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Idiopathic Lesions and Visual Deficits in the American Lobster (*Homarus americanus*) From Long Island Sound, NY

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Abstract. In 1999, a mass mortality of the American lobster (*Homarus americanus*) occurred in western Long Island Sound (WLIS). Although the etiology of this event remains unknown, bottom water temperature, hypoxia, heavy metal poisoning, and pesticides are potential causal factors. Lobsters from WLIS continue to display signs of morbidity, including lethargy and cloudy grey eyes that contain idiopathic lesions. As the effect of these lesions on lobster vision is unknown, we used electroretinography (ERG) to document changes in visual function in lobsters from WLIS, while using histology to quantify the extent of physical damage. Seventy-three percent of lobsters from WLIS showed damage to photoreceptors and optic nerve fibers, including necrosis, cellular breakdown, and hemocyte infiltration in the optic nerves, rhabdoms, and ommatidia. Animals with more than 15% of their photoreceptors exhibiting damage also displayed markedly reduced responses to 10-ms flashes of a broad-spectrum white light. Specifically, maximum voltage (V_{max}) responses were significantly lower and occurred at a lower light intensity compared to responses from lobsters lacking idiopathic lesions. Nearly a decade after the 1999 mortality event, lobsters from WLIS still appear to be subjected to a stressor of unknown etiology that causes significant functional damage to the eyes.

Introduction

The American lobster, *Homarus americanus* (H. Milne Edwards, 1837), is the basis of an important commercial

fishery on the east coast of the United States. In 2007, landings were 34,107 metric tons and were valued at \$349 million (National Marine Fisheries Service, 2008). Historically, 16% of the total lobster landings were in Long Island Sound (LIS). Within LIS, 50%–60% of that catch was taken in western Long Island Sound (WLIS) (Fig. 1) (Howell *et al.*, 2005). In the fall of 1999, a mass mortality of lobsters occurred in WLIS, which markedly reduced landings. By 2006 the fishery in LIS contributed only 5% of the total landings on the east coast of the United States. The etiology of the mortality remains unknown but, elevated bottom water temperature, hypoxia, heavy metal poisoning, increased pesticide use, and an outbreak of the facultative parasite *Neoparamoeba* sp. have all been implicated as causal or contributory agents (Mullen *et al.*, 2004; Pearce and Balcom, 2005; Maniscalco and Shields, 2006). Lobsters suffering mortality exhibited cellular infiltrates in the central and peripheral nervous system, immune system depression (Deguise *et al.*, 2005; Factor *et al.*, 2005), and alteration to the architecture of the compound eye (Mullen *et al.*, 2004; Maniscalco and Shields, 2006). Moribund lobsters were found to harbor infections of a species of *Neoparamoeba* associated with hemocyte infiltration in nerves and ganglia (Mullen *et al.*, 2004), though no direct correlation has been found between the mass mortality event and infections with this species.

After the 1999 mortality event in the WLIS, surviving lobsters continued to display lesions of varying intensities within the compound eye. Surveys conducted in 2001 and 2004 showed the prevalence of lesions in the eyes or eye-stalks to be over 50% (Maniscalco and Shields, 2006). Afflicted lobsters exhibited clumping of eye pigment, damage to the basement membrane of the ommatidia, and ne-

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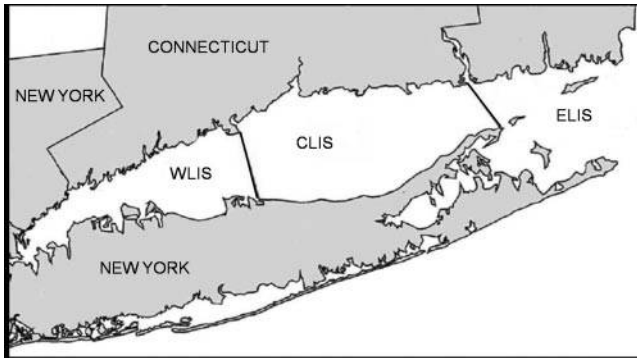


Figure 1. Map depicting the western (WLIS), central (CLIS), and eastern (ELIS) portions of the Long Island Sound. All animals from within the Sound were collected from WLIS. Figure provided by C. Giannini, Connecticut Department of Environmental Protection, and adapted from the *Journal of Invertebrate Pathology*, Vol. 91, A. M. Maniscalco and J. D. Shields, Histopathology of idiopathic lesions in the eyes of *Homarus americanus* from Long Island Sound, pp. 88–97, ©2006, with permission from Elsevier.

crisis of the ommatidia and optic nerve fibers. In extreme cases, the basement membrane was disrupted and hemocytes had infiltrated into the ommatidia region, with a loss of the general internal structure and morphology of the eye (Maniscalco and Shields, 2006). The lesions in the eyes of lobsters are of unknown etiology; hence the term idiopathic. More importantly, until now, it was not known whether the lesions affected vision. As in other crustaceans (Cronin, 1986; Warrant and Nilsson, 2006), lobsters have a reflecting superposition eye that is highly sensitive to light (Barnes and Goldsmith, 1977; Warrant, 1999). In this type of compound eye, individual ommatidia collect light, and the output from individual photoreceptors is then integrated into an image in the brain. Any change to the compound eye due to damage may adversely affect how the eye collects sensory information (Loew, 1976; Nilsson and Lindström, 1983).

Vision is not the primary sensing mechanism for *H. americanus*, which relies on its chemosensory and mechanosensory organs to perform complex social behaviors (Johnson and Atema, 2005), foraging (Breithaupt *et al.*, 1999), and social interactions (Breithaupt and Atema, 2000). Although considered supplemental in lobsters, vision may function in obstacle avoidance (Snyder *et al.*, 1992) and prey capture (Shelton *et al.*, 1985). However, even with light-induced damage to their superposition eyes (Chapman *et al.*, 2000), adult Norway lobsters (*Nephrops norvegicus*) exhibit no change in survival and growth. Combined with other sensory mechanisms, however, vision is a powerful tool to acquire information from the environment, and any diminution can significantly affect how an organism collects external sensory information (Meyer-Rochow, 1994). Degradation of the visual system in *H. americanus* may have been associated with the lobster mortality event in WLIS observed in 1999. However, if these two events are unre-

lated, lobsters from LIS may still be undergoing significant environmental stress.

Our study objective was to document changes in the visual response of lobsters from WLIS possessing idiopathic lesions by comparing their responses to those caught outside WLIS and lacking such lesions. We used electroretinography (ERG) to measure changes in the gross retinal response and used matching histological methods to assess the extent and intensity of idiopathic lesions. We then correlated the retinal responses with the extent of the associated histological damage.

Materials and Methods

Lobsters (*Homarus americanus*) were collected from western Long Island Sound (WLIS), as well as Rhode Island (RI), and Virginia (VA) in 2006 and 2007 by using standard pot gear. All lobsters were shipped to the Virginia Institute of Marine Science and maintained in a 500-liter recirculating tank chilled to 15 °C for a minimum of 2 weeks before use in an experiment. Holding conditions included a 12:12-h light-to-dark cycle for all lobsters in the study. During this period, they were fed squid (*Loligo sp.*) *ad libitum*. In total, 20 lobsters were processed: 14 from WLIS, five from VA, and one from RI.

To eliminate any bias in assessing results, ERG experiments were performed without prior knowledge of the condition of the eyes. Lobsters were placed in a custom-designed acrylic plastic chamber, restrained on a small platform, and allowed to dark-adapt for 30 min. The chamber was supplied with chilled (15 °C), aerated seawater with the water level maintained about 1 cm below the eyes of the lobster. The eye to be tested was immobilized at the base of the eye stalk by using quick-setting glue. Platinum-wire needle electrodes (Grass Technologies, Warwick, RI) were used to record retinal responses. The recording electrode was inserted perpendicular to the cuticle surface at a point about 2 mm back from the distal end of the eyestalk, such that the electrode was posterior to but in proximity to the photoreceptive cells. The reference electrode was inserted into the dorsal musculature between the pleomeres of the abdomen. Responses were amplified 10,000 times and filtered at 200-Hz high-pass and 3-Hz low-pass using a DAM-50 amplifier (Tucker-Davis Technologies, Gainesville, FL). The responses were further conditioned using a Humbug active electronic filter to remove 60-Hz noise (Quest Scientific, North Vancouver, BC, Canada). To minimize ambient electrical noise, the amplifier and the chamber holding the lobster were both placed in a Faraday cage grounded to the seawater with a stainless steel bolt. Data digitization (2-kHz sampling rate) and stimulus presentation were controlled using Bio-Sig software ver. 2.22 (Tucker-Davis Technologies).

ERG responses were recorded to 10-ms flashes of broad-

spectrum white light in increasing light intensities (I , in log candela m^{-2}). The light source (model SL2420, Advanced Illumination, Rochester, VT) consisted of 20 white-light-emitting diodes that created a 3.5-cm uniform field. Light intensity was regulated using an intensity controller (model CS410, Advanced Illumination), which in turn was governed by the voltage output of a digital-to-analog signal from a programmable attenuator (model PA5, Tucker-Davis Technologies), controlled by Bio-Sig software (Tucker-Davis Technologies). The range of light output was expanded through the use of neutral density filters (Kodak Optical Products, Rochester, NY). Light intensities ranged from $3.16 \times 10^1 \text{ cd cm}^{-2}$ to $6.31 \times 10^3 \text{ cd cm}^{-2}$. V-log I response curves were constructed from the ERG responses to 10-ms-duration light flashes, which increased in steps of 0.1 log unit from light intensities producing no measurable response to intensities producing a maximum response. Light stimuli were presented every 5 s, and an average was calculated for five ERG responses at each light intensity. Data were analyzed as either a raw voltage or normalized as a fraction of the maximum response observed in each experiment.

After the ERG experiments, lobsters were euthanized with an overdose of cold saturated potassium chloride injected into the ventral nerve cord, as in Battison *et al.* (2000). The animals were assessed for any signs of external damage, then the eyes were removed at the base of the eyestalk, using a scalpel. The eyes were placed in Bouin's fixative for 48 h and stored in 70% ethanol. They were subsequently decalcified overnight using a sodium-citrate-EDTA decalcifying solution (Luna, 1968), cut in half with a razor blade, dehydrated, cleared, and infiltrated with paraffin wax. Tissues were embedded in paraffin wax blocks, cut in sections of 5–6 μm , and stained with Mayer's hematoxylin and eosin (Luna, 1968).

Sections were examined using a compound light microscope and photographed with an attached digital camera (Nikon DM1200, Nikon Inc., Melville, NY). Areas of specific interest included the ommatidia and rhabdoms, basement membrane of the ommatidia, optic nerve fibers, and connective tissues near the lamina ganglionaris. One eye from each lobster was selected for further analysis. Specifically, the basement membrane between the ommatidia and the optic nerve fibers was measured using a calibrated optical micrometer at $10\times$ magnification, where one optical unit on the micrometer equaled 98.9 μm . The measurement of the basement membrane (a lineal arc) was used as a proxy for the effective surface area available to collect light. Each optical unit was assessed, and any apparent damage was documented. Data were recorded as the percentage of healthy tissue in the total arc of the basement membrane measured in each eye. Lobsters were classified as affected if more than 15% of the total measured surface area had some form of histological damage.

Paired histological and physiological data were analyzed using Minitab 15 (Minitab Inc., State College, PA), Microsoft Excel 2003 (Microsoft Corporation, Redmond, WA), and Systat 12 (Systat Software, San Jose, CA, USA). Linear regressions were used to quantify the relationship between the extent of histological damage and the ERG retinal responses. A repeated-measures ANOVA was used to assess any differences in V-log I responses between healthy and affected lobsters. Student's t tests were used to compare voltage data between healthy and affected lobsters. Values with P values less than 0.05 were considered statistically significant.

Results

Histological damage in the compound eye was quantified for 14 lobsters from western Long Island Sound, New York (WLIS). All lobsters were in good physical condition with no apparent exterior damage to the carapace or eyes. The average (\pm SEM) measurement of the basement membrane was 35.2 (\pm 2.35) mm, with 40.0 (\pm 0.08) ommatidia per millimeter.

The histopathology of the eyes of lobsters from WLIS was similar to that reported by Maniscalco and Shields (2006). Briefly, cellular change caused by lesions included changes to the cellular structure of the eye with necrosis of the optic nerves. Damage was not noticeable histologically within the lamina ganglionaris—the first large ganglion through which the optic nerves pass after leaving the basement membrane and photoreceptors. Histopathologic changes including necrosis were most apparent in the ommatidia, rhabdoms, basement membrane of the ommatidia, and optic nerve fibers anterior to the lamina ganglionaris. Pigment in the ommatidia lost its characteristic organization and uniform displacement (Fig. 2a). Pigment was often clumped and displaced from normal locations surrounding the optic nerves (Fig. 2b). The rhabdom lost its linear spindle shape (Fig. 3a), and the basement membrane often appeared ragged and degraded (Fig. 3b). The basement membrane, normally situated between the rhabdom and optic nerve fibers (Fig. 3a), was frequently disrupted or separated from the ommatidia. This separation allowed hemocytes to infiltrate through the basement membrane into the rhabdoms (Fig. 3b). In virtually all affected eyes, optic nerve fibers were degraded to differing degrees, ranging from localized damage of a few fibers to extensive damage encompassing all of the fibers. Normal healthy nerve fibers have a linear and fibrous organization in well-organized nerve tracts (Fig. 3a). In extreme cases, optic nerve fibers had lost their organized linear appearance and were necrotic, replaced with vascular tissue, or lost completely (Fig. 3b).

Idiopathic eye lesions were observed in 10 of 14 animals from the WLIS. Lesions occurred in the ommatidia region,

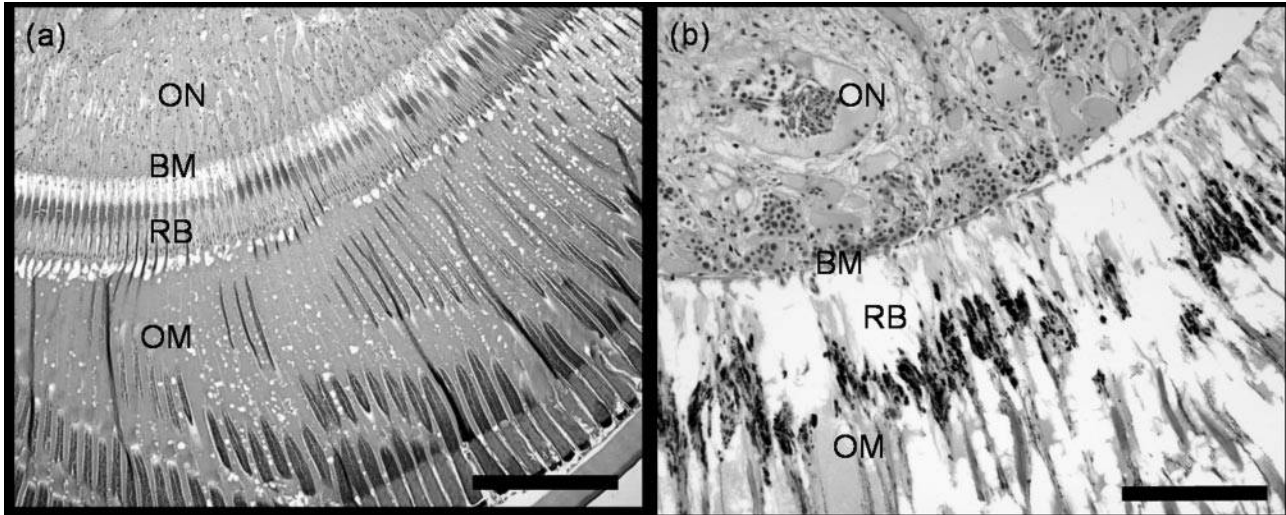


Figure 2. Distal portion of a healthy (a) and affected (b) eye stalk of *Homarus americanus*. Note that affected eye stalks lose general structure and often exhibit clumped pigment in the ommatidia (OM), breakdown of the rhabdom (RB) and basement membrane (BM), and necrosis of optic nerve fibers (ON). Damage was not noticeable histologically beyond the lamina ganglionaris (not shown) Scale = 500 μm (a), 250 μm (b).

basement membrane, and optic nerve fibers and were essentially identical to those previously reported by Maniscalco and Shields (2006). Four lobsters from WLIS and five lobsters from VA and RI had no (or only minimal) eye damage.

Physiological data were collected from the 10 affected and 9 unaffected lobsters. In all of these animals, ERG responses to light flashes increased with increasing light intensity (Fig. 4). Mean ERG responses of healthy and affected lobsters became statistically significantly divergent

at 2.1 log I light intensity units (repeated measures ANOVA, $P < 0.05$). Animals with healthy eyes responded to a greater range of light intensities and had a larger maximum voltage response (V_{max}) to the brightest experimental light intensity. Lobsters with eye lesions reached 100% of their V_{max} (*i.e.*, normalized voltage response) at a significantly lower light intensity compared to animals with healthy eyes (Fig. 5). Three lobsters from WLIS had 100% damage and showed no appreciable ERG responses to any of the experimental light intensities.

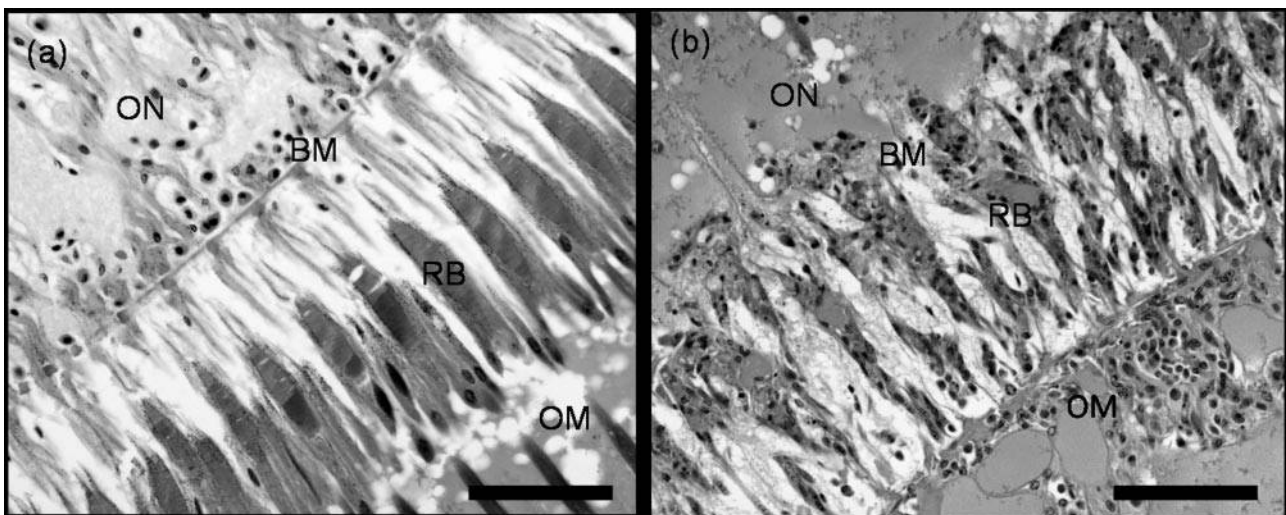


Figure 3. Magnified image of healthy (a) and damaged (b) rhabdom in the eye of *Homarus americanus*. A healthy rhabdom has a distinct spindle shape and forms a connection to optic nerve fibers through the basement membrane. Damaged rhabdom becomes ragged and loses its spindle shape. Optic nerve fibers become necrotic. Basement membrane is ruptured, allowing hemocyte infiltration into the photoreceptor space. Scale = 100 μm .

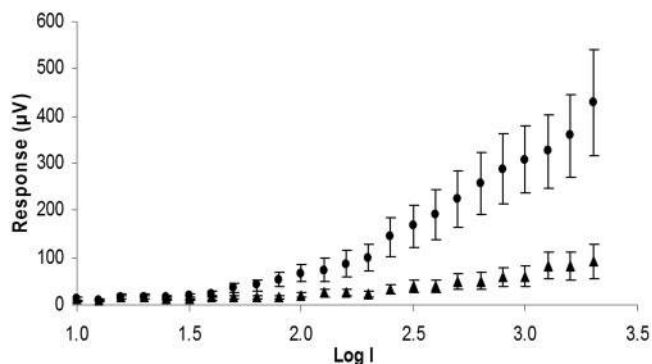


Figure 4. V-Log *I* response curves constructed from retinal responses (μV) to increasing light intensities (*I*, in log candela/ m^2) in *Homarus americanus*. V-log *I* curves were constructed using the mean value of five ERG responses to a 10-ms-duration flash of light increasing in 0.1 log unit steps from 0.0 (no measurable response) to 3.4 (maximum response). Light stimuli were presented every 5 s. Data are presented as voltage (μV). Data points are means \pm standard error. Circles indicate data from healthy lobsters; triangles indicate data from affected lobsters.

Healthy lobsters (<15% damage) had a significantly higher average V_{max} ($485 \pm 15 \mu\text{V}$; range: $198 \mu\text{V}$ – $1083 \mu\text{V}$) than lobsters with affected eyes ($79 \pm 27 \mu\text{V}$; range: $14 \mu\text{V}$ – $278 \mu\text{V}$) (*t* test, $P < 0.05$). One affected lobster had an unusually large response ($278 \mu\text{V}$ maximum response, shown by the filled circle in Fig 6), though only 32% of the observed ommatidia in the eye was deemed healthy. All other responses from affected lobsters were below $155 \mu\text{V}$ (Fig. 6). For lobsters with affected eyes, the fraction of undamaged ommatidia was not significantly correlated with the physiological response data ($y = 0.213x + 71.244$, $R^2 = 0.0063$, $S = 89.288$) (Fig. 6).

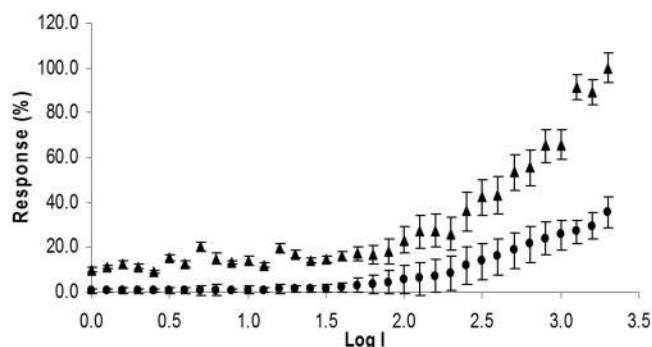


Figure 5. V Log *I* curves constructed from retinal responses (%) to increasing light intensities (*I*, in log candela/ m^2) in *Homarus americanus*. V-log *I* curves were constructed using the mean value of five ERG responses to a 10-ms-duration flash of light increasing in 0.1 log unit steps from 0.0 (no measurable response) to 3.4 (maximum response). Light stimuli were presented every 5 s. Data presented were normalized as a fraction of the maximum observed response for each experiment. Each data point is a mean \pm standard error. Circles indicate data from healthy lobsters; triangles indicate data from affected lobsters.

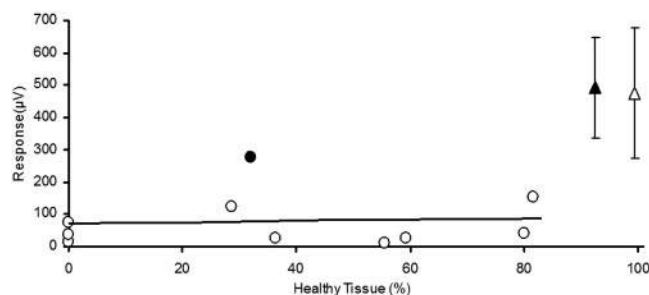


Figure 6. Data correlating maximum voltage response (μV) to the percentage of healthy eye tissue (%) in *Homarus americanus*. *T*-test analysis shows no difference between healthy animals within (open triangle) and outside (closed triangle) Long Island Sound, New York. There is also no relationship between V_{max} and the percentage of healthy tissue in lobsters with eye lesions (circles). $y = 0.2134x + 71.244$, $R^2 = 0.0063$, $P > 0.10$.

Discussion

Electroretinograms from lobsters with greater than 15% damage indicated that retinal responses were reduced both in maximal response (V_{max}) at the brightest light intensity and in percent maximum response over the V-log *I* response curve. Crustaceans with a loss of some visual function would not necessarily be blind, although vision would be greatly reduced (Meyer-Rochow, 2001). Superposition compound eyes act as light collectors and integrate an image over the entire working area of photoreceptors (Wald, 1968; Cronin, 1986; Cronin and Marshall, 2001). The advantage of a superposition eye is its sensitivity at lower light intensities, albeit at the expense of speed of vision (Warrant *et al.*, 1996; Warrant, 1999; Warrant and Nilsson, 2006). Damage to the eye can cause a loss in sensitivity. Therefore, if a lobster loses any portion of its photoreceptors, it will lose not only light-collecting organs, but also some of the neural architecture necessary for integrating images. In extreme cases (*i.e.*, 100% damage), lobsters would be completely blind as there would be no working photoreceptors to collect sensory information or optic nerves to process and integrate photoreceptor information.

A reduced V_{max} may indicate that a reduced number of photoreceptors successfully collected sensory information, and that small amounts of histological damage can cause a drastic reduction in function. Interestingly, lobsters with eye lesions also reached V_{max} at lower light intensities compared to lobsters without lesions. Fewer functioning photoreceptors likely photosaturate at lower light intensities and, therefore, would be unable to collect sensory information at brighter light intensities. Our research results confirm conclusions of Maniscalco and Shields (2006) that lesions cause pronounced changes to vision in *Homarus americanus* and that lobsters with these lesions still exist in WLIS.

We have two reasons for believing that light-induced damage was not a factor causing lesions and reduced visual

sensitivity in WLIS lobsters. First, light-induced damage has not been reported in *H. americanus* (Goldsmith and Bruno, 1973), unlike that reported in Norway lobster (*Nephrops norvegicus*) (Loew, 1976; Meyer-Rochow, 1994). Second, all the lobsters used in our experiments were exposed to light intensities (3.16×10^1 cd cm⁻² to 6.31×10^3 cd cm⁻²) similar to those encountered throughout the distributional range of *H. americanus*. Holding conditions also included a 12:12-h light-to-dark cycle. Control lobsters, therefore, experienced the same light levels as lobsters from WLIS but exhibited no evidence of damage to their eyes.

Judging from their carapace length, lobsters used in our study were about 5 years old (Hughes and Matthiessen, 1962; Hartnoll, 2001). All animals met minimum market requirements. We are therefore confident that none of the lobsters in the study was present during the 1999 WLIS mortality event and that the lesions we discovered in this study were the result of a stressor still present in WLIS.

Similar eye lesions have been reported in another crustacean, the Australian prawn *Penaeus monodon* (Smith, 2000; Callinan *et al.*, 2003), but the condition in this species was linked to a viral infection related to Australian gill-associated virus (GAV). GAV typically causes lethargy, shell discoloration, and retinopathy including necrosis of the eye and optic nerves. Though the GAV lesions and eye damage appear similar to those observed in our study, the lobsters from WLIS showed no lethargy or shell discoloration and were generally in good health prior to the start of any experiments. A few of the lobsters from WLIS did possess cloudy grey eyes (Magel and Shields, pers. obs.), and subsequent analysis showed that these eyes had extreme damage.

Conclusions from the initial lobster mortality reports in 1999 suggest that hypoxia and pesticides were major factors in the mortality of *H. americanus* in WLIS (Pearce and Balcom, 2005). Hypoxic events can leach heavy metals (*i.e.*, Mn²⁺ and Co²⁺) from pore waters, and the accrual of these metals has been suggested as a proxy for exposure to hypoxic water that may affect lobsters, especially those within WLIS (Draxler *et al.*, 2005a, b). Manganese (Mn²⁺) is readily found in tissues of *H. americanus* and *N. norvegicus*, and due to elimination kinetics may be an indicator of hypoxia. Mn²⁺ affects the chemosensory organs of *N. norvegicus* and its food-locating ability (Krång and Rosenqvist, 2006), but it is not known whether Mn²⁺ affects other sensory mechanisms in this species or in *H. americanus*. Furthermore, Mn²⁺ can also accumulate in the nerves of *N. norvegicus* exposed to hypoxia (Baden and Neil, 1998). In high concentrations, Mn²⁺ and other heavy metals may also affect escape behavior in *N. norvegicus* (Baden and Neil, 1998). Though the specimens of *H. americanus* examined in our study were not tested for high Mn²⁺ concentrations, heavy metals like Mn²⁺ may affect the sensory mechanisms

of this species, although the role (if any) of these metals in the degradation of optic nerves remains to be determined.

During 1999, pesticides were used to control mosquitoes in the LIS region to thwart outbreaks of West Nile virus (Pearce and Balcom, 2005). Pesticides have been linked to increased stress and mortality in lobsters (Burrige and Haya, 1997; Burrige *et al.*, 1999; Walker *et al.*, 2005). Specifically, methoprene affects juvenile hormone agonists and cuticle production and post-molt shell quality in adult lobsters (Walker *et al.*, 2005). Methoprene also accumulates in the hepatopancreas, gonad, epithelial tissue, and eyestalk nerve tissue (Walker *et al.*, 2005). Pesticides are volatile and may occur below detection limits (De Guise *et al.*, 2004). However, no appreciable levels of methoprene or other pesticides were detected in WLIS after the 1999 lobster mortality event (Zulkosky *et al.*, 2005). Although biochemical analyses were not conducted to confirm any accumulation of pesticides in our research, pesticides cannot be ruled out as a potential cause of damage and lesions in the eyes of *H. americanus*.

Acknowledgments

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