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EPIZOOTIC SHELL DISEASE IN AMERICAN LOBSTER (*HOMARUS AMERICANUS*) IN MASSACHUSETTS COASTAL WATERS: INTERACTIONS OF TEMPERATURE, MATURITY, AND INTERMOLT DURATION

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A B S T R A C T

An outbreak of shell disease in American lobster (*Homarus americanus*) over the past several years has generated a great deal of attention and concern regarding its causes and spread into New England coastal waters. An examination of the disease within Massachusetts state waters was conducted from 2000 through 2004. The overall average incidence observed in our data was 3% males and 8% females. Female lobsters were more likely to exhibit disease symptoms than males, and larger lobsters were more likely to possess disease symptoms than smaller lobsters. Disease incidence was highest in May and June, immediately prior to the molting period, and decreased dramatically through the molting season. We detected a north to south latitudinal gradient of increasing shell disease prevalence along the Massachusetts coast. Over the course of our study period, we found relatively constant low levels of shell disease in both the Gulf of Maine and Outer Cape Cod regions. However, a significantly higher level of shell disease was recorded in the Buzzards Bay region, indicating important differences in regional prevalence of disease. Our data suggest that this gradient in shell disease may be related to an interaction between water temperature, sexual maturity, and intermolt duration. There is a significant correlation between disease incidence in Buzzards Bay and a series of warmer than average water temperatures from 1999 to 2003, which suggests that temperature may be a primary factor related to the recent outbreak of epizootic shell disease.

INTRODUCTION

An outbreak of shell disease in American lobster (*Homarus americanus*, H. Milne Edwards, 1837) over the past several years has generated a great deal of attention and concern regarding its causes and spread into New England coastal waters. Between 1998 and 2004 shell disease has been observed to varying degrees in lobsters from eastern Long Island Sound to mid-coast Maine. This form of shell disease is characterized by lesions penetrating inwards from the carapace surface. Bacteria are seen at the leading edge of lesions and have been identified as the primary causative organism (Smolowitz et al., 2005). Chistoserdov et al. (2005) have described similar microbial communities in lesions of lobsters from different locations, and several investigators have suggested that the bacterial activity may be interacting with environmental factors (Chistoserdov et al., 2005; O'Kelly, 2005; Shiaris, 2005; Smolowitz et al., 2005). The high prevalence of disease symptoms observed in some regions, and the wide scale geographic distribution of disease symptoms has led researchers to label this disease as epizootic.

The Massachusetts Division of Marine Fisheries (MADMF) has observed shell disease in the catches of commercial lobstermen since the inception of its coastwide lobster trap sampling program in 1981. Estrella (1984, 1991) characterized shell disease in two separate studies and reported a gradient in Massachusetts coastal waters from north to south of increasing prevalence and severity. In both of these earlier studies the disease symptoms were described as localized pitting and light to moderate shell erosion.

In the late 1990s, commercial lobstermen started reporting a high incidence of lobsters with heavily eroded shells in southern Massachusetts waters. This recent outbreak of disease spread northwards in subsequent years reaching the northern most portion of the Massachusetts coast by the summer of 2003 (Glenn and Pugh, 2005).

With growing reports of disease throughout southern New England waters, MADMF initiated a standardized shell disease sampling protocol in the winter of 2000. Using this protocol, we examined shell disease in Massachusetts coastal waters from 2000 to 2004. In this paper we report on the incidence and severity of shell disease, and relate disease patterns to environmental variables and lobster life history.

METHODS

Sampling for shell disease took place during regular commercial lobster trap sampling trips conducted by MADMF staff. These trips were conducted bi-monthly in six regions (Fig. 1), from May through November. Standard sea sampling protocol involved recording the following attributes of the catch: number of lobsters, number of trap hauls, soak time, trap and bait type, carapace length (to the nearest mm), sex, shell hardness, culls and other shell damage, external gross pathology, mortality, and presence of extruded ova on females. The sampling location was recorded using LORAN or GPS. Shell disease sampling entailed inspecting the shell of all lobsters from trawls chosen haphazardly throughout the course of a sampling trip, until a total of at least 50 lobsters were examined per trip. Each lobster was categorized by an index based on the percent coverage of shell disease symptoms on the total surface area of the shell (all dorsal and ventral surfaces). Shell disease symptoms included pitting, shell erosion, and ulceration. The categories were broad to help reduce the subjectivity in assigning an index. Four categories of shell coverage were defined: 0% = No shell disease symptoms; Low = 1-10%; Moderate = 11-50%; Severe = 51-100%. Shell disease data were analyzed spatially and temporally with respect to water temperature and life history parameters. Females and males showed similar spatial and temporal trends, as such, sexes were pooled for analysis.

Temperature data were obtained from the National Oceanic and Atmospheric Administration's (NOAA) sea-surface temperature time series, and the MADMF bottom water temperature monitoring program. To gauge the cumulative effect of temperature on a lobster over the course of a year, we calculated the number of days the water temperature was above a threshold within each year. We chose 20°C as the threshold representative of the upper temperature range lobsters typically prefer

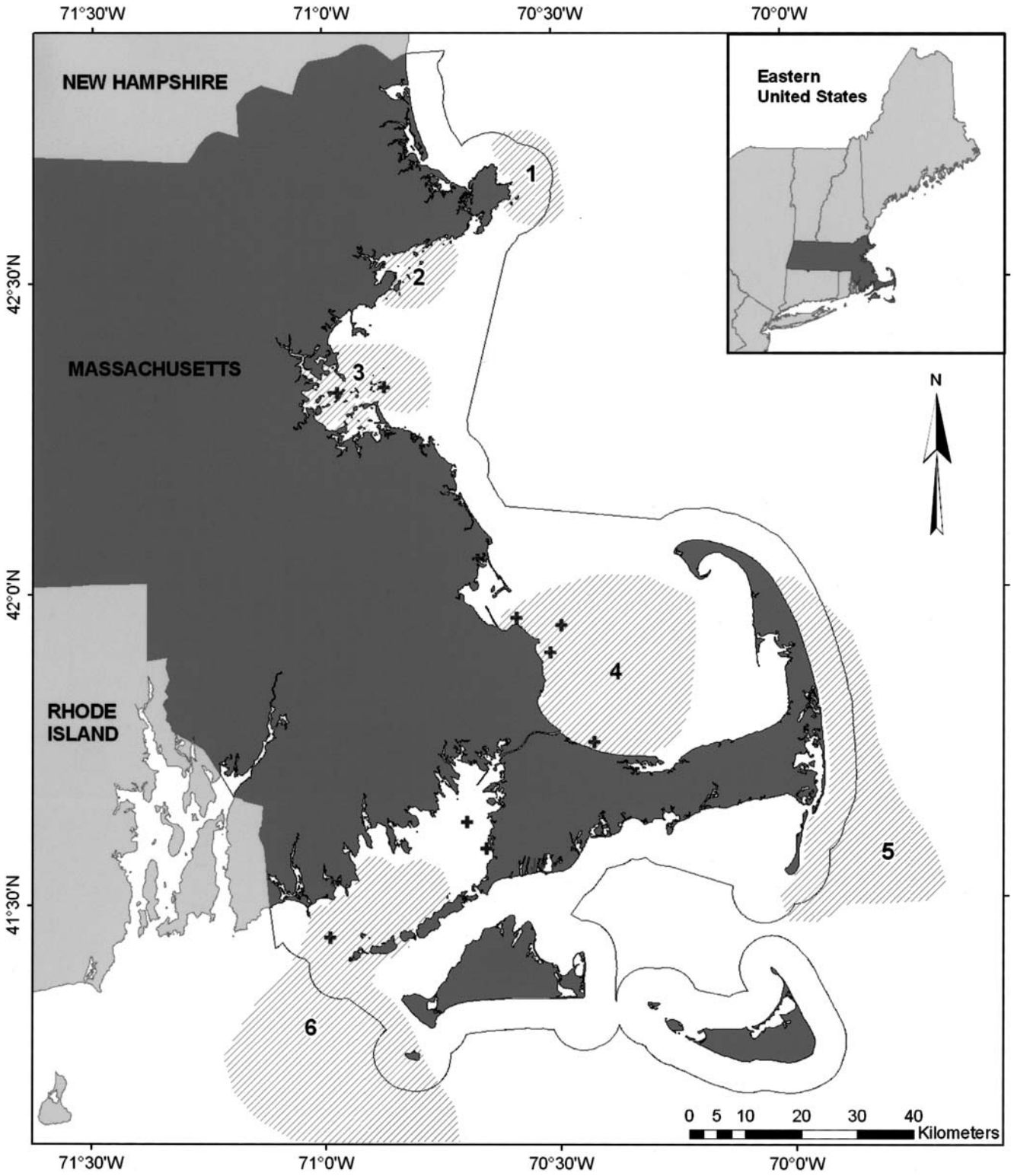

Fig. 1. Map of coastal Massachusetts. Hashed areas represent sampling areas covered by Marine Fisheries Commercial Lobster Trap Sampling Program. Sampling regions are; 1) Cape Ann, 2) Beverly/Salem, 3) Boston Harbor, 4) Cape Cod Bay, 5) Outer Cape Cod, 6) Buzzards Bay. Crosses represent locations of bottom water temperature monitors.

(Aiken and Waddy, 1986). The number of days above 20°C was calculated for each year and then subtracted from the time series mean to develop an annual deviation from the average number of days above 20°C.

The NOAA sea-surface temperature time series from Woods Hole, MA dates back to 1945. This time series was examined in relation to the threshold temperature in order to identify any potential anomalies in environmental conditions.

We used a time series of bottom water temperature collected by MADMF in Buzzards Bay to examine the relationship between shell disease incidence and temperature. These data were collected bi-hourly from a temperature

Table 1. Percent incidence of shell disease by symptom category in Massachusetts coastal waters.

	2000	2001	2002	2003	2004	Series mean
No Symptoms						
female	94.0	91.2	87.3	92.2	96.7	92.6
male	98.8	96.4	95.6	95.9	98.0	96.9
Low						
female	4.1	4.0	5.7	4.1	2.1	3.9
male	0.2	1.6	1.5	1.7	1.0	1.3
Moderate						
female	1.5	2.7	4.4	2.0	0.8	2.2
male	0.4	1.0	1.2	1.3	0.3	0.8
Severe						
female	0.3	2.1	2.5	1.7	0.4	1.4
male	0.6	1.1	1.6	1.2	0.6	1.0

logger that was deployed at 18.3 meters. Bottom water is more appropriate to relate to lobster life history parameters than surface water, because seasonal stratification of the water column causes a difference between surface and bottom temperatures. Disease incidence and temperature were analyzed by applying a regression to the number of days above 20°C in year (t) and disease incidence in the following year (t + 1).

Results

Sex-specific Differences

From May 2000 to November 2004 a total of 21,089 lobsters were sampled for shell disease in Massachusetts waters (Fig. 1). Female lobsters were more likely to have shell disease symptoms than males. Three percent of the males and 8% of the females showed symptoms of the disease. Egg-bearing female lobsters were 7.5 times more likely to have shell disease than females without eggs. Of the lobsters that did have shell disease, half had low symptoms (50%), the rest had moderate (29%) or severe (21%) symptoms. These sex-specific patterns were similar across all years (Table 1).

Spatial Patterns

Shell disease prevalence increased from north to south along the Massachusetts coast (Fig. 2). In Cape Ann, our northern-most sampling region, disease prevalence was very low, it increased slightly toward the south, dropped substantially in Outer Cape Cod, and increased sharply in our southern-most region Buzzards Bay. Regional annual mean bottom water temperature along the Massachusetts coast also increased toward the south (Fig. 3).

Temporal Patterns

The prevalence of shell disease by Massachusetts coastal region from 2000 to 2004 is depicted in Fig. 4. The incidence of shell disease in Outer Cape Cod and the three northern most regions, Cape Ann, Beverly/Salem, and Boston Harbor, remained below 5% throughout the entire 5-year period. The Cape Cod Bay region had moderate levels of shell disease in 2000 and 2001 (11%), but steadily declined through 2004. In contrast, Buzzards Bay had a 9% incidence of shell disease in 2000, increased to a high of 28% in 2003, and then dropped sharply to 12% in 2004.

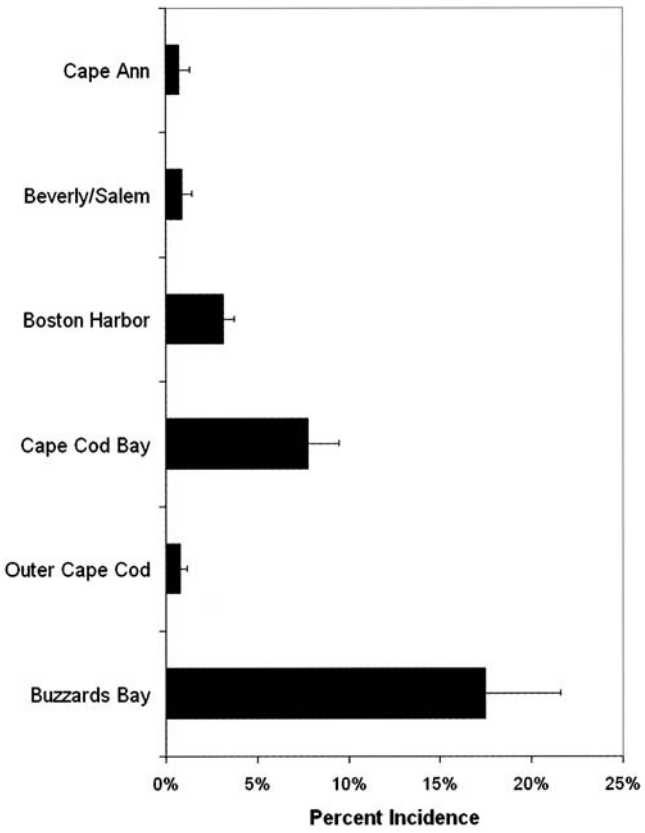

Fig. 2. Mean incidence (± SE) of lobster shell disease in Massachusetts coastal waters, 2000 to 2004.

The onset of shell disease in the late 1990s in southern Massachusetts corresponds to a period of warmer than average years. Historically, Woods Hole, MA has experienced an average of 73 days a year with surface water temperatures warmer than 20°C (1945-2004 records; http://co-ops.nos.noaa.gov). The deviations from this mean have fluctuated from positive to negative over the course of the time series until 1997, when a run of eight consecutive above average years occurred (Fig. 5). At no other period in this time series has there been a consecutive string of positive deviations of this duration. This time period corresponds to the shell disease outbreak in southern New England. In Buzzards Bay, the regions most affected by the disease, the prevalence of shell disease is significantly related to the number of days above 20°C in the previous year (Fig. 6; $R^2 = 0.79$, $P = 0.04$). These data should be interpreted cautiously because of the short time series for which we have overlapping temperature and shell disease observations. Nonetheless, they suggest that temporal trends in shell disease prevalence may be strongly related to temperature.

Discussion

Although bacteria have been implicated as the proximate cause of epizootic shell disease, the reason for the recent outbreak remains unclear. It appears that the bacteria found on the leading edge of lobster shell erosions both north and south of Cape Cod are identical, and that these bacteria

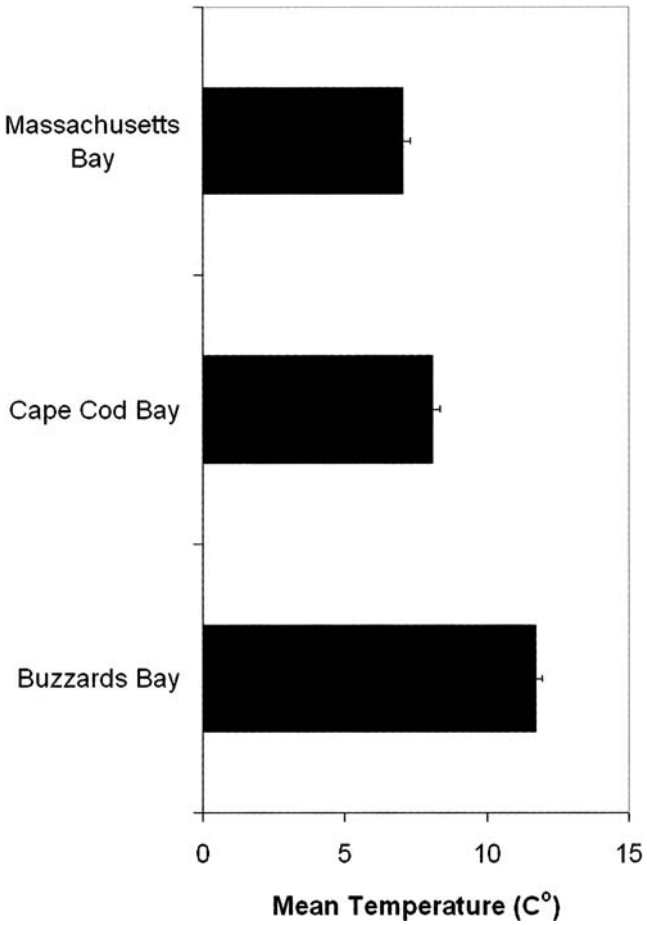

Fig. 3. Annual mean bottom water temperature (± SE) for the period 1998 to 2003 in three Massachusetts regions (18.3 meter depth strata). Temperatures measured bi-hourly with underwater loggers.

occur at background levels throughout the region (Smolowitz et al., 2003). The lack of geographical differences in presence and abundance of these bacteria suggest that the spatial and temporal patterns in shell disease prevalence are influenced by environmental factors.

The patterns of shell disease prevalence reported here are not consistent with the hypothesis that pollution is the primary factor affecting the occurrence of shell disease. Boston Harbor, which is one of the country's most polluted urban embayments, has maintained a very low prevalence of shell disease throughout the time series. Conversely, the area around the Elizabeth Islands in the southern portion of Buzzards Bay is not adjacent to any major population or industrial centers, nor is it immediately down-current of such influences, yet this region has consistently had the highest prevalence of shell disease within Massachusetts coastal waters. Similarly, Howell et al. (2005) reported lower incidences of shell disease in the more polluted waters of Western Long Island Sound and higher incidences of disease in the less polluted Eastern Long Island Sound. While we cannot rule out the role of anthropogenic influences on shell disease, the absence of a spatial link between major population centers is not consistent with the pollution hypothesis. More testing is necessary to determine if pollution plays a role in shell disease occurrence.

Our data are more consistent with the hypothesis that shell disease is linked to periods and locations of elevated water temperature, and that it is manifested most in lobsters with longer intermolt periods. Our data suggest that the spatial and temporal patterns of shell disease prevalence may be related to the interaction of temperature, sexual maturity, and intermolt duration.

Shell disease prevalence in Massachusetts increases dramatically from north to south. A comparison of shell disease prevalence data from Massachusetts coastal waters in 1983 and 1989 reveals a similar pattern (Estrella, 1984, 1991). The similarity in spatial patterns over time, in combination with similar microbial communities in lesions on lobsters both north and south of Cape Cod (Smolowitz et al., 2003; Chistoserdov et al., 2005), suggest environmental influence on the observed spatial pattern.

Cape Cod serves as a distinct geographic barrier between the cold waters of the Gulf of Maine and the warmer waters of southern New England. As such a north to south latitudinal gradient in bottom water temperature occurs similar to the pattern observed in shell disease along the Massachusetts coast. A bottom water temperature time series is not available for Outer Cape Cod, however this area is known to be a cold water environment dominated by mixing currents from the Gulf of Maine and Georges Bank.

The apparent influence of temperature on the geographic patterns of shell disease prevalence leads us to propose two mechanisms through which temperature may act independently or synergistically on disease prevalence.

Mechanism 1: It is possible that the cold water environments north and east of Cape Cod retard the growth of the bacteria suspected to cause shell disease. Conversely, the warmer waters south of Cape Cod may foster bacterial growth. Sustained periods of warmer than average temperatures may have promoted favorable conditions for disease-causing bacteria. Continued research is needed to examine the relationship between bacterial growth and water temperature.

Mechanism 2: Temperature has a strong influence on the growth and reproductive cycles of lobsters (Waddy et al., 1995), thus impacting the length of intermolt duration and subsequent exposure to disease-causing agents. Lobsters in warmer waters reach sexual maturity at a much smaller size than those in colder waters (Aiken, 1980). Estrella and McKiernan (1985) showed regional differences in size at sexual maturity along the Massachusetts coast. At Cape Ann, the northern most region, female size at 50% sexual maturity is 90 mm carapace length (CL), versus 76 mm in the southern most region, Buzzards Bay. Once female lobsters attain sexual maturity the average intermolt duration increases significantly (Aiken, 1980). The average commercial sized female lobster in southern New England retains its shell for approximately 175 more days than a similar sized female lobster in the Gulf of Maine (ASMFC, 2000). This would, in turn, increase their length of exposure to disease-causing agents.

We believe these two mechanisms explain the timing and geographic pattern of shell disease prevalence observed in recent years along the Massachusetts coast. In addition to affecting regional patterns of shell disease prevalence, water

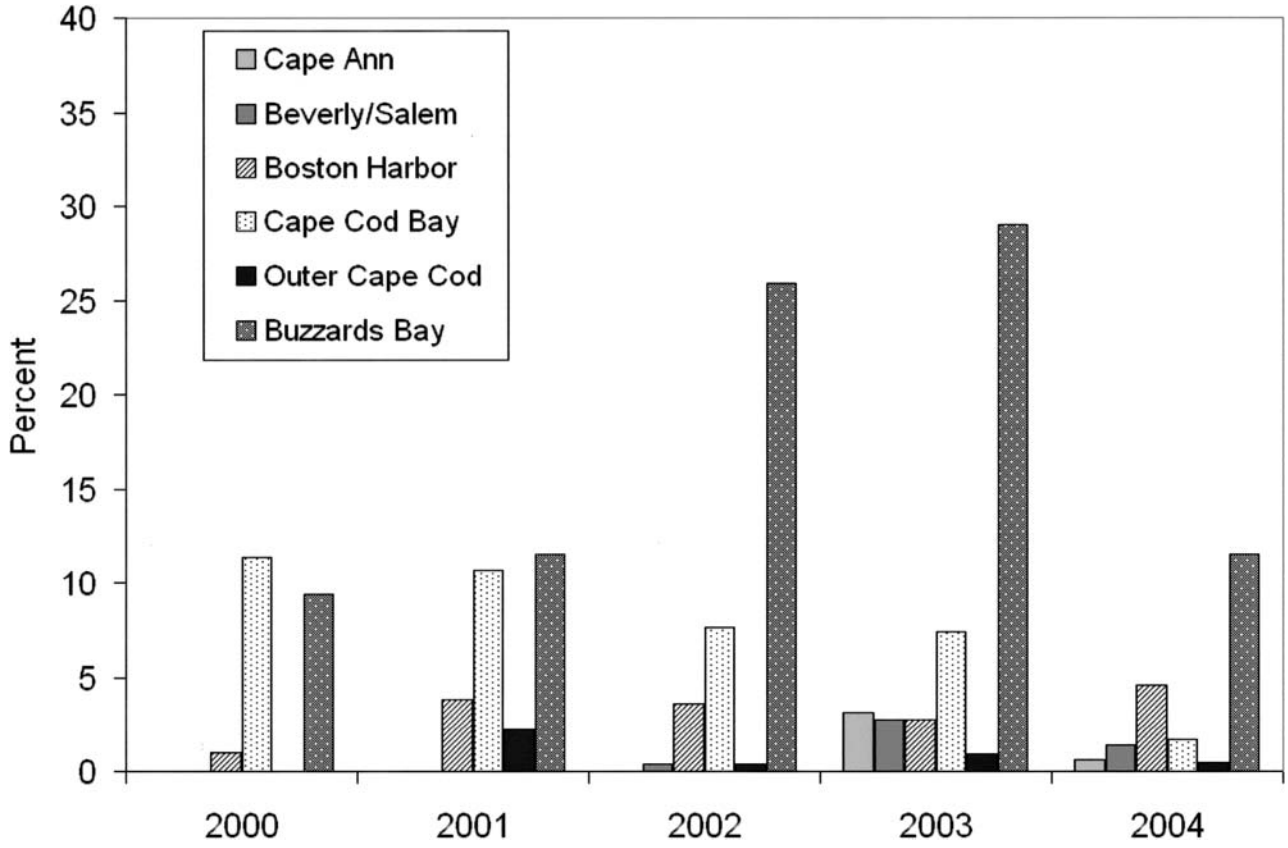

Fig 4. Prevalence of shell disease in six Massachusetts coastal regions, 2000 to 2004. Regions arranged north to south.

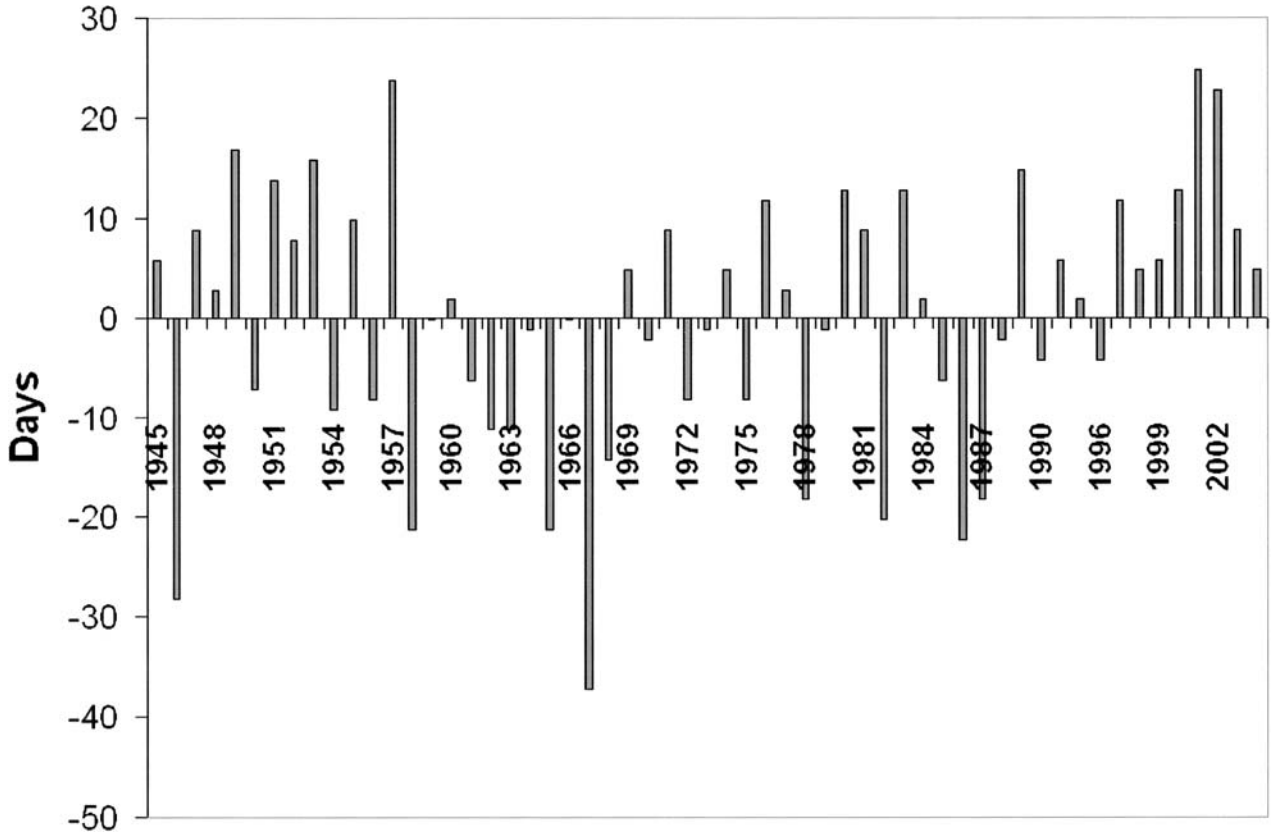

Fig. 5. Annual deviations from the time series mean number of days above 20°C. Time series represents sea-surface temperatures in Woods Hole, Massachusetts from 1945 to 2004. Data from the years 1993 through 1995 were not included due to incomplete data sets.

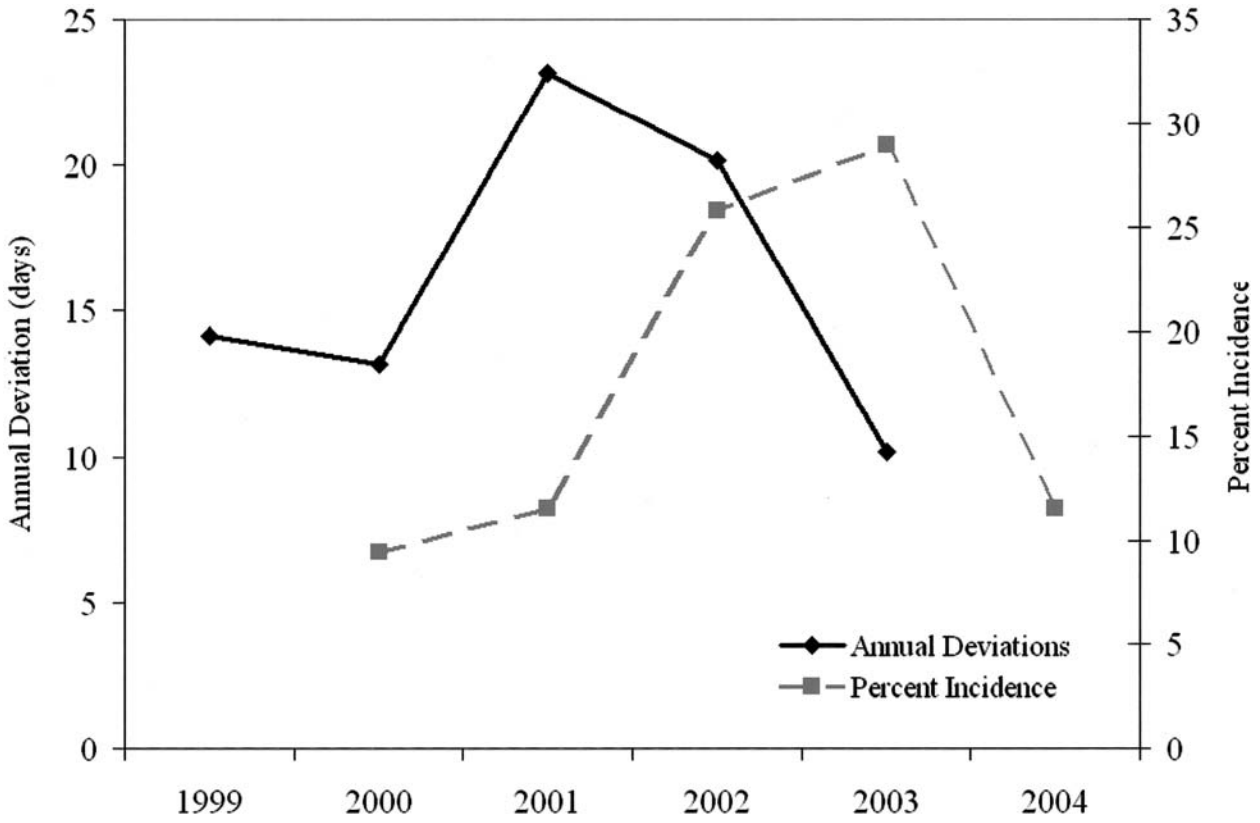

Fig. 6. Annual deviations from the time series mean number of days above 20°C from bottom water temperatures (30 ft.) in Buzzards Bay, Massachusetts, and percent incidence of lobster shell disease in Buzzards Bay.

temperature appears to have a strong influence on the observed temporal patterns of the recent outbreak. We propose that our data illustrate a lag in the relationship between water temperature and disease incidence, due to the correspondence of warm summer water temperatures and the development over time of disease symptoms. The visible symptoms of shell disease, pitting and erosion of the carapace, accumulate during the intermolt period. Recall that disease incidence is highest in May and June, before the major molt occurs, and lowest in August after most lobsters have molted into new, ''clean'' shells. If an increase in the number of days above 20°C is acting as an environmental stimulus for the development of shell disease, one would expect the summer water temperature in a given year to be related to the prevalence of shell disease in the following year, prior to the molt. The strong relationship we have demonstrated with our data supports this hypothesis.

Lobsters at temperatures greater than 20°C are subject to greater physiological stress. Laboratory studies have demonstrated increased oxygen consumption in lobsters exposed to temperatures $\geq$ 20°C for extended periods of time (Powers et al., 2004). Crossin et al. (1998) demonstrated that lobsters behaviorally thermoregulate and actively avoid waters warmer than 19°C. In a Long Island Sound experiment, lobsters v-notched and held in water warmer than 20°C died at a higher rate that non-notched lobsters, and at a higher rate than lobsters notched and held at temperatures less than 18°C (Howell et al., 2005). Results from these studies and others (McLeese, 1956; McLeese and Wilder, 1958) suggest that prolonged exposure to warm water ($\geq$ 20°C) exerts physiological stress on American lobster which could lead to increased susceptibility to disease. Exposure to warm water has also been linked to elevated mortality in lobsters due to other diseases, such as gaffkemia (Stewart et al., 1969) and excretory calcinosis (Dove et al., 2004). The direct link between shell disease and warmer temperatures is less conclusive, but appears to be related to how temperature affects the onset of maturity and the intermolt duration in lobsters.

Conclusions

In conclusion, while the precise mechanism behind infection remains unclear, our data suggest that temperature and time of exposure are likely to be key factors in lobster shell disease. The prevalence of symptoms as they vary by size, gender, and geographical location appear to be linked to the influence temperature exerts on growth and sexual maturity. The increasing trend in shell disease in the waters of southern New England appears to be related to a period of warmer water in the region. We also hypothesize that the greater prevalence among mature females is related to their tendency to have a longer intermolt period than males of the same size.

We have presented an argument linking warmer water temperatures to the prevalence and spatial distribution of shell disease in Massachusetts waters. Further empirical testing should be conducted in order to verify these hypotheses. Possible avenues for further exploration into

the causative mechanisms and effects of lobster shell disease include the mechanisms of infection, pathogenicity of bacteria, temperature as it relates to infection and symptom progression, and ramifications of the disease with regards to behavior and mortality.

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