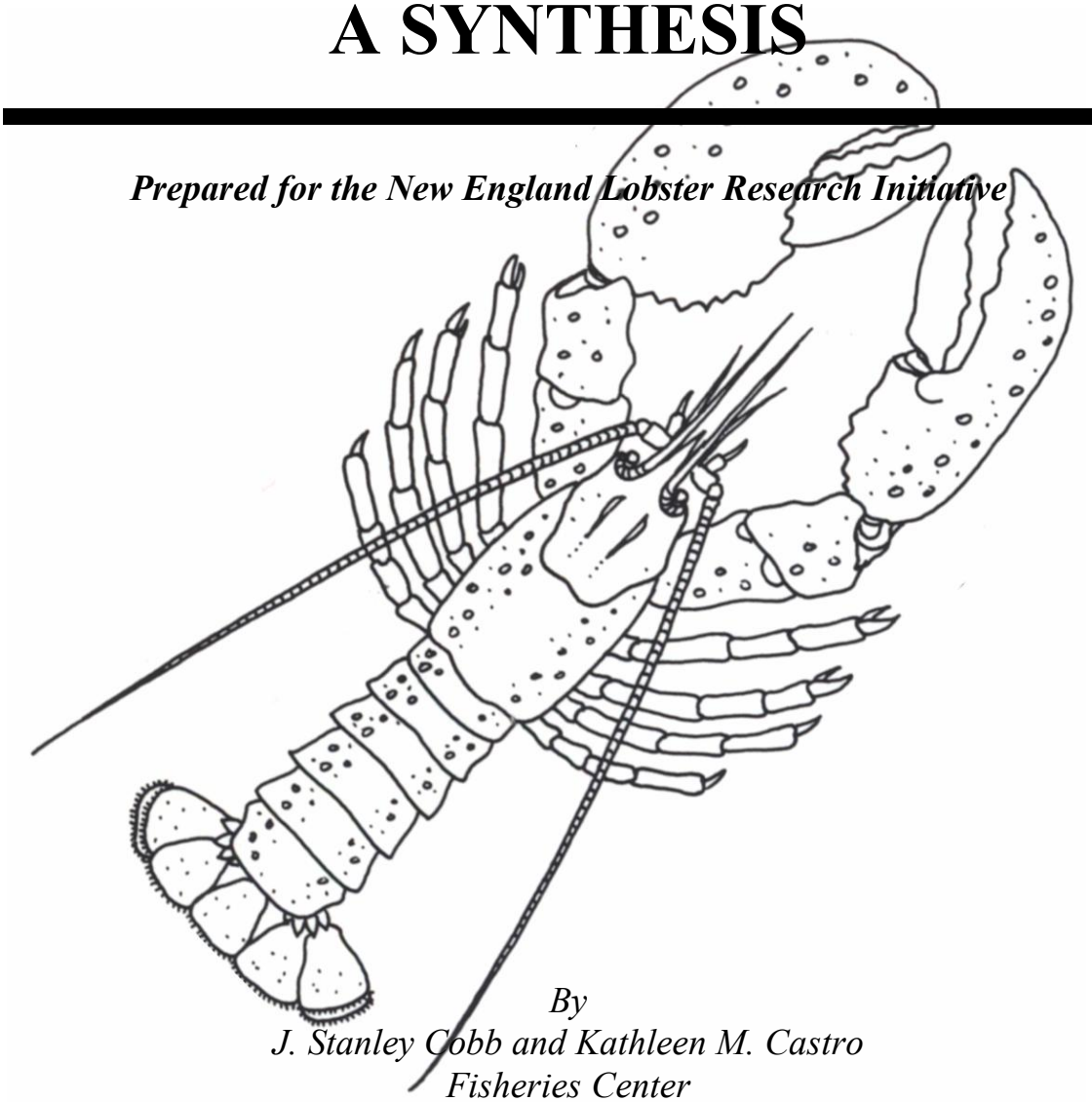
SHELL DISEASE IN LOBSTERS: A SYNTHESIS

Prepared for the New England Lobster Research Initiative



By
J. Stanley Cobb and Kathleen M. Castro
Fisheries Center
University of Rhode Island

May 2006

INTRODUCTION

The emergence of a virulent form of shell disease in the southern New England population of the American lobster (*Homarus americanus*) has raised concern among fishermen, managers, and scientists. Shell disease, particularly in Rhode Island, Connecticut, and southern Massachusetts waters, appears to be widespread. It may change the rate of natural mortality and egg production and certainly makes the afflicted individuals less saleable. It poses a real challenge to the management of the fishery, which, as noted by Duff (2005) at the recent Lobster Shell Disease Workshop hosted by the New England Aquarium, tries to strike a balance among the three objectives of biological sustainability, economic efficiency, and social equity. Presently, all three are of considerable concern in the southern New England region. Disease, declining recruitment, and high fishing mortality threaten sustainability; a rapid increase in the price of fuel affects economics; and the imposition of limits and allocations changes the social contract to which fishermen were accustomed.

Shell disease is common in crustaceans. Generally thought to be bacterial in origin, it is identified by a discolored or eroded exoskeleton. Shell disease is caused by chitinoclastic or lipolytic bacteria resulting in rust disease in king crabs and tanner crabs, burnt spot disease in the European crab, brown spot disease in shrimp, and just plain "shell disease" in the blue crab, the American lobster and several other species (Sindermann, 1989a). In most cases, the number of affected individuals in any population has been relatively few, and the impact assumed to be small. An exception is the report of Baross et al. (1978) of prevalence of diseased individuals being up to 76 percent in a population of tanner crab in the northeast Pacific.

Here, we address the history, prevalence and severity, and population-level effects of epizootic shell disease in *H. americanus* in New England waters.

DESCRIPTION

The term "shell disease" is a general one, used to describe a wide range of necrotic lesions, pits, and/or discolorations in the exoskeleton of crustaceans. Typically, the bacteria associated with the lesions are rod-shaped, chitinolytic, and Gram-negative (Porter et al., 2001). Sindermann (1989b) emphasized that "shell disease is not a discrete disease entity but instead can best be described as a 'disease syndrome' … characterized by progressive exoskeletal erosion resulting from activity of chitinoclastic microorganisms—often affecting stressed crustaceans …"

Shell disease in the clawed lobster *H. americanus* was first described 80 years ago in lobsters stored at high densities in tidal impoundments for later sale (Hess, 1937). *Vibrio* is the group of bacteria most commonly associated with shell-diseased lobsters in impoundments. Mortality of infected lobsters may be very high in pounds.

The epizootic shell disease of the American lobster that emerged in southern New England waters in the mid-1990s appears to be very different from the impoundment variety. *Vibrio* are rarely isolated from lesions of the exoskeleton; rather, members of the *Flavobacteriaceae* appear to be the dominant members of the microbial community of the shell (Chistoserdov et al., 2005a) of lobsters showing the symptoms and have been implicated as a possible culprit. There is no evidence that the disease is contagious; healthy lobsters held in laboratory tanks in close proximity to shell-diseased lobsters did not contract it. Koch's postulates have not been fulfilled for this "disease," thus, perhaps, it is best to think of shell disease as a "syndrome" as Sindermann suggests.

EMERGENCE AND SPREAD

Shell disease of *H. americanus* in natural conditions (i.e., not held in pounds) was recognized as early as 1981 (Glenn and Pugh, 2005). It probably was endemic and present earlier, although infrequent and in a mild form. Little attention was paid to the disease until 1983 when a survey of several locations in Massachusetts (Estrella, 1984) discovered a coastwise incidence of 12 percent. The low overall incidence and symptoms of minor pitting raised no alarm, even though a quarter of Buzzards Bay lobsters exhibited the symptoms. It probably is safe to say that the Massachusetts findings were mirrored in other areas, with low severity masking a moderate prevalence south of Cape Cod, as suggested by the 26 percent incidence in Buzzards Bay (Estrella, 1984), and nearly no shell disease in Cape Cod Bay and farther north.

In the 1990s, the endemic form of shell disease gave way to a much more aggressive form characterized by rapid infection and extensive, melanized, and deep lesions of the carapace. The symptoms appear to start in the dorsal area of the carapace just behind the rostrum and along the midline where it is more difficult for the lobster to groom. Entry to the shell appears to be through the pore canals. In the most severe cases, the lesions spread to cover the entire body. The claws are the last to be affected. (Castro and Angell, 2000). This new and aggressive disease was first seen in Rhode Island waters in 1997 (Castro and Angell, 2000) and in southern Massachusetts in 1997 (Glenn and Pugh, 2005). By 2000, it was clear that the disease had spread far enough and affected enough individuals to be classified as an epidemic or epizootic disease (Smolowitz et al., 2005b). In this paper we will distinguish between these two forms of shell disease as *endemic* and *epizootic* with the understanding that the etiology of the two diseases is most probably different, and that our knowledge, particularly of the endemic form, is not good enough to distinguish between the causative agents of the diseases.

PREVALENCE AND SEVERITY

Origin, Prevalence and Spread

Smolowitz et al. (2005b) recognized four types of shell disease in *H. americanus*: impoundment shell disease, burnt-spot shell disease, and the two diseases we call *endemic* and *epizootic* shell disease. Estrella (1984, 1991) described a rapidly expanding mild form of endemic shell disease (Figure 1) in Massachusetts. Between 1983 and 1989 the incidence of Buzzards Bay lobsters with endemic shell disease doubled from 26 percent to 52 percent, while in Cape Cod Bay the incidence went from 5 percent to over 40 percent.

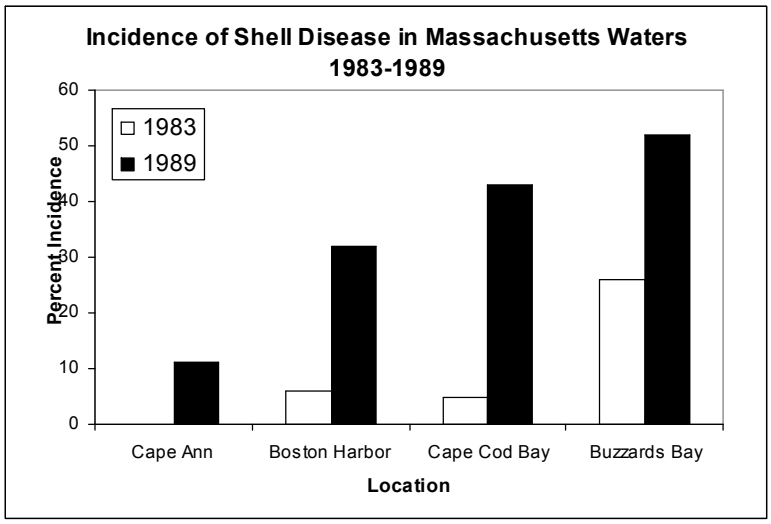


Figure 1. *Progression of endemic shell disease at four locations in Massachusetts (1983–1989) (Estrella 1984, 1991; Glenn and Pugh, 2005).*

A clear north-to-south gradient in incidence was seen in both years—Buzzards Bay to the south had the highest incidence of endemic shell disease, while the northernmost station in Massachusetts (Cape Ann) had the lowest. Severe epizootic shell disease was first reported by fishermen in Buzzards Bay in 1997.

Figure 2 shows the geographic and temporal spread of shell disease. From humble beginnings in 1997, it has become a major force in southern New England, while only a few cases have been noted in the Gulf of Maine.

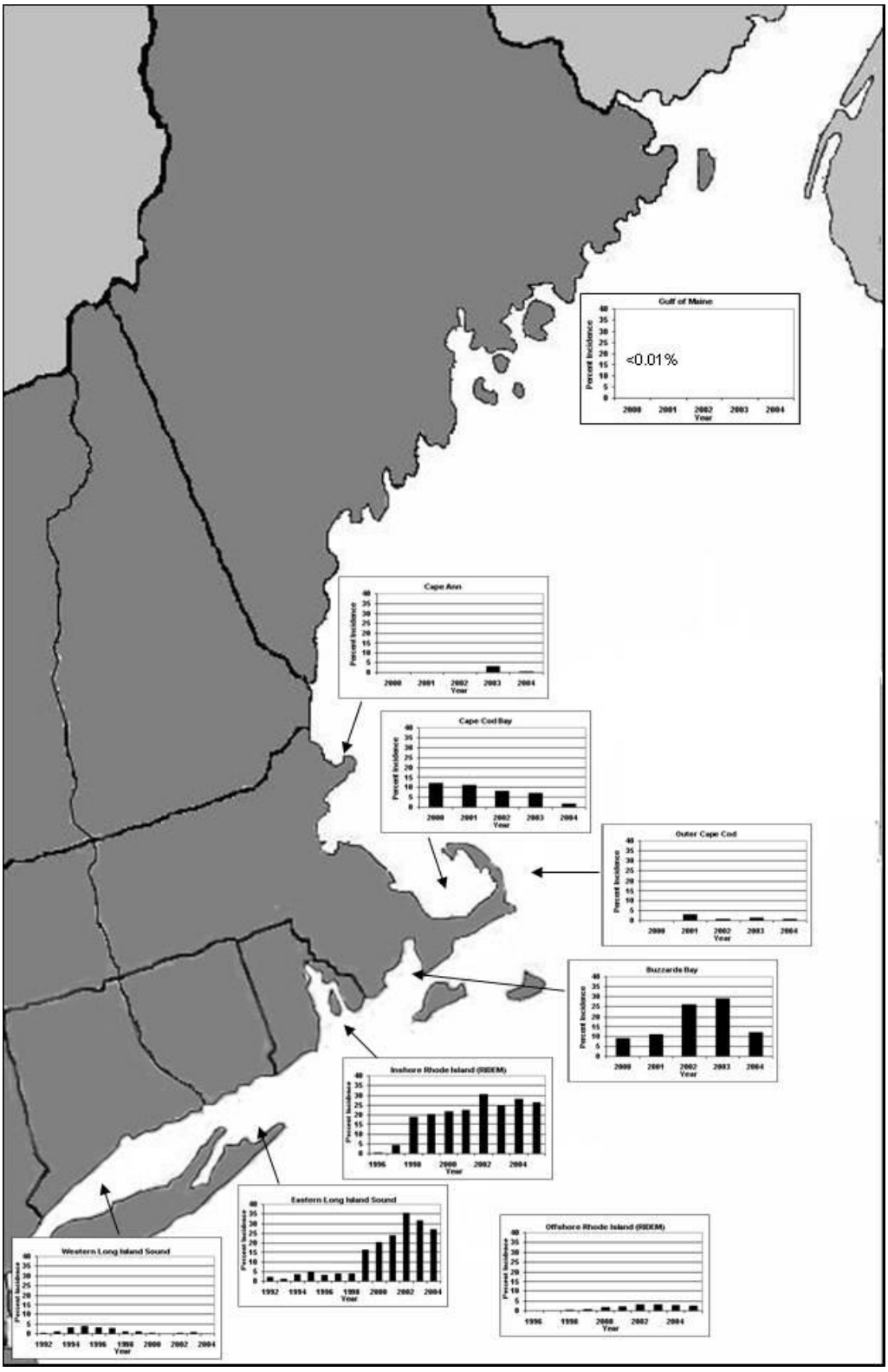


Figure2. *Incidence of shell disease over time at nine locations in New England. Data from reports by Castro et al., 2005; Glenn and Pugh, 2005; Howell, 2005; and Landers, 2005.*

In eastern Long Island sound, a lobster monitoring program included notations on the presence and severity of shell disease beginning in 1984 (Landers, 2005). Between 1984 and 1997 only seven of 115,800 lobsters examined showed signs of endemic shell disease. In 1998, 1.5 percent of the examined lobsters had shell disease, but it was not clear whether it was the endemic or the epizootic disease. Clear evidence of the severe, epizootic disease was first noted in 1999.

In Narragansett Bay, monitoring began in 1994 and the first symptoms of endemic shell disease were found in two lobsters in the fall of 1995 (Castro and Angell, 2000). The first individuals with epizootic shell disease were seen in 1997.

Shell disease in some form, while present, has not had an incidence greater than 5 percent overall in western Long Island Sound, the Gulf of Maine, or in the offshore canyons at any time (Landers, 2005; Castro and Angell, 2000; Wilson, 2005).

These comprehensive monitoring programs in southern New England pinpoint the emergence of epizootic shell disease in 1997 to Narragansett Bay and Buzzards Bay. (Castro and Angell, 2000; Glenn and Pugh, 2005). Since there was no report, formal or informal, of epizootic shell disease prior to 1996, it appears that the emergence of the disease was monitored from the very beginning. The location of emergence appears to be the region including eastern Long Island Sound, Block Island Sound, and Narragansett Bay. Very quickly, the disease was reported in Long Island Sound, Block Island Sound, and Cape Cod Bay. The spread of epizootic shell disease from 1997 to 2004 (or 2005) is shown on the chart in Figure 2. As with the less virulent endemic form, there is a north-to-south cline in prevalence of epizootic shell disease, with the Gulf of Maine showing very few diseased individuals, while south of Cape Cod, the disease affects 20 to 30 percent of all the animals sampled except in western Long Island Sound.

Severity

The severity of epizootic shell disease varies from barely noticeable (a few pits or a couple of small lesions on the carapace) to deeply eroded, melanized lesions covering nearly the whole body. If the disease is mild, the lesions are small and most likely found on the dorsal carapace just behind the rostrum or along the midline of the carapace. These are areas where the grooming with the legs is more difficult and probably is where the disease originates on the individual. Bacteria, probably of the *Flavobacter* group, destroy the hard exoskeleton gradually, from the outside in, progressing through the several layers of the shell. A cross-section of the exoskeleton is found in Figure 3 for reference. As the disease

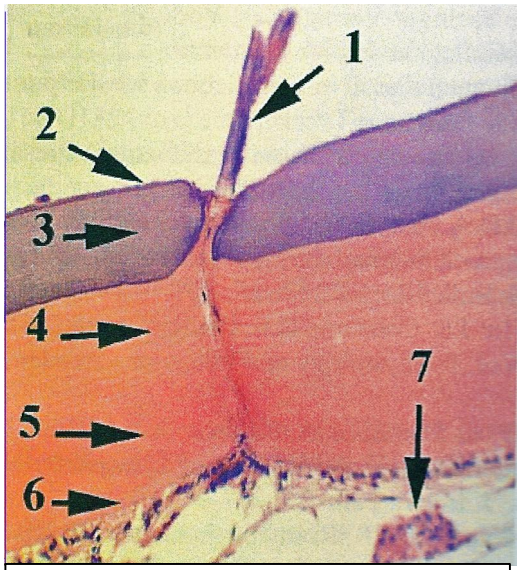


***Figure 3**. Cross-section of normal crustacean exoskeleton. 1. seta. 2. epicuticle. 3. exocuticle; 4. calcified endocuticle; 5. uncalcified endocuticle; 6. membranous layer; 7. tegmental gland. Source: Smolowitz et al., 2005a.*

progresses, it goes through four stages identified by Smolowitz, et al. (2005a):

1. Shallow and infrequent lesions of the epicuticle and exocuticle. The infection seems to extend from the surface of the exoskeleton to the exocuticle through the pores.
2. Frequent moderately deep lesions from the epicuticle penetrate into the calcified endocuticle. Pillars of remaining lattice crystals project from the leading edge of the lesions. An inflammatory membrane produced by the cuticular epithelium appears between the epithelium and the uncalcified endocuticle acting as a barrier against the disease.
3. Lesions penetrate deeply into the uncalcified endocuticle to the inflammatory membrane. Lesions are found over most of the carapace and start to appear on the dorsal surface of the abdomen.
4. Ulceration. Some of the lesions show loss of all the cuticular material and exposure of the connective tissue of the individual to the environment. Lesions cover most of the body.

The monitoring of shell disease by fishermen and biologists employs a scale of severity that is based wholly on the percent coverage of the body by lesions. There are four levels of severity (Table 1). All shell-disease monitoring efforts have used this scale since it was adopted by the Atlantic States Marine Fisheries Commission in 2000.

Table 1. The shell disease scale used for the American lobster to standardize data collection.

Shell Disease Index	% Coverage of shell
0 Disease free	No symptoms
1 Mild	1 – 10%
2 Moderate	11 – 50%
3 Severe	51 – 100%

Castro and Angell (2000) described the beginnings of the outbreak in Narragansett Bay, R.I. There, as the incidence of shell disease increased markedly from 1997 to 1999, the proportion of individuals classified in the lowest category of the severity increased from 3 percent to 14 percent, while the highest category showed little change, ranging between 1 and 2 percent. The severity of the disease seems to have peaked from 2000 to 2002 (Figure 4). The reason for the decline in severity (note, not incidence) after 2002 is not easily explained. Hypotheses include mortality of the most severe cases, or perhaps accommodation to the disease via some change in the innate immune system, and changing environment. None of these have been tested.

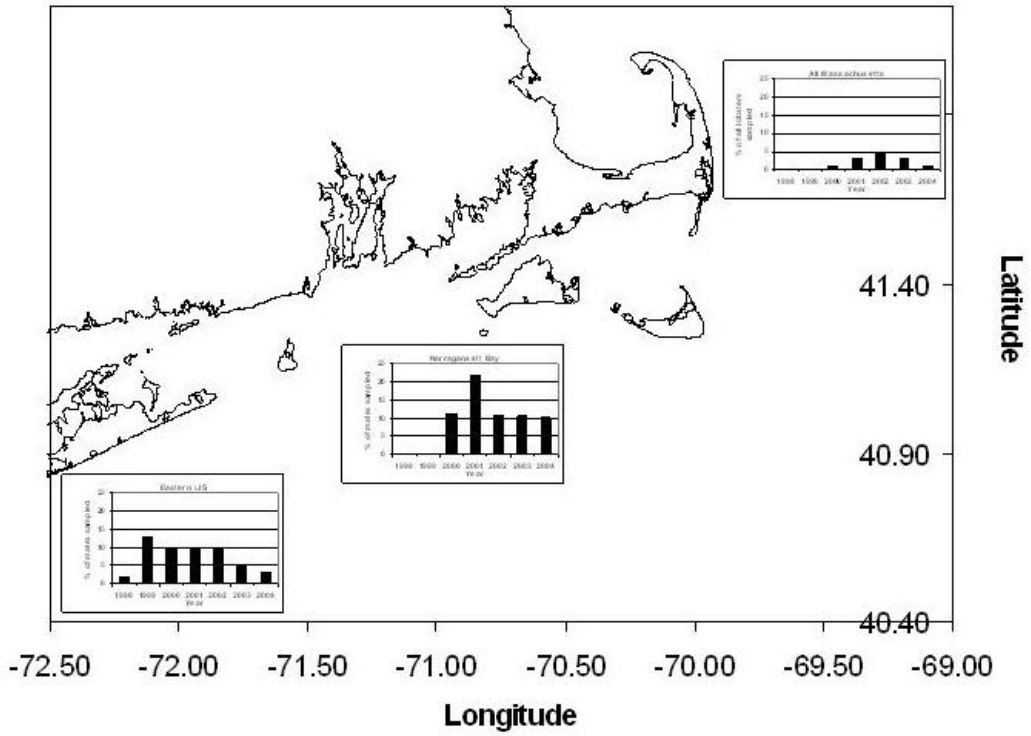


Figure 4. *The incidence, over time, of the severest form of epizootic shell disease (Category 3). Data extracted from Glenn and Pugh, 2005; Castro, et al., 2005; Landers, 2005.*

PROGRESSION OF THE DISEASE

The development of shell disease can be inferred from tagging studies. Castro et al. (2005) tagged lobsters in Narragansett Bay and examined the 107 female and 242 males that were recaptured more than once. Shell disease symptoms could appear or worsen within a week. Of the diseased lobsters that molted, the majority remained disease-free after ecdysis, while a few recontracted the disease. Of the disease-free lobsters that molted, nearly all became shell diseased after molting. A 41-month tagging study in eastern Long Island Sound (Landers, 2005) found 2,674 returns useful for analysis of shell-disease progression. Of those lobsters that were disease-free when tagged, 8.2 percent were diseased when recaptured. Conversely, of those diseased when tagged, 37 percent were free of shell disease when recaptured. There were no differences in patterns of movement between shell-diseased lobsters and disease-free lobsters in this tagging study.

When lobsters with shell disease molt, the lesions caused by the disease disappear and the newly molted lobster may or may not be disease-free. A common assumption has been that molting was a "get out of jail free" card, but it may not be the case. The assumption that molting makes an individual free of shell disease has been contradicted by the tagging studies of Landers (2005) and Castro et al. (2005). A very high proportion of lobsters with shell disease that molted when tagged had it again when recaptured.

Because development of the lesions and erosion of the shell progresses over time, one would expect a correlation between severity and the time elapsed

since the most recent ecdysis. Since molting is seasonal, the incidence of shell disease may be a function of size (but see later) and of season. Incidence and severity of shell disease would be predicted to be highest just before the molt and lowest just after it. Data in Table 2 illustrate the seasonality. In eastern Long Island Sound where the major molting period is in July, Landers (2005) reported the highest levels of shell disease May and October, and the lowest incidence just after the molts in August and September. The same pattern held in Narragansett Bay (Castro et al., 2005); where the shed occurs in June and October–November, incidence peaked in May–June and September–October. In Massachusetts, the statewide averages of incidence peaked in May-June and had only a small rise in November (Glenn and Pugh, 2005).

Table 2. Average seasonal incidence (proportion of total examined) of shell disease in three locations: Massachusetts (MASS)—average incidence all lobsters, all stations, 2000–2004; Rhode Island (RI)—research traps in Narragansett Bay, 2003; eastern Long Island Sound (ELIS)—average incidence in all lobsters, research traps 2003; see text for citations.

	May	June	July	Aug	Sept	Oct	Nov
MASS	.13	.13	.07	.04	.03	.01	.04
RI	.43	.20	.13	.15	.33	.58	--
ELIS	.35	.23	.08	.05	.18	.42	--

DEMOGRAPHY

Age (Size)

The more frequently the animal molts, the less likely it is to have shell disease. This would suggest that smaller lobsters are less likely than large ones to exhibit symptoms of shell disease simply because they molt more frequently, shedding the presumptive causative agent with the shell. Although logical, the data published to date do not support this explanation. Castro and Angell (2000) report significant correlations between size and shell-disease incidence in lobsters caught in monitoring trawls, (Figure 5) but the correlation coefficients are low. Castro et al. (2005) indicate that size matters in females over the average

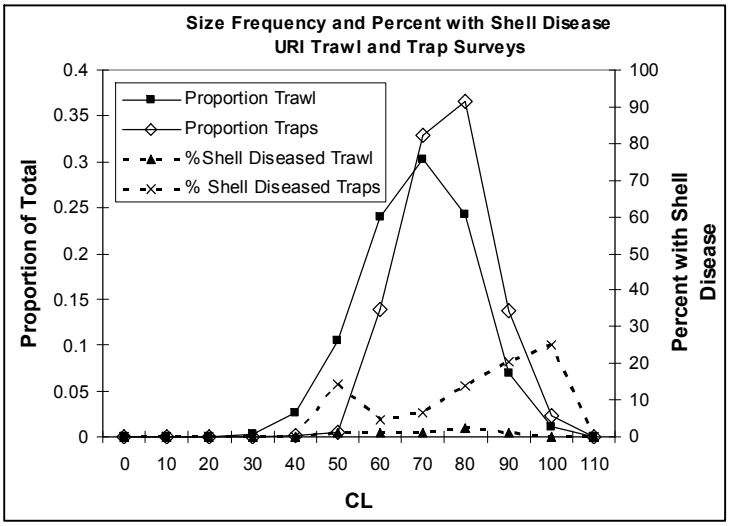


Figure 5. *Size frequency of shell disease lobsters from URI trawl survey (Narragansett Bay) and trap survey (Dutch Harbor).*

size at maturity in Narragansett Bay (Figure 6) , but an alternate explanation is that rather than size *per se*, the mature females molt less frequently due to their schedule of egg bearing and molting. This does not explain, however, the much higher incidence of ovigerous females with moderate to severe shell disease.

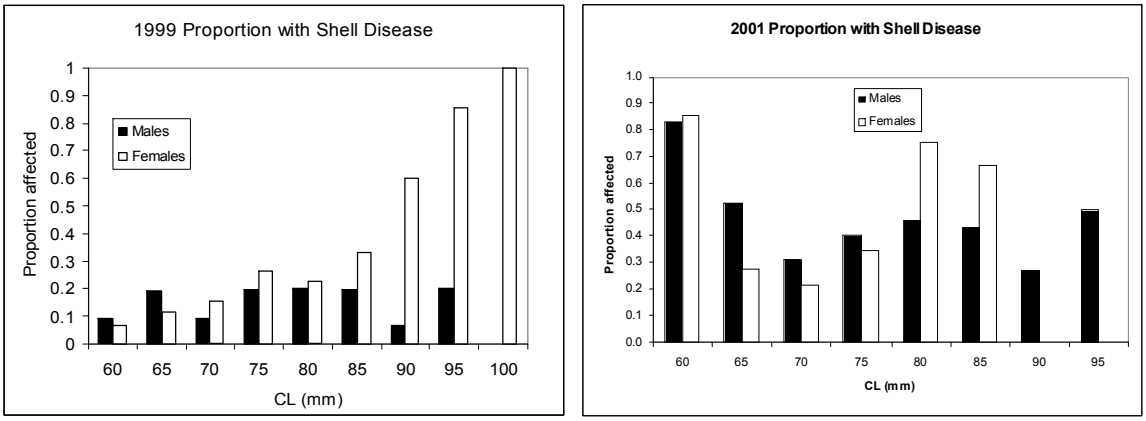


Figure 6. *Incidence of shell disease by size in Narragansett Bay in 1999 and 2002. Note that there appears to be no effect of size on incidence of shell disease in males, while more females get shell disease after maturity. Source: Castro et al., 2005.*

Evidence for size being a contributing factor would best come from records of males only. Castro et al. (2005) found no effect of size for males (Figure 6). Glenn and Pugh (2005) found higher incidence of shell disease in recruit-sized males than legal males (Figure 7). They offer several plausible explanations for this, including higher stress in the recruit group from handling by fishermen or competition with larger lobsters in traps, making individuals more susceptible to shell disease, inflation of the count (multiple counting) since recruit lobsters are returned to the seafloor to be caught again, or the next larger size category (legals) is underrepresented since these animals are removed from the population almost as soon as they molt so are present and available to be sampled only part of the year.

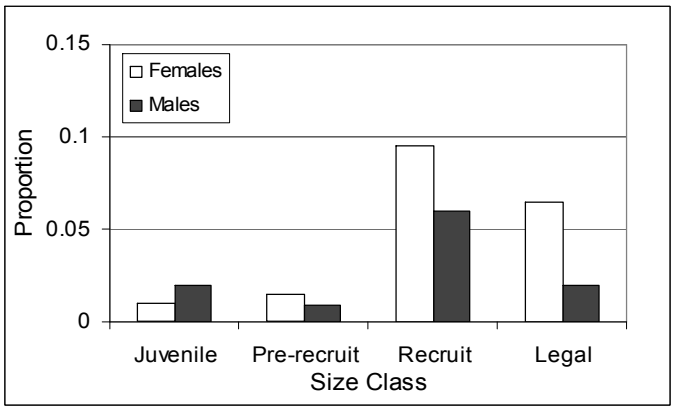


Figure 7. The proportion of diseased lobster in four size classes. Size classes can be described as follows: Juveniles (30 to 58 mm), pre-recruits (59 to 70 mm), recruits (71to 82mm) and legals (>83 mm).

Gender and Egg-bearing

Females and males have approximately the same incidence of shell disease until they reach the size of maturity (Figure 7). Subsequently, the proportion of females with shell disease becomes much larger than that for males. An explanation is found

in the interaction between the female's reproductive cycle and her molting cycle. Once mature, females bear eggs one year and molt the next, making the molting frequency of females about half that of males. The interval between ecdyses effectively doubles, permitting shell disease greater opportunity for infection and progression to greater severity. This explains the greater proportions of shell-diseased females over 80 mm in Figure 6, and the much higher proportion of afflicted females in the legal size class in Figure 7. In Massachusetts, ovigerous females were 7.5 times more likely to have shell disease than females without eggs (Glenn and Pugh, 2005). While this extreme value is not found in all regions, it is indicative of how important the length of time since ecdysis is to the probability of an individual becoming infected with shell disease. A dramatic example of the elevated incidence of shell disease in ovigerous females is seen in eastern Long Island Sound (Figure 8). As the disease emerged in 1998 and subsequently became epidemic, the proportion of berried females with shell disease increased from near zero in 1998 to 70 to 80 percent of the population two years later (Howell, 2005).

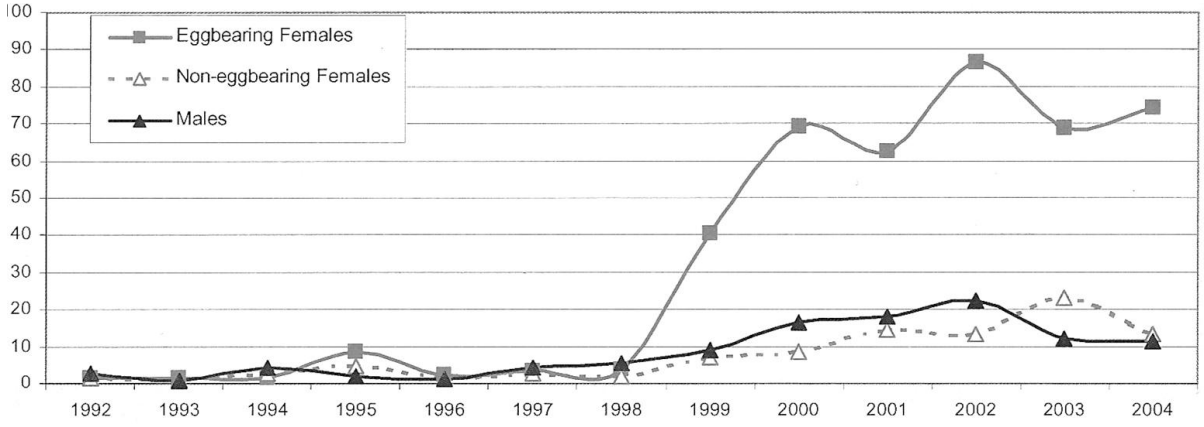


***Figure 8**. Eastern Long Island Sound. Incidence of endemic and epizootic shell disease by year. Note that after the emergence of epizootic shell disease in 1998 the proportion of ovigorous females with shell disease quickly climbed to over 70 percent.*

Females bearing eggs occasionally have been reported to molt before hatching their eggs. When this happens, all the eggs are lost. Egg production by the population thus suffers in two ways from shell disease. First, shell disease–related mortality removes females from the population. Second, molting when ovigerous lowers egg production without an increased mortality. The evolutionary implications of this should be considered.

BEHAVIOR

Little is known of the effects of shell disease on the behavior of the afflicted. Diseased lobsters are caught in traps, so presumably continue to walk, explore, and feed. Lander's (2005) tagging study reported above showed that diseased lobsters were no different than healthy ones in distance or direction

traveled. Castro et al. (2005) did behavioral, time-budget studies on diseased and clean lobsters in laboratory tanks. Of three behaviors recorded, only sheltering was different, with diseased lobsters spending more time in contact with shelter materials.

EFFECTS OF EPIZOOTIC SHELL DISEASE AT THE POPULATION LEVEL

Epizootic shell disease clearly has effects on individuals that will be felt at the population level. A slower growth rate, increased risk of death, and a disproportionate effect on ovigerous females will translate into lower productivity, higher natural mortality, lower abundance, and decreased population egg production. While it is clear that these effects are present, the magnitude of each is unknown.

In southern New England, the fishery is heavily dependent on new recruits and thus may react more strongly; that is, show larger change in abundance and catch to changes in fishing rate, mortality of juveniles, or supply of larvae (Gibson and Wahle, 2005). In fact, a steep decline in landings by the fishery was seen coincident to the rise in shell disease (Gibson and Wahle, 2005). The question asked by Gibson and Wahle was, what is the relationship between young of the year (YoY, 5–15 mm carapace length (CL)) and pre-recruits (52 – 72 mm CL, which are two molts away from entering the fishery at 83 mm)? And has that been altered by the addition of shell disease to the mix? They used the Ricker stock-recruitment relationship:

$$\mathbf{R = \alpha\, S \exp(-\beta S)}$$

that can be linearized to:

$$\mathbf{\ln(R/S) = \ln(\alpha) - \beta S + \varepsilon}$$

In these equations, **R** = recruitment to a particular life stage (pre-recruit in this example), **S** = spawning stock abundance (YoY for this example), α = maximum rate of recruitment; β = coefficient of compensatory mortality; and ε = lognormal error term. The terms α and β are density independent and density dependent factors, respectively. If specific factors other than α and β are likely to affect the stock – recruit relationship (by changing juvenile mortality in an epidemic, for example), an additional term can be added to the equation to take this into account. To include the effects of epizootic shell disease, Gibson and Wahle (2005) added the term $\gamma\mathbf{D}$ where γ reflects mortality due to shell disease, and **D** is an index of disease severity, to make the equation:

$$\mathbf{\ln(R/S) = \ln(\alpha) - \beta S - \gamma D + \varepsilon}$$

The time to grow from YoY to pre-recruit is about three years, so a three-year lag was used in linking YoY abundance (Wahle's settlement index, personal communcation) with pre-recruit abundance from trawl surveys. When the basic

two-parameter Ricker model is applied to data from 1990 to 1996, it produced a significant regression explaining 88 percent of the variance (Figure 9, left). However, when data from the years 1997–2001 (1997 marked the beginning of epizootic shell disease) were also used in the equation, the regression no longer was significant, indicating that something had a great impact on

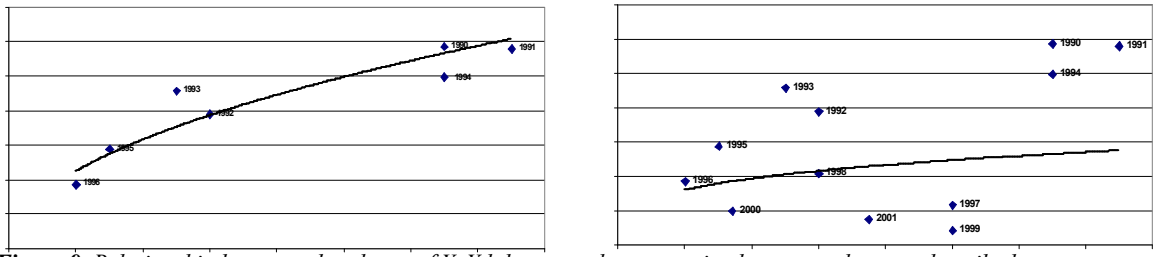


Figure 9. *Relationship between abundance of YoY lobsters and pre-recruits three years later as described by a three-parameter Ricker stock recruitment model (see text). Left panel: Data from before epizootic shell disease. Right panel: All data 1990 to 2001. From Gibson and Wahle (2005).*

the relationship starting in 1997 (Figure 9, above). When the term for shell disease was added to the model, the regression using all the data again became significant, explaining 86 percent of the variance. This is a remarkable result, indicating the importance of shell disease in the population dynamics of the lobster. It suggests that the status of the population was precarious before the appearance of the disease, and that the disease had a major effect on mortality in the late 1990s and early 2000s. For some time, fishery managers recognized the possibility that a subsidy in larval supply from the offshore population to the inshore southern New England population (Katz et al., 1992; Fogarty, 1998) was keeping the inshore population productive in the face of extremely high fishing pressure and rates of fishing mortality.

SHELL DISEASE: FROM ETIOLOGY TO MANAGEMENT

Factors Influencing Shell Disease Development

The primary categories of factors that may play a role in the expression of shell disease are the *environment*, the *host*, and the *pathogen*. Many have suggested that a stressful environment may facilitate the disease. Stress may cause pre-molt development of new cuticle to be faulty, it may compromise the immune system of the lobster, it might change the characteristics of the pathogen, or it might change the dynamics of the bacterial community on the lobster exoskeleton.

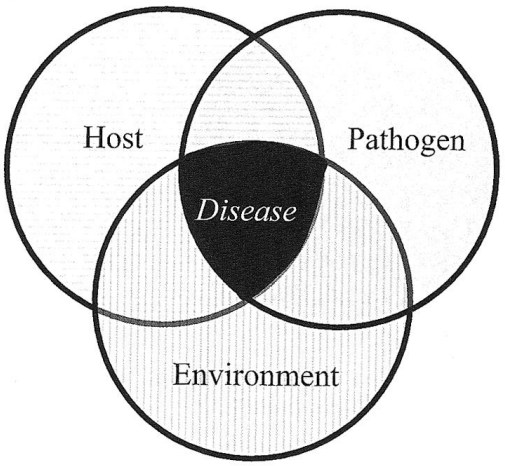


Figure 10. *Interaction of the environment, host, and pathogen. Source: Tlusty et al. (2005).*

Duboise and Moulton (2005) were unable to effect transmission of shell disease from one individual to another. They concluded that, "Lack of transmission from diseased to unaffected lobsters in this initial study suggests the etiology of the disease may depend on the interaction of environmental and genetic factors in a complex polymicrobial context."

Several studies (review by Sindermann, 1989a) implicated members of the Vibrionaceae. Porter et al. (2001) report that bacterial flora on the shell of the spiny lobster *Panulirus argus* are primarily heretofore unknown species of *Vibrio*, and that the bacterial community in the biofilm on the shell is not markedly different in shell-diseased or healthy lobsters. *Vibrio* also is an important component of the bacterial flora of *H. americanus* found in stressful conditions. These appear to be normal members of the biofilm community found on the surface of the shell. Sindermann (1991) hypothesized that shell disease occurs when, for some reason, the process of chitin deposition fails to keep pace with the normal processes of the surficial microbial community, and lesions follow. This hypothesis is strengthened by the observation that the most severe cases are found on lobsters that molt less often—primarily ovigerous females. A number of investigators have suggested that poor environmental quality (extreme temperatures, pollution, hypoxia, excess organic matter) may contribute to the high incidence of shell disease in lobsters held in captivity (Martin and Hose, 1995).

A Conceptual Model

Building on earlier conceptual models of shell disease (Sindermann, 1991; Castro et al., 2000), Castro et al., (in review) link environmental stress, physiological response, shell disease, and the population-level factors into a single model (Figure 11).

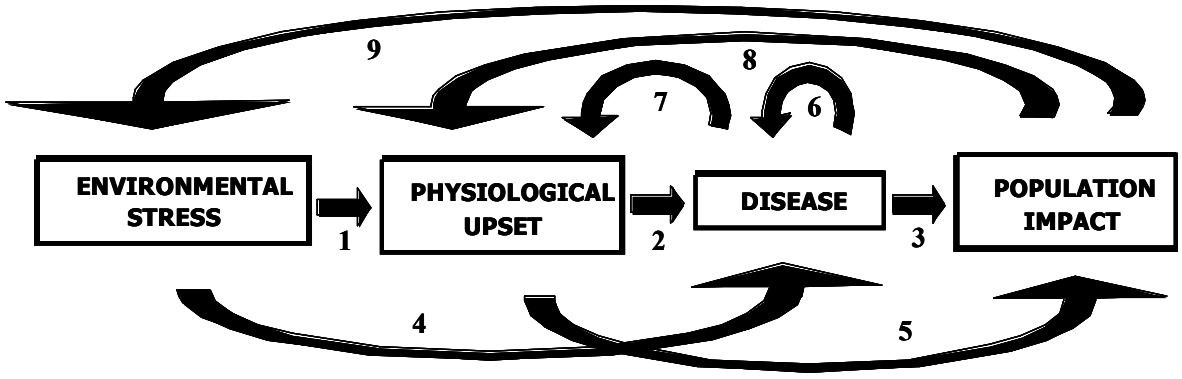


Figure 11. *Flow chart of interactions postulated in a conceptual model of shell disease. Source: Castro et al., in review.*

Environmental stressors such as elevated temperature, hypoxia, toxic substances, and endocrine mimics may initiate or potentiate the disease, or upset some aspect of the homeostatic regulatory mechanisms of the lobster, perhaps compromising its immune system and allowing shell disease to gain a foothold. Once diseased, the lobster's physiology may be upset further, setting up a positive feedback loop (arrows 2 and 7). Another possibility is that changing environmental conditions have altered the community dynamics of the biofilm on the surface of the lobster—to the advantage of the bacteria causing the disease. In any case, the effects of shell disease on the individual will have consequences at the population level. Changes in abundance, in natural mortality, egg production, and in size at maturity all have been debated as resulting from a large number of diseased individuals and the consequences to those individuals. Castro et al. (in review) discussed their model in the context of three hypotheses:

1. The prevalence of shell disease is increasing in the natural population;
2. Shell disease has population level consequences;
3. Stress, either natural or anthropogenic in origin, affects infection and progress of the disease.

These broadly stated general hypotheses, and others predicted by the model, provide a framework for the investigation of epizootic shell disease. Information is needed at all levels, from molecular to community, and description of the links among them is critical.

Managing with Shell Disease

Gibson and Wahle (2005) were able to begin making linkages between disease and population by including a disease parameter in the Ricker stock-recruit model (reviewed above). They noted the possible importance of a supply of larvae from outside the population to the continued viability of an overfished population vulnerable to recruitment failure. A decade ago, shell disease was not anticipated to be a factor in the population dynamics of *H. americanus* in southern New England. However, it is possible that the additional mortality

imposed by shell disease in an already vulnerable population has overwhelmed the benefits of the larval subsidy. As with all the hypotheses that may be generated from the model described by Castro et al. (in review), a great deal of research is needed for adequate testing. In the meantime, the temptation simply to harvest everything before the disease gets it may arise. Nevertheless, the daunting nature and long timeline of the task should not rule out management continuing to advocate a precautionary approach (Fogarty and Gendron, 2004) in confronting the collapse rather than retreating into regulatory paralysis in the face of pessimistic news (Gibson and Wahle, 2005).

LITERATURE CITED

Baross, J.A., P.A. Tester, and R.Y. Morita (1978) Incidence, microscopy and etiology of exoskeleton lesions in the tanner crab (*Chionoecetes tanneri*). *Journal of the Fishery Research Board of Canada* **35**:1141–1149.

Bullis, R.A. (1989) Shell disease in impounded American lobsters, *Homarus americanus*. *Biological Bulletin* **177**:327.

Castro, K.M. and T.E. Angell (2000) Prevalence and progression of shell disease in American lobster, *Homarus americanus*, from Rhode Island waters and the offshore canyons. *Journal of Shellfish Research* **19**:691–700.

Castro, K.M., T.E. Angell, and B. Somers (2005). Lobster shell disease in Southern New England: Monitoring and research. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 165–172. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Castro, K.M., J.R. Factor, T.E. Angell, and D.F. Landers (2006) A model of lobster disease: The conceptual approach to shell disease revisited. *In review.*

Chistoserdov, A.Y., R. Smolowitz, F. Mirasol, and A. Hsu (2005a) Culture-dependent characterization of the microbial community associated with epizootic shell disease lesions in American lobster, *Homarus americanus. Journal of Shellfish Research* **24**:741–747.

Christoserdov, A.Y., S.L. Gubbala, R. Smolowitz, F. Mirazol and A. Hsu. (2005b) A microbiological assessment of epizootic shell disease in the American lobster indicates its strictly dermal etiology. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 12–20. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Duboise and Moulton (2005) Defining the etiology of epizootic shell disease: the importance of genetic investigations of the associated bacterial and viral ecology. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 36–41. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Duff (2005) Management implications of lobster disease: How do you manage what you don't understand? **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 162–164. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Estrella, B.T. (1984) Black gill and shell disease in the American lobster (*Homarus americanus*) as indicators of pollution in Massachusetts Bay and Buzzards Bay. Massachusetts Division of Marine Fisheries Publication # 14049-19-125-5-85-CR. 17 pp.

Estrella, B.T. (1991) Shell Disease in the American lobster (*Homarus americanus,* H. Milne Edwards, 1837) from Massachusetts coastal waters with considerations for standardized sampling. *Journal of Shellfish Research* **10**:483–488.

Fogarty, M. (1998) Implications of migration and larval interchange in American lobster (*Homarus americanus*) stocks: Spatial structure and resilience. *Canadian Special Publication Fisheries and Aquatic Science* **125**:273–283.

Fogarty, M. and L. Gendron (2004) Biological reference points for American lobster (*Homarus americanus*) populations: Limits to exploitation and the precautionary approach. *Canadian Journal of Fisheries and Aquatic Sciences* **61**:1392–1403.

Gibson, M. and R. Wahle (2005) Reduced recruitment of inshore lobster in Rhode Island in association with an outbreak of shell disease and management implications. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 115–139. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Glenn and Pugh (2005) Observations on the chronology and distribution of lobster shell disease in Massachusetts coastal waters. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 141–155. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Hess (1937) A shell disease in lobsters (*Homarus americanus*) caused by chitinovorous bacteria. *Journal of the Fisheries Research Board of Canada* **3**:358–362.

Howell, P., C. Giannini, and J. Benway (2005) Status of shell disease in Long lsland Sound. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 106–114. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Hsu, A.C., R.M. Smolowitz, A.Y. Chistoserdov, and H.M. Chikarmane (2003) Comparison along the New England coast of epidemic shell disease in the American lobster, *Homarus americanus*. *Journal of Shellfish Research* **22**:335.

Katz, C.H., J.S. Cobb, and M. Spaulding (1992) Larval behavior, hydrodynamic transport, and potential offshore recruitment in the American lobster, *Homarus americanus*. *Marine Ecology Progress Series.* 103:265–273.

Landers, D.F. (2005) Prevalence and severity of shell disease in American lobster *Homarus americanus* from eastern Long Island Sound, Connecitcut. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 94–97. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Hose, J.E. and G.G. Martin (1995) Circulation, the Blood and Disease. **In:** *Biology of the Lobster,* Homarus americanus. Edited by J.R. Factor. Academic Press, San Diego, Calif. Pp. 465–495.

Porter, L., M. Butler, and R. Reeves (2001) Normal bacterial flora of the spiny lobster *Panulirus argus* and its possible role in shell disease. *Marine and Freshwater Research* **52**:1401–1405.

Sindermann, C.J. (1989a). *Principal Diseases of Marine and Fish and Shellfish.* Volume 2, Second edition.

Sindermann, C.J. (1989b) The shell disease syndrome in marine crustaceans. *NOAA Technical Memorandum* NMFS-F/NEC-64. 43pp.

Sindermann, C.J. (1991) Shell disease in marine crustaceans—A conceptual approach. *Journal of Shellfish Research* **10**:491–494.

Smolowitz, R., A.Y. Chistoserdov, and A. Hsu (2005a) A description of the pathology of epizootic shell disease in the American lobster, *Homarus americanus*, H. Milne Edwards 1837. *Journal of Shellfish Research* **24**:749–756.

Smolowitz, R. et al. (2005b). Epizootic shell disease in the American lobster, *Homarus americanus*. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 2–11. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, Mass.

Tlusty, M. (2005). New in vivo methods to study shell formation and possible implications for shell disease. **In** : *Lobster Shell Disease Workshop Forum Series 05-1*. Pgs 68-71. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, MA

Wahle, R., L. Incze, and M. Fogarty (2004) First projections of American lobster fishery recruitment using a settlement index and variable growth. *Bulletin of Marine Science* **74**:101–114.

Wilson, C. (2005) Observation of shell disease in coastal marine water: 2003 and 2004. **In:** *Lobster Shell Disease Workshop Forum Series 05-1.* Pp. 156–161. Edited by Tlusty, M.F., Halvorson, H.O., Smolowitz, R., and U. Sharma. New England Aquarium, Boston, MA