GROWTH AND MORTALITY PATTERNS OF THE EASTERN OYSTER CRASSOSTREA VIRGINICA IN IMPACTED WATERS IN COASTAL WATERS IN NEW YORK, USA

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ABSTRACT We monitored cage-based populations of the eastern oyster Crassostrea virginica in coastal waters of New York Harbor in 2 phases of sampling, 1 with localities spread out over the New York–New Jersey Harbor area (started 2008) and another with 3 localities within Jamaica Bay (started 2010), all impacted by high nitrogen input, low dissolved oxygen, but over a water quality gradient. Patterns of growth, mortality, condition, and disease were compared with a clean-water site in Shelter Island, NY, sampled in parallel with both sampling phases. In both studies, oyster mortality in the urban sites increased dramatically during and after the second summer growth season. Mortality also increased at the same time period at the clean-water site, but to a much smaller degree. One instance of high mortality in the Lower Hudson was caused by MSX; but, otherwise, no known diseases were identified as the main cause of the sudden mortality increases. Our results suggest that a general effect of reduced water quality had a cumulative effect on the New York Harbor-emplaced oysters, which culminated in high mortality, mainly at the end of the second summer growing season. Despite the increased mortality, other factors such as soft tissue growth and reproduction were not reduced in the harbor sites relative to the clean-water control site. The vulnerability of oysters grown in impacted waters may have to be factored in attempts to restore oysters to impacted harbor waters.

KEY WORDS: oyster, Crassostrea virginica, mortality, restoration, stressors

INTRODUCTION

The eastern oyster Crassostrea virginica is a classic case of a once-abundant resource organism now in a steep and widespread decline along the east coast of the United States (Mckenzie 2007). Indeed, oyster reefs in bays throughout the world have declined to less than a quarter of their historical occurrence (Beck et al. 2011). Overexploitation, disturbance from dredging, pollution, and disease are likely the major components of loss of eastern oyster reefs. When an oyster reef is nearly removed by dredging, the balance of shell formation and degradation tips in the direction of shell breakdown, which reduces the amount of hard substratum available for colonization by oyster larvae (Mann & Powell 2007, Powell & Klinck 2007). It is possible that, globally, this balance point has been surpassed to the degree that restoration on a large scale is not generally possible. Local restorations, however, have been successful where tall layers of shell have been placed in areas where natural larval supplies are still available (Schulte et al. 2009).

Although exploitation, dredging, and disease may be the major factors in oyster decline, pollution has reinforced the declines initiated by exploitation (Lenihan & Peterson 1998), especially in New York Harbor (Franz 1982). The eastern oyster has wide physiological limits, and can survive and reproduce in salinities from open marine to less than 10 (Shumway 1996). Oyster gill function can be retained at temperatures as high as 37°C and salinities as low as 4 (Vernberg et al. 1963). Oysters are also rather tolerant of hypoxia (Stickle et al. 1989), but larval settlement and juvenile growth are strongly affected at hypoxic oxygen concentrations of 1.5 mg O2/L (Baker & Mann 1994). Low oxygen stress on oyster reefs is especially important in stratified waters, especially in deeper areas where circulation may be sluggish (Lenihan & Peterson 1998).

Oyster disease has been a major factor in oyster mortality in the middle Atlantic states. Dermo (Perkinsus marinus) was first described in Louisiana waters (Mackin et al. 1950) and in Texas (Ray et al. 1953), but it has become a major factor in northeastern U.S. waters (Dungan & Hamilton 1995, Mccollough et al. 2007) and appears to be moving northward as sea surface temperature has increased in recent decades (Ford & Smolowitz 2007). The agent (Haplosporidium nelsoni) for another disease, MSX, was transported to eastern U.S. waters along with an introduced oyster, and caused devastating mortality in Delaware Bay, followed by the local evolution of resistance (Haskin & Ford 1979, Ford & Haskin 1987, Ford & Tripp 1996). In both the cases of Dermo and MSX, parasite effects are strongest in higher salinities, corresponding to the seaward portions of estuaries and within estuaries during droughts (Soniat 1996), and minimal in salinities less than 10 (Haskin & Ford 1982, Kraeuter et al. 2003). Climate change involving increased rainfall may cause significant shifts in estuaries, balancing low growth versus low disease occurrence in expanded estuarine ranges of low salinity (Najjar et al. 2010, Levinton et al. 2011). Cycles such as the North Atlantic Oscillation influence climate in estuaries such as Delaware Bay, causing shifts in salinity and disease susceptibility (Bushek et al. 2012). In the Gulf of Mexico, disease has changed in prevalence in approximate synchrony with el Niño–la Niña cycles, although population responses may more be affected by salinity change and recruitment success during these cycles (Soniat et al. 2012).

The interaction of stressors such as pollution and disease may be a major obstacle to oyster restoration in impacted estuarine waters (Breitburg & Riedel 2005). Extensive evidence exists for interaction effects where infections of Perkinsus marinus increase in prevalence and intensity when oysters are exposed to a variety of toxic pollutants (Chu & Hale 1994, Anderson et al. 2002, Chu et al. 2002, Morely 2010). The ultimate decline of oysters in New York Harbor and the lower Hudson was accelerated by changes in water quality, which also was
accompanied by the spread of human pathogens (Franz 1982). During the 1970s *Vibrio cholerae* was detected in oysters from Louisiana (Blake et al. 1980), and species of *Vibrio* known to infect humans have been found in many areas including New York waters (Depaola et al. 2000, Lyons et al. 2007). In recent decades, water quality in New York Harbor has improved substantially, especially with regard to increased dissolved oxygen and reduced sewage input (Brosnan & O'Shea, 1998).

Oyster reefs, once plentiful from New York Harbor to lower Haverstraw Bay in the Hudson, are now missing, and opportunities for oyster reef reestablishment are few, except for rare occurrences of shell piles in Jamaica Bay and Haverstraw Bay (pers. obs., pers. comm.). Current oyster rarity means that recovery likely can only be achieved by human intervention via aggressive restoration programs (Bain et al. 2007).

Our objective, in conjunction with future plans for experimental reefs (Bain et al. 2007), was to investigate growth and mortality of oysters placed in aquaculture cages in a variety of coastal sites in New York Harbor waters and compare them with oysters placed in relatively clean water. Our working hypothesis was that oyster growth and survival in impacted harbor waters was equal to that in an unpolluted site, and that oyster disease would not be a factor in oyster mortality or reduced growth. If results were consistent with this hypothesis, one might argue that oyster restoration on a large scale was justified, because provision of substratum might result in rapid growth and recovery of oyster reefs by the establishment of a sustainable reproducing population.

MATERIALS AND METHODS

Study Regions

We studied growth, condition, and survival of oysters in impacted areas in 2 phases, with a different set of localities in each phase. Phase 1, initiated in June 2008, involved transplanting juvenile oysters (Fig. 1) to Raritan Bay (RB; Keyport, Brown's Point Marina), Lower Manhattan (P40; Pier 40), central Jamaica Bay (JBC; Broad Channel, private dock), and to a clean-water control site at Shelter Island (SI; Mashomack Preserve, Log Cabin Creek). Phase 2, initiated in June 2010, involved 3 sites (Fig. 1), spread along a water-quality gradient in Jamaica Bay, New York. The site near open ocean water was located at Gateway Marina (Jamaica Bay West (JBW)), a central Bay site was identical to the phase 1 study (Jamaica Bay Central (JBC); Broad Channel, private dock), and an eastern site (Jamaica Bay East (JBE)) was located at Inwood Marina, Hempstead, New York. The Shelter Island site was used again as an unpolluted control site. GIS locations are as follows: RB, Keyport NJ: 40.435929° N, 74.213071° W; P40, north side, Manhattan Hudson River: 40.730708° N, 74.013240° W; JBW Gateway Marina: 40.583279° N, 73.900469° W; JBC, Broad Channel (phases 1 and 2): 40.607083° N, 73.821776° W; JBE Inwood Marina, Hempstead, New York: 40.617382° N, 73.757888° W; SI, Mashomack Preserve: 41°02.832' N, 072°18.020' W.

Oyster Growth and Survival Monitoring

Oyster Source

Oysters were purchased from the Fishers Island Oyster Farm, Fishers Island, New York. We obtained oysters that were produced in a hatchery in late summer 2007 (phase 1) and 2009 (phase 2). These newly settled oysters were overwintered in the oyster farm’s nursery area located in Ocean Pond, an 85-acre brackish salt pond, and were then obtained the following late spring and transplanted to cages the following June (phase 1, 2008; phase 2, 2010). Experimental oysters were maintained in polyethylene mesh grow-out bags with 13-mm mesh, supported in wire mesh cages. The semirigid grow-out bags are shaped like rectangular boxes, with dimensions 94×43×7.6 cm. Each cage held 2 grow-out bags, 1 above the other in a vinyl-clad,
heavy-gauge wire cage (phase 1, 3 cages per locality and 2 bags per cage; phase 2, 2 cages per locality and 2 bags per cage). Each grow-out bag was stocked with 300 oysters.

We visited all cages every 2 wk to monitor mortality and growth. All oysters were counted to tally live and dead oysters; also, 20 live oysters were taken at random from each bag to measure shell height and then were returned to the bag. The individual bags and cages were cleaned of biofouling organisms, as well as feces and sediment buildup. Every second sampling, we also removed permanently 20 oysters to measure soft tissue dry mass, condition index (CI), and gonad condition. Oysters brought to the laboratory were placed in a temperature-controlled aquarium until they started pumping, typically within several hours, and were made to close their valves underwater before being removed to measure the whole live weight. Soft tissues were removed and oven-dried to constant weight at 75°C, and weighed using an analytical balance (±1 mg). Shell weight was measured immediately after removing tissue (i.e., towel and air-dried only) as recommended by Abbe and Albright (2003). Condition index was calculated as (Higgins 1938)

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CI = \frac{\text{Dry meat weight (g) } \times 100}{\text{Internal capacity volume (cm}^3\text{)}}
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The internal capacity volume is determined by subtracting the weight in air of the oyster’s valves from the weight in air of the oyster live weight. This is a valid method under the assumption that the effective density of the cavity contents is approximately 1 g/cm³ (Lawrence & Scott 1982).

To determine gonad ripeness and sex of the sampled oysters, the gonad tissue was examined closely before being dried with other soft tissue. The visceral mass was dissected and the relative amount of gonad tissue inside was assessed visually. A gonad tissue smear was prepared and observed under a compound light microscope, and 60 oysters were examined for presence of gametes. We report as an index of reproductive activity the percent of 60 oysters in which gametes (eggs or sperm) were present.

**Disease Monitoring**

Once a year (September 2008, 2009) for phase 1 we sampled 30 experimental oysters from each location for pathology. For histological evaluation, sections were prepared from bivalves using standard procedures (Howard et al. 2004). Tissues examined typically included the stomach, digestive gland, intestine, kidney, gill, and mantle. All stained tissues were evaluated microscopically for abnormal conditions, and the prevalence and intensity of infecting agents were determined when applicable (e.g., *Haplosporidium nelsoni*). For Dermo infection in oysters, pathogen burdens were assessed using standard Ray’s fluid thioglycollate medium technique. This culture-based technique provides better accuracy and quantification of Dermo infection than histological techniques. Limited funds during the phase 2 study restricted us to 1 assessment of disease in November 2010, following the second growing season. Because effects of Dermo persist in northeast oysters several months after summer (Ford & Smolowitz 2007), this sampling would be appropriate to evaluate the potential impact of Dermo disease at these sites. Infection intensities were ranked from 0 (no parasite cells detected) to 5 (heavy infection) as described by Ray (1954). Prevalence of *Perkinsus marinus* infection (percent infected divided by total examined) and weighted prevalence (sum of individual intensities divided by the total number of oysters examined, including negative oysters) were determined as described by Ray (1954).

**Monitoring of Water Quality**

Temperature, salinity, and dissolved oxygen were monitored biweekly with the aid of a YSI 85 TSO meter (YSI Corporation, Yellow Springs, OH). We also did high-resolution monitoring (every 15 min) with Onset HOBO temperature data loggers, which were attached to 1 cage at each site. We monitored chlorophyll biweekly, spring to fall, and less frequently in winter by collecting water samples with the aid of a Niskin bottle. The general method for analysis followed that of Strickland and Parsons (1968). Samples were passed through a glass fiber filter and extracted with 100% acetone, and filtered extracts were analyzed in a Turner Trilogy fluorometer (Turner Designs, Sunnyvale, CA). In most cases, we also filtered water on 20-, 5-, and 2-µm polycarbonate filters, and also glass fiber filters with a 0.7-µm retention to examine size-structured chlorophyll. We took the fraction of total chlorophyll trapped on a 5-µm filter as that size fraction that could be retained by eastern oysters, because particle retention declines as size decreases to less than 3–4 µm (Haven & Morales-Alamo 1970).

**Statistical Analysis**

All statistical analyses were done using JMP (SAS Corporation, Cary, NC). Tests of difference used the Kruskal-Wallace test, with significance set at *P* = 0.05. This included a general correlation analysis examining relations between mortality and salinity, temperature, dissolved oxygen, disease, and chlorophyll concentrations.

**RESULTS**

**Survivorship**

**Survival During Phase 1**

Figure 2A shows survival of transplanted oysters to different localities in phase 1, starting in June 2008. In fall 2008, an obvious major mortality event occurred at P40, with more than 35% mortality. Histological analysis demonstrated that these oysters were severely infected with MSX (not shown). Such an outbreak was not found in any of the other transplant localities during phase 1 and phase 2. During the summer and early fall 2009 (year 2 of the study), there was a more than 50% decrease in population size simultaneously in Jamaica Bay (JBC) and western RB, with a distinct pulse of mortality even at P40 (Fig. 2B). A marked increase in mortality rate also occurred at the SI control site during this time (Fig. 2A, B), but to a much smaller degree than the New York–New Jersey Harbor sites (~10% mortality). During the third year of the study (2010), an increase in mortality was again observed during the summer and fall at the 2 sites we continued to monitor (JBC and SI). Once again, the increase in mortality was greater at the New York Harbor site (JBC; ~20%) than at the clean-water control site (SI; ~10%). We followed the experimental populations through June 2011, but there was little subsequent mortality.
Survival During Phase 2

Figure 2C, D shows patterns of survival and mortality rates during the phase 2 study, which commenced in June 2010. Mortality was 5% or less for all sites through spring 2011. However, mortality at all 3 Jamaica Bay sites increased dramatically in summer to fall 2011, during and just after the second growth season (Fig. 2C, D). Mortality increases were not synchronous at the 3 sites, and the more oceanic site showed the first signs of increased mortality. However, the Jamaica Bay JBC site showed the greatest decrease, of about 70% of the starting population, through November 2011. Mortality also increased at the SI control site in July 2011, but the decrease in percent of the starting population was far less (about 15%) than that of any of the Jamaica Bay sites, which ranged from 45–75% during the summer to fall period.

Soft Tissue Growth

Figure 3A shows soft tissue dry mass for phase 1 localities. During the first season, oysters at Jamaica Bay added tissue far more than the other localities. The RB and SI control site were about the same. Notable was the very low growth observed at P40, located within the lower Hudson River. During the second season, Jamaica Bay sites still had the greatest mass, although SI oysters appeared to catch up. Oysters at P40 still did not gain mass. In phase 2 sites (Fig. 3B), soft tissue growth was much less at the SI control site relative to the 3 Jamaica Bay sites, where growth at the Jamaica Bay East locality was somewhat less than the other 2 sites.

Condition Index

Condition index is shown in Figure 4. During the phase 1 study, CI was consistently lower at P40 and at SI, and higher in Jamaica Bay and RB. For Jamaica Bay and RB, CI was highest in summer 2008 and lower 2009, when rains were very heavy (Levinton et al. 2011). During the phase 2 sampling, CI at SI was, for most of the period, less than at the 3 Jamaica Bay sites. This may be explained by the high shell growth rate at this site (Levinton et al. 2011).

Gamete Presence

Percent of oysters with gametes in phase 1 and phase 2 localities is shown in Figure 5. During the phase 1 study, gonad ripeness was high for all localities for summer 2008 and summer 2009, and low in winter, with little difference among localities. Similar results were obtained in the phase 2 study.

Water Column Chlorophyll

Figure 6 shows chlorophyll in particles larger than 5 μm for all sites during the phase 1 (left panel) and phase 2 (right panel) studies. During phase 1, summer and late-winter peaks in chlorophyll are apparent and consistently highest in Jamaica
Bay. The SI control site is generally low in chlorophyll, as is the P40 Hudson River site. Raritan Bay is more variable, with summer low peaks and a strong winter peak. Jamaica Bay total chlorophyll regularly exceeded 60 μg/L and often 100 μg/L. Raritan Bay was often greater than 10 μg/L, but the SI control site and P40 were usually less than 5 μg/L, although in the late summer a bloom at SI reached values of more than 40 μg/L.

During the phase 2 study, summer and winter peaks in chlorophyll were apparent at all 3 Jamaica Bay sites (Fig. 6). During summer in Jamaica Bay, the east and central sites had total chlorophyll that regularly exceeded 60 μg/L, whereas the west site only reached summer levels of 20–40 μg/L, probably as a result of greater mixing with ocean water. In contrast, the SI control site usually did not exceed in total chlorophyll (5 μg/L), and never exceeded 50 μg/L. Of great interest were the generally high percentages of chlorophyll in particle sizes larger than 5 μm in both phase 1 and phase 2 samples. During the phase 1 study, these percentages were generally more than 50% (JBC, 72.9 ± 4.0% (SE); P40, 59.9 ± 4.0%; RB, 66.4 ± 4.0%; SI, 49.2 ± 4.0%), although among-locality variation was significant (Kruskal-Wallace test, $P < 0.0009$), with SI and P40 the lowest. This was also true of the phase 2 study (JBE, 64.4 ± 4.2%; JBC, 71.7 ± 3.9%; JBW, 72.5 ± 4.5%), but among-locality variation was not significant (Kruskal-Wallace test, $P > 0.05$).

**Dissolved Oxygen.**

Figure 7 shows dissolved oxygen at the depths of the cages at all sites for phase 1 (Fig. 7A) and phase 2 (Fig. 7B). During the first growing season of the phase 1 study, dissolved oxygen was often less than 4 mg/L at both the RB and Jamaica Bay sites. Raritan Bay dissolved oxygen was consistently at 2 mg/L during the second growing season. Otherwise, concentrations ranged between 4 mg/L and 6 mg/L during both growing seasons and were much greater in the colder winter–spring seasons. At the SI control site, dissolved oxygen in the growing season was usually 6 mg/L or greater, although it was less than 4 mg/L for 1 sampling period during late summer 2008. During the phase 2 study, dissolved oxygen at all Jamaica Bay sites was generally more than 4 mg/L during the first season, but a strong anoxic event hit JBC at the end of July 2011 (Fig. 5B). This anoxic event did not occur at the 2 other sites and it corresponded in time to the strong mortality event at the JBC site (Fig. 2B). Dissolved oxygen at the SI control site was

![Figure 3. (A, B) Soft tissue mass (mean ± SE) for phase 1 sites (A) and phase 2 sites (B). Phase 1 sites: Pier 40, square; Jamaica Bay Central, circle; Raritan Bay, triangle; Shelter Island, diamond. Phase 2 sites: Jamaica Bay East, diamond; Jamaica Bay Central, square; Jamaica Bay West, triangle; Shelter Island, circle.](image)

![Figure 4. (A, B) Condition index for phase 1 sites (A) and phase 2 sites (B). Phase 1 sites: Pier 40, square; Jamaica Bay Central, circle; Raritan Bay, triangle; Shelter Island, diamond. Phase 2 sites: Jamaica Bay East, diamond; Jamaica Bay Central, square; Jamaica Bay West, triangle; Shelter Island, circle.](image)
Salinity data are shown in Figure 8. For phase 1, salinity at the SI control site and JBC were consistently between 25 and 30. However, salinities at P40 and RB were less, fluctuating around 20, but ranging from 8–25. During phase 2, salinities at all 3 Jamaica Bay sites fluctuated around 25, ranging from 22–30 and overlapping with the SI control site.

Disease

During the phase 1 study, Dermo infections were common and similar in prevalence and intensity in oysters in Jamaica Bay, RB, and the SI unpolluted site (Fig. 9). The prevalence and intensity of Dermo infections increased in year 2 of the study at these 3 sites. Dermo was much less prevalent at the lower Hudson River site, P40. However, infections with MSX were highly prevalent at P40 in 2008, causing substantial mortality, as mentioned earlier (Fig. 2A, B). The prevalence of MSX declined sharply at the lower Hudson site in 2009, as did mortality. In 2008, MSX was not detected in oysters in Jamaica Bay, RB, or the Shelter Island, but did appear in 2009 in RB (21% prevalence) and Jamaica Bay (3% prevalence). No other significant disease-related conditions were detected.

During phase 2 sampling within Jamaica Bay, we only sampled for diseases (Dermo and MSX) in November 2011 (end of second season). Dermo prevalence (Fig. 10) was high in the SI sample (>90%), average to low in the JBC Broad Channel site and at the JBE Inwood Marina site, and very low in the JBW Gateway Marina site. The disease MSX was detected only in the JBW Gateway sample (13% prevalence). No other significant conditions were detected. The order of prevalence shows that mortality was low (SI) where disease prevalence was very high, and vice versa for the Jamaica Bay sites, which suggests that disease prevalence measured in November alone cannot explain mortality patterns.

Multivariable Considerations

Combining localities for 2 y, it is possible to consider multivariable correlations between mortality and salinity, oxygen, chlorophyll a, and disease (weighted prevalence). To do this, we combined data from phase 1 and phase 2 sampling. Mortality peaks occurred at different temperatures, but the correlation between degree of mortality and temperature at the time of the peak was very low ($r = -0.14$). Mortality showed no strong correlation with any of these variables except weighted prevalence of Dermo (partial correlation $r = -0.773$, $P < 0.05$). However, this negative correlation (which would mean that disease is associated with low mortality) is explained by time,
because values from phase 1 and phase 2 clustered separately at relatively high mortality–low disease and low mortality–high disease, respectively. Mortality was highest at the impacted Jamaica Bay sites during the phase 2 sampling, but weighted prevalence of Dermo was very low, especially relative to the clean-water site at SI (Figs. 9B and 10B). In any event, they show no correlation that suggests that disease is causing mortality differences among sites. Overall, any analyses have limited ability to achieve statistically significant relationships, with only 7 sites to compare.

**DISCUSSION**

Oysters are a cultural legend in New York Harbor and Lower Hudson waters (Kurlansky 2006), but they disappeared by the early 20th century, a victim mainly of pollution, with a source from sewage that resulted in very low oxygen concentrations and even anoxia in summer (Franz 1982). Management with the objective of reducing biological oxygen demand has resulted in strong improvements in dissolved oxygen through the latter half of the 20th century (Suszkowski 1990, Brosnan & O’Shea 1998), but New York Harbor and lower Hudson estuary waters are still strongly impacted by hypoxia, high nutrient loading (Howarth et al. 2006), and the presence of a wide range of toxic substances that can be accumulated readily by oysters (Steinberg et al. 2004). Oysters are found in scattered populations in New York Harbor and the lower Hudson, but they are in very low abundance and never in the form of oyster reefs (Medley 2010). Oyster recruitment is evidenced by the scattered appearance of oysters in the intertidal (Medley 2010, pers. obs.), and occasional recruitment in areas such as the Lower Hudson. Three years of placement of shell bags failed to result in oyster settlement in a range of sites in Jamaica Bay (Levinton & Doall, unpubl. data), where our evidence demonstrates that oysters can grow well and spawn.

Environmental stressors such as anoxia (Shumway & Scott 1993, De Zwaan & Eertman 1996, Shumway 1996), extreme temperature (Vernberg et al. 1963), toxic substances (Roberts 1976, Calabrese et al. 1977), and even invasive species (Thieltges 2005) can increase mortality of marine bivalves, sometimes by competitive interactions (Morales et al. 2006). In Jamaica Bay, hypoxic and anoxic events are common, and bivalves take up metals from the water column (Rodney et al. 2007). However, the interactions of stress factors may elevate the probability of
mortality. Exposure to cadmium reduces survival of eastern oysters exposed to elevated temperature stress (Lannig et al. 2006). The eastern oyster may be more susceptible to infection by diseases such as Dermo when exposed to low flow (Lenihan et al. 1999) or toxic substances in polluted sediments (Chu & Hale 1994). Marine mussel mortality is enhanced when mussels exposed previously to hypoxia are exposed to toxic substances such as metals. In the case of PCBs, this effect may be observed, but only after several months. In Raritan Bay, which is about 38 km apart, Jamaica Bay and RB, which are about 38 km apart, Jamaica Bay and RB are both strongly impacted by a range of toxic substances and hypoxia (Steinberg et al. 2004), although improvements in dissolved oxygen have been observed during recent decades (O’Shea & Brosnan 2000).

During our phase 2 study, we also saw a striking increase in mortality during the second warm growing season at 3 sites in Jamaica Bay, ranging from 1 that is close to a high exchange with open coastal shelf waters and another that is quite distant from such an exchange. Unexpectedly, mortality commenced earlier in the eastern high-exchanging site, although ultimately total mortality was less than in JBC and JBE. In both studies, we observed an increased mortality at the clean-water control site, but mortality never was as high as the impacted sites. In both phase 1 and phase 2 studies, mortality at the SI site amounted to about 15% of the starting population and was never above a few percent during a 2-wk period. In contrast, mortality at the phase 1 sites was 40% or more, and mortality during the phase 2 Jamaica Bay sites was 50–70%. Disease prevalence alone could not explain differences in mortality patterns among sites because there was no strong difference between the control site and the impacted sites during phase 1, and prevalence of Dermo was much greater at the SI control site during the phase 2 study relative to all 3 impacted Jamaica Bay sites. One can argue that our sampling schedule for disease prevalence during phase 2 sampling (done in November) may have missed peak MSX and Dermo prevalence.
weighted prevalence in the Northeast (and in New York in particular) peak in fall, lagging 2–3 mo after temperature maxima (Ford & Smolowitz 2007). It is also possible that high mortalities of infected oysters may have resulted in lower disease prevalence after periods of high mortality, resulting in the negative relationship observed between disease prevalence and mortality. This interpretation is not consistent, however, with the high weighted prevalence of Dermo found at the SI site (phase 2) with very low mortality, which was estimated at the same time as the sites with high mortality.

Despite the great increase of mortality rate in impacted sites in the second summer, we found vigorous rates of soft tissue growth at the impacted sites, especially in Jamaica Bay. This is likely associated with the high proportion of food particles larger than 5 μm. Thus, until the great increase of mortality during the second warm season, oysters were growing and apparently spawning.

We explored the hypothesis that differences in mortality patterns at the different sites could be the result of differences in environmental conditions. During phase 2, the water at the JBC site was anoxic at the end of July 2011, and mortality was stronger at this site than at the other 2 Jamaica Bay sites, where oxygen remained above 4–5 mg/L (Fig. 5). Jamaica Bay West, the putatively cleanest water site in Jamaica Bay, had the lowest mortality, close to that of SI (Fig. 2). Salinity does not appear to be related directly to the observed differences in mortality patterns, because mortality was observed during the second warm season in high-salinity sites (e.g., Jamaica Bay) and lower salinity sites (e.g., RB, P40). Mortality in our control high-salinity site was elevated during the second year, but the peak was very small and far less than that of the impacted areas during both phase 1 and phase 2 samplings. Our study suffers for having had only 1 “clean” control site, but the low mortality during the second warm season at this site (SI), was quite similar from phase 1 to phase 2 sampling.

The second-year mortality syndrome we observed is clearly associated with summer, which is the time of maximum water temperature and also corresponds to spawning season. As spawning proceeds, oysters are probably weakened physiologically (Li et al. 2007). However, we did not observe high mortality during the first reproductive season, and mortality was only high in presumably environmentally impacted sites during the second warm season, as evidenced by only slightly increased mortality at the control site at SI, suggesting no relationship between reproductive status and mortality. Reproductive status also did not correlate significantly with mortality over the 7 sites (P > 0.05). Summer mortality has been observed in a number of studies of the Pacific oyster Crassostrea gigas, the cause of which is likely multifactorial and perhaps related to pathogens (Samain et al. 2007). Growth and even gonad maturation may be diminished (Soletrchner et al. 2006). In an Irish Sea site, sea temperatures warmer than 21°C were associated with compromise of the immune system, and experimental studies suggested that high nutrient concentrations affected physiological conditions at high seawater temperatures (Malham et al. 2009). Strong phytoplankton blooms occurred in Jamaica Bay during both winter and summer, but mortality was elevated only in summer. Even though the mortality patterns described here in Crassostrea virginica are similar to those described during summer mortalities of Pacific oysters, it remains speculative to suggest similar underlying mechanisms for both species. Our study shows no strong association between total chlorophyll and mortality.

Our results show that oysters at several impacted sites in New York experience higher levels of mortality than oysters at SI, a clean site. These observations have important implications for oyster population dynamics of impacted sites and also for the fate of oyster restoration projects in impacted waters. Oyster restoration projects in New York Harbor and coastal waters are designed mainly to restore the ecosystem services formerly performed by dense oyster populations, especially when they are in the form of well-developed reefs. Oysters can be restored only by the installation of a sustainable population. The large-scale mortality we observed occurred under what might otherwise be considered ideal conditions, because oysters were caged from predators, and culture conditions prevented excessive resuspension, turbidity, and strong crowding. These conditions allowed high survival among oysters maintained in a clean-water control site through the second year, but resulted in very high mortality at all impacted sites commencing during the second summer. We conclude from this that oyster mortality resulting from disease and other unexplained conditions in impacted waters may be a major impediment to maintenance of long-term sustainable oyster populations that can replace themselves with juvenile recruitment. High mortality during the second year constitutes a potential major loss of reproductive contribution to a sustainable population.

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LITERATURE CITED


