

## HISTORY OF *PERKINSUS MARINUS*, A PATHOGEN OF OYSTERS IN CHESAPEAKE BAY 1950-1984



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**ABSTRACT** The pathogen *Perkinsus marinus* (Dermo) was discovered in Chesapeake Bay in 1950. It was already widely distributed in the Bay and caused annual mortality below the mouth of the Rappahannock River. Annual mortality in trayed oysters at the Virginia Institute of Marine Science (VIMS) varied annually from 24% to 57% at this most favorable site for the disease. Over 2 million bushels of seed oysters from the James River public beds were transplanted annually to private beds in 4 major growing areas. These were Hampton Roads, lower Bay proper, Mobjack Bay at mouth of York River, and the Rappahannock River. The introduction of *Haplosporidium nelsoni* (MSX) in 1959 resulted in killing most oysters throughout the Bay, and private planting was abandoned. Extreme dry weather during the decade of the 1980s allowed both diseases to spread widely throughout the Bay, and the oysters became scarce everywhere. MSX retreated to its endemic area below the mouth of the Rappahannock River when salinities returned to average levels. Dermo destroyed oysters in the seed area of the James River, and it has persisted there tenaciously with low mortality. Market-oyster production dropped from 2 to 3 million bushels annually during the 1950s to 6,000 in 1993. No seed oysters are available, and planting of private beds has ceased. Recovery is slow, and the oyster industry in Virginia was destroyed.

**KEY WORDS:** History, diseases, Chesapeake Bay, pathogen, mortality, distribution, oyster culture

### ORIGIN AND LIFE CYCLE OF *PERKINSUS MARINUS*

The origin of *Perkinsus marinus* is obscure. The pathogen is widely spread throughout SE Asia; possibly it was introduced by ship transport during World War II, but mortality of oysters was reported before 1940 in Virginia. Numerous small introductions of Pacific oysters (*Crassostrea gigas*) have been made along the east coast of North America from the west coast (Andrews 1979). The disease has not been a problem along the west coast of North America, or in Europe where oceanic climates and upwelling keep waters much cooler than on east coasts. The Pacific oyster was introduced along the west coast of North America before 1900; seed oysters in commercial quantities were imported regularly from Japan to Washington and California after World War II (Andrews 1980). Dermo has not occurred along the western shores of Europe despite many tons of introductions of Japanese oysters in late 1960s and early 1970s.

Dermo causes a warm-season disease of eastern oysters (*Crassostrea virginica*) in Chesapeake Bay (Andrews 1988). At tem-

peratures above 20°C, the pathogen multiplies and kills oysters about a month after infection. For rapid proliferation, the disease requires temperatures of 25°C which prevail for about 5 months in Chesapeake Bay waters (Andrews and Hewatt 1957). Mortality ceases by 1 November when water temperatures decline below 20°C; during the 1950s, oysters gradually expelled infections, and from February through April most samples showed no infections by Ray's FTM test (Ray 1952). However, oysters placed in 25°C water during late winter and spring revealed about 20% infection within a month (Andrews and Hewatt 1957). These hidden infections became patent in June when temperatures reached 25°C. These over-wintering infections caused deaths by 1 August, and two more generations of infections occurred before mid-October with prevalences of Dermo often at 90% to 100%.

A comparison of the life cycle of Dermo in the Gulf of Mexico and in Chesapeake Bay is revealing (Andrews and Ray 1988). Higher winter temperatures in the Gulf allow the pathogen to persist in oysters with patent infections throughout the winter, although intensity and prevalence decline. In Louisiana where most Gulf oysters are grown, salinities fluctuate widely depending upon Mississippi River flow, resulting in wide fluctuations of the disease by years and areas. Planters there must search for disease-

<sup>1</sup>VIMS Contribution No. 1884.

free oysters in low-salinity areas to transplant into high-salinity areas for growth, fattening, and early marketing.

Dermo had a wide distribution in Chesapeake Bay at the time of its discovery in 1950. It spread more widely into marginal salinity areas during the mid-1960s' invasion of upper Virginia and Maryland oyster beds. It spread into the James River only during the dry period of the 1980s. It persisted tenaciously at low levels of infection most winters. Only during dry summers did the pathogen kill oysters in areas with late-summer salinities <20 ppt. Scarcity of oysters in the lower bay limited the distribution of Dermo to manmade structures and creeks which had regular recruitment of new year-classes. Beds leased by private growers became barren because those bottoms are soft and oysters sink in time. This fact is important to any efforts to grow oysters in isolation once disease-free seed is available.

#### TRANSMISSION OF *P. MARINUS* DISEASE

Transmission of Dermo is direct from infected dying oysters to other hosts of the species (Mackin 1962). Proximity to gapers (dying oysters) is necessary because large dosage is required to achieve rapid infection ( $1 \times 10^5$ ) zoospores (Roberts, Virginia Institute of Marine Science [VIMS], pers. comm. 1984). All stages appear to be infective or become so when the host dies and prezoosporangia are released into marine waters. From 1,000 to 2,000 zoospores are estimated to be produced by one large sporangium (Perkins 1966). Zoospores are produced from prezoosporangia after culture in thioglycollate medium for 24 to 48 hours. Feeding or injecting small amounts of macerated gaper tissues produces infection in nearly all oysters. Infection occurs apparently through the digestive tract as indicated by the location of foci of infection in sectioned live oysters. The role of zoospores in open water infections is unknown. They must be an infective stage, but difficulty in production of this stage in the laboratory has prevented completion of the life cycle for the pathogen after 45 years of research. Distances for isolation of oysters from the disease are speculative, which hampers planning for repopulation of oysters in Chesapeake Bay.

A host of scavengers live on oyster beds to feed on oysters killed by predators and diseases (Andrews 1988). Blue crabs and mud crabs (Xanthids) kill small oysters whereas nereid worms, spider crabs, and several small fishes such as blennies, gobies, and clingfish are scavengers quick to snatch bits of loose flesh from gaping oysters (SCUBA observations).

#### OVER-WINTERING OF *P. MARINUS* IN CHESAPEAKE BAY

The level of over-wintering infection is critical to the infectivity and mortality caused by *P. marinus* disease the following summer. There is a 5-month period of temperatures above 20°C that favors multiplication by the pathogen. If there were no over-wintering infections, the disease would die out; no alternate host has been identified. During the early 1950s, Delaware Bay planters imported oysters from the eastern shore peninsula of Virginia and introduced the disease there (Ford 1992, 1996).

During the 1950s, when Dermo was monitored without interference by *Haplosporidium nelsoni* (MSX) disease, Ray's FTM tests showed low levels of over-wintering infection at VIMS pier in samples from February through April. Yet development of a few infections was found in June and July after temperatures reached 20°C to 25°C. Oysters placed in warm water for a month in April had about 20% infection. Disease-free oysters imported in April from the upper James River did not develop infections until August, and mortalities were far less than those of acclimated

oysters from a previous year's transplanting. This pattern of infection and mortality was derived from 30 years of FTM tests on Virginia oysters. It was apparent that hidden infections were over-wintering, but the stage and site in oysters were not known (Andrews 1988).

#### SPREAD OF *P. MARINUS* DURING DROUGHT OF 1980S

The droughty decade of the 1980s allowed Dermo to spread widely into Maryland and up the James River seed area which is vital to oyster culture in Virginia (Andrews 1988, Burreson and Andrews 1988) (Fig. 1). Record high salinities and well-populated contiguous oyster beds allowed the disease to spread rapidly and to kill most oysters in the James River except on a couple of upriver beds. Continued harvesting by oystermen helped to deplete the 15-mile-long area of seed oysters and broodstock. Yet the pathogen persisted by wintering in quite low-salinity waters with temperate winters. Over-wintering prevalences of 100% were found at Point of Shoals, a rather upriver site (Ragone Calvo and Burreson 1994). Probably planters, who transported infected oysters from lower river beds to their upper river private beds, helped spread the disease. For a low price, these planters bought market-size oysters in late spring and held them on upriver beds through the summer for high fall prices.

The spread of Dermo into the James River seed area during the 1980s, after 30 years of freedom from disease, was unprecedented (Andrews 1988). The prolonged dry weather during the decade, as well as the complete absence of hurricanes to depress salinities during summers, was critical. Each of three earlier decades had one or more hurricane flooding periods. Salinity regimes in Maryland were very high with a one-time record of 25 ppt at the Bay Bridge above the oyster-growing area. Dry summers increased salinity levels.

Many years of observation of coastal plains estuaries, such as the Great Wicomico and Piankatank Rivers, show that Dermo can persist indefinitely in rather low-salinity rivers once established; only when bay waters are salty from low runoff do the two pathogens, Dermo and MSX, cause appreciable mortality. These estuaries are dependent on the bay for their salinity regimes, and little freshwater runoff is available to reduce salinity. Typically, these estuaries get to 15 ppt only in late summer and fall when time for disease development is limited. Importantly, these estuaries are effective in producing seed oysters with quite regular spatfalls. The diseases have not affected setting rates in the coastal plain estuaries because low populations of broodstocks are adequate. The James River requires very high oyster populations for production of seed oysters.

#### SUSCEPTIBILITY OF OYSTERS TO *P. MARINUS* BY SIZE AND ORIGIN

Few seed oysters have been imported commercially from the Carolinas into Chesapeake Bay; however, in the early 1950s when New Jersey planters were transplanting oysters from eastern shore of Virginia, a scarcity for local planters induced a trial of South Carolina oysters. Seaside (Virginia) oysters were found to be highly susceptible to MSX, but Dermo was not found on these beds perhaps because fast growth allowed early harvesting. South Carolina oysters matured rapidly in Virginia waters and were somewhat resistant to Dermo. Native yearling oysters from James River, where no selection had occurred, showed strong resistance to Dermo infection in open waters. Heavy dosage in aquaria produced infections.

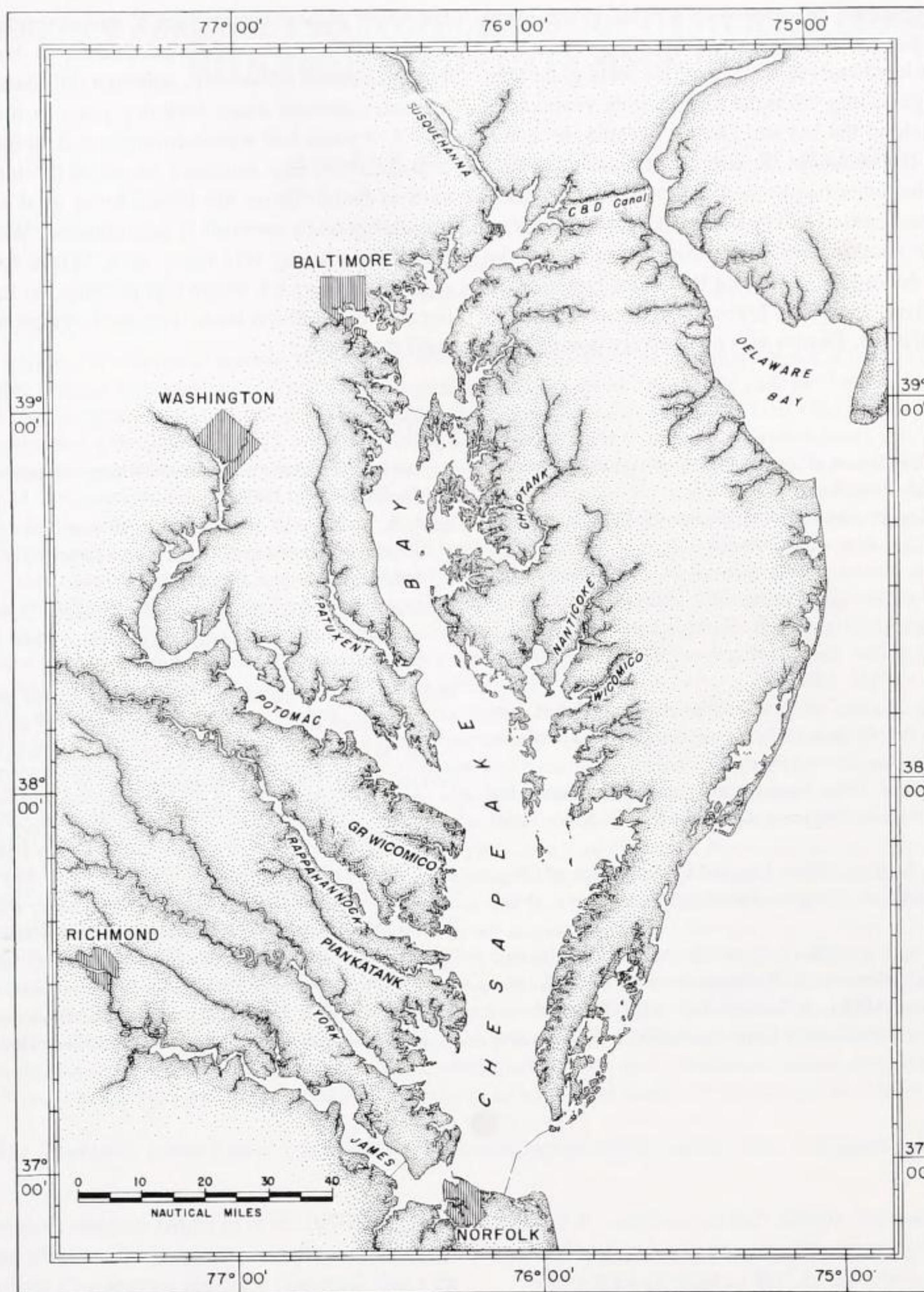


Figure 1. Map of Chesapeake Bay showing major rivers where oysters were grown as discussed in text.

This resistance of yearlings to Dermo may allow production of oysters on isolated beds if moved before their second summer of exposure. If a coastal plain estuary were declared strictly for production of young seed oysters, by prohibiting private plantings along the shores, a disease-free supply of oysters could be produced annually for transplanting to barren areas in higher salinity waters. Early harvesting would be necessary. This method should be tried because recovery of setting and decline of Dermo in the James River seed area are unpredictable. The price of a pint of west coast oysters in local stores is \$8 now, which deprives the author of a seafood that was cheap throughout most of his 40 years of study of oyster diseases. I miss them.

#### INTRODUCTION OF *H. NELSONI* DISEASE IN 1959

The introduction of *H. nelsoni* (MSX) (Ford and Haskin 1982) complicated disease problems in Chesapeake Bay. This disease

moves rapidly up and down the bay with changes in salinities. It requires salinities of 12 to 15 ppt to infect oysters, but it is easily discharged at 10 ppt. This disease invaded the Maryland part of the bay in the mid-1960s, causing heavy mortality (Andrews 1967), and again in the mid-1980s. The source of infection by MSX is unknown. Fresh-water flows from the large drainage areas of the James, Potomac, and Susquehanna Rivers reduce salinities in winter and spring, and that sets the patterns of disease for different areas. MSX invades the upper bay rapidly in one year and is usually discharged the following winter. Hurricanes play an important role in controlling MSX by lowering bay salinities during summers, and it is expelled in winters. There have been no significant hurricanes in the Chesapeake area since 1973.

*H. nelsoni* became the dominant pathogen during the 1960s and 1970s in lower Chesapeake Bay. It kills quicker than Dermo, and it has no need for proximity to infected oysters to produce infec-

tions. Dermo was suppressed by scarcity of oysters in the lower bay, but given 2 or 3 years of exposure, it eventually got into trays and oyster beds. Populated beds in the lower bay were gone after 1961 from MSX ravages. Only when the high-salinity years of the 1980s occurred throughout the bay did Dermo become the dominant disease in upper bay estuaries. Sparse populations of oysters were being killed in the lower bay by both diseases. Oyster planting in the lower bay had ceased in 1961 after MSX was imported in 1959. The endemic zone for MSX was from the mouth of the Rappahannock River down-bay, including the lower James River and all of the York River. In up-bay low-salinity areas some oysters were still being planted. Dermo was explosively dominant in

the upper James River where it had never appeared before. The quick invasion and strong persistence in James River have not been explained adequately, although the cause was definitely high salinities through many very dry years in the 1980s. During the 1990s, 5 years had winter-spring runoff in the bay less than half-average flow. Dry summers sustained the high salinities. Expulsion of Dermo from the James River seed area is slow and the possibility of its removal is questionable. Wet, cold winters may be helpful. It may take many years before the river develops adequate broodstock to begin repopulating the famous river that produced 2 to 3 million bushels of seed oysters annually without fail until the 1980s.

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