LONG ISLAND SOUND

Lobster Health Symposium

Immune & Endocrine Compromise in American Lobsters

As cold-blooded animals, the metabolism of lobsters is extremely sensitive to prevailing environmental conditions. Here in Long Island Sound (LIS) at the southern edge of their inshore range, this is especially true for temperature, which is at the upper margin of their tolerance limits in any given summer. During unusually warm summers, lobsters may become stressed when their temperature tolerance is exceeded, and lose their internal equilibrium. This effect is exacerbated during low oxygen concentrations in sea water (hypoxia), which tend to occur at the same time as temperature is at its highest and, therefore, when the lobsters need oxygen the most. The responses of lobsters to this sort of stress could include death directly from the environmental stressor, reduced immunity to disease and disruption of hormonal and other systems that lobsters use to maintain their equilibrium. At the beginning of this research, we lacked the research tools necessary to explore these issues, and significant time and effort was devoted to develop a tool kit of laboratory techniques that we could use to answer these questions. Application of these tools, similar to many medical and veterinary tests, showed that lobsters are indeed stressed by the environmental conditions in LIS in late summer. They show reduced immune function, disrupted hormone systems (including the molting hormone) and, when temperature and hypoxia combine, can die directly from environmental stress. These results suggest that environmental inputs can have a dramatic effect on lobster health and survival both directly and indirectly, by reducing their ability to fight infectious pathogens and tolerate chemical contamination.

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Disease in American Lobsters

A small group of bacteria are consistently identified in lesions of lobsters infected with epizootic shell disease, and this form of the disease has characteristic gross and microscopic features that distinguish it from shell disease of impoundment lobsters. There is evidence of an appropriate inflammatory response and healing in lobsters with epizootic shell disease, but when the carapace is breached (ulcerated), internal lesions may lead occasionally to death. The research indicates that shell disease is not a cause for lobster mass mortalities.

A subpopulation of lobsters from the 1999 die-off were infected with paramoebae without consistent evidence of other disease-causing infectious agents, and this fact must be accounted for in an understanding of that mortality event. Lobsters since the die-off lobsters have been identified as infected with paramoebae, although at much reduced levels. Genetics of the paramoebae from 1999 and 2000-2002 lobster populations indicate they are strains of *Neoparamoeba pemaquidensis*, which has been demonstrated to be an environmental inhabitant of LIS and a facultative parasite in sea urchins and salmon, although recent attempts to recreate infection using a strain of *Neoparamoeba pemaquidensis* isolated from sea urchin were unsuccessful. One possible explanation for the presence of neoparamoebae in 1999 lobsters is to postulate that high water temperatures, which are known to support the growth of natural populations of *N. pemaquidensis* and other amoebae, combined with record-high population densities of temperature-challenged lobster under hypoxic conditions, promoted infection of a subpopulation of lobster that contributed to the die-off.

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