Lethal Parasites in Oysters from Coastal Georgia with Discussion of Disease and Management Implications

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Introduction

Oysters in many parts of the world have declined in abundance since the 19th century, probably in large part from indiscriminate harvesting and destruction of beds (Sindermann, 1970). Lyles (1969) published data showing a similar decline in the U.S. commercial harvest of oysters, *Crassostrea virginica*, in this century. During the last several decades, an increasing awareness of the role shellfish pathogens play in the population dynamics of oysters has been reflected in publications of Sindermann and Rosenfield (1967), Sindermann (1970, 1990), Kinne (1983), Farley et al. (1988), Fisher (1988), and Farley (1989, In press). Disease is clearly a major factor affecting the abundance of shellfish stocks. Malpeque Bay disease (Needler and Logie, 1947) and other distinct diseases caused by the parasites *Perkinsus marinus* (Mackin et al., 1950; Levine, 1978), *Haplosporidium nelsoni* (Haskin et al., 1966; Sprague, 1978), and *H. costale* (Wood and Andrews, 1962) have been reported to cause mortalities in the oyster, *C. virginica* (Sindermann, 1990; Farley, In press).

In 1966 and 1968, one of the authors (Rosenfield) made a histological study of oysters from coastal Georgia, as part of an Atlantic coastal survey to determine the status of oyster disease agents and parasites. At that time, *H. nelsoni* was causing mortalities of oysters from Delaware and Chesapeake Bays, and *P. marinus* was causing serious losses to oysters in the Gulf of Mexico region. Results of this study (unpubl.) document the occurrence of *P. marinus* in Georgia waters as early as 1966; however, *H. nelsoni* was not observed at that time.

In November 1985, the Georgia Department of Natural Resources (DNR) began receiving complaints from the shellfish industry of widespread mortalities of oysters, *C. virginica*, from commercial leases. Initial mortalities were attributed to natural causes, alarming neither industry nor the DNR. However, as additional shellfishermen began to report increased mortalities, the DNR initiated field surveillance at selected areas. Results of the survey showed that where mortalities occurred they ranged from 40 to 100 percent. Most locations exhibited mortalities of 60-70 percent.

To determine the cause of mortalities, NOAA's National Marine Fisheries Service (NMFS) and the Georgia DNR began a cooperative study in January of 1986. Data in this report suggest that epizootic levels of infection with *P. marinus* ("Dermo") are the most probable causes of the mortalities. The report also documents the occurrence of *H. nelsoni* ("MSX") in the coastal waters of Georgia for the first time.

Methodology

Samples of apparently healthy oysters were collected for histological examination from 17 sites throughout coastal Georgia during two studies (Fig. 1). The same protocols were followed for both studies, with the exception of the number of oysters examined, and quantifying the disease intensity of the earlier unpublished work by Rosenfield.

In January 1966, samples of 25 oysters each were collected from Wassaw Creek and the Duplin and Woodbine Rivers, and processed for histological examination. Subsequently, 15-20 animals were collected and processed for a follow-up evaluation in April of 1968 from each of the following four sites: Eagle Creek and the Darien, Wilmington, and Brickhill Rivers. Intensities of infection were not recorded.

Beginning in January of 1986, oysters were collected from Mud Creek

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ABSTRACT— Extensive mortalities of oysters, Crassostrea virginica, *occurred from 1985 through 1987 in coastal waters of Georgia. Fluid thioglycolate cultures of oysters collected from 16 of 17 locations revealed infections by the apicomplexan parasite* Perkinsus marinus. *An ascetosporan parasite,* Haplosporidium nelsoni, *was also observed in histopathological examination of oysters from 4 of the locations. While the range of* H. nelsoni *currently is recognized as the east coast of the United States from Maine to Florida, this is the first report of the parasite in Georgia waters. This paper documents the occurrence of these two lethal parasites in oysters from coastal waters of Georgia, along with potential disease and management implications. Results of an earlier independent and previously unpublished survey are also discussed which document the presence of* P. marinus *in Georgia as early as 1966.*

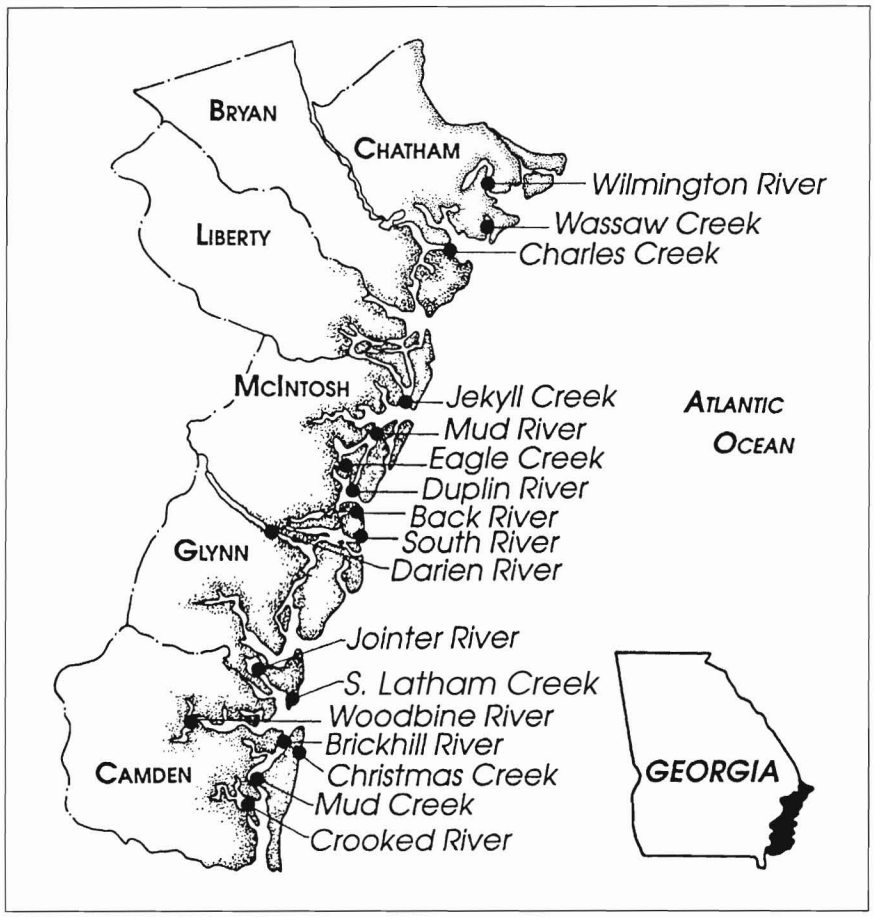

Figure 1.—Distribution of sample sites in coastal Georgia (1966-87).

and Crooked River. During November 1986, oysters were collected from Jekyll, South Latham, and Charles Creeks, and the Jointer River. In November 1987, oysters were gathered from Christmas Creek, as well as Back, Mud, and South Rivers. One hundred oysters were collected at each site, of which 50 were selected randomly and processed for histological examination.

In each study, collections were made by personnel of the Georgia DNR and sent to the NMFS Laboratory at Oxford, Md., for processing and detection of the epizootic agent(s). DNR personnel also provided salinity and temperature data for 1986-87.

Animals were macroscopically examined and processed for histology using standard Oxford Laboratory histological techniques (Howard and Smith, 1983). Each oyster was measured, examined visually for gross abnormalities, and assigned a condition based on a scale of 1 (very watery) to 9 (very fat), as explained in Table 1. Fluid thioglycolate cultures of oyster rectal tissue (Ray, 1966) were made to diagnose *P. marinus* infection.

Light microscopy was used to determine the presence and intensity of infections in individual animals. In the 1986-87 study, infections were assigned numerical values of intensity from 1 to 9, as indicated in Table 2.

Results of the thioglycolate cultures and histopathology were used to determine disease intensities of *P. marinus* and *H. nelsoni* in each of the sample populations. This value is calculated by the summation of disease intensity in all animals from a site, divided by the number of animals tested for that site (Ray, 1954).

Table 1.—Field survey and macroscopic oyster examination data, 1986-87 survey.

Sample area	Salinity ‰	Water temp. (°C)	Mean length (mm)	Condition range[1]	Mean condition[1]
Jan. 1986					
Mud Creek	28[2]	13.0	89.4	1-4	3.0
Crooked R.	28[2]	13.0	86.4	1-5	2.1
Mean	28	13.0	87.9	1-5	2.6
Nov. 1986					
Jointer R.	32	23.0	68.4	4-6	4.9
S. Latham Cr.	31	23.0	65.0	2-5	4.1
Jekyll Cr.	29	22.0	52.8	2-6	4.5
Charles Cr	32	22.0	53.1	2-6	3.5
Mean	31	22.5	59.8	2-6	4.3
Nov. 1987					
Christmas Cr.	30	16.0	78.4	2-6	4.6
Mud R.	30	16.5	92.4	1-5	2.5
Back R.	32[2]	18.8[2]	88.0	1-5	3.6
South R.	32[2]	18.8[2]	72.0	2-6	4.1
Mean	31	17.5	82.7	1-6	3.7

[1]Criteria for condition based on visual observations and rated on a scale of 1-9 as follows: 1 = watery(-), 2 = watery, 3 = watery(+), 4 = medium(-), 5 = medium, 6 = medium(+), 7 = fat(-), 8 = fat and 9 = fat(+).
[2]Estimated values based on information of the area. This portion of data for sample was destroyed in shipment.

Table 2.—Prevalence and intensity of oyster parasites *Perkinsus marinus* and *Haplosporidium nelsoni* in coastal waters of Georgia.

Sample area and survey date	*P. marinus*[1] *n*	Percent prevalence	Sample population intensity[3]	*H. nelsoni*[2] *n*	Percent prevalence	Sample population intensity[3]
Jan. 1966						
Woodbine R.	25	12	NA[4]	25	0	NA
Wassaw Cr.	25	12	NA	25	0	NA
Duplin R.	25	44	NA	25	0	NA
Mean	25	23		25	0	
April 1968						
Darien R.	10	10	NA	20	0	NA
Eagle Cr.	10	10	NA	15	0	NA
Wilmington R.	15	0	NA	15	0	NA
Brickhill R.	10	10	NA	20	0	NA
Mean	11	8		18	0	
Jan. 1986						
Mud Cr.	25	100	NA	50	6	0.4
Crooked R.	25	100	3.6	50	0	0.0
Mean	25	100	3.6	50	3	0.2
Nov. 1986						
Jointer R.	22	88	3.6	50	0	0.0
S. Latham Cr	25	100	3.9	50	6	0.3
Jekyll Cr.	25	96	3.4	50	0	0.0
Charles Cr.	25	100	3.8	50	0	0.0
Mean	24	96	3.7	50	2	0.1
Nov. 1987						
Christmas Cr	25	100	4.9	50	2	0.1
Mud R.	25	100	4.2	50	0	0.0
Back R.	25	100	3.8	50	0	0.0
South R.	25	100	4.1	50	2	0.2
Mean	25	100	4.3	50	1	0.03

[1]*P. marinus* results based on thioglycolate cultures of rectal tissue.
[2]*H. nelsoni* results based on histological observations.
[3]Criteria for intensity data based on a numerical scale from 1 to 9: 1 = very light, 3 = light, 5 = moderate, 7 = heavy, 9 = very heavy.
[4]NA = Data not available.

Results

During 1986-87, oysters were obtained from high salinity waters, between 28 and 32‰, with temperatures of 13°-23°C. Oysters ranged in length from 25 to 143 mm, and their mean visually assessed condition varied from watery(–), or very poor, to medium(+) (Table 1). No fat oysters were found in any of the samples. Similar data were not available from 1966 or 1968 samples.

Thioglycolate cultures and histopathological examination of oysters revealed the presence of *P. marinus* from 16 of the 17 sites sampled, and *H. nelsoni* in oysters from 4 sites (Table 2). Parasites were found in oysters throughout coastal Georgia, without indication of a regional distribution.

Oysters collected from Georgia in 1966 and 1968 revealed the presence of *P. marinus* in 0-44% of the oysters examined. *Perkinsus marinus* was observed in 12-44% of oysters sampled in January of 1966, but no mortalities were reported. Two years later, *P. marinus* was found in 0-10% of the oysters examined, again with no reports of associated mortalities. Examination of oysters in 1986-87 revealed *P. marinus* occurred at all sites sampled. Prevalence of the disease ranged from 88 to 100%, with intensities of infection ranging from very light (1) to very heavy (9). Infections showed an increase in sample population intensity from a mean of 3.6 in January 1986 to 4.3 in November 1987. Mean intensity of infection for the entire study was 3.9.

Haplosporidium nelsoni was not observed in either of the 1966 or 1968 samples; however, the parasite was diagnosed in a total of eight animals from four sites during 1986 and 1987. Prevalence of *H. nelsoni* from these sites ranged from 2 to 6%, with sample population intensities ranging from 0.1 to 0.4. Intensity of disease among individual infected animals varied from very light (1.0) to heavy (7.0).

Discussion

Since the turn of the century, annual U.S. oyster production has fallen from about 158 million pounds of meats (Lyles, 1969) to 29.9 million pounds of meats in 1989 (USDOC, 1990). While overharvesting, predation, and deteriorating water quality are believed to be responsible for much of the decline (Leonard et al., 1989), diseases have also been a significant factor.

Traditionally, Chesapeake Bay led the United States in oyster production prior to *H. nelsoni* and *P. marinus* becoming endemic to the area (Anderson and Power, 1957). Following several decades of severe losses due to oyster diseases, there has been a complete reversal in oyster production in the nation. Chesapeake Bay now contributes only 14 percent of the annual U.S. oyster harvest, compared to 50 percent produced in the Gulf of Mexico region, principally by Louisiana (USDOC, 1990). Commensurate with this change, oyster imports since 1986 have consistently exceeded the U.S. commercial landings (USDOC, 1990), to meet the American consumer demand.

Perkinsus marinus, first described in oysters from the Gulf of Mexico by Mackin et al. (1950), causes a chronic disease which can be fatal in *C. virginica* (Mackin, 1951). Some confusion apparently exists regarding the parasite's northern range and its presence in coastal bays of Maryland and Virginia. Quick (1977) reported its range from Massachusetts south into the Gulf of Mexico. Kern et al. (1973) also reported the parasite to occur in *C. virginica* growing in Hawaii. However, later publications (Andrews, 1988; Sindermann, 1990) cite Delaware Bay as the northern boundary. In an unpublished report, Farley and Plutschak[1] observed possible, very early infections of *P. marinus* in 8% of oysters diagnosed by rectal thioglycolate cultures from three sites in Massachusetts. This suggests that oysters in waters as far north as Massachusetts may indeed experience *P. marinus* infections, as earlier publications suggested. The most serious effects of *P. marinus* infections, however, occur in the Gulf of Mexico, where mortalities are estimated at 50% or more annually (Craig et al., 1989). The occurrence of *P. marinus* in oysters north of Delaware Bay apparently remains a rare event at this time. Andrews (1988) claimed that seaside bays of the eastern shore of Virginia, and usually Maryland, were free of *P. marinus*. Lewis and Kern (independent personal observations) found the parasite in oysters from Maryland and Virginia portions of Chincoteague Bay. Prevalences as high as 96% were observed, of which 76% were judged to be heavy or very heavy infections. Associated mortalities of up to 84% were reported as being caused primarily by *P. marinus*.

In Georgia, *P. marinus* was found as early as 1966 when it was observed in oysters from Wassaw Creek and the Duplin and Woodbine Rivers. With the exception of oysters from the Duplin River (44%), the prevalence of infection from *P. marinus* never exceeded 12% in the 1966-68 survey. Any mortality which might have occurred as a result of *P. marinus* infections at these low prevalences would likely have been masked by what are considered normal losses to natural causes, and thus never reported.

Perkinsus marinus is, however, considered to be the etiological agent responsible for Georgia oyster mortalities observed in 1985-87, because of its high prevalence and intensity in thioglycolate cultures and histological sections of the oysters examined. Sampling in 1986 and 1987 showed continued high prevalence of *P. marinus* infections, with a slight but progressive increase in intensities.

Disease prevalence and intensity of *P. marinus* reflect seasonal parasite activity which strongly correlate with water temperature (Quick and Mackin, 1971). Infections are most severe during summer and early fall, then decrease in intensity as water temperatures drop. Results of this study failed to demonstrate statistically any correlation between water temperature and disease intensity. This is attributed to a lack of seasonal data and does not dispute the existence of the relationship. However, the trend of our disease in-

[1]C. A. Farley, National Marine Fisheries Service, NOAA, Northeast Fisheries Science Center, Oxford, MD 21654, and D. L. Plutschak, Maryland Department of Natural Resources, Oxford, MD 21654. Personal commun.

tensity data graphically resembles that of Quick and Mackin (1971) for Florida oysters infected by *P. marinus* at comparable temperatures.

Gross condition observed in 1986-87 reflects a lack of stored glycogen or "fatness." Low gross conditions are typical of oysters stressed by high water temperature, disease, spawning, and other events causing an animal to expend disproportionate amounts of energy on self maintenance. This results in depletion of glycogen reserves which translates into a poor condition. At the time of the year samples were taken, oysters would be expected to have a condition above medium (5). In all cases, the mean condition was below 5 and no oysters above a value of 6 were observed.

Oyster mortalities were first reported from Camden County, Ga., early in November 1985, and subsequently from McIntosh County in the beginning of January 1986. Reports occurred at a time when water temperatures were abnormally high and following a period of extensive rainfall associated with several tropical storms and hurricanes.

Typically, the intensity of *P. marinus* infections seen in the surviving oysters sampled in January 1986 would not be considered sufficient in itself to cause the extensive mortalities experienced in November 1985. However, by the time samples were taken the most heavily infected oysters had died. Quick and Mackin (1971) reported mortality in Florida oysters infected by *P. marinus* to begin at a medium infection intensity under normal circumstances. That would approximate a moderate infection intensity, or stage 5, in the current study. While infection intensity of individual oysters exceeded a stage 5, sample population intensity for sites ranged from 3.4 to 4.9. The added stress of extended periods of abnormally high water temperatures and unusually heavy rainfall are considered contributing factors to the mortalities. The same pressures likely continued during 1986, as climatic conditions were reported to have mirrored those of 1985 (Stevens, Personal commun.).

Since *H. nelsoni* was established as the causative agent of the Delaware Bay oyster epizootic (Haskin et. al., 1966), cyclic periods of oyster mortalities caused by *H. nelsoni* have continued to occur along the Atlantic coast in Massachusetts, Connecticut, New York, New Jersey, Delaware, Maryland, Virginia, and, to a limited extent, North Carolina (Haskin and Andrews, 1988). With the exception of New Hampshire and Rhode Island, *H. nelsoni* has been observed in oysters from each state along the east coast from Maine to Florida (Haskin and Andrews, 1988; Sindermann, 1990). To date, however, parasite-induced mortalities have not been reported from Maine or states south of North Carolina.

The most severe effects of *H. nelsoni* are seen in Delaware and Chesapeake Bays (Haskin et. al., 1965; Andrews and Wood, 1967; Farley, 1975; Lewis, 1988). As a direct result of *H. nelsoni* infections, oyster production in Delaware Bay dropped from about 8 million pounds of meats in 1953 to 167,000 pounds by 1960 (Sindermann and Rosenfield, 1967). Likewise, oyster production in Chesapeake Bay fell from 39.2 million pounds of oyster meats in 1955 (Anderson and Power, 1957) to less than 4.1 million pounds of meats in 1989 (USDOC, 1990). Disease caused by *Perkinsus marinus* and *H. nelsoni* has had a combined effect in reducing oyster production from Chesapeake Bay.

In January 1986, *H. nelsoni* was detected for the first time in Georgia in three oysters from Mud Creek. Although very heavy levels of intensity were observed in a few oysters (7.0), *H. nelsoni* is not believed to be the principal agent responsible for mortalities, because of its low prevalence and intensity in the sample population (0.0-0.4). Examination of the November 1986 and 1987 samples also revealed the presence of *H. nelsoni* in oysters from three additional locations in coastal Georgia.

Management Implications

Minimizing the effect of disease caused by *P. marinus* involves several key strategies which include: 1) Avoiding the transplantation of diseased seed stock, 2) reducing the time oysters are exposed to the disease (this may involve reducing the legal harvest size of oysters, and planting seed in the fall and winter after the disease process has been slowed by decreased water temperatures), and 3) isolating grow-out areas from known diseased areas (Andrews and Ray, 1988).

The cross-infection of oysters by *P. marinus* from other mollusks is a management concern with regard to the isolation of shellfish beds from infectious sources. Ray (1954), Andrews (1955), Andrews and Hewatt (1957), Perkins (1988), and McGladdery et. al. (1991) reported observations of *Perkinsus* and *Perkinsus*-like organisms in many other mollusks along the east coast of the United States. Although their taxonomic identity has not always been established, the organisms are apparently ubiquitous and easily transmitted. Results of earlier, unsuccessful work to cross-infect mollusks with *Perkinsus* spp. isolated from other molluscan species led to belief in the host specificity of parasites. Goggins et al. (1989), however, demonstrated cross-infection of Australian *Perkinsus* spp. from 6 molluscan sources to 10 species of mollusks; they concluded they were dealing with at least 2 species of *Perkinsus* and low levels of host specificity. The potential for cross infection, along with the observation of another species of *Perkinsus* found in scallops on the east coast, may be a concern where oysters are not infected by *P. marinus*. McGladdery et al. (1991) recently described a new species, *Perkinsus karlssoni*, in the bay scallop, *Argopecten irradians*, which was observed in specimens from the Gulf of St. Lawrence and Atlantic Nova Scotia, Can., as well as Rhode Island, Connecticut, and Cape Cod, Mass., in the United States. Whether *P. karlssoni* or other *Perkinsus*-like organisms may cross-infect *C. virginica* is yet to be demonstrated.

Mortalities are increased in areas, such as the Chesapeake Bay, where high levels of both *P. marinus* and *H. nelsoni* coexist. In these situations, Andrews (1979) believes *H. nelsoni* outcompetes *P. marinus*. This information highlights a potential danger to

Georgia shellfisheries, considering the recent discovery of *H. nelsoni* in Georgia oysters.

Responding to scientific evidence that *H. nelsoni* is intolerant of salinities below 10‰ (Andrews, 1964; Ford, 1985; Ford and Haskin, 1988a), oyster management officials now emphasize the use of lower salinity growing areas to avoid, or at least minimize, the effects of *H. nelsoni* on oysters (Ford and Haskin, 1988b). Because the transplantation of seed and shell stock is a vital component of management, the introduction of infected animals into previously unaffected systems is of great concern (Rosenfield and Kern, 1987; Sindermann, In press). Mortalities, with long-lasting consequences, can be linked to the movement of shellfish stocks (Farley, In press). It is likely, to some degree, that the progressive spread of diseases within Chesapeake Bay may be linked to the movement of infected seed stock.

Another management strategy involves the development of disease-resistant stocks through selective breeding techniques (Haskin and Ford, 1979; Ford, 1987). While some success has been made along these lines with regard to *H. nelsoni*, several problems remain. First, under intense infection pressures, even resistant strains succumb to *H. nelsoni* (Ford and Haskin, 1988b). Second, disease resistance against *P. marinus* has not been achieved. It is unknown at this time if animals resistant to *H. nelsoni* are resistant to other fatal oyster disease agents. Although disease resistance is viewed as a valuable asset in mariculture operations, it is not readily applicable to a wild fishery. In other than strictly controlled mariculture operations, diminished resistance by interbreeding with wild shellfish stocks may likely result over time.

Management of *P. marinus* has been shown to be more complex in the Gulf of Mexico than in the Chesapeake Bay region, largely because of elevated southern water temperatures (Andrews and Ray, 1988). It also may be premature to assume that management strategies devised to deal with *H. nelsoni* in the northeastern United States will apply in Georgia; climate and marsh ecology of coastal Georgia contrast greatly with the remainder of the eastern United States. This is reflected in the biology of local shellfish (Walker and Humphrey, 1984; Walker and Tenore, 1984; Walker, 1985; Heffernan et al., 1989a,b). For example, distribution, growth rates, and reproductive patterns for Georgia oysters and hard clams, *Mercenaria mercenaria*, contrast greatly when compared with other areas of the Atlantic coastline. Georgia oysters have extended reproductive periods, while polymodal reproductive cycles occur in hard clams. In Georgia, oysters primarily occur intertidally and spawn from April to October (Harris, 1980; Heffernan et al., 1989b); whereas, in most of their distribution, oysters occur subtidally. It may be reasonable to expect differences in the ecology and dynamics of pathogens in coastal Georgia as compared with the northeastern United States. Based on the uniqueness of the Georgia habitat, studies are in progress to develop approaches that will reduce mortalities from the effects of *H. nelsoni* and *P. marinus*. These include allowing the earlier harvest of marketable oysters, prior to the onset of mortalities resulting from parasitic infections.

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