Delaware Bay Oyster Seedbed Disease Monitoring Program 2005 Status Report

Prepared by

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To

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New Jersey Delaware Bay Seedbeds

Introduction

Oyster mortality on the Delaware Bay seedbeds is caused by a variety of factors including predation, siltation, and freshets, but since the appearance of *Haplosporidium nelsoni* (the agent of MSX disease) and *Perkinsus marinus* (the agent of dermo disease), disease mortality has been the primary concern. Fortunately, MSX disease has not been problematic for native oysters in Delaware Bay for several years, particularly on the seedbeds. Hence, recent efforts have focused on dermo disease.

Since the appearance of dermo disease in 1990, average mortality on the seedbeds, as assessed by total box counts during the fall survey, has fallen into 3 major groups: Upper, Upper Central and Central/Lower regions (Figure 1). Salinity increases from Upper to Lower regions and each region receives fresh water inputs from different tributaries along the Jersey Shore. The inputs and the geomorphological configuration of the coves influence salinity, circulation and flushing, which in turn influences the spatial and temporal prevalence and intensity of dermo disease on the seedbeds and ultimately oyster mortality. Through continued temporal and spatial sampling efforts, we are developing a better understanding of dermo disease patterns and processes that should ultimately improve management efforts. For 2005, with funding from the State of New Jersey and directed by the Delaware Bay Shellfisheries Council, the Disease Monitoring Program was instructed to complete the following objectives:

1. Continue monthly monitoring from March to November of size, mortality and disease at the five long-term seedbed stations (see Figure 1 for locations):
    a. New Beds grid 26
    b. Bennies grid 110
    c. Shell Rock, corner of grids 10, 11, 19 and 20
    d. Cohansey grid 44
    e. Arnolds grid 18

2. In coordination with directive 1, continue monthly monitoring of size, mortality and disease on the 2003 Bennies Sand surf clam plant demonstration project, and the 2004 Middle to New Beds transplant (see Figure 1 for locations).

3. In coordination with the annual stock assessment survey, conduct a spatial survey of dermo disease covering all beds sampled by the survey, sampling selected sites for MSX.

4. Conduct a preliminary experiment to assess the relative MSX resistance of upper seedbed oysters.

Objectives 1, 2 and 3 comprise the basis of the long-term monitoring program that is essential to track disease problems and alert the industry. At the 2005 Delaware Bay Stock Assessment Workshop, discussion of moving oysters from the upper seedbeds down bay in order to replenish dwindling stocks due to lack of recruitment lead to concerns over the potential for development of an MSX epizootic. Over the last decade or so, MSX infections have been rare and generally light when detected. As a result, MSX data has not been presented nor discussed in recent annual reports. There is fairly good evidence that much of the oyster population in Delaware Bay has developed relatively high levels of resistance to MSX following a severe epizootic in the mid-1980s. The spatial extent of this resistance, particularly up bay into the lower salinity areas, remains unclear. Hence, oysters from low salinity areas might risk the re-introduction of susceptible individuals and hence their genes into the larger Bay population. This hypothesis precipitated the request for a preliminary MSX challenge study (objective 4).

In addition to the four objectives listed above, between September and December 2005, the Disease Monitoring Program measured spat recruitment, growth, mortality and background dermo levels on seven shell planting sites, a control site, and a transplant site that relocated oysters from Middle to Shell Rock. These additional efforts were supported with funds provided by New Jersey, Delaware and the US Army Corps of Engineers and will be reported in detail elsewhere. HSRL staff, especially Iris Burt, and NJDEP Bureau of Shellfisheries staff, especially Jason Hearon, provided technical support for much of this work. Dr. Susan Ford, assisted by Emily Scarpa, conducted the MSX challenge experiment and MSX survey.

Methods

Monthly measurements related to objectives 1 and 2 included oyster density estimates, size frequencies, box count mortality estimates, and prevalence and intensity of dermo disease.

Samples were collected from the NJDEP R/V Zephyrus captained by Jason Hearon. Three one-minute dredge hauls with a 0.81 m oyster dredge were collected at each site with a set length of cable, regardless of water depth. GPS readings were recorded to estimate individual tow distances. Two separate composite one-bushel samples were created from the three replicate tows at each site. The first bushel was used explicitly to estimate oyster densities relative to boxes and cultch material. For this bushel, a 12-14 quart sub-sample of cultch, boxes and oysters was combined from each of the three one-minute dredge hauls to form a composite bushel that was subsequently sorted on deck to determine numbers and volumes of oysters, boxes and cultch present. Because this first bushel may not always yield a high number of oysters and boxes to provide a precise estimate of mortality, a second composite bushel consisting of randomly collected oysters and boxes from the three each replicate dredge hauls (approximately a third of a bushel from each haul) was created and then sorted to enumerate boxes and oysters to provide a more precise measure of mortality. One hundred randomly selected oysters from this second bushel were returned to the laboratory for size frequency determination. Twenty of those individuals were used in the standard RFTM dermo disease assay to determine prevalence and intensity of dermo infections. Dermo was diagnosed using the standard RFTM tissue assay with rectal and mantle tissues. The percent of oysters in the sample with detectable infections is termed the prevalence. Infection intensity is scored along the "Mackin scale" from zero (= pathogen not detected) to five (= heavily infected) and then averaged among all oysters in the sample to calculate a weighted prevalence. From June to September, a subsample of the oysters returned to the laboratory were inspected for reproductive maturity and assigned ranks of 0 to 3 based on the fullness of the gonad. Gametes from each individual inspected were also examined microscopically to determine sex.

Completion of objective 3 was coordinated with the annual Fall seedbed stock assessment survey. Samples of at least 20 oysters from every bed surveyed were processed for dermo prevalence and intensity. Mortality estimates were calculated as the number of boxes and gapers divided by the total boxes, gapers and live oysters (excluding oysters less than 20 mm in shell height). Dermo was diagnosed as described above. Histological analyses were performed on selected beds to determine prevalence and intensity of MSX disease using standard histological procedures for bivalve molluscs. Dermo disease data have been collected since 1990 and MSX disease data since 1958, providing insight into year-to-year variation and long-term cycling.

To complete objective 4, a preliminary analysis of MSX resistance of upper seedbed oysters, samples of 400 oysters each were collected from Arnolds, Shell Rock, the Cape Shore flats and Maine. These oysters respectively represent three stocks that historically experienced increasingly greater selection by MSX (maximum MSX infection prevalence in most of Delaware Bay has been ≤30% since 1989, and <10% on the upper beds, e.g. Cohansey and Arnolds, since 1992) and a naïve stock that has never been exposed to MSX. Each sample was divided equally between two replicate plastic mesh bags and deployed on the Rutgers lease in Cape May Harbor in May 2005. In recent years, oysters in Cape May Harbor had been regularly exposed to MSX infections, although dermo infections have been very light. An initial sample of 20 individuals from each location was collected prior to deployment and processed for both MSX and dermo. Mortality was monitored throughout the summer and oysters were sampled (n = 20 per stock) in July, September and October 2005 for both pathogens.

Results and Discussion

During 2005, temperature and salinity showed normal seasonal fluctuations (Figure 2). There was virtually no difference in temperature across the seedbeds. Temperatures generally considered warm enough to trigger widespread spawning (26ºC) were reached by July, contrasting 2004 when these temperatures were not observed. Oysters were reproductively mature by mid-June and remained at least partially ripe into August, but most had spawned by September. Salinity followed the general up to down bay gradient with bay-wide lows occurring in April and October (Figure 2), but no dramatic freshets occurred as experienced in the previous year. Dermo prevalence was negligible in late spring, but increased substantially by August (Figure 3) in response to the warm summer temperatures. Fortunately, infection intensities remained relatively low except on New Beds, which was the only bed monitored that incurred a subsequent increase in mortality (Figure 3). On all other beds monitored monthly, box-counts remained relatively constant at background levels of 5-15% throughout the year.

In contrast to recent years, the average size of oysters began to decline during 2005 on some of the seedbeds, namely New Beds and Bennies (Figure 5). The absence of significant mortalities (Figure 3), reduced fishing pressure resulting from reduction in harvest quotas or bed closures, and the appearance of many young oysters, particularly on lower bay beds indicated that this reduction in average size was a result of recruitment of smaller oysters into the population rather than removal of larger oysters by fishing or natural mortality. Young oysters were noticeably abundant during monthly sampling efforts on these beds. Interestingly, the same was not true for Shell Rock or Cohansey, and oysters actually increased slightly in size during the year on Arnolds. These observations suggest little recruitment has occurred on Shell Rock, Cohansey or Arnolds.

As noted last year, oysters transplanted down bay from Middle to New Beds rapidly developed heavy dermo infections and immediately began to die. This pattern continued during 2005 (Figure 4). Assuming many of these were larger oysters, it may partially account for the striking decrease in mean size of oysters on the 2004 Middle to New Beds transplant ground (Figure 5). Visual inspection of dredge samples indicated there was good recruitment on this ground and this certainly also accounted for part of the decrease in mean size. In contrast, the spatted clamshell transplanted up bay in 2003 from Reeds Beach to Bennies Sand as part of the NJDEP Bureau of Shellfisheries demonstration project did not begin to accumulate more than light dermo infections until this year. Furthermore, those infections were still less prevalent and less intense than dermo infections in the Middle to New Beds transplant oysters. One reason may be that the oysters from Middle were older and contained latent infections when transferred whereas the younger oysters planted on Bennies Sand had not yet acquired infections. In addition, Bennies Sand is further up bay at a lower salinity site that typically experiences slightly lower dermo levels (Figures 9 and 10). Nevertheless, given that the past two years have been relatively low years for Dermo disease, these observations indicate that moving seed oysters down to New Beds is not a viable strategy unless the oysters will be harvested later in the same season. This would require only moving market-size oysters. If they are not harvested shortly after being moved, dermo is likely to proliferate and cause significant mortality, risking additional spread of the disease to other areas of the bay. In contrast to the Middle to New Beds

transplant, results from the spatted clamshell transplant project from Reeds Beach indicate that this is a viable strategy that can yield high quality, marketable oysters within two years, at least in low dermo years. It is not clear whether these oysters possess any increased heritable genetic resistance to dermo and there is no data to suggest this was the case. Instead it may simply be that the Middle oysters were exposed to dermo and transplanted with latent infections whereas the Bennies Sand oysters were not.

Figures 6 through 8 depict annual fall dermo prevalence, dermo infection intensity (= weighted prevalence) and box-count estimated mortality from 1989 to 2005. In each figure, a red horizontal line indicates the long-term average for the time period displayed. In 2005, the prevalence and intensity of dermo were below long-term means on the upper-central, central and lower beds, but were higher than last year (Table 1, Figures 6-8). In contrast, mortality levels were lower than last year, indicating that the increase in dermo infections was not increasing mortality. The exception to this pattern was in the upper seedbeds where mortality was higher than average while disease was lower than average and lower than 2004. Freshwater kill that occurs on the upper seedbeds, reducing dermo levels as it also kills oysters, explains this apparent anomaly. The long-term dermo patterns on all but the upper beds indicate a possible cyclical periodicity of about seven or eight years. Such a cycle could be driven by larger regional climate patterns such as those driven by the North Atlantic Oscillation (NAO), but this requires further investigation and probably a longer time series for validation of any relationship. This periodicity would also suggest that dermo and possibly dermo-related mortality will increase next year. Alternatively, more local conditions could be determining interannual fluctuations and a long, cool, wet spring could suppress dermo disease as it appears to have done in recent years. Examination of dermo prevalence, dermo intensity and box-count mortality estimates on a bed-by-bed basis continues to reflect the overall positive correlation with increasing salinity from up bay sites to down bay sites (Figures 9-11). These data also show that dermo prevalence on many beds exceeds long-term means even though dermo intensity and mortality do not. The increase in prevalence may, unfortunately, forecast increases in dermo intensity and dermo-related mortality for 2006, unless, as noted above, local conditions inhibit the development of dermo disease.

A plot of long-term mean fall box-count mortality estimates against long-term mean dermo infection intensities (Figure 12), shows how the seedbeds can be demarcated into three or four disease and mortality zones. The upper beds fall into a low disease zone with weighted prevalence of dermo generally below 1.0 on the Mackin Ranking Scale. This zone generally experiences a background mortality of 11 to 12 percent. As dermo intensities increase above 1.0, they begin to generate higher levels of mortality. Dermo intensities between 1 and 2 occur on the upper-central beds (Middle to Shell Rock) and generate mortality that averages between 15 and 20 percent. Once dermo levels exceed 2.0, average mortality increases to between 25 and 50%. Figure 13 updates the plot originally developed by Dr. Susan Ford to predict fall mortality from May prevalence data. The relationship is positive and significant, but only explains about 50% of the variation. Clearly, many other factors such as seasonal temperature, freshwater inflow, predation, etc, influence this relationship and account for the remaining variation.

Finally, the MSX challenge study indicated that a high degree of MSX resistance had developed on upper-central and upper seedbeds (Table 2). The oysters from Arnolds and Shell

Rock were exposed to natural levels of MSX beside lower Bay MSX-resistant Cape Shore native oysters and susceptible controls from Maine in Cape May Harbor where *H. nelsoni* pressure has been relatively heavy in recent years. After six months, prevalence of susceptible Maine oysters was 90%, with most infections being advanced. At the same time, prevalence in the two upper bay groups was only 10 to 20% with mostly light, localized infections. These observations support the hypothesis that a high degree of MSX resistance is now present across the New Jersey Delaware Bay seedbeds. In contrast, there is little evidence that significant resistance to infection by *Perkinsus marinus* has developed despite continuous exposure and consequent mortality over the past 15 years.

Table 1. Comparisons of 2005 mortalities, dermo prevalence and dermo weighted prevalence with long-term averages (1989-2005) by seedbed region. Numbers are means ±95% CI.

	Percent Mortality		Prevalence		Weighted Prevalence	
Region	**2005**	**long-term**	**2005**	**long-term**	**2005**	**long-term**
Upper	14 (2)	11 (2)	0 (0)	31 (19)	0.0 (0.0)	0.3 (0.3)
Upper-Central	7 (5)	15 (2)	63 (27)	75 (11)	1.1 (0.3)	1.5 (0.3)
Central-Lower	18 (4)	35 (3)	81 (22)	92 (3)	2.0 (0.4)	2.6 (0.2)
All regions	**14 (4)**	**25 (2)**	**60 (23)**	**81 (5)**	**1.4 (0.4)**	**2.0 (0.4)**

Table 2. MSX prevalence / percent advanced / and weighted prevalence (scale of 0 to 4) in oysters undergoing natural challenge at Cape May Harbor in 2005. N = 20 for each sample.

Source of Oysters	**Time 0, May 3-12**	**July 7**	**August 8**	**October 13**
Maine (Susceptible)	0 / 0 / 0	0 / 0 / 0	30% / 20% / 1.0	90% / 80% / 3.3
Arnolds	0 / 0 / 0	0 / 0 / 0	0 / 0 / 0	20% / 5% / 0.5
Shell Rock	0 / 0 / 0	0 / 0 / 0	5% / 0.1	10%/ 5% / 0.3
Cape Shore	0 / 0 / 0	--	0 / 0 / 0	0 / 0 / 0

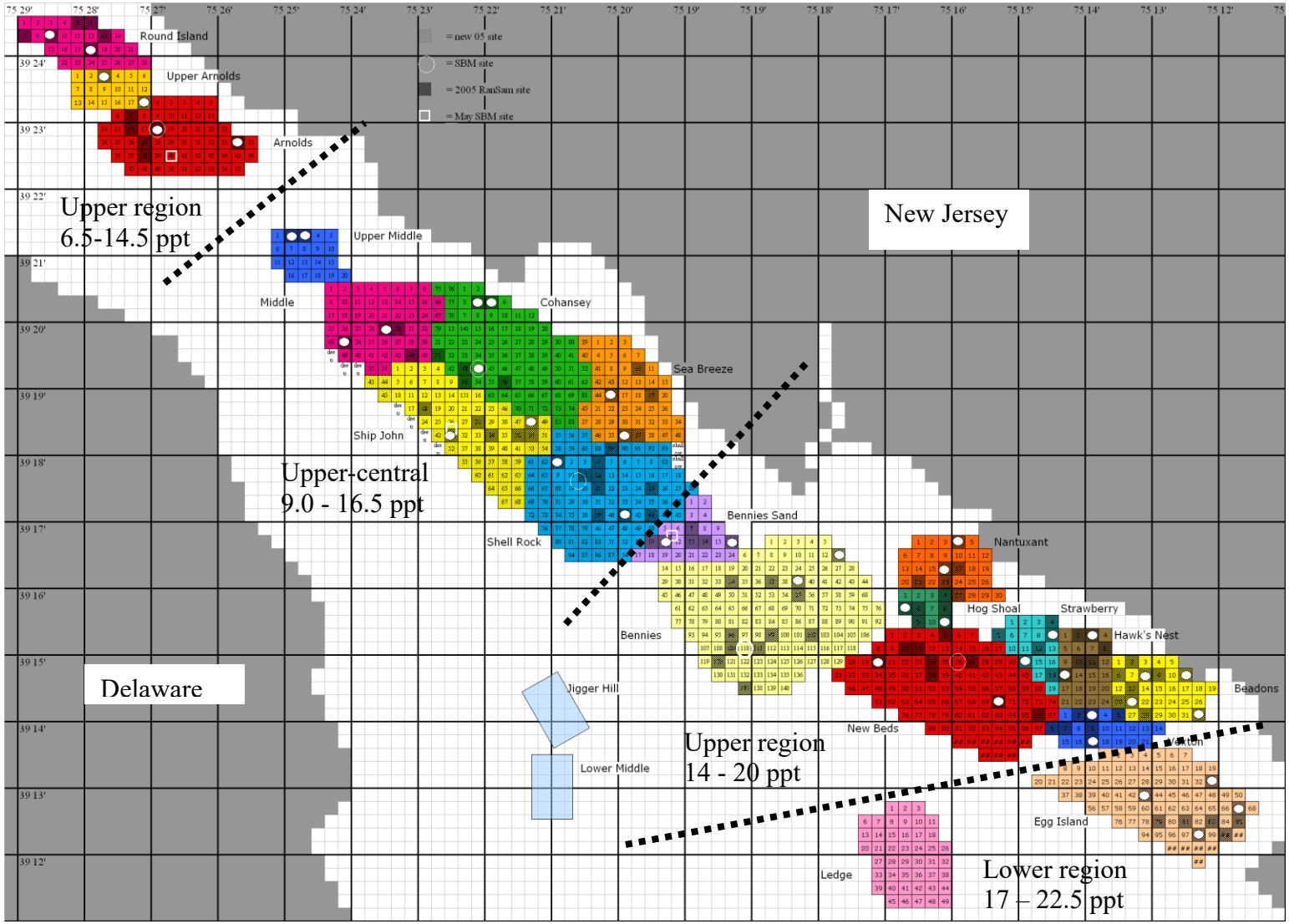

Figure 1. Delaware Bay New Jersey Natural Oyster Seedbeds and two shell plant sites in Delaware waters. Dotted lines separate regions. Salinity regimes for upper, upper-central, central and lower regions are indicated in parts per thousand.

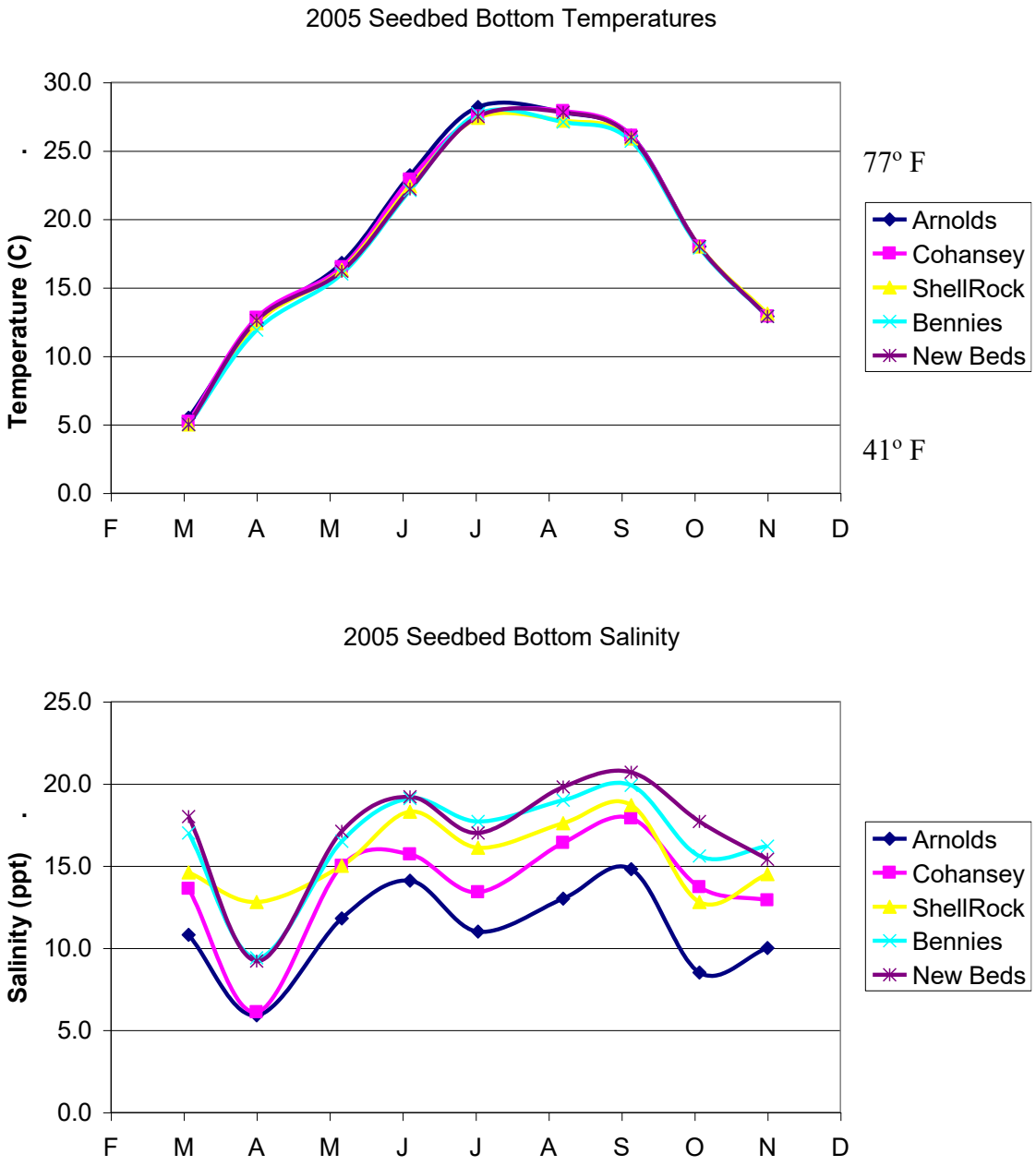

Figure 2. New Jersey Delaware Bay oyster seedbed temperatures and salinities during 2005

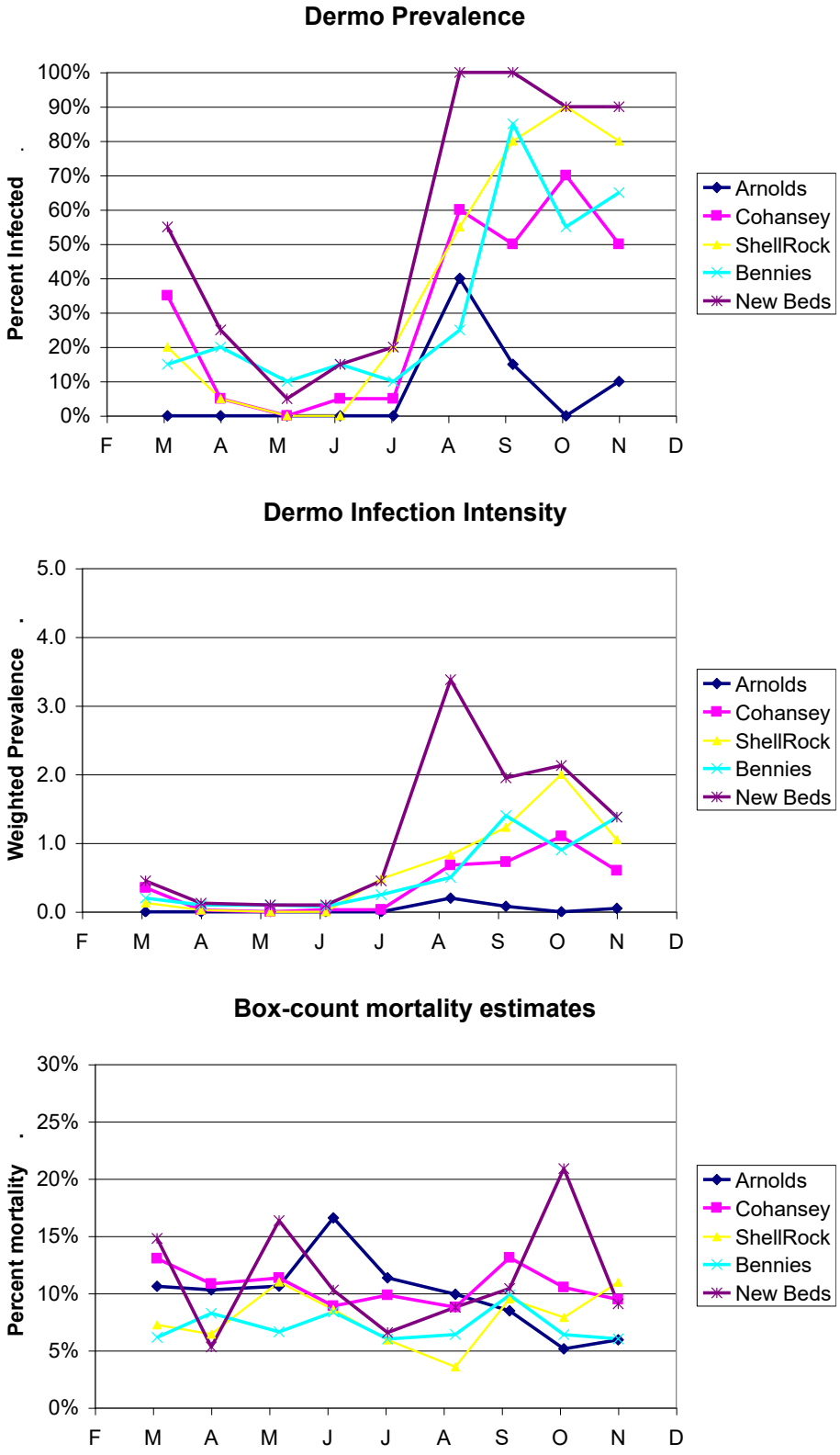

Figure 3. Monthly New Jersey Delaware Bay seedbed dermo disease and mortality data for 2005.

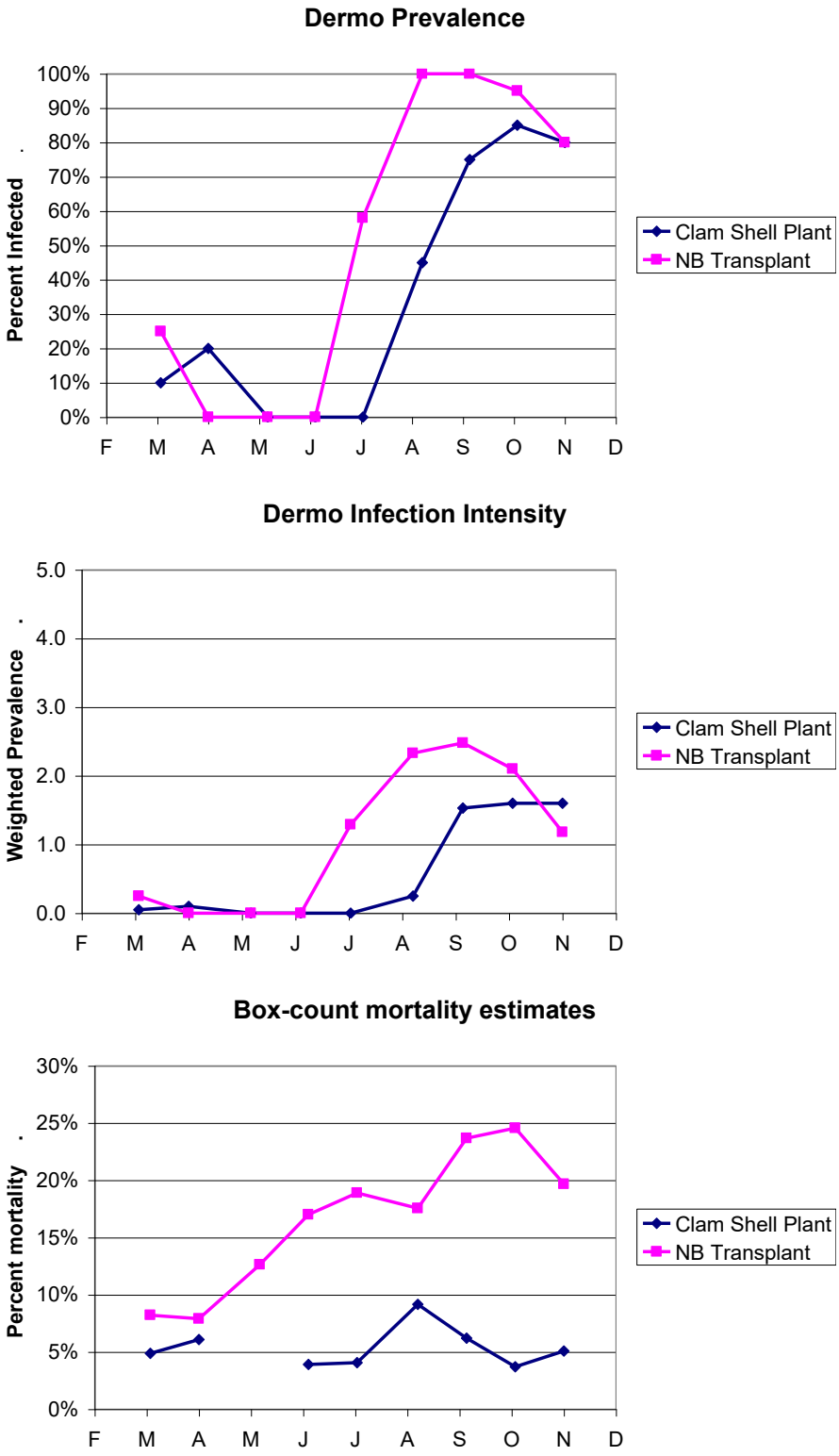

Figure 4. Monthly dermo disease and mortality data for 2005 on two grounds employing different planting strategies described in text.

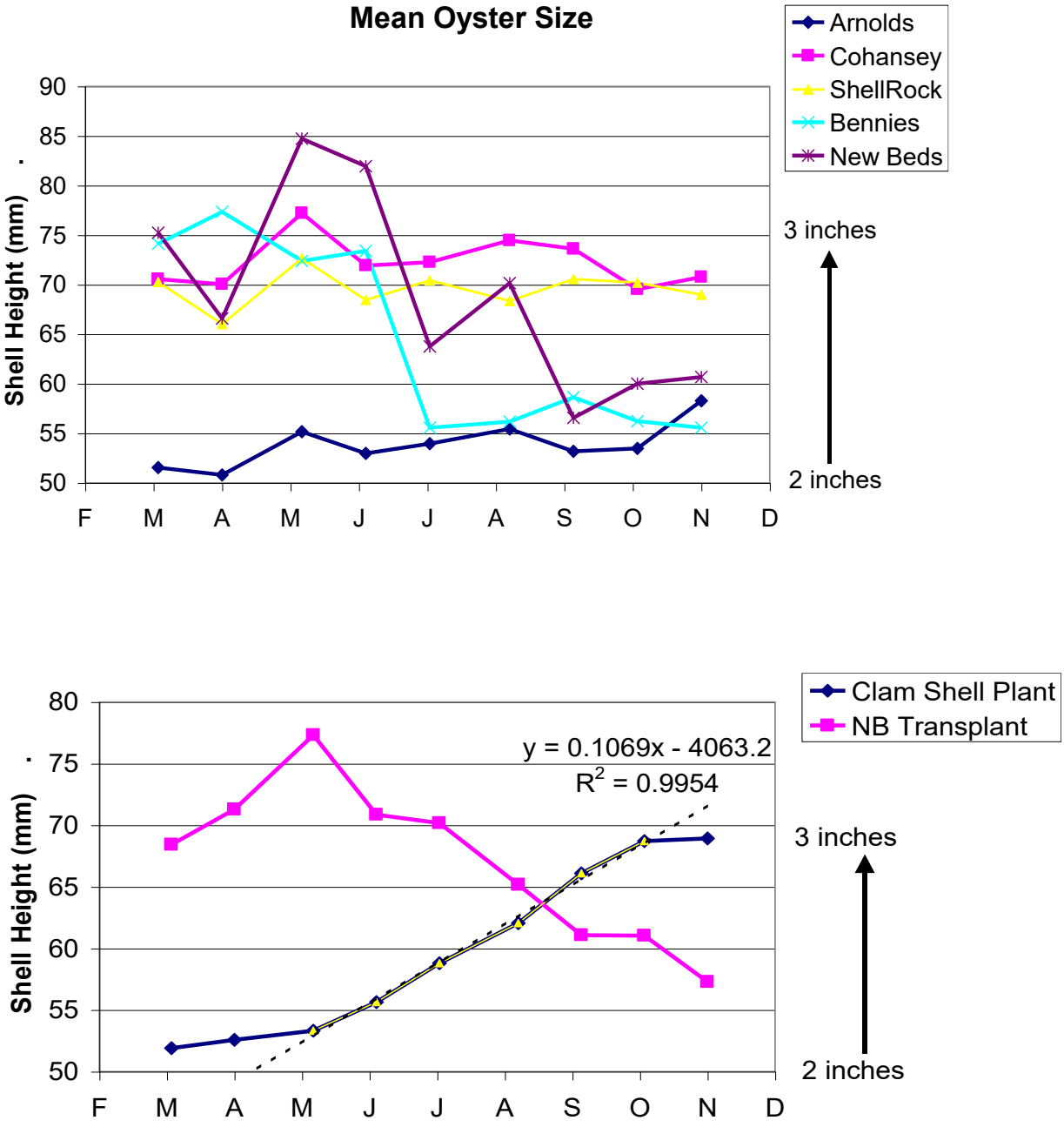

Figure 5. Monthly New Jersey Delaware Bay oyster seedbed mean size data for 2005. Upper panel shows standard monitoring sites. Lower panel shows the 2003 NJDEP clamshell demonstration planting on Bennies Sand and a 2004 transplant from Middle to New Beds. Growth of oysters on the clamshell plant was nearly linear from May to October at a rate of 0.107 mm d^{-1}. The growth rate of these oysters during the previous year was 0.168 mm d^{-1}.

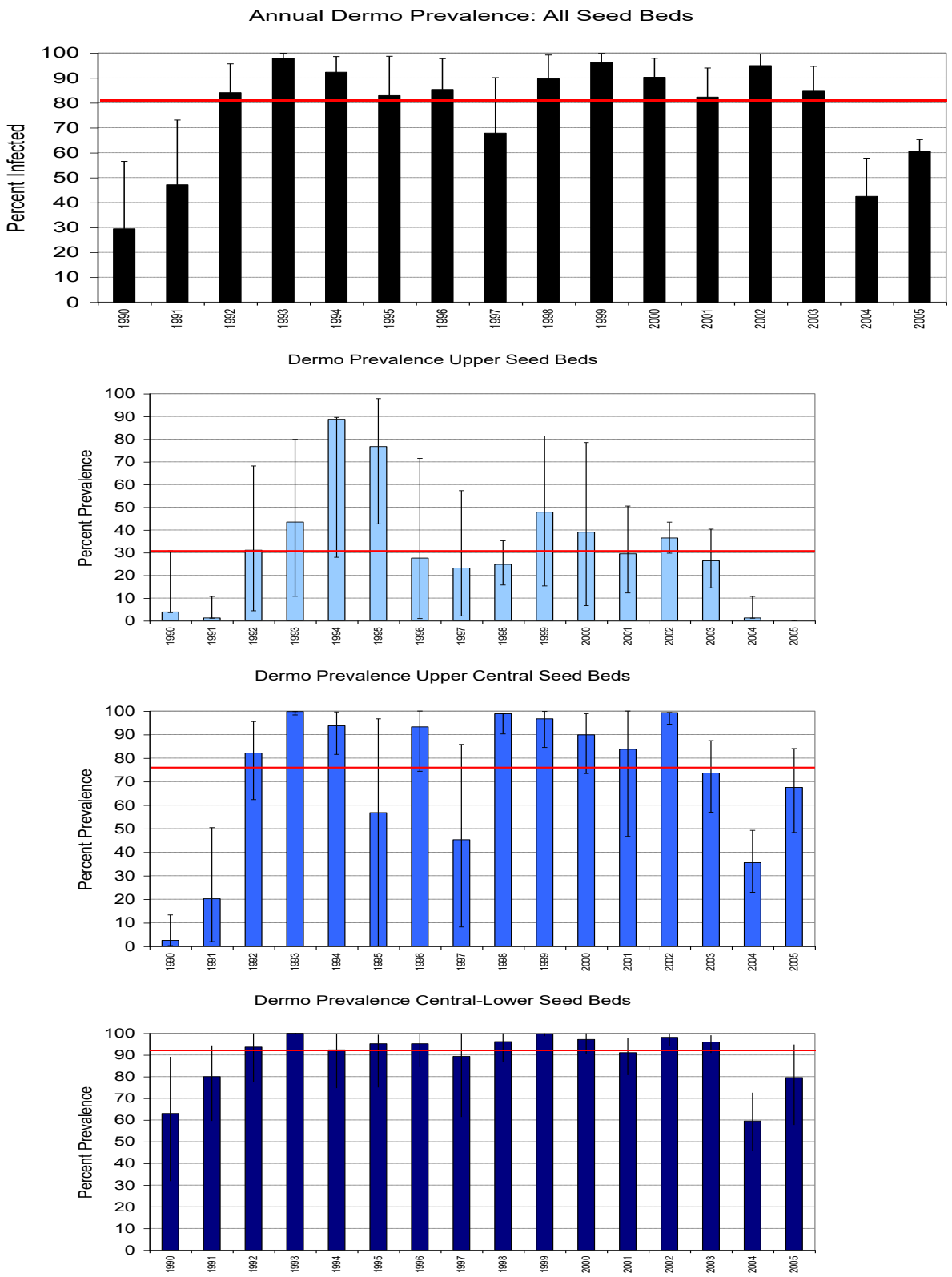

Figure 6. Annual mean fall dermo prevalence on New Jersey Delaware Bay seedbeds. Red line in each plot indicates average of all years.

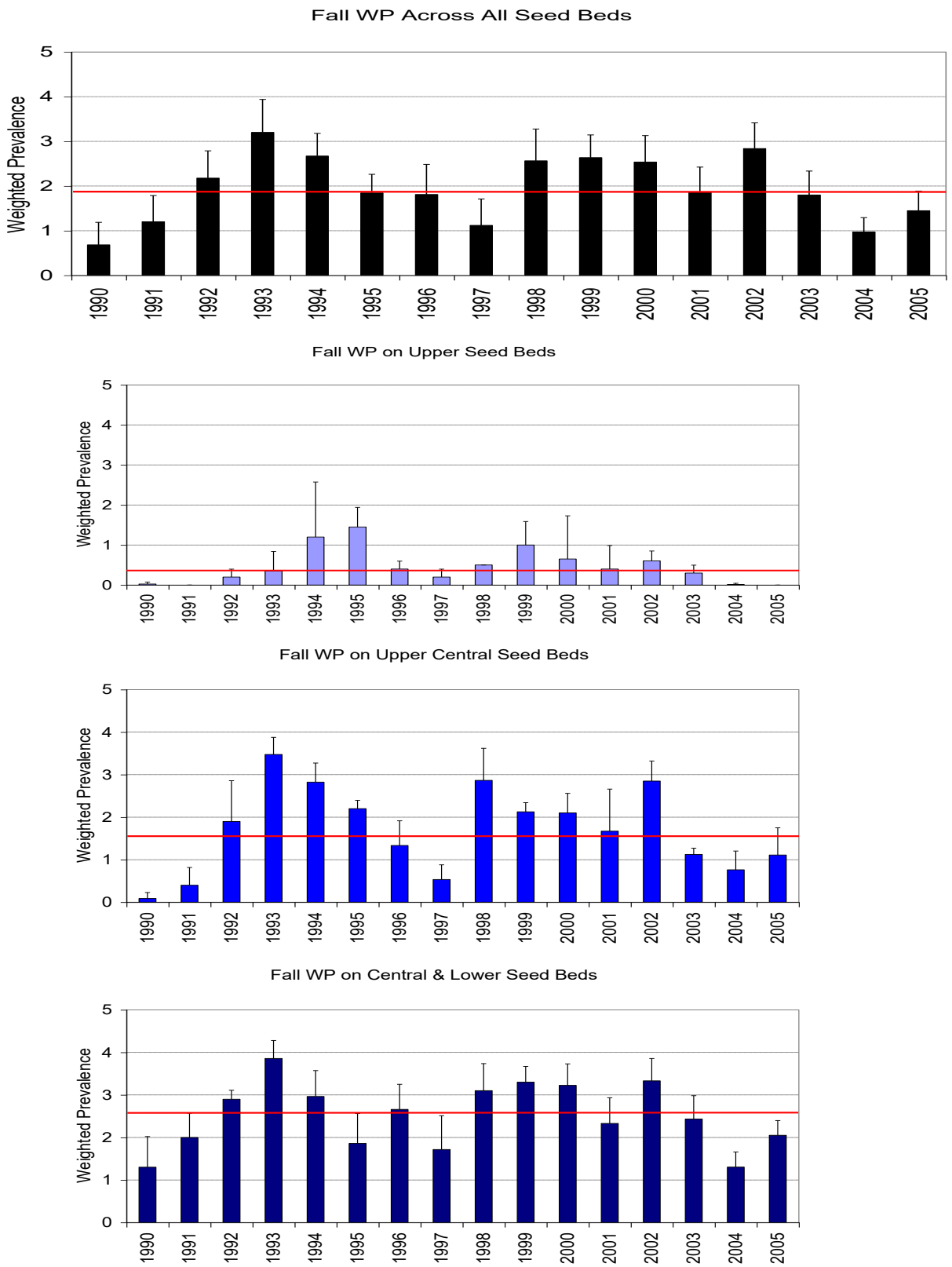

Figure 7. Annual mean fall dermo intensity on New Jersey Delaware Bay seedbeds. Red line in each plot indicates average of all years.

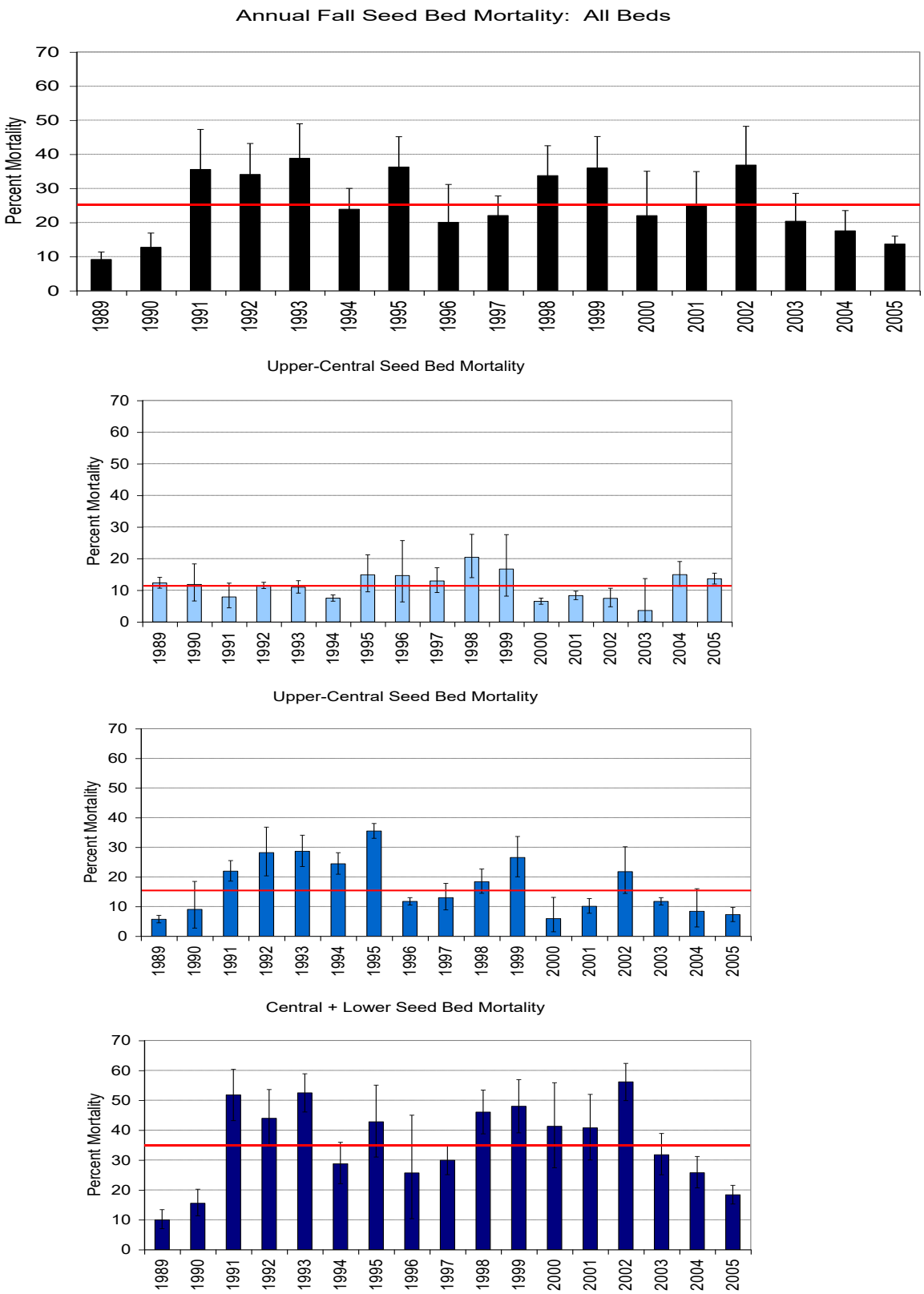

Figure 8. Annual mean fall box-count estimated mortality on New Jersey Delaware Bay Seedbeds. Red line in each plot indicates average of all years.

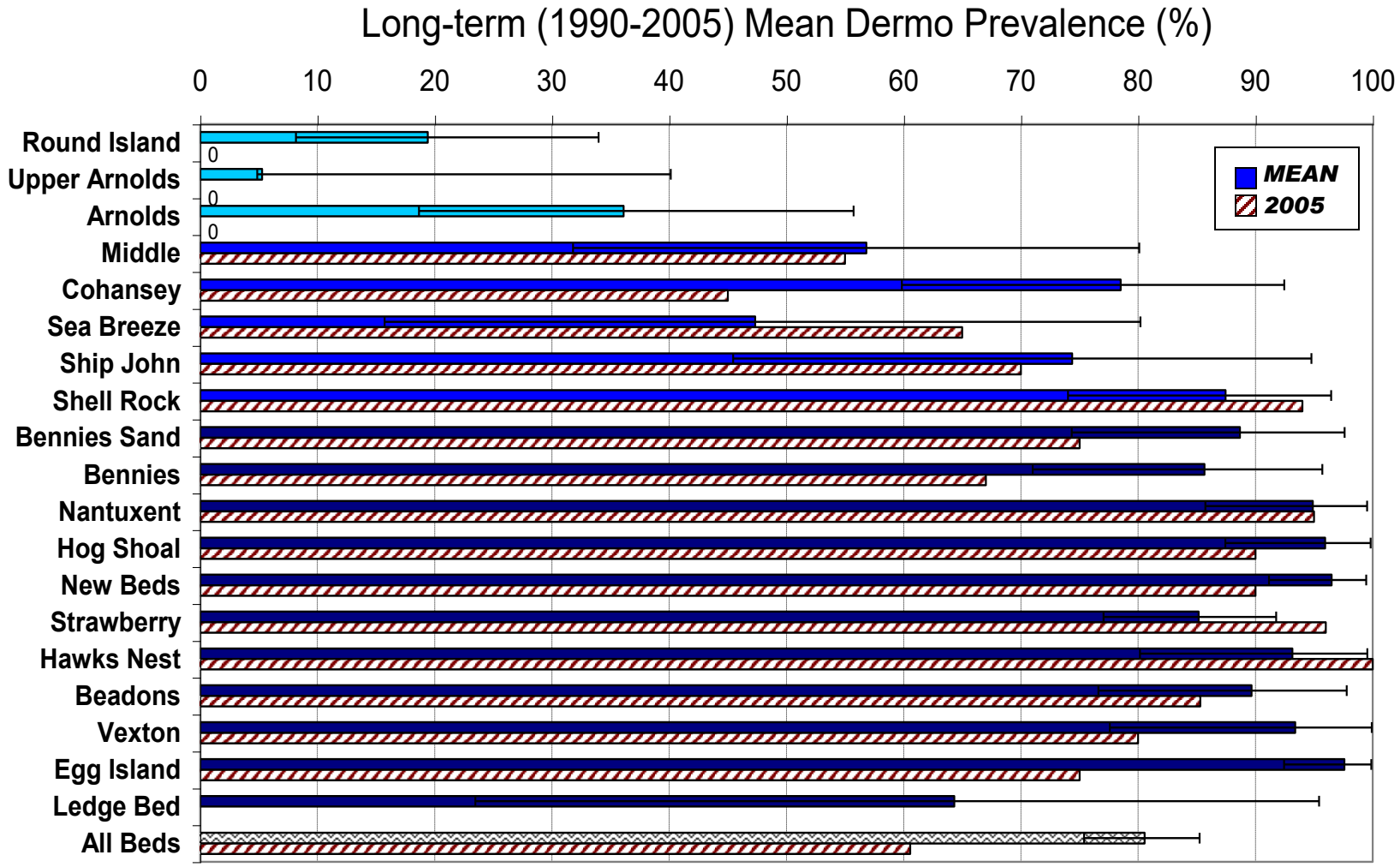

Figure 9. Comparison of average fall *Perkinsus marinus* (dermo) prevalence in oysters on New Jersey Delaware Bay seedbeds since 1990 (solid bars) with 2005 levels (hatched bars). Bar shading grades from light in upper region beds to dark for lower bay beds. Not all beds were sampled every year. Zeros indicate no dermo was detected in Fall 2005.

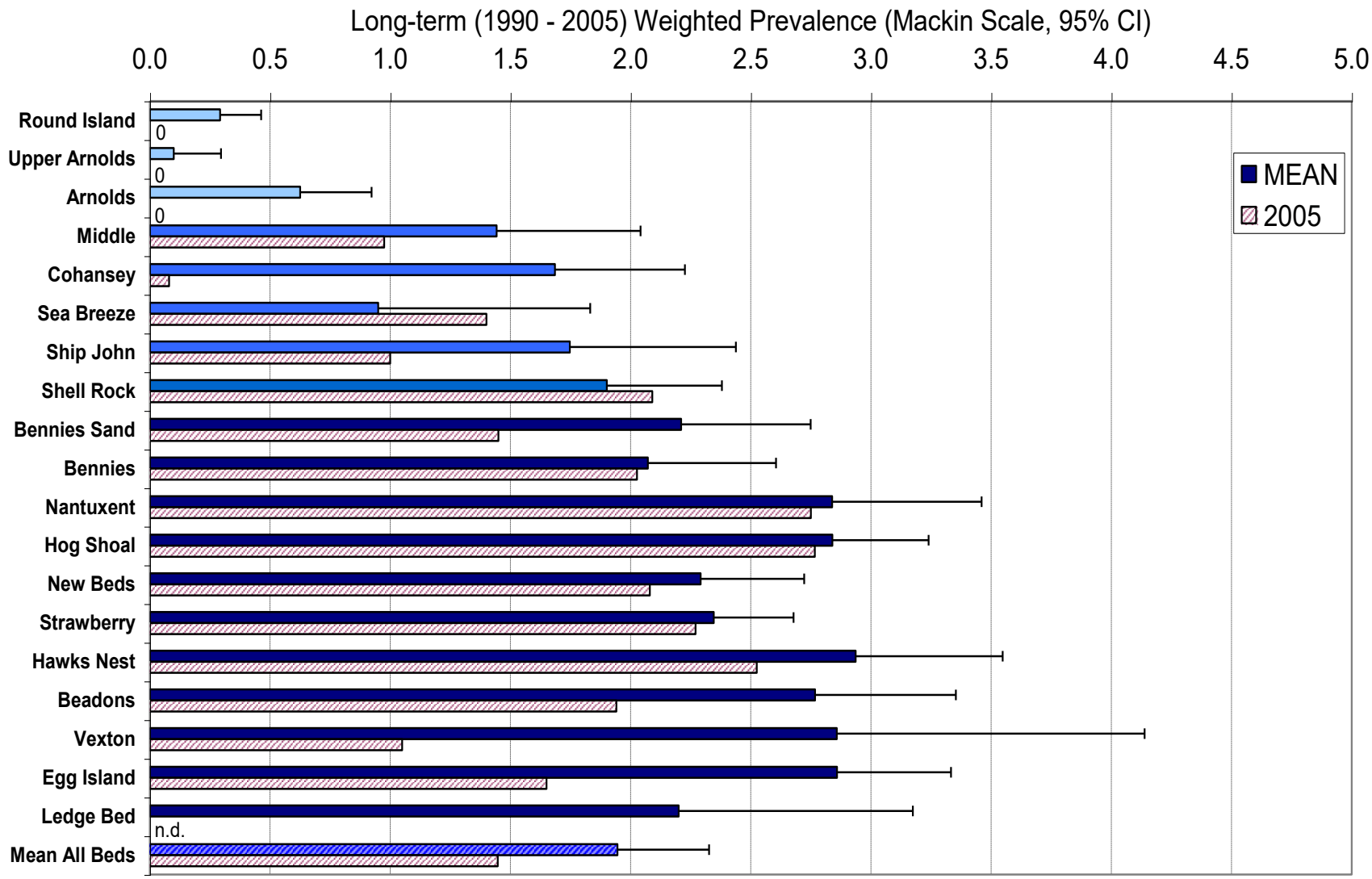

Figure 10. Comparison of average fall dermo infection intensities (weighted prevalence) in oysters on New Jersey Delaware Bay seedbeds since 1990 (solid bars) with 2005 levels (hatched bars). Bar shading grades from light in upper region beds to dark for lower bay beds. N.d. = no data, 0 = no dermo detected.

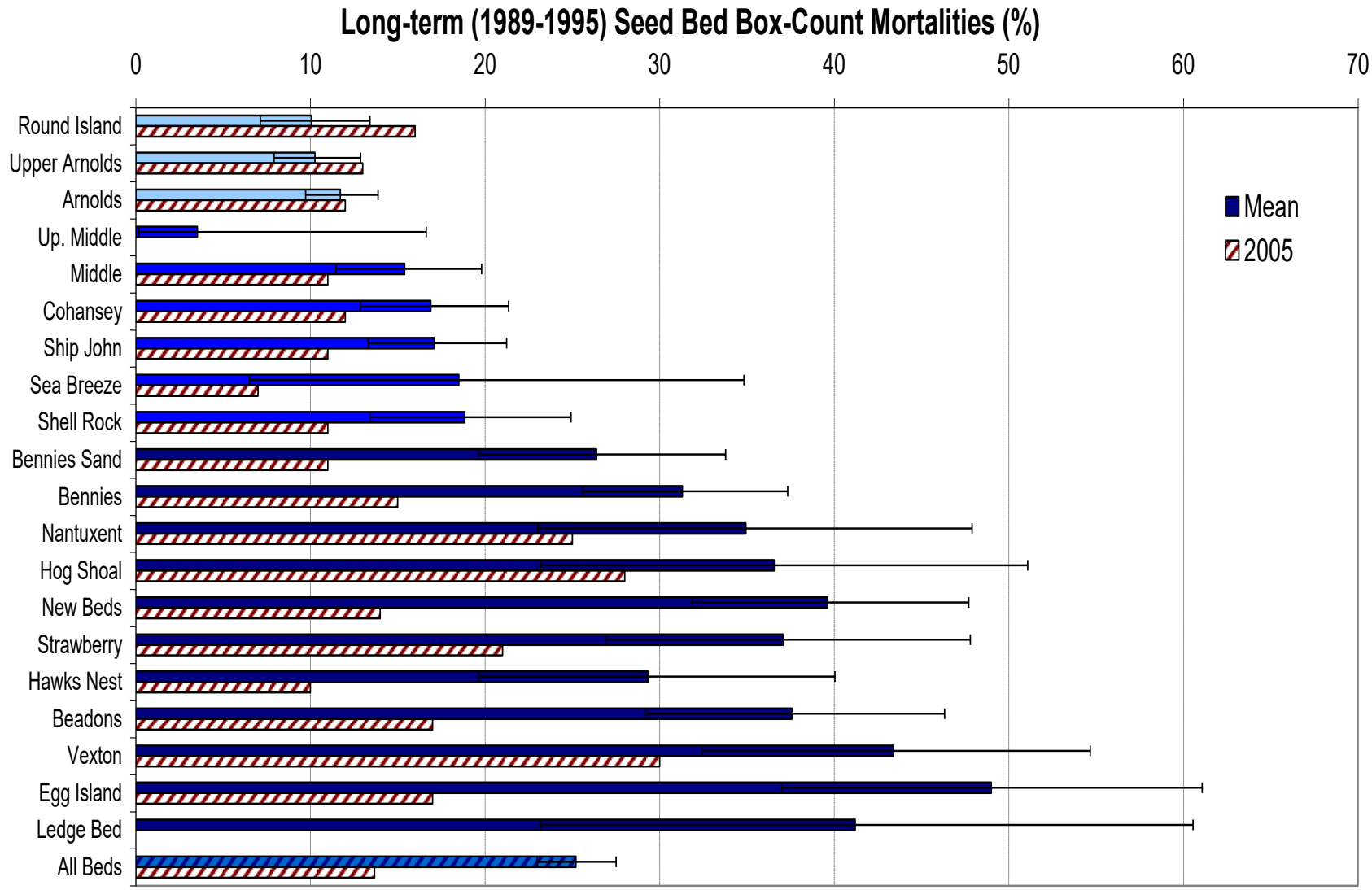

Figure 11. Comparison of average annual fall estimated box-count mortality of oysters on New Jersey Delaware Bay seedbeds since 1989 (solid bars) with 2005 levels (hatched bars). Bar shading grades from light in upper region beds to dark for lower bay beds. Not all seedbeds were sampled every year.

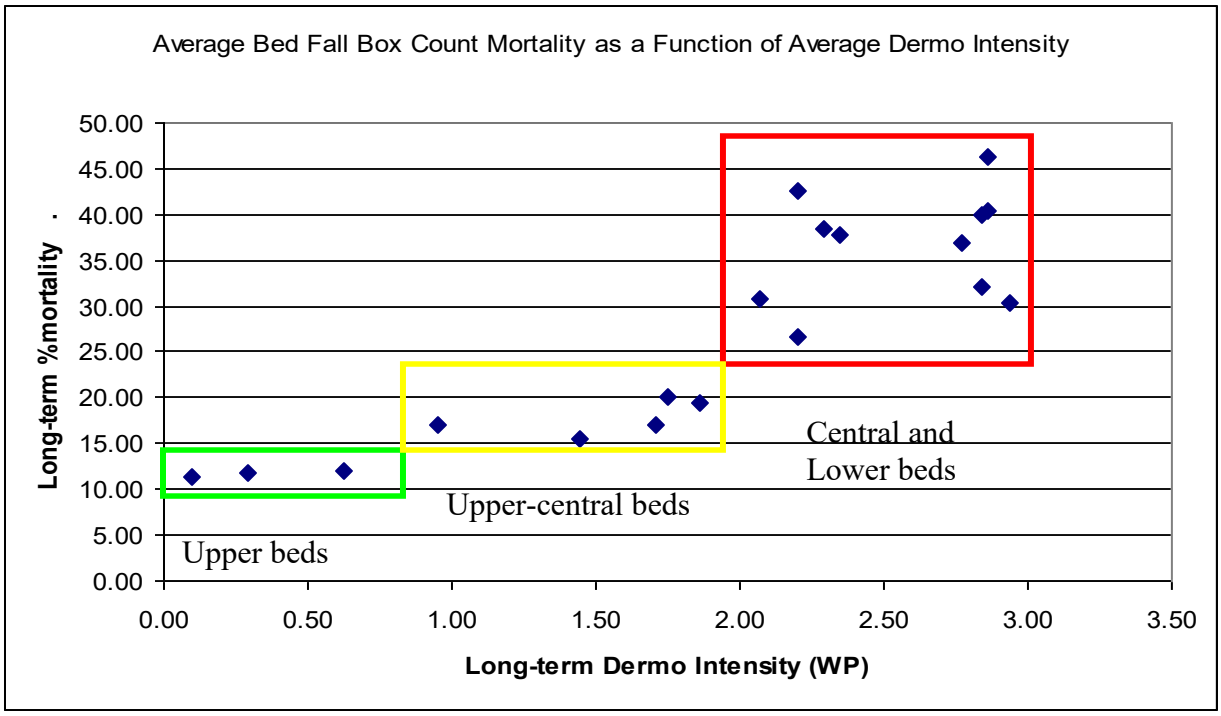

Figure 12. Relationship between long-term mean percent mortality estimate based on fall box-counts and the long-term mean intensity of dermo infections since 1990. Data are individual bed estimates. Note increase in mortality appears to be a step function with thresholds at weighted prevalences of about 1 and 2 on the standard 0-5 Mackin Rank scale.

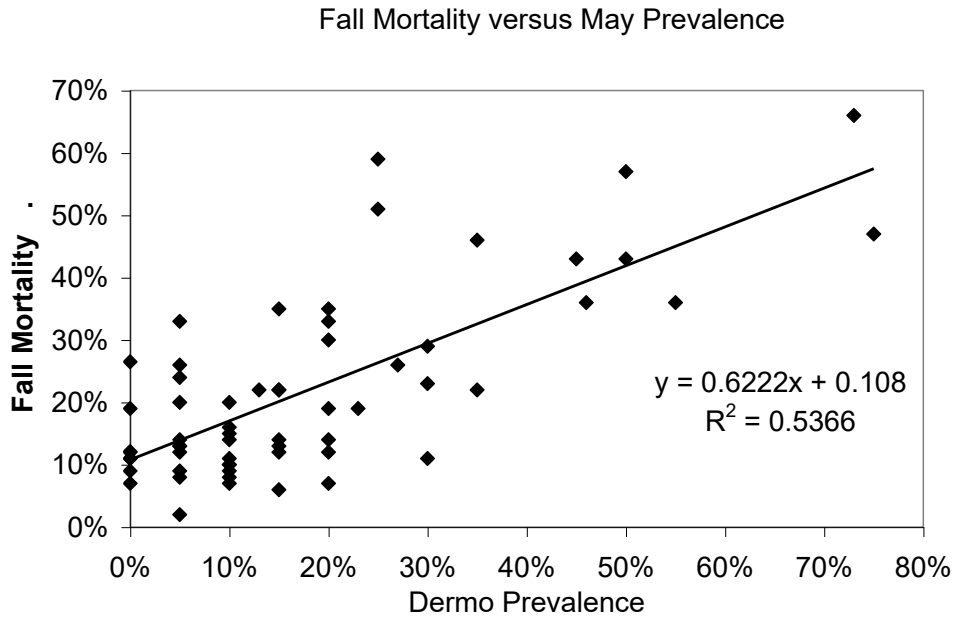

Figure 13. Plot of fall box-count estimated mortality on selected seedbeds against corresponding May dermo prevalence. Linear regression is highly significant ($p < 0.001$), but only explains about half (54%) of the variation.