andrews, J. D. & Frierman, M., 1974. Epizootiology of *Minchinia costalis* in susceptible wild oysters in Virginia, 1959–1971. – J. Invertebr. Pathol. *24,* 127–140.

Andrews, J. D. & Hewatt, W. G., 1957. Oyster mortality studies in Virginia. II. The fungus disease caused by *Dermocystidium marinum* in oysters of Chesapeake Bay. – Ecol. Monogr. *27,* 1–25.

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

Andrews, J. D., Wood, J. L. & Hoese, H. D., 1962. Oyster mortality studies in Virginia. III. Epizootiology of a disease caused by *Haplosporidium costale* Wood & Andrews. – J. Insect. Pathol. *4,* 327–343.

Banning, P. van, 1977. *Minchinia armoricana* sp. nov. (Haplosporida), a parasite of the European flat oyster, *Ostrea edulis.* – J. Invertebr. Pathol. *30,* 199–206.

Barrow, J. H., Jr., 1965. Observations on *Minchinia pickfordae* (Barrow, 1961) found in snails of the Great Lakes region. – Trans. Am. microsc. Soc. *84,* 587–593.

Becker, C. D. & Pauley, G. B., 1968. An ovarian parasite *(Protista incertae sedis)* from the Pacific oyster, *Crassostrea gigas.* – J. Invertebr. Pathol. *12,* 425–437.

Caullery, M., 1953. Appendice aux sporozoaires. Classe des haplosporidies (Haplosporidia Caullery et Mesnil, 1899). In: Traité de zoologie. Ed. by P. P. Grassé. Masson, Paris *1* (2), 922–934.

Couch, J. A., Farley, C. A. & Rosenfield, A., 1966. Sporulation of *Minchinia nelsoni* (Haplosporida, Haplosporididae) in the American oyster *Crassostrea virginica*. – Science, N.Y. *153*, 1529–1531.

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

Engel, W. A. van, Dillon, W. A., Zwerner, D. & Eldridge, D., 1966. *Loxothylacus panopaei* (Cirripedia, Sacculinidae) an introduced parasite on a xanthid crab in Chesapeake Bay. – Crustaceana *10*, 110–112.

Farley, C. A., 1967. A proposed life cycle of *Minchinia nelsoni* (Haplosporida, Haplosporididae) in the American oyster *Crassostrea virginica*. – J. Protozool. *14*, 616–625.

Farley, C. A., 1975. Epizootic and enzootic aspects of *Minchinia nelsoni* (Haplosporida) disease in Maryland oysters. – J. Protozool. *22*, 418–427.

Ford, S. E. & Haskin, H. H., 1982. History and epizootiology of *Haplosporidium nelsoni* (MSX), an oyster pathogen in Delaware Bay, 1957–1980. – J. Invertebr. Pathol. *40*, 118–141.

Grizel, H., Comps, M., Cousserans, F., Bonami, J. R. & Vago, C., 1974. Etude d'un parasite de la glande digestive observé au cours de l'épizootie actuelle de l'huître plate. – C. r. hebd. Séanc. Acad. Sci., Paris *279*, 783–84.

Haskin, H. H. & Ford, S. E., 1979. Development of resistance to *Minchinia nelsoni* (MSX) mortality in laboratory-reared and native oyster stocks in Delaware. Bay. – Mar. Fish. Rev. *41* (1–2), 54–63.

Haskin, H. H. & Ford, S. E., 1982. *Haplosporidium nelsoni* on Delaware Bay seed oyster beds: a host-parasite relationship along a salinity gradient. – J. Invertebr. Pathol. *40*, 388–405.

Haskin, H. H., Stauber, L. A. & Mackin, J. A., 1966. *Minchinia nelsoni* n. sp. (Haplosporida, Haplosporidiidae): causative agent of the Delaware Bay oyster spizootic. – Science, N.Y. *153*, 1414–1416.

Hillman, R. E., Maciolek, N. J., Lahey, J. I. & Belmore, C. I., 1982. Effects of a haplosporidian parasite, *Haplosporidium* sp., on species of the molluscan woodborer Teredo in Barnegat Bay, New Jersey. – J. Invertebr. Pathol. *40*, 307–319.

Hopkins, S. H., 1957. Our present knowledge of the oyster parasite *Bucephalus*. – Proc. natn. Shellfish Ass. *47*, 58–61.

Lauckner, G., 1983. Diseases of Mollusca: Bivalvia. In: Diseases of marine animals. Ed. by O. Kinne. Biologische Anstalt Helgoland, Hamburg, *2*, 467–1038.

Mackin, J. G., 1962. Oyster disease caused by *Dermocystidium marinum* and other microorganisms in Louisiana. – Publs Inst. mar. Sci. *7*, 132–229.

Mackin, J. G. & Loesch, H., 1955. A haplosporidian hyperparasite of oysters. – Proc. natn. Shellfish Ass. *45*, 182–183.

Marchand, J., 1974. Présence de Minchinia sp. (Haplosporida, Haplosporidiidae) chez le xanthide *Rhithropanopeus harrisii* (Gould) tridentatus (Maitland) dans le canal de Caen à la mer. – Revue Trav. Inst. Pêch. marit. *38*, 209–213.

Marteil, L., 1969. Données générales sur la maladie des branchies. – Revue Trav. Inst. Pêch. marit. *33*, 145–150.

Perkins, F. O., 1971. Sporulation in the trematode hyperparasite, *Urosporidium crescens* deTurk, 1940 (Haplosporida, Haplosporidae) – an electron microscope study. – J. Parasit. *57*, 9–23.

Perkins, F. O., 1975. Fine structure of *Minchinia* sp. (Haplosporida) sporulation in the mud crab *Panopeus herbstii*. – Mar. Fish. Rev. *37* (5–6), 46–60.

Perkins, F. O., 1979. Cell structure of shellfish pathogens and hyperparasites in the genera Minchinia, Urosporidium, Haplosporidium, and Marteilia – taxonomic implications. – Mar. Fish. Rev. *41*, (1–2), 25–37.

Perkins, F. O. & Banning, P. van, 1981. Surface ultrastructure of spores in three genera of Balanosporida, particularly in *Minchinia armoricana* van Banning, 1977 – the taxonomic significance of spore wall ornamentation in the Balanosporida. – J. Parasit. *67*, 866–874.

Ray, S. M., 1954. Biological studies of *Dermocystidium marinum*, a fungus parasite of oysters. Rice Inst. Pam., 114 pp. (Monograph in Biology. Spec. Issue)

Ryan, E. P., 1956. Observations on the life histories and the distribution of the Xanthidae (mud crabs) of Chesapeake Bay. – Am. Midl. Nat. *56*, 138–162.

Sindermann, C. J., 1976. Oyster mortalities and their control. – FAO Tech. Conf. Aquaculture, Kyoto, Japan, 25 pp.

Sprague, V., 1963. *Minchinia louisiana* n. sp. (Haplosporida, Haplosporidiidae) a parasite of *Panopeus herbstii*. – J. Protozool. *10*, 267–274.

Sprague, V., 1964. *Nosema dollfusi* n. sp. (Microsporidia, Nosematidae) a hyperparasite of *Bucephalus cuculus* in *Crassostrea virginica*. – J. Protozool. *11*, 381–385.

Sprague, V., 1965. Observations on *Chytridiopsis mytilovum* (Field) formerly *Haplosporidium mytilovum* Field (Microsporida). – J. Protozool. *12*, 385–389.

Sprague, V., 1967. Identification and taxonomy of disease organisms. Natl. Resources Inst., Univ. Md., Chesapeake Biological Lab., Solomons, Md, Ref. 67–31 (Mimeogr.).

Sprague, V., 1970. Some protozoan parasites and hyperparasites in marine bivalve molluscs. – Spec. Publ. Am. Fish. Soc. *5*, 416–430.

Sprague, V., 1971. Diseases of oysters. – A. Rev. Microbiol. *25*, 211–230.

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

Sprague, V., 1979. Classification of the Haplosporidia. – Mar. Fish. Rev. *41* (1–2), 40–44.

Sprague, V. & Orr, P. E., 1955. *Nematopsis ostrearum* and *N. prytherchi* (Eugregarinia: Porosporidae) with special reference to the host-parasite relations. – J. Parasit. *41*, 89–104.

Tennent, D. H., 1909. An account of experiments for determining the complete life history of *Gasterostomum gracilescens*. – Science, N.Y. *29*, 432–433.

Wolf, P. H., 1972. Occurrence of a haplosporidan in Sidney rock oysters *(Crassostrea commercialis)* from Moreton Bay, Queensland, Aust. – J. Invertebr. Pathol. *19*, 416–417.

Wood, J. L. & Andrews, J. D., 1962. *Haplosporidium costale* (Sporozoa) associated with a disease of Virginia oysters. – Science, N.Y. *136*, 710–711.