Histopathology of idiopathic lesions in the eyes of *Homarus americanus* from Long Island Sound

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Abstract

In 1999, American lobsters, *Homarus americanus*, from western Long Island Sound (WLIS) experienced a significant mortality. In 2001 and 2004, the eyes and eyestalks of lobsters from WLIS and central LIS were examined for histopathological changes. Idiopathic lesions were identified in the ommatidia and optic nerve fibers proximal to the ommatidia in 29 (56%) of the lobsters from LIS. Lesions were categorized as either moderate or severe. Moderate lesions had altered rhabdoms, clumped pigment, and altered optic nerve fibers. Severe lesions were marked by absent rhabdoms, clumped pigment in both the ommatidial region and in the optic nerve region; and optic nerve fibers that had been completely destroyed and were replaced by vascular tissue. Idiopathic lesions occurred primarily in the central and ventral regions of the eye, and with much less frequency in the dorsal region. In addition, damage to the dorsal area tended to occur only when the severity of lesions was high, indicating a spatially progressive pattern to the lesion development. The lesions occurred in both western and central Long Island Sound, with no significant differences in severity between locations. The prevalence of lesions did not vary between years, but in 2004, several eyes had less severe pathology than those from 2001. These data indicate that the etiological agent is present throughout a large portion of the Sound, and that lobsters are probably continually exposed to it.

**Keywords:** Lobster; Eyestalk; Eye; Ommatidia; Optic nerve; Blindness; Compound eye; Nerve

1. Introduction

The American lobster *Homarus americanus* is an important commercial species in the United States. Landings of *H. americanus* for 2002 were recorded at 82.3 million pounds with an economic value of $293.3 million (National Marine Fisheries Service, 2003) and worldwide landings are valued at over $750 million (National Marine Fisheries Service Annual Landings Query; Fisheries and Oceans Canada: State of Canada’s Fishery 2002 Fact Sheet). Prior to 1999, the *H. americanus* fishery for New York in Long Island Sound was the third largest in the country, with annual landings of 8.5 million pounds; the Connecticut LIS fishery brought in 2.5–3.7 million pounds. For 1998, the combined landings for New York and Connecticut were valued at over $40 million (Connecticut Department of Environmental Protection, 2000). The lobster fishery is a significant resource from Long Island Sound accounting for over 90% of the value of commercial landings there.

In fall 1999, a mortality of *H. americanus* occurred in western Long Island Sound (WLIS). Surveys of local lobster fishers taken after the mortality event found that sporadic mortalities occurred in 1998 and to a lesser degree in 1997, but that the 1999 event was severe and widespread (Connecticut DEP, 1999). The same surveys also reported that there had been delayed molting for *H. americanus* in the Sound in 1999—the fall molt did not occur until mid December rather than in September–October, when it normally occurs there (Connecticut DEP, 1999).

Symptoms in affected lobsters were reported as lethargy and a pink discoloration to the ventral surface of the abdomen. The disease was classified as a systemic inflammatory...
disease that primarily affected the nervous system of the lobster: there was damage to areas such as the ventral nerve cords, brain, and neurosecretory portions of the eye. More specifically, pathology associated with infection included discoloration of hemolymph and muscle, granulocytopenia, granulomas in connective tissues, hypertrophy and necrosis in nerve tissues, and altered hemolymph clotting ability (French, 2000; Russell et al., 2000). Further study by Mullen et al. (2004) found hemocytic infiltrates in nerves and ganglia of infected lobsters, as well as in the retina, around the tegumental glands and within the muscular interstitium of the eyestalks. A parasitic amoeba, Paramoeba sp., was found within the hemocytic infiltrates. The same parasite was found in neural tissue, within the cytoplasm of neurons, and between nerve fibers of the ventral nerve cord, antenna, and eye, even when no hemocytic infiltration was observed. The parasite also occurred in the tegumental glands and nerves of the antennae and eyestalks, where degenerate epithelium and nerves, respectively, were present in hemocytic infiltrates. The parasite was also found with less frequency in foci of hemocytic infiltration outside of the nervous system (Mullen et al., 2004). Mullen et al. (2005) have now characterized the agent as a species of Neoparamoeba. No infections have been reported since 1999.

In an effort to identify possible chemical causes for the lobster die-off, the Department of Environmental Protection, Water Bureau and the Marine Fisheries Office (Connecticut) conducted water quality analyses for 30 sites in WLIS. They tested for the presence of 66 volatile organic compounds, 137 semi-volatile organics, 30 organochlorine pesticides, 18 chlorinated herbicides, and toxic algae, and found no abnormal levels for any of the compounds (Connecticut DEP, 1999). However, the lobster mortality coincided with the spraying of malathion, an organophosphate pesticide, and the use of other pesticides (sumithrin, permethrin, and methoprene) to combat the spread of mosquito-carrying West Nile Virus. Low levels of malathion have recently been shown to depress phagocytosis in lobster hemocytes (DeGuise et al., 2004). Mullen et al. (2004) noted that smaller mortalities of lobsters were reported in 1998, before pesticides were used in New York and toxicological testing did not find measurable amounts of target pesticides—such as malathion, methoprene, and resmethrin—in experimentally exposed lobster tissue (Mullen et al., 2004). However, malathion has a short half life in lobster tissues (DeGuise et al., 2004).

In 2001, while studying the aftereffects of the initial mortality in WLIS, moribund lobsters from the Sound had “cloudy” gray eyes (J. Shields, personal observation). The eyes from these lobsters were pathologically altered in a manner unlike that reported for blue crabs infected with Paramoeba perniciosa (Johnson, 1977). Thus, the goal of this research was to understand the underlying abnormalities in the eyes of American lobsters H. americanus from Long Island Sound and where possible to determine their cause. We examined the level of severity of the eye pathology as well as the spatial extent of the damage, and attempted to determine the potential causes of the pathology.

2. Materials and methods

2.1. Collection of animals

American lobsters used in this study were collected from Long Island Sound in September 2001 and in June and July 2004. In 2001, lobsters were taken from commercial lobster pots in western Long Island Sound (WLIS), southeast of Stamford, CT (Fig. 1). Overall, 31 lobsters were taken, with eyes being collected from 23 animals. In 2004, 10 lobsters were taken from commercial lobster pots in WLIS and 20 were collected from pots in central Long Island Sound (CLIS). Lobsters collected in 2004 were shipped overnight from Connecticut to the Virginia Institute of Marine Science (VIMS) in Gloucester Point, Virginia. Ten additional lobsters were collected from a lobster facility in Nova Scotia that had been sent to a holding facility in Kentucky, USA.

2.2. Dissection and histology

Lobsters from 2001 were processed for histology dockside, following their collection; the lobsters from 2004 were processed immediately upon their arrival at VIMS. Notes were taken on the general physical appearance and behavior of lobsters prior to dissection. The hepatopancreas, hindgut, hemopoietic tissue, heart, gill, brain, skin, and eyestalk were fixed in Bouin’s solution for approximately 48 h and then held in 70% EtOH. Eyestalks were decalciﬁed using the formic acid–sodium citrate method (Luna, 1968). They were then cut in half in the sagittal plane using a single-edged razor. The eyestalks and other tissues were processed using standard histological techniques and stained with hematoxylin and eosin (Luna, 1968).

2.3. Histology of the eye

All of the tissues were microscopically examined, but the eyestalks were studied in detail. Areas of interest in the eyestalks were the ommatidia, basement membrane, optic nerve fibers, lamina ganglionaris (Fig. 2), tegumental glands, the three medullar areas, sinus gland, neurosecretory cells, connective tissues, and hemal spaces. Tissues were examined using an Olympus BX51 compound microscope and photographs were taken using a Nikon DXM1200 with the aid of the ACT-1 computer program (Nikon).

Damage found in the eye was subjectively rated for both severity and spatial extent. The scale for severity ranged from 0 to 2, with 0 = no damage, 1 = moderate damage, and 2 = severe damage (see below). Spatial extent was also assessed with a scale between 0 and 2, where 0 = no damage, 1 = 1–49% of the area damaged, and 2 = 50–100% damaged. The total area of the eye was evaluated, as were ventral, central, and dorsal
regions, which were simply defined as each third of the eye (ventral, central, dorsal) from the lamina ganglionaris distally to the lens as an arc. These ratings are referred to as the total area index (TA), ventral area index (VA), central area index (CA), and dorsal area index (DA). The sagittal halves of each eye were rated for severity and spatial extent of damage. The size of lobsters was categorized by carapace length (CL) into three groups <77.5 mm CL, 77.5–80 mm CL, and >80 mm CL. These rankings were chosen based on sample size.

2.4. Statistics

Data were analyzed with Microsoft Excel and SYSTAT (SPSS Inc.). Contingency tables were used separately to examine differences in prevalence of the lesions in 2001 and 2004, severity between two eyes within individuals, severity between years, and severity between animals collected in 2004 from WLIS and CLIS. The spatial extent of eye damage and severity of damage were analyzed with contingency tables to determine whether there was a region of the eye, central, ventral, or dorsal—where the lesions preferentially occurred. In addition, the relationships between lobster size and lesion severity, as well as size and the total area of the eye affected were also examined by contingency table.

3. Results

3.1. Idiopathic lesions in the eyes of H. americanus

The severity of the pathology was evaluated in the eyes of 52 lobsters from LIS. One lobster had to be removed from the study because it exhibited pathology that was obviously different from that described here (see below). Idiopathic lesions were observed in the ommatidia and in their associated optic nerve fibers that run proximal to the ommatidia to the lamina ganglionaris.

Moderate damage was defined by subtle to pronounced changes within the ommatidia and the optic nerve. Hemocytes were observed within the ommatidia, having crossed the basement membrane, a condition not seen in healthy eyes. Hemocyte aggregations were observed in the blood vessels of the optic nerve area of the eye, distal to the lamina ganglionaris (Fig. 3). One of the most obvious changes to the ommatidia was the clumped pigmentation (Fig. 4). The shift in pigmentation revealed slight changes in the appearance of the retinular cell layer, most noticeable by the absence or relocation of retinular cell nuclei, and the
loss of the spindle shape of the rhabdoms (Fig. 5). Occasionally, the crystalline tracts of the damaged regions appeared to be shortened, but this pathology was rare. The optic nerve fibers of moderately affected eyes had lost their well ordered, straight, and fibrous appearance; instead appearing as basophilic, loose fibers with ragged edges, perhaps indicative of necrosis (Fig. 6). The fibers, while still present, no longer converged into taut, well-defined tracts.

Severely damaged eyes had noticeable changes in the ommatidia and in the optic nerve region. In the eyes of some of these lobsters, hemocyte infiltration occurred into the ommatidial region, and hemocyte aggregations could be observed in the enlarged hemal spaces of the optic nerve region. Pigment was clumped on both sides of the basement membrane (Fig. 7). The retinular cell layer had lost its structure, and was either filled with cellular debris (Fig. 8) or was free of any remnants of the retinular cells or their rhabdoms (Fig. 7). In the ommatidial region, damage ranged from destruction of the retinular cell layer, to obliteration of the ommatidia. The crystalline tracts of severely damaged lobsters became vacuolated (Fig. 8), and occasionally the distal pigment that surrounded the crystalline cone cells of healthy and moderately damaged ommatidia was dispersed throughout the clear zone. Material that appeared to be remnants of the crystalline tract had moved proximally into the former retinular cell layer in some lobsters (Fig. 9A). Perhaps, the most striking pathology was the complete loss of optic nerve fibers along the damaged tracts (Fig. 9). Increased presence of vascular tissue occurred in the region where the optic nerve fibers previously existed. This could either be the result of new tissue growth, or an increased hemolymph flow causing the already existing blood vessels to enlarge into the
newly vacant areas. In severely damaged eyes, no remnants of the optic nerves were visible.

In none of these cases was there apparent pathology to the other structures in the eyes, including the lamina ganglionaris, medullar areas, tegumental glands, and sinus gland.

3.2. Analysis of data from one eye vs. two eyes

Only one eye was taken from most lobsters collected in 2001. In 2004, two eyes were taken from each lobster. In one of the 2004 samples, only one eye could be included in the study because the second eye was pathologically altered by previous mechanical damage. The total number of lobsters from which both eyes were examined was 29. Of these, 18 animals had lesions in their eyes, and 72% (13/18) of these animals had idiopathic lesions in both eyes. Five lobsters exhibited lesions in only one eye.

When lesions were present in both eyes, the severity ranking between eyes was identical. Therefore, examining only one eye was a reasonable indicator of the prevalence of eye lesions, as well as the overall severity of the lesions, although it probably underestimated the prevalence of the condition. For this assessment, 11 lobsters had no lesions in both eyes, nine had moderate lesions in both eyes, and four had severe lesions in both eyes. For the five lobsters that had lesions in only one eye, three had lesions ranked as moderate, and two had lesions ranked as severe. In these five lobsters only, the other eye did not have idiopathic lesions.
3.3. Prevalence and severity between years and locations

Idiopathic lesions were found in 10 of 23 (43%) lobsters sampled in 2001 and 19 of 29 (66%) lobsters sampled in 2004, for an overall prevalence of 56%. Prevalence was not significantly different between years ($\chi^2 = 2.53$, df = 1, $p > 0.05$). However, severity varied significantly between years ($\chi^2 = 13.88$, df = 2, $p < 0.001$). In 2004, 13 lobsters had moderate lesions and six had severe lesions, whereas in 2001, no lobsters had moderate lesions and 10 lobsters had severe lesions. Further, there were no differences in severity between lobsters taken from both WLIS and CLIS in 2004 ($\chi^2 = 1.07$, df = 2, $p > 0.05$). Of the 10 lobsters from WLIS, five had moderate and one had severe lesions compared to the eight animals with moderate and five with severe lesions from CLIS.

Although sample size was low, there were no apparent trends in the severity of lesions at the different locations.

3.4. Spatial extent of lesions

The spatial extent of eye damage was variable between animals, ranging from 1 to 100% of the total area of the eye examined (Fig. 10). The spatial extent was also variable in different sections of the same eye. For example, one half of the eye may have been rated at 50% damage to the optic field, whereas the other half of the same eye may have been rated at 25% damage to the optic field; and in some cases, lesions were present in one half of the eye, but not in the other half.

Each eye was divided into ventral, central, and dorsal areas to further examine the spatial extent of the lesions. The ventral area index (VA) showed a significant difference from the total area index (TA) ($\chi^2 = 10.71$, df = 2, $p < 0.01$). Those lobsters without apparent lesions (i.e., TA equal to zero) were
excluded, leaving 29 of the initial 52 lobsters for comparison. When the TA was high, damage was likely to occur in the ventral region, but when TA was low, damage was only sometimes associated with the ventral region (Table 1). The central area index (CA) showed similar results; there was a significant difference between the indices ($\chi^2 = 15.65, df = 2, p < 0.001$). When the TA was high, the CA was likely to be high as well, but when TA was low, damage also occurred in the central region, but this damage was more likely to cover a smaller area (7/12 lobsters with CA < 50) than a larger area (4/12 lobsters with CA ≥ 50) (Table 1). Only one affected lobster (of 29) had no damage to the central region. The dorsal area index (DA) was also significantly different from the TA ($\chi^2 = 6.7, df = 2, p < 0.05$), but with a different pattern ($n = 28$ eyes). In 12 affected lobsters, no damage occurred in the dorsal region (Table 1). Furthermore, when damage occurred in the dorsal region, it was more likely to occur when the TA was high; i.e., a ranking of ≥ 50 or more of the total area damaged. Therefore, the lesions occurred more frequently in the ventral and central regions of the eye, and occurred less frequently in the dorsal region.

Severity was unrelated to TA, VA, and CA (Table 2), but there was a significant relationship between DA and severity ($\chi^2 = 8.49, df = 2, p < 0.05$). That is, when there was no damage to the dorsal region, severity tended to be lower, and when there was damage to the dorsal region, severity tended to be higher.

### 3.5. Relationship between eye lesions and size

Lobster size was not related to the total area affected ($\chi^2 = 2.20, df = 2, p > 0.05$). However, there was a significant difference between size and severity ($\chi^2 = 12.42, df = 2, p < 0.01$), with larger, and therefore older, lobsters having more severe lesions than younger lobsters. Of the smaller (68 ≤ CL < 77.5 mm) animals with lesions, six had moderate and three had severe rankings. Of the lobsters in the 77.5 ≤ CL ≤ 80 mm CL size class, seven had moderate and three had severe rankings. Of the larger (80 ≤ CL ≤ 93 mm) animals with lesions, all 10 had severe rankings.

### 3.6. Other eye pathologies

In many of the lobsters examined, non-specific granulomas were observed in various regions of the eye, including connective tissues, tegumental glands, medullar areas, lamina ganglionaris, and the optic nerve region. There was no association between the presence of granulomas and the severity of lesions in the eyes ($\chi^2 = 2.77, df = 2, p > 0.05$).

### 3.7. General condition of lobsters

In general, the lobsters were in good physical condition prior to dissection. In 2004, two lobsters exhibited lethargy; one of these lobsters had no idiopathic lesions and the other had moderate lesions in their eyes. One lobster without eye lesions was missing a claw and one lobster with severe lesions had two legs missing. Histological examination of other tissues, exclusive of the eyes, revealed no other

### Table 2

Frequency of lobsters with lesions in various regions of the eye in relation to the severity of the lesions; total area (TA), ventral (VA), central (CA), and dorsal (DA)

<table>
<thead>
<tr>
<th>% Total area of the eye with lesions</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50%</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>≥ 50%</td>
<td>6</td>
<td>11</td>
</tr>
</tbody>
</table>

$\chi^2 = 1.51, df = 1, p > 0.05$

<table>
<thead>
<tr>
<th>Ventral area (VA)</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50%</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>≥ 50%</td>
<td>7</td>
<td>9</td>
</tr>
</tbody>
</table>

$\chi^2 = 0.64, df = 2, p > 0.05$

<table>
<thead>
<tr>
<th>Central area (CA)</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50%</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>≥ 50%</td>
<td>10</td>
<td>11</td>
</tr>
</tbody>
</table>

$\chi^2 = 0.89, df = 2, p > 0.05$

<table>
<thead>
<tr>
<th>Dorsal area (DA)</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50%</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>≥ 50%</td>
<td>1</td>
<td>8</td>
</tr>
</tbody>
</table>

$\chi^2 = 8.49, df = 2, p < 0.05$

Only lobsters with eye lesions were assessed for extent of damage. Area is 0%, <50%, ≥ 50%. Lesions with moderate severity had altered rhabdoids and optic nerve fibers, and clumped pigment. Severe lesions were marked by absent rhabdoids, clumped pigments in both the ommatidial and optic nerve regions, and the complete loss of optic nerve fibers.
pathological alterations, with the exception of non-specific granulomas which were not associated with eye lesions and were distributed primarily in connective tissues. Shell disease was observed on two animals from 2001 and no animals from 2004.

4. Discussion

Idiopathic lesions of the eye were common in lobsters from Long Island Sound, occurring in 56% of the 52 lobsters examined. Because of the destruction of the visual receptor cells, the lesions in these lobsters can render them effectively blind. This blindness can be extensive, as 59% of the lobsters had at least half of their retinular cell layer and optic nerve fibers affected, and 2 lobsters had all of their retinular cells and optic nerve fibers destroyed. In other lobsters, the area affected was variable between the eyes and between sections of a single eye, but at least 56% exhibited impaired vision. These lesions did not evoke an all-or-nothing type of blindness; some lobsters would have retained limited vision in the dorsal or peripheral ommatidia. However, because all of the ommatidia in the eye combine to form a mosaic image (Barnes et al., 1993; Pearse et al., 1987), it is likely that the affected lobster experienced blind spots in their visual field. Ommatidia are connected by interneurons that can influence each other. That is, due to lateral inhibition, a single ommatidium will have a greater level of response when it alone is exposed to light than when it is exposed to light as part of a group of ommatidia (Barnes et al., 1993). Therefore, lesions in the ommatidia may lead to altered responsiveness in the remaining healthy portions of the eye.

*Homarus americanus* may use their visual sense to aid in shelter acquisition and feeding. While some lobsters frequently change shelters, others may remain in a single shelter for up to 10 weeks (Karnofsky et al., 1989), probably because they act as central place foragers (Lawton and Lavalli, 1995). American lobsters use vision as a supplement to their mostly chemosensory foraging methods (Atena and Voigt, 1995; Lawton and Lavalli, 1995). They often act as ambush predators, and respond only to prey that are moving rather than stationary (Hirtle and Mann, 1978). Large blind spots could make this feeding technique less successful, since lobsters rely on successive stimulation of ommatidia to see moving objects (Pearse et al., 1987). However, they feed on bivalves too, and likely rely on olfaction to find sedentary prey (Hirtle and Mann, 1978). In addition to shelter and feeding, lobsters also use vision and light detection to determine time of day, as they tend to be active only at night (Karnofsky et al., 1989). Light may also be important as a seasonal cue for molting and migration. However, lobsters do not use vision for mating or agonistic behaviors (Snyder et al., 1992). In addition, Chapman et al. (2000), using a mark-recapture study, found that light-induced eye damage did not influence long term survival, growth, and reproduction in *Nephrops norvegicus*.

A systemic exposure or etiology is indicated as the cause of the idiopathic lesions because 72% of animals with lesions had them in both eyes (when both eyes were examined); and they presented with the same degree of severity in both eyes. No pathology was observed in the eyes of the 10 lobsters collected from Nova Scotia, ruling out staining or fixation artifacts. Further, the disease agent was present throughout a large portion of the Sound, and the lobsters in WLIS and CLIS were probably simultaneously exposed to it because affected lobsters were found at the same level of severity from both regions in 2004. These data suggest that the etiology of the lesions caused a progressive disease in lobsters with moderate lesions advancing to severe lesions over time. Additionally, larger and presumably older lobsters had more severe lesions supporting the hypothesis that lesions increase in severity over time or that older lobsters are more susceptible to the etiological agent.

The causal agent apparently affects the central and ventral portions of the ommatidia first before progressing to the dorsal portion. Damage to the dorsal portion occurred only when the severity was high, indicating a spatially progressive pattern to lesion development. That is, as the lesions become more severe, they extend into the dorsal region. When decapods molt, they add additional rows of ommatidia mainly at the dorsal eye margin (Parker, 1890; Shelton et al., 1981). Therefore, it seems possible that unaffected dorsal regions are the result of new ommatidial development. It is also suggestive that the damage to the eye may occur prior to the latest instar.

4.1. Possible causes of lesions

There are a number of possible etiologies for these idiopathic lesions in the eyes of *H. americanus*, including, but not limited to, microbial or parasitic infection (e.g., paramoebiasis), excretory calcinos is, light exposure, or chemical exposure.

Paramoebiasis, although implicated in the 1999 mortality event, is probably not the etiological agent for this eye pathology. Mullen et al. (2004) examined the eyes of lobsters infected with paramoebiasis, but the only pathologies they reported were hemocytic infiltrates in the retina, in connective tissues around the tegumental glands, and within the muscular interstitia of eyestalks. In blue crabs infected by *P. perniciosa*, Johnson (1977) reported amoebae within the glia and in the hemal spaces of infected crabs. The eyes were not examined in that study, but nerve cells were invaded and damaged by the parasite. However, the eyes of a blue crab that was infected by *P. perniciosa* did not show damage comparable to that seen in lobsters (Maniscalco and Shields, personal observation). The type of nerve pathology associated with paramoebiasis (Mullen et al., 2004) was not observed in the present study. Furthermore, no amoebae were seen in any tissues from lobsters in the current study, including the skin, nerve, hepatopancreas, heart, gill, antennal gland, and eyestalks. Because there was no evidence that the lobsters in this study had
amoebic infections, and no mention of the lesions was made in prior studies of paramoebiasis, it is doubtful that Paramoeba sp. caused the lesions in H. americanus eyes.

Microbial agents can cause eye damage in crustaceans. Smith (2000) reported suppurrative inflammation (edema, hemocyte infiltration, and localized abscesses) in the eyes of the shrimp Penaeus monodon with vibrosis and viral infections. The abscesses, containing necrotic and pyknotic cells were frequently found in the dippotric (ommatidia, cornea, and lens) region. There were also granulomas affecting the ommatidia, ganglia, and other internal structures of the eye (Smith, 2000). Malacia, characterized by necrosis of nervous tissue, vacuolation, and vascular proliferation in the medullar ganglia was also observed. Callinan et al. (2003) reported on a disease, peripheral neuropathy and retinopathy (PNR), that caused minor to heavy mortalities in P. monodon on an Australian shrimp farm. In affected shrimp, peripheral nerve axons and their sheaths were degenerated and necrotic, with apoptosis of associated glial cells, and the reticular cells and their axons were degenerated and necrotic. In addition, the retinular cells that were not destroyed were often fused together. Edema occurred in the fasciculated zone and the lamina ganglionaris, and dilated blood vessels with hemocyte aggregations were present in the fasciculated zone, replacing the necrotic axons (Callinan et al., 2003). In advanced cases, there was fragmentation and liquefaction of the crystalline tracts overlying the necrotic retinular cells. The pathology reported by Callinan et al. (2003) is very similar to the pathology in the eyes of the lobsters from LIS. The fasciculated zone is proximal to the basement membrane, where the optic nerve fibers occur in American lobsters, and the increased vascularization observed in the region by Callinan et al. (2003) is similar to what was observed in the optic nerve region of LIS lobsters.

Although the reported pathologies in these two studies appear similar, it is doubtful that the lobsters in this study had viral infections. Unlike the shrimp in the other study, most lobsters in the present study were not moribund and did not present with any signs of disease. There was no microscopic evidence of viral, bacterial, or protozoan pathogens in the lobsters. In fact, other tissues from the affected lobsters, including the eyestalk ganglia, showed no pathological alterations, with the exception of granuloma formation, which was not associated with the severity of the lesions in lobster eyes. However, no specific stains for viruses or bacteria were utilized.

Excretory calcinosis, a disease of LIS lobsters (Dove et al., 2004), can also be ruled out as the causal agent for the eye lesions. Lobsters from the current study did not exhibit the lethargy and orange discoloration associated with excretory calcinosis, nor did they exhibit coagulopathy. Microscopically, the gills and antennal glands of affected lobsters from this study did not have the multifocal or diffuse granulomatous inflammation that is characteristic of excretory calcinosis.

Light damage, although possible, is an unlikely etiological agent for the idiopathic lesions observed in H. americanus. To date, there are no reports of light damage in the eyes of American lobsters and Shelton et al. (1985) state that it does not occur in this species. In Norway lobsters, N. norvegicus, with light damage, both the left and right eyes of the same animal were consistently damaged (Shelton et al., 1985). However, in at least five H. americanus from the current study, one eye had lesions while the other did not. In addition, the center of the retina is always affected in Norwegian lobsters exhibiting light damage (Shelton et al., 1985); whereas, in the present study, the central region was not always affected and the ventral region, that most shaded from light, was affected almost as frequently as the central region. Finally, 44% of lobsters collected in LIS for the present study had no damage to their eyes and they were collected from the same habitat as those with damage.

Chemical exposure is a possible cause of lesions in the eyes of American lobsters from LIS. Long Island Sound is not a pristine environment, and its inhabitants are exposed to a wide range of chemical contaminants (Robertson et al., 1991). Biggers and Laufers (2004) reported the presence of alkylphenols in the hemolymph and tissues of H. americanus from LIS. These compounds are juvenile hormone (JH) analogs in bioassays, and may result in serious toxic and endocrine-disrupting effects in lobsters. Because JH analogs are believed to increase respiration in crustaceans, it is possible that the alkylphenols contributed to the lobster mortality in LIS by making the lobsters more susceptible to hypoxia (Biggers and Laufers, 2004). However, it is unlikely that JH analogs contributed to the idiopathic lesions reported here, although they may affect the x-organ sinus-gland complex by stimulating molting.

In recent years, malathion, an organophosphate pesticide, has been used in the LIS watershed to combat the spread of mosquitoes carrying West Nile Virus. Methoprene, another pesticide used in the region, was found to bioaccumulate in the eyestalks of adult lobsters, much more so than in other tissues that were examined (Walker et al., 2005). Although immunotoxicity studies have been done on malathion (DeGuise et al., 2004; Mullen et al., 2004) and methoprene (Walker et al., 2005) exposure in lobsters, no histopathological work has been done. Therefore, chemical intoxication cannot be ruled out as an etiological agent for these idiopathic lesions in the eyes. In addition, manganese, which is released from sediments during periods of hypoxia (Hernroth et al., 2004), has been found to accumulate in the nervous tissue and hemolymph of exposed N. norvegicus (Baden and Neil, 1998). Manganese exposure in lobsters results in hemocytopenia, reduction in degranulation responses of hemocytes, and blocking of the prophenoloxidase system (Hernroth et al., 2004), thereby suppressing the immune system of lobsters. Manganese exposure through hypoxia could potentially play a role in the presence of idiopathic lesions in H. americanus.

Lobsters from Long Island Sound have faced a number of threats in the past few years, including the paramoebiasis that likely contributed to the 1999 mortality event, elevated bottom water temperatures, and introduction of pesticides.
and other pollutants into the Sound. The idiopathic eye lesions described in this paper are likely to be indicators of the continuous exposure to stressors faced by lobsters in Long Island Sound.

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References


