Suffolk County Vector Control & Wetlands Management Long Term Plan & Environmental Impact Statement

Task 3 Literature Review
Book 8: Marine and Non-Target Impacts
Part 2: Mosquito Control Pesticides and Lobster Mortality

Prepared for:

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SUFFOLK COUNTY VECTOR CONTROL AND WETLANDS MANAGEMENT LONG - TERM PLAN AND ENVIRONMENTAL IMPACT STATEMENT

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LIST OF ACRONYMS

CTDEP	Connecticut Department of Environmental Protection
LIS	Long Island Sound
LISS	Long Island Sound Study
MF	Methyl farnesoate
NOAA	National Oceanic and Atmospheric Administration
NYSDEC	New York State Department of Environmental Conservation
PBO	Piperonyl butoxide
SUNY	State University of New York
UCONN	University of Connecticut
USEPA	US Environmental Protection Agency
WNV	West Nile Virus

EXECUTIVE SUMMARY

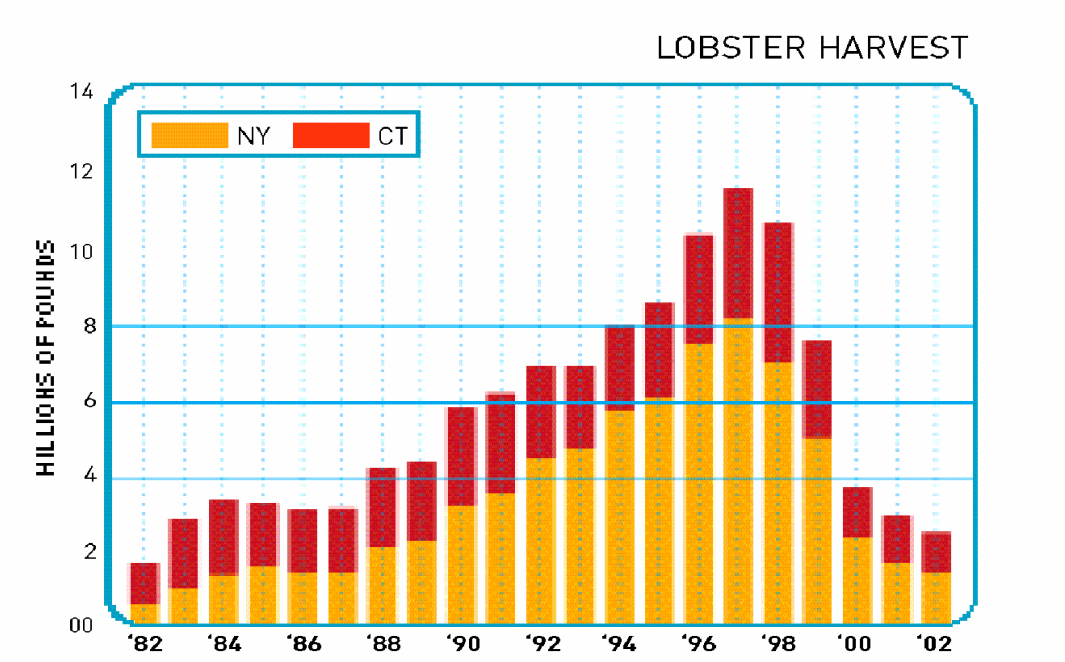
A legal case has been filed against Suffolk County that alleges that the use of pesticides to control a mosquito-borne West Nile Virus (WNV) outbreak in 1999 was responsible for a concurrent die-off of the lobster fishery in western Long Island Sound. Federal and state funds have been made available to researchers to examine the causes of the die-off separately from the legal matter. New York and Connecticut Sea Grants have organized a Long Island Sound Lobster Research Initiative, which sponsored targeted research on the issues of concern, and hosted symposia to disseminate preliminary research results.

The reports from the sponsored research make clear the difficulty of identifying a single causative event, agent, or environmental factor related to the mortality. Although lobsters are sensitive to mosquito control pesticides under laboratory conditions, it is not yet known whether they are exposed to these pesticides in deleterious concentrations. Most indications from modeling exercises are that critical concentrations were not reached, especially in the more central areas away from the shorelines. Modeling was the only means of determining the level of exposure, since water samples were not taken at the time of the die-off. There is strong evidence that other factors have had negative effects on lobster populations. They include elevated water temperature, low dissolved oxygen concentrations, storm-related salinity effects, and possibly diseases such as shell disease, calcinosis, and parasites. These factors, alone or combined, may have been the primary or secondary agent responsible for the population decline. The 1998 fishery data can be interpreted as showing a decrease in the lobster population, which was prior to the increased use of mosquito control pesticides to combat the initial outbreak of WNV in September of 1999. However, the large-scale population die-off in western Long Island Sound was not noticeable until 1999. To date, the fishery, especially in the western Long Island Sound, has not recovered.

Most research cited for this report is preliminary. A summary conference for the research initiative was held on October 4, 2004, at Stony Brook University. Although the reports have not yet been finalized or published, the consensus of the researchers was that the mosquito control pesticides were not the cause of the die-off. It is likely no single cause was responsible, however, environmental factors seem to be the catalyst of the mortalities.

1. Introduction

Lobstering in the Long Island Sound (LIS) has a long history dating back to the colonial times. LIS lobster harvests reached record highs by the late 1990s (Figure 1-1). At that time, the lobster industry supported approximately 760 lobstermen in New York and Connecticut (Haberstroh, 2002). Annual revenues were estimated at approximately $100 million. However, the steep rise in lobster harvests was followed by a similarly steep decline in catches (LISS, 2003a).



Long Island Sound Study, Sound Health 2003

Figure 1-1 – Lobster Harvests 1982 - 2002

Industry harvests dropped from more than 10 million pounds in 1998 to 3.6 million pounds in 2000 and to only 2.5 million pounds in 2002 (Gimple, 2003). Although there had been a decline in landings from 1997 to 1998, 1999 was marked by widespread discoveries of dead and dying lobsters in the traps and the decrease in sales was dramatic. Sales of lobsters in western LIS[1] fell to almost zero, while the decline in central and eastern LIS was approximately 30 percent of 1998 levels. The lobster die-off led the states of New York and Connecticut to submit

[1] Western LIS, technically, is the narrow portion of LIS found west of the New York City line. The eastern portion of LIS is found east of the Mattituck Sill. Practically, these definitions are more fluid, and no grave error would ensue if the reader understood LIS to be composed of approximately equal thirds, designated western, central, and eastern LIS.

documentation to the Department of Commerce requesting a fishery disaster declaration, which was subsequently granted by the Secretary of Commerce. This declaration made the LIS lobster fishery a marine resource disaster (Clemetson, 2001).

According to a Connecticut Department of Environmental Protection (CTDEP) update at the 4th Lobster Research Initiative Symposium of 2004, the industry has not recovered from the die-off and what may be a coincidental outbreak of shell disease. Although the die-off hit western LIS harder than eastern LIS, lobsters in the eastern LIS were afflicted by shell disease, an unsightly but relatively benign condition that reduces market value because of consumer resistance. The percentage of fishing trips with dead lobsters in traps was three percent in 2003, compared to 14 percent in 2002. The incidence of shell disease was 31 percent in eastern LIS, 7 percent in central LIS, and only 0.8 percent in western LIS. Although no legal size female lobsters were taken in the Narrows, the area in the far western end of LIS hardest hit by mortalities, in 2001 and 2002, 33 percent of lobsters collected there in the fall of 2003 were female. However, none of the females were egg bearing, as had also been the case in 2001-2002. Lobster migration data showed that lobsters on average travel less than 10 km, suggesting that migration from outside of the area is unlikely to address the current woes.

Reporting by the New York Department of Environmental Conservation (NYSDEC) for 2003 at the same conference echoed the CTDEP data. Catches in all areas declined sharply in late August - early September 2003, which was coincident with a decline in dissolved oxygen. When oxygen levels rose in late September, so did the catch. On Long Island's north shore, the incidence of shell disease has increased substantially, from four percent in 2000 to 19.6 percent in 2003. Total New York LIS lobster landings declined in 2003 to levels of the early 1980s.

Some lobstermen link the decline of the lobster population to the use of mosquito-control pesticides by Suffolk County. Three lobstermen from Connecticut and New York filed a lawsuit in August 2000 against various manufacturers of pesticides that were sprayed in the New York metropolitan area in 1999 to combat the outbreak of West Nile Virus (WNV), which first appeared in August, 1999. The $125 million case alleges that pesticides were responsible for the lobster decline (*John Fox et. al. vs. Cheminova et. al*. CV 00-5145(TCP) (E.D.N.Y. Feb. 28, 2003)). The lobstermen suggest that the stormwater runoff from a summer tropical storm

introduced mosquito control pesticides into the Sound, which lead to the lobster mortalities. The case achieved class action status in 2003.

Because of the tremendous loss of the fishery, funding was made available from a variety of sources to see if the cause of the problem could be determined. The following State and Federal agencies are currently supporting research into LIS lobster mortalities:

- The Environmental Protection Agency (USEPA)
- National Oceanic and Atmospheric Administration (NOAA)
- Connecticut Department of Environmental Protection (CTDEP)
- New York State Department of Environmental Conservation (NYSDEC)
- New York and Connecticut Sea Grant (Sea Grant)
- The University of Connecticut (UCONN)
- The State University of New York (SUNY)

The sum of research efforts investigating the causes of the dramatic decline in the Long Island lobster population has been named the 'Long Island Sound Lobster Research Initiative.' The research focus of each of the laboratories is listed in Table 1-1; research summaries are listed in the Appendix.

Table 1-1 - Laboratories Conducting Lobster Mortality Research

IMMUNOLOGY & ENDOCRINOLOGY			
PRINCIPAL INVESTIGATOR	**DEPARTMENT**	**INSTITUTION**	**RESEARCH FOCUS**
Robert S. Anderson	Chesapeake Biological Lab	University of Maryland	Immunological Health of Lobsters
Ernest S. Chang	Bodega Marine Laboratory	University of California, Davis	Environmental and Physiological Stresses in Lobsters: Effects on Crustacean Hyperglycemic Hormone and Heat-Shock Proteins.
Richard Robohm	Fisheries Laboratory	NOAA	Effects of Environmental Stressors on Disease Susceptibility in Lobsters: A Controlled Laboratory Study
PHYSICAL/CHEMICAL ENVIRONMENT			
PRINCIPAL INVESTIGATOR	**DEPARTMENT**	**INSTITUTION**	**RESEARCH FOCUS**
Hans Laufer	Molecular and Cellular Biology	UCONN, Storrs	Hormonal Responses of Lobsters to Stresses
Robert E. Wilson	Marine Sciences Research Center	SUNY at Stony Brook	Prevailing Water Column Conditions in Long Island Sound and the Relationship to Lobster Mortality Events
Glen Lopez	Marine Sciences Research Center	SUNY at Stony Brook	Effects of Temperatures and Body Size on Metabolic Stress in LIS Lobsters
SHELL DISEASE & PARAMOEBA			
PRINCIPAL INVESTIGATOR	**DEPARTMENT**	**INSTITUTION**	**RESEARCH FOCUS**
Salvatore Frasca, Jr.	Pathology	UCONN, Storrs	Development of Polymerase Chain Reaction- and *in situ* Hybridization-based Tests for the Specific Detection of the Paramoeba Associated with Epizootic Lobster Mortality by Determination of the Molecular Systematics of the Genus Paramoeba
MONITORING & ASSESSMENT			
PRINCIPAL INVESTIGATOR	**DEPARTMENT**	**INSTITUTION**	**RESEARCH FOCUS**
Carl LoBue	Marine Resources	NYSDEC	Fishery Dependant Monitoring of the Long Island Sound Lobster Resource
PESTICIDES			
PRINCIPAL INVESTIGATOR	**DEPARTMENT**	**INSTITUTION**	**RESEARCH FOCUS**
Anne E. McElroy	Marine Sciences Research Center	SUNY at Stony Brook	Effects of Pesticides of Lobster Health: Trace Level Measurements and Toxicology Assessment at Environmentally Realistic Concentrations
Sylvain DeGuise	Pathology	UCONN, Storrs	Malathion immunotoxicity in the American Lobsters, *Homarus americanus,* upon experimental exposure
Michael N. Horst	School of Medicine	Mercer University	Acute Effects of Methoprene on Survival, Cuticular Morphogenesis and Shell Biosynthesis in the American Lobster, *Homarus Americanus.*

2. Lobster Biology

Lobsters are invertebrates, part of the phylum Arthropoda (from the Greek, arthron, meaning joint and pous, meaning foot), animals with jointed appendages. Lobsters are in the class Crustacea because of their flexible shell (from the Latin, crusta). As they have ten legs, lobsters are placed in the order Decapoda (from the Latin meaning ten feet). Lobsters of the Long Island Sound are members of the species *Homarus americanus*. *H. americanus* are found in marine, coastal waters along the continental shelf from Labrador to North Carolina. The following sections on lobster biology, growth, and development were compiled from information provided in the Lobster Conservancy Organization's website (Lobster Conservancy Organization, 2004).

2.1. Anatomy

Lobsters have two primary body parts, the cephalothorax and the abdomen. The cephalothorax is comprised of the head and the thorax. The abdomen is the tail portion of the lobster. The lobster head contains the eyes, antennules, antennae, mandibles, and the first and second maxillae. The antennules have chemosensory organs. The antennae provide the lobster with touching sensation, while the maxillae act as the jaws or mouth of the lobster. The thorax contains the primary walking legs for the lobster. The abdomen contains the pleopods, which are used for reproduction.

Lobster organs come in pairs located on each side of the body (bilaterally symmetrical), with the exception of the heart. A lobster's heart is a single-chambered sac that contains striated muscles and openings called ostia. A blood sinus called the pericardium positions the heart. The blood, or hemolymph, which is clear in color, passes from the pericardium, through the ostia, and into the heart. Blood is pumped through the major arteries supplying the organs.

2.2. Respiration

Lobster respiration starts with water entering openings between the thoracic legs into two branchial chambers, which consist of forty gills, or twenty pairs. Water passes through the gills and out toward the lobsters' head, which allows oxygen to diffuse out of the water and into the

lobster. The gill bailer of the second maxillae causes this flow. The flow is reversed every few minutes to clear the gills of debris and flush the debris out of the branchial chamber.

2.3. Reproduction

During reproduction, the male transfers a spermatophore to the female. LIS lobsters mate while the female is soft shelled (after molting her hard shell). The male supports the female's body above the ground while he climbs on her and inserts his first pair of pleopods into the seminal receptacle of the female while the female is turned over. The female fertilizes the eggs using the spermatophore and carries them on her abdomen until they hatch the following spring or early summer. The hatched eggs are free-swimming larvae for approximately one month before permanently settling to the bottom.

2.4. Growth and Development

The lobster has four growth phases beginning with the larval phase and ending with the adult phase. Lobster life begins with the free-swimming larval phase, growing from one-sixth of an inch to about half an inch. This phase encompasses three larval stages, which are based on the size and development of the larvae. At end of the larval phase, the lobster goes through a metamorphosis into the postlarval phase, where it takes a form similar to an adult and begins living on the benthos. The postlarval phase is the onset of the juvenile phase where, like an adult, the lobster is a bottom feeder. Between each phase, the lobster sheds its exoskeleton. Lobsters grow by producing a new exoskeleton that is larger than the one it shed. Methyl farnesoate (MF) is a chemical that stimulates the release of the hormone ecdysone, which initiates the molting process by softening the shell. The lobster may molt up to ten times during their first growing season and only once or twice yearly as adults. After the juvenile stage, the adult lobster acquires its two distinct claws. Only during the adult phase does the lobster have the ability to reproduce.

2.5. Migration

Lobsters are found from the intertidal zone to a depth of up to 480 meters, but are most commonly found at depths ranging from four to 50 meters (Lobster Conservancy, 2004). Long Island Sound lobsters are "inshore" lobsters, whereas the "offshore" lobsters are found on the continental slope at considerably greater depths. Cooper (1977) suggested that approximately 20 percent of the offshore lobster population migrates the long distance to the warmer inshore waters in the summer and back to the deeper waters in the winter.

Lobsters migrate in large numbers in patterns that have been shown to depend in part on water temperature and salinity (Clauss, 2001; Watson *et al.*, 1999). Inshore lobsters typically have a short migration of 1 to 10 km (Campbell and Stasko, 1986) whereas offshore lobsters have been shown to migrate 100-150 km in shore and then back (Pezzack and Duggan, 1986). Tagging studies have shown an annual migration from offshore to inshore in the late winter and spring and a return migration in the late fall and winter (Cooper and Uzmann, 1971). Watson *et. al.* (1999) found that of the 1212 lobsters they tagged in the Great Bay estuary, most moved less than five km to or from the coast, with most movement into the estuary occurring in the spring, while the rest of the year lobsters moved toward the coast. The researchers postulated that lobster migration was driven by a desire to avoid low salinity events in the spring and fall, and to accelerate growth in warmer waters of the estuary in the summer. Watson *et al.* cite research by Thomas and White (1969) that demonstrated that "heavy mortalities occur during years with a large spring runoff.." The authors found that 2 percent of the tagged lobsters moved long distances, with 30 lobsters traveling more than 16 km. They cite other research that found lobsters able to travel over 100 km. The researchers found that the lobsters that migrated the farthest were those that were subjected to the greatest fluctuations in temperature and salinity. Large environmental fluctuations can sometimes result from storms. Jury *et al.* (1995) demonstrated that lobsters migrate offshore in response to large storm events. They suggested that the movement was in response to avoidance of low salinity.

3. Long Island Sound and Hypoxia

LIS is approximately 110 miles in length and 21 miles at its widest point. With an average depth of approximately 63 feet, the Sound's volume is approximately 18 trillion (18×10^{12}) gallons. It is frequently divided into eastern and western portions. The area adjacent to the Sound is home to over 8 million people. LIS is both directly and indirectly responsible for billions of dollars of business annually from activities such as recreational boating, commercial and sport fishing, swimming, and other shoreline activities (LISS, 2003a). Environmental, recreational, and business concerns were raised in 1985 when serious water quality declines were recognized. The Federal government along with the states of New York and Connecticut initiated a program known as the Long Island Sound Study (LISS). The LISS has funded research to determine the causes of the "Sound's most pressing environmental problems" (LISS, 2003a). The program has documented past and present quality indicators such as nitrogen pollution, hypoxia, toxic contaminants, and pathogens.

The environmental problem that has been highlighted by LISS as the most serious problem for the LIS is hypoxia, low levels of dissolved oxygen. The LISS has concluded that excess nutrients, such as nitrogen, results in eutrophication, high rates of high primary productivity, and that these conditions lead to hypoxia. . Excess nutrients may result in dense production, blooms, of microscopic floating plants known as phytoplankton. When the bloom abates, the microalgae sink to the bottom of the waterbody where their decomposition by bacteria utilizes dissolved oxygen. This oxygen usage by bacteria lowers dissolved oxygen in the waterbody, which can ultimately lead to hypoxia. A dissolved oxygen level of less than 3 milligrams per liter (mg/l) has been defined as hypoxic in the LIS (LISS, 1994). Cleorn (2001) points out that not all estuarine systems react similarly to excess nutrients, and that particular local factors determine the impact of the nutrient input.

Sewage effluent is seen as one easily controllable source of nitrogen inputs into LISS, and one that clearly is not a natural nutrient source for the region. There are 105 New York and Connecticut plants discharging treated sewage effluent into the Long Island Sound (IEC, 2004). Sewage effluent is high in nutrients, particularly nitrogen. Since 1994, the LISS has been implementing a phased plan to reduce nitrogen loads and thereby raise oxygen levels in the

Sound (LISS, 1994). In 1998, Connecticut, New York, and the Federal government targeted a 58.5 percent reduction in nitrogen loading to the Sound by the year 2014. Two other very significant sources of nitrogen are stormwater runoff and groundwater discharge. Taken together, they may represent an equal or greater source of nitrogen than the wastewater plants. Efforts are underway to control these discharges by better managing stormwater runoff and improving land use practices to reduce nitrogen use and discharges (LISS, 2003a).

Hypoxia occurs each year in the western LIS. Factors that have been shown to affect the spacial and temporal extent of hypoxia include the presence and strength of a density difference between the top of the water column and the bottom of the water column, major storms that stir the water column, and combined sewer overflow events (Swanson and Tonjes, 2001). Other research seems to show that the intensity of algal blooms may be inversely related to dissolved oxygen concentrations in the Sound. Cores from bottom sediments have been interpreted as showing changes in the ecological communities in the LIS following European settlement in the area. These changes are most marked in the 20th Century, and seem to imply that algal blooms, and, perhaps, associated hypoxia are relatively recent phenomena and are not natural occurrences for the LIS (Varekamp et. al., 2003).

Hypoxic events generally occur during the late summer into the early fall and are more severe in the western LIS, both in terms of lower decreased oxygen concentrations and the duration of the events, than elsewhere in LIS. Figure 3-1 shows the historical distribution of hypoxia over a nine-year period.

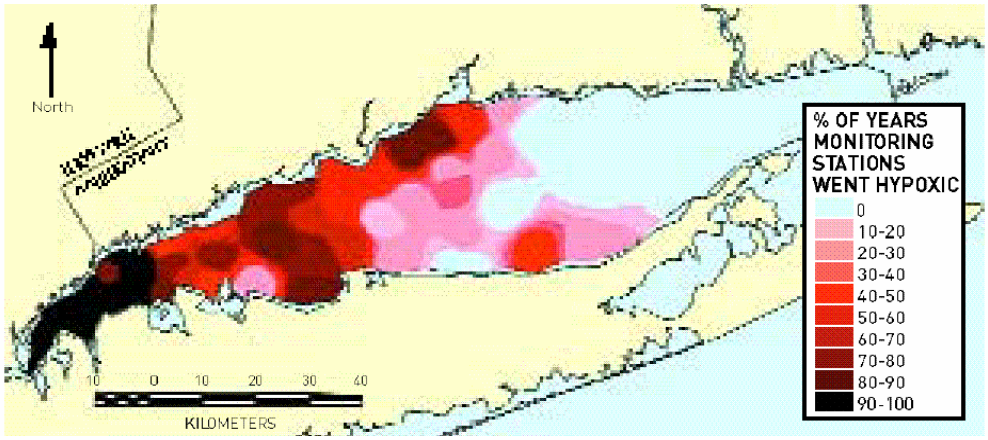


Figure 3-1 – Summer Hypoxia (O_2 < 3 mg/l) in the Long Island Sound between 1994 and 2002

4. Lobster Mortality Research

Different factors have been implicated in the lobster mortality. Attention first focused on the relationship between adulticiding for WNV control in 1999 and the sharp decline in lobster catches that year. Lobster parasites and other lobster diseases have also been implicated as well as changes in environmental conditions such as water temperature, salinity, and hypoxia.

4.1. Pesticides

4.1.1. Pesticide Use and Detection

At the May 2004, LI Sound Lobster Research Initiative meeting, Chytalo *et. al.* (2004) reported on pesticide applications in 1999, the first year of serious lobster mortalities. Although the catch was lower in 1998, large numbers of dead and dying lobsters in traps were commonly observed in 1999, defining the event now known as the lobster die-off. Their information is summarized in tabular format below:

Table 4-1 - 1999 Mosquito Control Pesticides Used by LIS Municipalities

Pesticide	NY Application Period	Weeks	NY Quantity	CT Application Period	CT Quantity	Weeks
Malathion	8/29/99-9/26/99	4	8,648 lbs	Not applied	0 lbs	--
Malathion	Week of 9/12/99	1	1,264 lbs	Not applied	0 lbs	--
Pyrethroids	8/1/99-10/9/99	10	3,560 lbs	7/25/99-9/28/99	15 lbs	9+
Pyrethroids	Week of 9/12/99	1	1,402 lbs	9/23	6 lbs	One day
Methoprene	8/1/99-9/19/99	7	5.8 lbs	4/15-9/27/99	2.8 lbs	24
Methoprene	Week of 8/15/99	1	2.6 lbs	9/27	1.5 lbs	One day

The absence of malathion use in Connecticut is notable as is the occurrence of Hurricane Floyd during the week of the heaviest use of malathion (the week of 9/12/99). Pyrethroid use in New York was significantly greater than in Connecticut. Methoprene usage was comparable between the states.

However, it is not known what concentration of each chemical resulted in the LIS from the applications above. Bruce Brownawell at the Marine Sciences Research Center of Stony Brook University has improved detection limits for vector control chemicals to the range of 100 to 200 pq/L (parts per quadrillion – per 10^{15}). Anne McElroy at the Marine Sciences Research Center of Stony Brook University and Brownawell tested for resmethrin following spray events in Connecticut in 2003. It was detected in 5 of 11 water samples immediately offshore of the spray site at concentrations ranging from 1 ng/l to 1 µg/L. They were not able to detect sumethrin in water after two spray events. Extensive testing for methoprene resulted in only one detection of that compound, at a concentration of 7 ng/L. The pyrethroid synergist, piperonyl butoxide (PBO), was detected after every pyrethroid spray event, at concentrations of 0.3 – 15,000 ng/L. Similarly, samples from the East River Narrows, taken 10 days after a sumithrin spray event and following a heavy rainstorm that would have promoted substantial runoff, found no parent compound, but did find PBO at concentrations of 0.2 –7 ng/L. Brownawell and McElroy did not have the opportunity to collect samples following a recent application of malathion. These data suggest it will be difficult to regularly directly detect pyrethroids in the marine environment following applications made on land.

At the request of the NYSDEC, Hydroqual, Inc. and a team at the Marine Sciences Research Center at Stony Brook University are modeling the fate, distribution, and concentrations of mosquito control pesticides entering the LIS (Karen Chytalo, NYSDEC, personal communication, 2004). Preliminary data from this effort will be available at the Lobster Research Initiative conference in October 2004.

4.1.2. Toxicity Testing

Malathion

Sylvain De Guise of the Department of Pathology at UCONN at Storrs exposed lobsters to a single dose of malathion for 96 hours (De Guise *et. al.*, 2003). The test resulted in 50 percent mortality (LC_{50}) at 33 parts per billion (ppb) of malathion. Additional testing showed that three days after a single exposure to 5 ppb of malathion, phagocytosis was significantly reduced. Phagocytosis is the process by which immune cells (phagocytes) fight disease by consuming

foreign particles (including microbes) that invade cells. The study also noted that the degradation of malathion is rapid. Between 65 and 77 percent of the pesticide in the lobster tissue was degraded within one day after the application and 98 percent was degraded within three days following its application. By the end of the 5-day exposure, no malathion was detected in the lobsters' tissues.

De Guise also performed a sub-acute exposure test. This study exposed lobsters to malathion for a period of a month. Lobsters exposed to 21 ppb of malathion experienced a considerable decrease in phagocytosis after one week. Similarly, lobsters exposed to 5 ppb of malathion experienced a significant reduction in phagocytosis after two weeks. The study also showed that after three weeks of exposure to all concentrations tested, phagocytosis diminished dramatically.

De Guise presented a research update at the May 2004 meeting of the LIS Lobster Initiative researchers (De Guise *et. al.* 2004). He stated, "The malathion LC_{50} for lobsters was revised to 38 ppb due to use of a slightly different calculation."

At the Long Island Sound Lobster Initiative researchers' meeting of May 2004, the laboratories of McElroy and Brownawell reported the 48-hour LC_{50} for lobster larvae exposed to malathion to be 3.5 ppb (McElroy *et. al.*, 2004). In other tests, they found that "in seven day continuous exposures to malathion at 16ºC and 22ºC at concentrations 25 percent of the 48-hour LC_{50}, phenyloxidase activity [a measure of immune response] was significantly elevated at high temperature, but was not influenced by pesticide exposure."

These studies were conducted under controlled conditions in the laboratory. It is not known what concentration of malathion lobsters may have been exposed to in the Long Island Sound.

Methoprene

LIS Lobster Initiative researchers investigated methoprene for its potential impact on lobsters. Methoprene is an insect growth regulator, preventing mosquito larvae from maturing. It is structurally similar to methyl farnesoate (MF), the crustacean juvenile growth hormone that regulates growth, molting, and reproduction. In crustaceans, such as lobsters, MF stimulates ecdysone, the hormone that induces molting. It has been postulated, "that pesticide exposure

may stimulate molting at inappropriate times under some circumstances" (LIS Lobster Initiative, 2003a).

Michael Horst of the Mercer University School of Medicine determined the toxicity of methoprene to various lobster life stages (Horst, *et. al.*, 2003). Stage I larvae exposed to 25 ppb of methoprene for 48 hours did not experience any mortality. Stage II larvae were exposed at varying concentrations (0.1 ppb, 0.5 ppb, and 10 ppb) for three days and mortalities were recorded after 24, 48 and 72 hours. No mortalities were observed after 24 hours at the various concentrations. At concentrations of 10 ppb, mortalities were recorded up to a maximum of 82 percent after 48 and 72 hours. Postlarval juveniles, and intermolt adults survived 24-hour exposure to methoprene at 25 ppb. Postmolt adult lobsters died 18 hours after exposure to 25 ppb methoprene.

Hans Laufer, Department of Molecular and Cellular Biology at UCONN, worked with a team whose research involves the effects of methoprene on lobster larvae. His team subjected larvae to methoprene at numerous concentrations and found that second stage larvae when exposed to 1, 10, and 100 ppm of methoprene had, on average, a six-day life span with 1 and 10 ppm having a slower killing rate than the 100 ppm exposure. Over the course of the week, no larvae had reached the third stage of development (Laufer *et. al.*, 2003).

At the LIS Lobster Initiative researchers' meeting of May 2004, Horst *et. al.* (2004) presented their most recent evaluation of the acute effects of methoprene on larval, juvenile, and adult lobsters. They found toxicity effects on larvae at concentrations as low as 2 ppb. Juvenile lobster synthesis of cuticular, shell, proteins was altered at 25 ppb. They also found alterations in the chitoproteins, shell proteins, of lobster tissue following methoprene treatment. In addition, the authors observed a "marked concentration of methoprene in neural tissues, such as the eyes, of adults at concentrations equivalent to a 1200-fold concentration over the surrounding seawater". Stress proteins were measured by the authors in juvenile and adult lobsters after exposure to methoprene. Organisms respond to unfavorable conditions, such as heat shock, environmental contaminants, and other stresses, increasing the rate of expression of specific genes, such as heat shock genes. Consequently, during heat shock or other stress, the products of these genes, heat shock or stress proteins, increase and accumulate in cells. Measurement of heat

shock proteins in the lobsters after methoprene exposure is an indication of stress. The authors are also developing a method that will enable them to "describe the molecular 'footprint' of methoprene's acute effect on both intermolt and postmolt lobsters". They believe that they will be able to detect impacts of methoprene exposure on lobsters, both in the generation of stress proteins and in the synthesis of specific enzymes by the lobster.

At the May 2004 meeting of the LIS Lobster Initiative, researchers McElroy and Brownawell reported that they had found no toxicity of methoprene to larval lobsters at the highest concentration tested, 10,000 ng/L (10 ppb) (McElroy, *et. al.*, 2004). Their results contradict those of Horst *et. al.* (2004), presented above.

De Guise (2004) presented a research update at the May 2004 meeting of Lobster Initiative researchers. The key findings related to methoprene exposure to adult lobsters were as follows:

- Acute (96 hour) exposure top concentrations of methoprene up to 221 ppb did not result in significant mortalities in adult lobsters, therefore preventing calculation of the LC_{50} in this species.
- Methoprene disappeared rapidly in seawater, with a half-life of less than a day.
- A single acute (5 day) exposure of lobsters to methoprene resulted in a significant increase in hemocytes phagocytosis on days 3 and 5 at 221 ppb (highest dose).
- Weekly exposure of lobsters to methoprene for 4 weeks resulted in a significant reduction in hemocytes phagocytosis at 33 ppb (week 1).
- Methoprene bioaccumulated in lobster tissues.
- Based on release data and application guidelines, methoprene should not be considered a high risk to adult lobsters.

Resmethrin

McElroy and Brownawell (McElroy, *et. al.*, 2004) reported on the effect of resmethrin exposure to lobster larvae at the May 2004 meeting of the LI Lobster Initiative researchers. They found the larvae to be "extremely sensitive to continuous exposure to resmethrin." LC_{50} values varied from 100 to 200 ng/L in 48 hour and 96 hour experiments at 16ºC. Little toxicity was observed prior to 24 hours of exposure. Elevated temperature (24ºC) significantly increased control

mortality, making it difficult to determine any synergistic effect of temperature on pesticide mortality.

De Guise presented a research update at the May 2004 meeting of Lobster Initiative researchers (De Guise, *et. al.*, 2004). The key findings were as follows:

- Acute (96 hour) exposure top concentrations of resmethrin up to 1 ppb did not result in significant mortalities in adult lobsters, therefore preventing calculation of the LC_{50} in this species.
- A single acute (5 day) exposure of lobsters to resmethrin resulted in a significant reduction in hemocytes phagocytosis at doses of 0.1 and 1 ppb.
- Weekly exposure of lobsters to resmethrin for 4 weeks resulted in a significant reduction in hemocytes phagocytosis at 0.1 ppb (week 3 and 4) and 0.01 ppb (week 4).
- Resmethrin did not bioaccumulate in lobster tissues.

4.2. Chemical Links to Shell Disease

At the May 2004 LIS Lobster Initiative research meeting, Laufer reported on his work with molting hormone concentrations of normal and shell diseased animals (Laufer, 2004). He found that lobster blood and tissue samples contained "substantial levels" of alkylphenols, which were also found in sediment samples. Alkylphenols are thought to be lobster endocrine disrupters at low concentrations that interfere with normal molting. Laufer found that alkylphenols are toxic to larval lobsters. He suggests that alkylphenols present in lobster tissue probably come from environmental contamination by industrial wastes such as detergents, lubricants, paints, and degrading plastics and rubber tires. Laufer suggests a link between alkylphenols and shell disease based on the increased molting frequency associated with shell disease and the ability of alkylphenols to interfere with lobster endocrine function.

4.3. Parasites and Diseases

Parasites or diseases may have caused some or all of the LIS lobster mortality. Paramoeba and bacteria have been linked to various diseases involving severe tissue and exoskeleton damage.

Some known diseases caused by parasites and bacteria within the LIS are limp lobster syndrome and Gaffkemia.

4.3.1. Paramoeba

It has not yet been determined whether parameoba infections are the primary or secondary cause of lobster mortality. Parameoba may infect lobsters that are weak or stressed by other environmental factors. Paramoeba have been shown to cause limp lobster syndrome (LIS Lobster Initiative, 2003b and 2003c). Paramoeba are a parasitic protozoa similar to amoeba but distinguished primarily by the presence of both a parasome and a nucleus. The parasome is a round object that contains DNA material. Lobsters affected by the syndrome have damaged and inflamed connective and nervous tissues from attack by paramoeba. Lobsters afflicted by this syndrome become severely weakened and die shortly after being landed.

Although research is underway to test the tissues of diseased lobsters, it has yet to be determined which paramoeba, if any, cause disease in the lobster. One laboratory conducting such research is Frasca, Nevis, and Mullen of the Department of Pathobiology and Veterinary Science at UCONN. They plan to identify the type(s) of parameoba causing the disease by taking DNA samples of infected lobster tissue.

The laboratory of Rebecca Gast (2004) is investigating a detection method for the pathogenic *Neoparamoeba* species that infects lobsters. The goal is to determine the distribution of the organism in the Long Island Sound. With that information, she intends to determine if it is an opportunistic pathogen normally found free-living in the Sound or if it was introduced from elsewhere.

A report was presented from a published paper by Peglar *et. al.* (2003) on techniques used to determine the phylogenetic relationship of the lobster paramoeba to other known paramoeba species. They are also developing molecular tests to detect the pathogenic paramoeba in host (lobster) tissue. Other researchers also reported on their efforts to develop a method to detect the paramoeba associated with the lobster mortality (Frasca *et. al.,* 2004)

At the May, 2004 Long Island Lobster Initiative meeting, researchers reported on the testing they are conducting on the infectivity of parasitic amoeba in lobsters (Robohm *et. al.*, 2004). They wish to determine if "the parasitic amoebae, found in lobsters during the 1999 mortality event, are the primary cause of lobster mortality or are only present in lobsters under severe immune-system stress from other environmental factors." To prove that a particular agent causes disease, the standard approach (known as Koch's postulates) is to culture the suspected disease organism, use it to cause the infection, and then re-isolate the organism that appears to be causing the infection. Another approach must be taken, as the lobster amoeba cannot be cultured *in vitro* (in an artificial environment outside a living organism). The agent can be proven as causative if it can be isolated from infected lobsters and used to infect healthy lobsters. The Robohm laboratory has used a modification of this approach. They injected lobsters with *Neoparamoeba pemaquidensis* from diseased sea urchins. Lobsters held six weeks did not die and did not show any evidence of amoebae in their tissues.

At the May, 2004 Long Island Lobster Initiative meeting, O'Kelly and Gillevet reported on *Paramoeba-Neoparamoeba* complex amoeba. They reached the following conclusions based on their investigations and those of the Frasca and Gast laboratories:

- *Paramoeba-Neoparamoeba* complex amoeba are abundant, widespread, and genetically diverse in shallow marine waters, and are a common component of the microbiota of the lobster carapace.

- Any strain within the *Paramoeba-Neoparamoeba* complex is potentially pathogenic to marine vertebrate and invertebrate animals. The factors inducing an amoeba to switch from preying on bacteria and/or algae to preying on animals are unknown, though elevated sea surface temperatures are correlated with outbreaks in sea urchin and salmon paramoebiasis outbreaks.

- The proximate cause of the 1999 lobster die-off in western Long Island Sound was massive infection by at least one strain of *Neoparamoeba pemaquidensis*. Satisfying Koch's postulates will be difficult given the documented loss of pathogenicity from amoeba cultured from urchin and salmon paramoebiasis lesions. An exploration of the cellular mechanisms associated with prey choice in the amoebae, for example an examination of gene expression patterns associated with environmental factors, especially temperature and prey availability, is highly desirable.

The current research does not seem to point to paramoeba as a primary causative disease agent, but rather as a secondary infectious agent, that might be present because of other stresses such as elevated temperature.

4.3.2. Bacteria

Bacteria have also been implicated as a potential cause of LIS lobster mortality. Bacteria known as *Aerococcus viridians* var. *homari* have been found in lobsters afflicted with Gaffkemia, also known as "red-tail disease." The bacteria infect the circulatory system, weaken blood circulation, and may cause hemorrhaging (LIS Lobster Initiative, 2003b and 2003c). Red tail disease has been less prevalent in recent years.

A lobster disease of more pressing concern is known as shell disease caused by chitinolytic bacteria. Chitinolytic bacteria are an assemblage of species so named because they attack and break down the chitin of the lobsters' shell. Shell disease is more common on the eastern end of the LIS. It is found in approximately 40 percent of the total catch east of the Connecticut River. Although shell disease does not necessarily cause death in lobsters or affect the meat in any way, it dramatically decreases the lobsters' market value.

At the May, 2004 meeting of LIS Lobster Initiative researchers, Chistoserdov *et. al.* (2004) reported on their investigation of bacterial assemblages involved in the development and progression of shell disease. The researchers identified different assemblages of bacteria associated with shell disease at each of the test sites in Maine, Massachusetts, and LIS. The researchers were unable to prove Koch's postulates using any of the bacterial assemblages. They were therefore unable to prove a primary causal relationship between the bacteria and the shell disease. The same researchers found "no other disease was identified in affected lobsters … [and] no correlation between rarely identified bacterial infection in lobster hemolymph and epizootic shell disease".

Like paramoeba, therefore, bacterial infections may be a secondary cause of lobster mortality. Bacteria may infect lobsters that are weak or stressed by other environmental factors.

4.3.3. Calcinosis

A more recently discovered syndrome is not caused by an infectious agent. It is instead a metabolic condition linked to the increased temperature of the Sound (see below). Lobsters with the condition known as calcinosis have "orange discoloration of the abdomen, lethargy, an excess of epibionts (organisms growing on the lobster's shell) and poor post-capture survival" (Dove *et. al.*, 2003). Calcinosis affects lobster respiratory functions and antennal glands, which aid in phagocytosis and filtering foreign items within the lobster. Calcinosis in lobsters is analogous to kidney stones in humans. Calcium deposits built up on the lobsters' gills, interfering with both respiratory and excretory functions, and eventually choking them to death.

Alistair Dove of the Cornell College of Veterinary Medicine hypothesized that temperature related respiratory stress causes excess carbon dioxide (CO_2) in the hemolymph. Excess blood CO_2 is referred to as hypercapnia. When lobsters experience hypercapnia, they react by releasing calcium carbonate ($CaCO_3$) into their blood. The $CaCO_3$ released into the blood is passed to the antennal glands for excretion. However, when the antennal glands become clogged, $CaCO_3$ is passed to the gills for excretion. Ultimately the hair-like filaments of the gills also become clogged with $CaCO_3$, which reduces their capacity for respiration and eventually results in death.

4.4. Temperature

Lobsters are extremely sensitive to increased temperature. According to Dove *et. al.* (2003), lobsters are adapted to water temperatures around 55°F (15°C). Lobsters have an estimated high temperature tolerance of 69°F (20.5°C) (Gimple, 2003). In 1999, when unusual lobster mortalities were first noted, the temperature at the bottom of the Sound was 72°F (22.2°C). Again, in 2002, when the second major lobster mortality was documented, the temperature had reached approximately 73°F (22.8°C). Laufer observed that lobsters die when temperatures exceed 71.6°F (22°C) under controlled conditions. He stated, "They [scientists] were saying, 'we don't know why they died', but they were reporting 60 days of high temperatures in Long Island Sound, more then I knew they could stand" (NY Times, 2001). Bottom temperature trends in the Long Island Sound are shown in Figure 4-1, below.

The CTDEP found a statistically significant linear relationship between bottom water temperature in the western part of the Sound and lobster mortality for the period 1996-2003 (as reported at the 4th Lobster Research Symposium, 2004).

Dying, dead and weak lobsters were harvested in mid-August 2002 just after bottom temperatures reached a high of 73°F. These lobsters "had a distinct orange coloring to their blood that showed through the undersides of their tails and leg joints" (LoBue and Howell, 2003). Lobster blood is normally clear. Affected lobsters were located throughout the Sound from Mattituck to Oyster Bay.

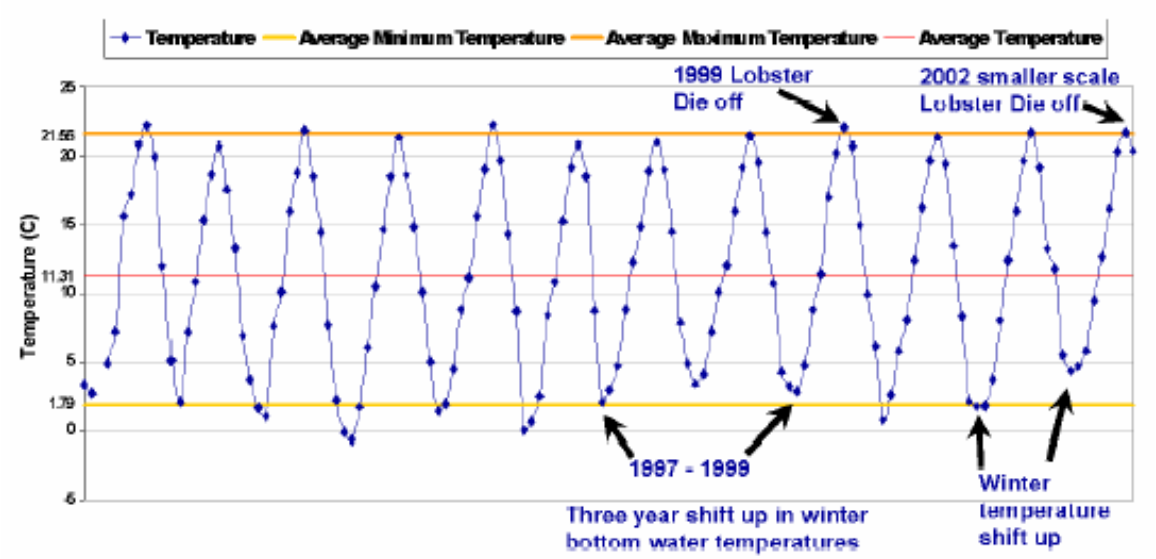


Figure 4-1 - Bottom Temperature Trends in Western Sound from 1991 to 2002

From CTDEP - Monitoring Long Island Sound Hypoxia 2002

Dove believes that abnormally high temperatures can cause calcinosis in the blood of lobsters, which leads to abnormal shell deposits (Rheault, 2002) and eventually asphyxiation. Dove stated that, "they were dying from the stress of the environment that had become hostile to their ancient internal thermostats. Climate is the killer here. The correlation is very strong. Not proven, but strong" (Gimple, 2003).

Glen Lopez of the Marine Sciences Research Center at Stony Brook University has been studying the effects of temperature and body size on metabolism of Long Island Sound lobsters. Lopez (personal communication) indicated that lobster metabolic function becomes stressed at

temperatures above 71.6ºF (22ºC) (as was the case during the summers from 1999 through 2002). He also stated that as temperature rises, stress increases dramatically. He added that at temperatures above 71.6ºF (22ºC) lobsters' buffering capacity (ability to regulate blood pH) and oxygen consumption are severely weakened. Lopez said that temperature may not be the reason for lobster mortalities, but may be a considerable factor.

Robert Wilson, at the Marine Science Research Center at Stony Brook University has been studying LIS water temperature and its relationship to lobster mortalities. He has evidence that bottom temperatures beginning in 1999 exceeded the ten-year average by as much as 1-2 degrees Celsius, and that this difference was sustained over a substantial period of time (Figure 4-2).

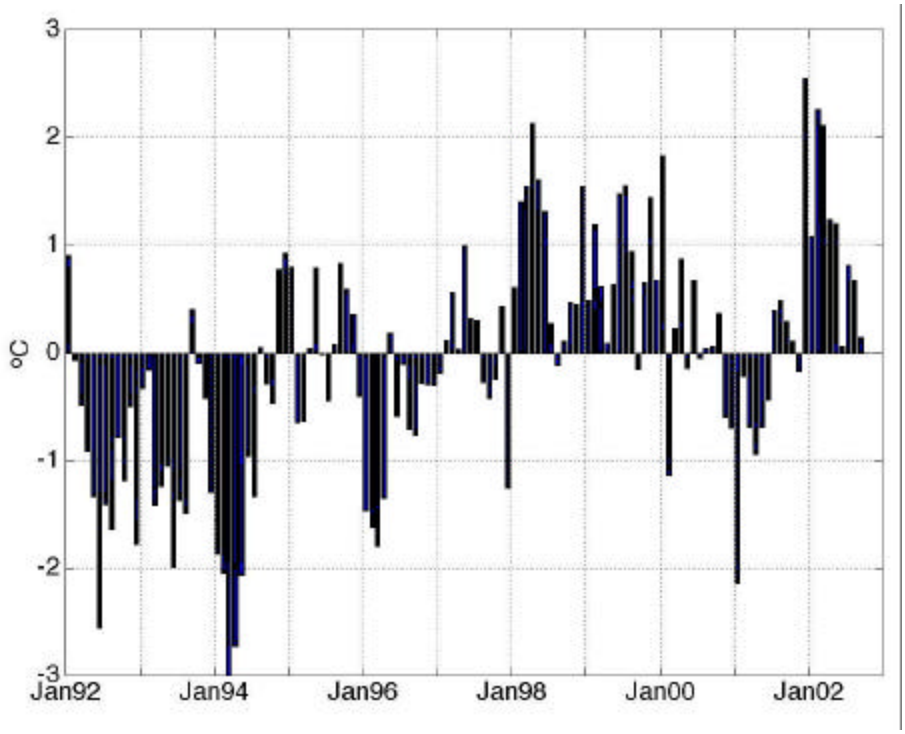


Figure 4-2 - LIS Bottom Temperature Differences from the Ten-Year Average
from (Wilson and Swanson, 2004)

Wilson also suggests that the initial lobster die-off may be related to the severe storm of August 2, 1999. He feels that the storm may have caused a mixing of surface and bottom waters leading to the increase in temperature for the normally colder bottom water from the sun-warmed surface layer. The surface and bottom temperatures shown in Figure 4-3 are relatively high for that day.

Additional information pertaining to that event is anticipated in the fall of 2004 (Wilson and Waliser, 2003).

A somewhat contradictory study was reported by Zajac *et. al.* (2004) during the May 2004 research update of the LI Lobster Initiative. Their spatial analysis of LIS lobster populations in relation to habitat found "weak relationships to contemporaneous dissolved oxygen and temperature data" and that most population variations were explicable by location. They believe these population centers are the result of habitat choices made in the early benthic stage, juvenile stage, and reproduction stage.

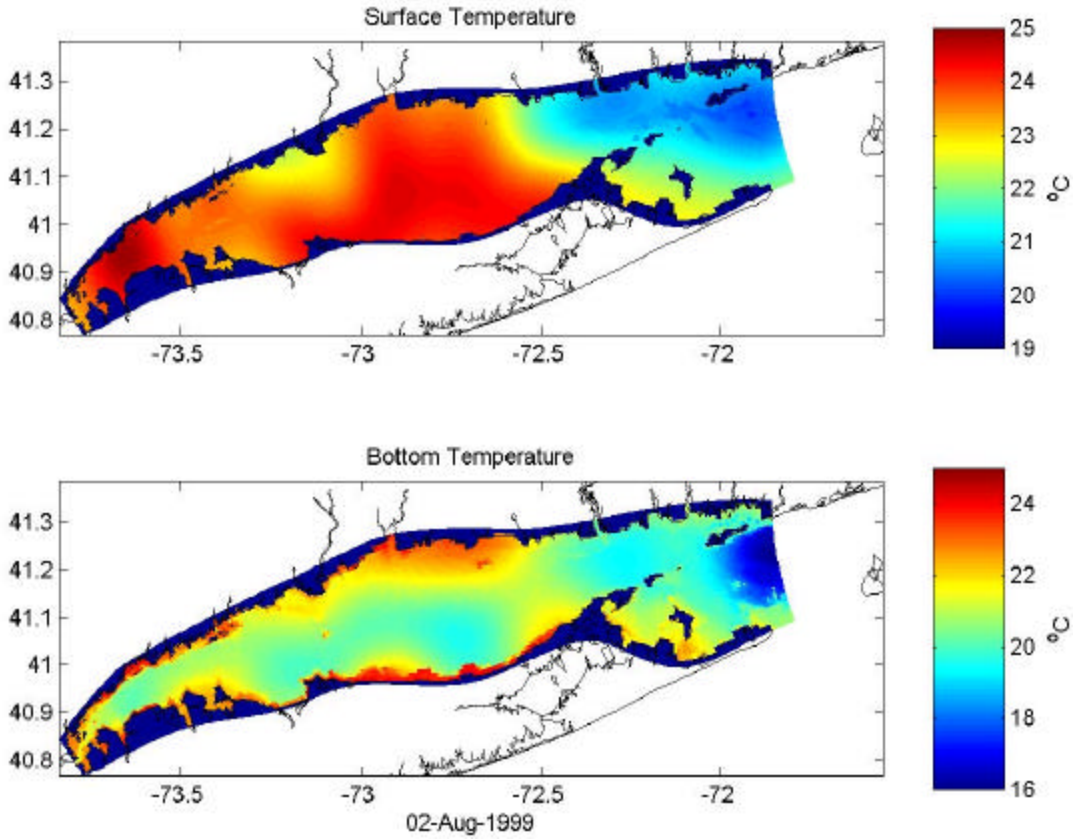


Figure 4-3 - LIS Bottom Temperature on August 2, 1999

from (Wilson and Swanson, 2004)

4.5. Salinity

Wilson suggested that the initial lobster mortalities of 1999 were related to higher bottom temperatures due to the severe storm of August 2, 1999 (see above). That may be part of the answer. Jury *et. al.* (1995) observed lobster movements in response to a hurricane in Florida. They found substantial migration of lobsters after the storm to deeper and more saline waters further offshore. They also speculated that some lobsters died because of storm, particularly those that were near molting (molting occurred over the period June through September). The researchers believe the primary trigger for the off shore movement was avoidance of low salinity. They suggest that there may be frequent migrations in response to storm runoff generated low salinity events. Another paper found that very few subadult and juvenile Florida spiny lobsters were caught for months on another occasion after a hurricane (Hurricane Alma) dramatically lowered the salinity in the St. Lucie estuary (Witham *et. al.*, 1968).

Lobsters exposed to lowered salinities experience increased oxygen consumption (Jury *et. al.*, 1994). It may be, therefore, that lobsters that were unable to migrate quickly enough to cooler and more saline waters suffered respiratory stress from the combined effects of higher bottom temperatures and lower salinity.

4.6. Hypoxia

Hypoxia has also been implicated in lobster illness and deaths. The lobster mortality of 1999 tended to coincide with the historical onset of hypoxia; hypoxia was recorded in the LIS in the early summer and generally persisted until Hurricane Floyd in mid-September 1999, as measured at the CTDEP monitoring stations (CTDEP, 2004). At the New York City Harbor Survey monitoring stations, bottom water hypoxia was measured at two western LIS stations (Stepping Stones and Hart Island) throughout most of July and August in 1999, with late July dissolved oxygen (DO) concentrations falling below 2.0 mg/l. Late August DO conditions were no longer hypoxic. However, the Harbor Survey annual report found statistically significant downward trends for summer-averaged bottom DO concentrations for both stations over a fifteen-year period, indicating that summer bottom DO conditions in western LIS have experienced a long-term deterioration in the late 20th Century (Swanson *et al*., 2000).

In 2000, the USEPA and the New England Interstate Water Pollution Control Commission (NEIWPCC) collected water samples at 36 sites, sometimes as little as five centimeters above the sediment surface, from August through November. Sediment chemistry was also measured at those sites. The majority of the tested sites coincided with the locations where sediments were sampled in October 1999. The sampling found "extremely black (anoxic/sulfidic) sediments and thin redox depths," especially in western LIS. The redox depth is the depth above which aerobic reactions occur, and below which anaerobic reactions predominate. Therefore, it describes the degree of oxygen penetration into the sediments, serving as an indicator of oxygen concentrations in the overlying waters. Moderate to severe hypoxia was observed at stations in the extreme western LIS during late August of 2000 (Cuoma *et. al.*, 2003). This research, coupled with mapping such as that in Figure 3-1, suggests that hypoxia is common in western LIS, and likely occurred in the areas most impacted by the die-off in 1999.

The effects of low dissolved oxygen have been well documented. Locally appropriate information has been developed by the USEPA Environmental Research Laboratory in Narragansett and the CTDEP Marine Fisheries Division. A summary of their findings is presented in Table 4-2, below.

Table 4-2 – Dissolved Oxygen Effects on Marine Animals (LISS, 2003B)

Dissolved Oxygen	Consequences
1.0 mg/l	Highly lethality (75-90%) in fishes: pipe fish, winter flounder, summer flounder, Atlantic menhaden.
	Lethality (~ 25%) in three additional fishes: windowpane flounder, tautog, fourspine stickle back.
	Increased lethality (50%) in juvenile crustaceans: American lobster, sand shrimp, grass shrimp.
1.5 mg/l	Lethality in some fishes: pipe fish, 50%; winter flounder, 35%; summer flounder, 25%; Atlantic menhaden, 20%.
	Lethal threshold for some juvenile crustaceans: American lobsters, sand shrimp, grass shrimp.
2.0 mg/l	Reduce growth (~ 50%) in juvenile summer founder and juvenile grass shrimp.
	Lowest safe dissolved oxygen for survival of juveniles of several fish and crustaceans.
2.5 mg/l	Lethality threshold (~ 15%) for the less sensitive planktonic larvae of crustaceans.
	Growth reduced (25%) in juvenile grass shrimp and summer flounder; 50% in American lobster.
	Additional species of bottom-living fishes show low dissolved oxygen avoidance.
3.0 mg/l	Greater lethality (~75%) among most sensitive planktonic crab larvae.
	Growth reduced (50%) in other, less sensitive planktonic crab larvae.
	Growth reduced in juvenile American lobsters by 30%.
	Bottom-living fishes begin to show low dissolved oxygen avoidance.
4.0 mg/l	May reduce survival (30%) of very sensitive planktonic larvae of some crabs.
5.0 mg/l	Few adverse effects expected.

4.7. Effects of Environmental Stressors on Lobster Disease Susceptibility

At the May 2004 meeting of the LIS Lobster Initiative researchers, Robohm *et. al*. (2004) reported on their work to determine whether increased temperature, hypoxia, sulfide, and ammonium alone or in combination, can increase the susceptibility of lobsters to microbial infections. Lobsters were injected with the bacterium *Arococcus viridans*, a pathogen that kills lobsters within 12 days at moderate temperatures. The bacterium was used in place of the paramoeba associated with the lobster mortalities, as the paramoeba cannot be cultured. The researchers reached the following conclusions:

- Temperature and bacterial dose levels had moderate effects on lobster mortality,
- At adequate oxygen levels, sulfide and ammonia strongly accelerated infected lobster deaths,
- Non-stressed lobsters tolerated high bacterial infection rates (as high as 1×10^9 g^{-1}),
- Lobsters stressed by low oxygen died sooner and at lower bacterial infection rates,
- With adequate oxygen, lobsters survived for an extended time (>14 days) at an elevated temperature of 24ºC,
- Survival was reduced to 3-12 days under hypoxic conditions at 24ºC,
- Survival was reduced to 3-12 days under high temperature, hypoxia, and elevated sulfide and ammonium

5. October 4, 2004 Symposium

On October 4, 2004, at Stony Brook University, the LIS Research Initiative held its summary symposium. The reports and papers comprising the final results are not expected to be released until 2005. The information presented at the symposium has been assembled from newspaper reports and notes from symposium participants.

Many of the preliminary research results were proven accurate, thus, little new information was presented at the symposium. The one new element of the research was modeling results. Both HydroQual and the Marine Science Research Center, Stony Brook University, modeled the transport of the reported pesticides released during the critical time period before Hurricane Floyd. It was assumed that essentially all of the pesticides applied the coast of the LIS reached the LIS via stormwater. There were some differences in the mass of pesticides used by each of the model teams, and in the hydrodynamic modeling approach used. When reporting on the maximum concentrations that would have reached the lower depths of the Sound away from the immediate shoreline, where lobsters are resident, both teams agreed. They reported that pesticide concentrations would have been many factors below the published critical concentrations where mortality might occur (Rather, 2004; Haberstroh, 2004; M. Dempsey, SCVC, personal communication, 2004; RL Swanson, Stony Brook University, personal communication; J. Mattice, New York Sea Grant, personal communication, 2004).

These modeling exercises confirmed the belief of the LIS Lobster Initiative scientists that the mosquito control chemicals had no role in the die-off event. Rather, the extended warm water, low DO, and potentially toxic, sulfidic environment of the bottom waters in September 1999, with opportunistic diseases apparently attacking the weakened shellfish, caused the event. Because these environmental conditions may be chronic, the researchers saw little chance of reestablishing the fishery, especially to the levels of its 1997-1998 harvest (Rather, 2004; Haberstroh, 2004; M. Dempsey, SCVC, personal communication, 2004; RL Swanson, Stony Brook University, personal communication; J. Mattice, New York Sea Grant, personal communication, 2004).

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APPENDIX

NY/CT Sea Grant's Long Island Sound Lobster Initiative Research Projects

The Long Island Sound Lobster Initiative encompasses 17 research projects and 2 monitoring projects. Investigation summaries below reflect the period between July 1, 2001 and June 30, 2003.

Immunology & Endocrinology Development of an Assay for Phagocytic Activity in the Immune System of Lobsters

Investigator:

Jan Factor, Division of Natural Sciences, SUNY Purchase
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Factor is examining how lobsters defend themselves against infection and disease. He is seeking to develop methods that will allow the assessment of cellular defenses against infection and disease after sublethal exposure to environmental stresses and toxic substances. Research may lead to an explanation of the recent mortalities by enabling assessment of impacts on the immune system that may lead to lethal infections.

Immunological Health of Lobsters: Assays and Applications

Investigator:

Robert S. Anderson, Chesapeake Biological Laboratory, Center for Environmental Sciences, University of Maryland.
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Anderson is using biotechnology tools to measure the blood cell-related defense system of the lobster against disease. This research will lay the groundwork for discerning changes in immune response due to toxicity or other environmental stressors.

Stress Indicators in Lobsters: Hormones and Heat Shock Proteins

Investigator:

Ernest S. Chang, Bodega Marine Laboratory, University of California Davis
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Chang is investigating the relative impacts of stresses from environmental factors (i.e., temperature and salinity), biological factors (i.e., bacteria and protozoa), and human-caused

stresses (i.e., pesticides). Lobsters exposed to these stresses are being examined for changes in stress proteins and steroid molting hormones.

Development of assays for the evaluation of immune functions of the American Lobster (*Homarus americanus*) as a tool for health assessment

Investigator:

Sylvain DeGuise, The Dept. of Pathobiology, University of Connecticut, Storrs
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Co-investigators:

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The University of Connecticut Pathobiology team is seeking to develop new tools to use in evaluating how immune systems work in both sick and healthy lobsters. They are systematically exposing lobsters to various chemicals and other stressors and measuring the response of the immune system in each case.

Effects of Environmental Stressors on Disease Susceptibility in Lobsters: A Controlled Laboratory Story

Investigator:

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Co-Principal Investigator:

Andrew F.J. Draxler, NOAA Fisheries, Howard Laboratory, Sandy Hook, NJ
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Robohm and Draxler are investigating the effects of environmental stressors on the susceptibility of lobsters to pathogens. Their work examines whether depressed habitat quality may have compromised lobsters' immune systems and contributed to the die-off. The researchers are exposing healthy lobsters to two bacterial pathogens in the presence of varying levels of environmentally relevant bio-geochemicals such as sulfide and ammonia as well as environmental conditions such as low oxygen and increased temperatures. After the exposures, changes in bacterial numbers and five, lobster, immune-system indices are measured. The protocol also allows for testing of stressors on the growth of a parasitic amoeba in lobsters, should the amoeba be cultured successfully by other researchers.

Pesticides

Effects of Pesticides of Lobster Health: Trace Level Measurements and Toxicological Assessment at Environmentally Realistic Concentrations

Investigator:

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Co-Investigator:

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McElroy and Brownawell are addressing the potential link between pesticide use and lobster mortality. They are measuring mortality and immune response in larval and juvenile lobsters exposed to environmentally realistic levels of pesticides (malathion, methoprene, and selected pyrethroids such as Anvil and Scourge). The team is also looking to develop ways to measure levels of these pesticides and their breakdown products in seawater, sediment, and possibly lobster tissues. They are particularly interested in sampling water after storm events when concentrations may be highest. The results of this study should provide a strong indication whether or not pesticide use is likely to contribute to degraded lobster health in Long Island Sound. This study will also shed light on the effects of temperature on the immune response of young lobsters.

Determination of the toxicity and sublethal effects of selected pesticides on the American Lobster (*Homarus americanus*)

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The research team is exposing lobsters to malathion, resmethrin, and methoprene, three pesticides used in the region to control mosquitoes after West Nile virus was found. The subtle effects of low levels of pesticides on the lobster immune system is being measured, in addition to high level exposures, to determine toxicity.

Acute Effects of Methoprene on Survival, Cuticular Morphogenesis and Shell Biosynthesis in the American Lobster, *Homarus americanus*

Investigator:

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Horst has hypothesized that methoprene causes biochemical changes in, and/or kills, juvenile and adult lobsters. His team is studying the effects that a range of doses has on nerve, skin, and pancreatic cells, and shell formation.

Hormonal Responses of Lobsters to Stresses of Western LIS

Investigator:

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Laufer and his co-investigators are assessing the effects of long-term stresses-heat, methoprene, and infection, on the growth, maturation and reproduction processes of lobsters. Western LIS lobsters exposed to known stress factors, as well as those infected by the paramoeba are being compared to field collected lobsters in the laboratory. Elevated temperature and pesticide levels could potentially alter lobster endocrinology, leading to effects on growth, molting and ultimately, survival. This project will evaluate several endocrine levels in response to laboratory manipulation of temperature and pesticide levels.

Physical/Chemical Environment

Relationship between American Lobster Mortality in LIS and Prevailing Water Column Conditions

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Wilson, Swanson and Waliser are examining water quality indicators, such as temperature, salinity, dissolved oxygen, and pollutants, with respect to the lobster mortalities. The lobsters are vulnerable to stress, and sometimes mortality, when exposed to unfavorable environmental conditions, especially during the molt cycle. Environmental factors can act singularly, or in combination, to cause sublethal stress that increases sensitivity to events that would otherwise be

tolerated. Significantly elevated bottom temperatures during the summer and fall of 1999 lead the team to focus primarily on co-variations in temperature and dissolved oxygen.

Effects of Temperature and Body Size on Metabolic Stress in LIS Lobsters

Investigator:

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Co-investigator:

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Lopez and Cerrato are trying to determine to what extent high summer temperatures in Long Island Sound's bottom waters have negative impacts on lobsters and whether larger lobsters are more susceptible to temperature stress than smaller ones. The results of their lab studies may be used to predict the effects of long term changes in summer temperatures on the health of the LIS lobster population. The study will shed light on normal patterns of lobster stress and mortality as well as the extraordinary mortality event of fall 1999.

Exposure of Lobsters to the Varied Chemical and Biological Environment of Long Island Sound

Investigators:

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Paulson, Deshpande, and Draxler are documenting lobster responses to the chemical and biological conditions of Long Island Sound in an attempt to uncover any direct relationship between lobster health and their environment. The experiment examines the health of lobsters under ambient Long Island Sound conditions during the time period when lobsters are believed to be most susceptible to these conditions. Lobsters, taken from uncontaminated regions are being evaluated, then placed in cages at six sites around western and central Long Island Sound. The sites have been chosen to represent a variety of environmental conditions. For four weeks, scientists will monitor the cages, routinely recover lobsters from each site, and evaluate them for changes attributed to exposure to naturally occurring biogeochemicals (such as ammonia and sulfide) as well as to contaminants. Lobster health will be assessed by bacterial determinations, and physiological condition. A limited number of the exposed lobsters will also undergo pathological examination.

Environmental Change in LIS in the Recent Past

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This project is based on an ongoing study, includes the development of a detailed time line of environmental changes in the LIS over the last decade through the evaluation of sediment cores. Evaluation of sediment will provide information on water temperatures, organisms within the food chain, dissolved oxygen levels, pollution and salinity. This study will help ascertain whether the lobster die-off is more strongly linked to global climate change or local contamination with pollutants or nutrients.

Shell Disease

Bacterial Assemblages Involved in the Development and Progression of Shell Disease in the American Lobster*, Homarus americanus*

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Co-investigator:

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By comparing shell disease in lobsters from Eastern Long Island Sound with those from Buzzards Bay, Massachusetts, Chistoserdov and Smolowitz seek to identify the strains of bacteria that cause lobster shell disease. The team is also designing a set of specific probes that are being used to test for such pathogens.

Paramoeba

Phenotypic and Molecular Identification of Environmental Specimens of the Genus Paramoeba Associated with Lobster Mortality Events

Investigator:

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Gillevet is using a combination of methods to isolate and characterize the paramoeba that has been identified in Long Island Sound lobsters. Gillevet and O'Kelly hope to develop a sensitive "fingerprinting" tool that will detect this paramoeba in the environment.

Oligonucleotide-based Detection of Pathogenic Paramoeba Species

Investigator:

Rebecca J. Gast, Woods Hole Oceanographic Institution, Woods Hole, MA.
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Gast is seeking to develop a method to facilitate the detection of paramoeba in animal tissues, water, and sediment samples. Using the new method, the researchers are performing a year-long sampling of Long Island Sound to analyze the paramoeba's fluctuation and distribution.

Development of polymerase chain reaction- and in situ hybridization-based tests for the specific detection of the paramoeba associated with epizootic lobster mortality by determination of the molecular systematics of the genus Paramoeba

Investigator:

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The purpose of this project is to produce and analyze gene sequences of a number of Paramoeba spp. and to develop a molecular diagnostic assay for the lobster Paramoeba sp.

Monitoring & Assessment

Two projects being conducted by NYSDEC and CTDEP are looked at as the glue that binds the 17 research projects together. Each is geared towards monitoring and assessing Long Island Sound's lobster population and its commercial lobster fishery.

Investigators:

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Carl Lo Bue, from NYSDEC's Division of Fish and Wildlife and Marine Resources, heads a study to combine a variety of lobster sea-sampling data to describe the existing population in the western Sound, document the commercial lobstering activities throughout the entire Sound, and develop methods to properly index the recruitment strength of young-of-year lobsters each year.

The second study, overseen by ***David Simpson*** from CTDEP's Marine Fisheries Division, addresses the need for expanded sea-sampling and trawl survey monitoring, sample collections for researchers, stock movements and identification, and habitat use.