NOAA Technical Memorandum NMFS-NE-105

Review of American Lobster (*Homarus americanus*) Habitat Requirements and Responses to Contaminant Exposures

U. S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
National Marine Fisheries Service
Northeast Region
Northeast Fisheries Science Center
Woods Hole, Massachusetts

July 1994

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Renee Mercaldo-Allen and Catherine A. Kuropat

Milford Lab., National Marine Fisheries Serv., Milford, CT 06460

U. S. DEPARTMENT OF COMMERCE
Ronald H. Brown, Secretary
National Oceanic and Atmospheric Administration
D. James Baker, Administrator
National Marine Fisheries Service
Rolland A. Schmitten, Assistant Administrator for Fisheries
Northeast Region
Northeast Fisheries Science Center
Woods Hole, Massachusetts

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NOTE ON SPECIES NAMES

The NMFS Northeast Region's policy on the use of species names in technical publications and reports is to follow the American Fisheries Society's (AFS) lists of scientific and common names for fishes (Robins *et al.* 1991)[a], mollusks (Turgeon *et al.* 1988)[b], and decapod crustaceans (Williams *et al.* 1989)[c], and to follow the American Society of Mammalogists' list of scientific and common names for marine mammals (Wilson and Reeder 1993)[d]. This policy applies to all issues of the *NOAA Technical Memorandum NMFS-NE* series.

Generally, the "sportsman's singular" style is used for the common names of all organisms (*i.e.*, the "s" or "es" normally appended to the plural form of a noun is omitted).

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[a] Robins, C.R. (chair); Bailey, R.M.; Bond, C.E.; Brooker, J.R.; Lachner, E.A.; Lea, R.N.; Scott, W.B. 1991. Common and scientific names of fishes from the United States and Canada. 5th ed. *Amer. Fish. Soc. Spec. Publ.* 20; 183 p.

[b] Turgeon, D.D. (chair); Bogan, A.E.; Coan, E.V.; Emerson, W.K.; Lyons, W.G.; Pratt, W.L.; Roper, C.F.E.; Scheltema, A.; Thompson, F.G.; Williams, J.D. 1988. Common and scientific names of aquatic invertebrates from the United States and Canada: mollusks. *Amer. Fish. Soc. Spec. Publ.* 16; 277 p.

[c] Williams, A.B. (chair); Abele, L.G.; Felder, D.L.; Hobbs, H.H., Jr.; Manning, R.B.; McLaughlin, P.A.; Pérez Farfante, I. 1989. Common and scientific names of aquatic invertebrates from the United States and Canada: decapod crustaceans. *Amer. Fish. Soc. Spec. Publ.* 17; 77 p.

[d] Wilson, D.E.; Reeder, D.M. 1993. Mammal species of the world: a taxonomic and geographic reference. Washington, DC: Smithsonian Institution Press; 1206 p.

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List of Acronyms

AEC	=	adenylate energy charge
BB	=	barite-bentonite
BKME	=	bleached kraft pulp mill effluent
CL	=	carapace length
DM	=	drilling mud
DO	=	dissolved oxygen
DSS	=	dodecyl sodium sulfate
EBP	=	early benthic phase
G6PDH	=	[indicator of pentose shunt activity]
HPCB	=	hexachlorobiphenyl
LC_{50}	=	[concentration at which 50% of experimental population dies]
LT_{50}	=	[time at which 50% of experimental population dies]
NTA	=	nitrilotriacetic acid
PAH	=	polycyclic aromatic hydrocarbon
PCB	=	polychlorinated biphenyl
TL_m	=	median tolerance limit
TPCB	=	tetrachlorobiphenyl

ABSTRACT

The American lobster (*Homarus americanus*) is a commercially important species found along the Northeast Coast of the United States and Canada. Lobsters respond differently to a variety of environmental conditions and contaminants based upon life stage. Larvae are generally less tolerant than juveniles and adults to environmental extremes or contaminant exposure. Understanding the complex interactions between the lobster and its environment can provide useful information for managing the fishery. This review summarizes much of the literature on: (1) habitat requirements of the American lobster, (2) effects of various contaminants on lobster biology as shown in laboratory and field exposures, and (3) contaminant concentrations measured in tissues of field-collected animals.

INTRODUCTION

The American lobster (*Homarus americanus*) occurs naturally along the East Coast of North America from Labrador to North Carolina, but is most common from Nova Scotia to New York (Herrick 1909; Cooper and Uzmann 1980). Lobsters are found from the relatively shallow intertidal zone to waters as deep as 700 m. They inhabit pristine oceanic environments as well as contaminated inshore locations (Aiken and Waddy 1986).

Habitat of the American lobster is defined by various environmental factors, including temperature, salinity, dissolved oxygen (DO), nitrogen, light intensity, current, substrate, and food. Addition of contaminants to the marine environment can affect the quality of lobster habitat. Contaminants are readily accumulated in lobster tissues, and can reach high concentrations in the hepatopancreas and eggs. Laboratory studies have determined tolerance limits for lethal concentrations of certain contaminants at various life stages, whereas field studies report contaminant concentrations in various tissues. Laboratory and field studies have also determined effects of contaminants on physiological and behavioral processes.

Biology of the American lobster has been reviewed extensively by Cobb and Phillips (1980a,b), but contaminant effects were beyond the scope of those volumes. Aiken and Waddy (1986) reviewed effects of environmental influences on larval recruitment. To date, no publication has reviewed habitat requirements of the lobster, as well as effects of contaminant exposure. This review describes the habitat of the American lobster and those environmental factors, both natural and human-induced, which may influence habitat quality. Emphasized are contaminant concentrations measured in lobsters during field studies and laboratory exposures, and, when possible, the effects of such exposures on the biology of the animal.

Environmental conditions and contaminant effects are discussed in terms of three life history stages: eggs/embryos, larvae/postlarvae, and juveniles/adults. These groupings were chosen to reflect developmental stages most commonly studied together, and are not meant to suggest discrete life stages. This is important in the case of the fourth-stage or postlarval lobster which is a transitional stage between a free-swimming and and benthic existence. In many respects, postlarvae are more similar to adults than larvae in their physiology, morphology, and behavior (Charmantier *et al.* 1991). Studies of environmental limitations or contaminant effects on larvae most often include the postlarval stage as well; postlarvae have been grouped with larvae for ease of discussion, but are also discussed in the juvenile/adult section when appropriate. Following is a broad overview environmental conditions and contaminant effects on: (1) eggs/embryos, (2) larvae/postlarvae, and (3) juveniles/adults.

EGGS/EMBRYOS

Laboratory studies have shown that environmental conditions influence the security of egg attachment to maternal pleopods, incubation success, rate of embryonic development, and survival of embryos. To survive the lengthy (10-12 mo) natural incubation period, embryos must have sufficient organic reserves, be securely attached, and remain free from disease, predation, and poor water quality (Aiken and Waddy 1985, 1986).

Adverse conditions tolerated by an egg-bearing female may threaten survival of the embryos she carries. The mechanisms allowing secure attachment of eggs to pleopods are not well known, but when conditions are unfavorable, eggs are lost from pleopods and die (Aiken and Waddy 1986). Loss of developing embryos between extrusion and hatching has been estimated at 36% (Perkins 1971). Attrition of embryos may be aggravated by frequent capture, handling, and release of berried female lobsters during fishing activity, and may be more dramatic late in development when embryos are less securely attached (Aiken and Waddy 1986).

LARVAE/POSTLARVAE

Physiological tolerances to environmental conditions must be such that larval recruitment to the juvenile stock is ensured (Phillips *et al.* 1980). Stressful or unfavorable environmental conditions can result in intermediate larval stages (Templeman 1936a). Molting to the postlarval stage can be delayed when larvae are exposed to unsuitable bottom conditions requiring the larvae to remain pelagic for a longer-than-average period (Cobb

1968). In both cases, metamorphosis is delayed and recruitment is threatened.

Larval recruitment can be altered by human activities that introduce contaminants to the environment or cause siltation and turbidity (Aiken and Waddy 1986). Planktonic larvae released in coastal waters can be carried through areas of high contaminant load where damage may occur (Johnson and Gentile 1979). Larvae, especially the first stage, are very sensitive to contaminants (Aiken and Waddy 1986). Larvae of the European lobster (*H. gammarus*) are 14-1000 times more susceptible to contaminants than adults of that species (Connor 1972). Exposure to contaminants can reduce survival and growth, increase incidence of malformation, delay development, alter metabolism and energetics, inhibit molting, and result in intermediate larval stages (Aiken and Waddy 1986). Limitations on contaminant levels in the environment designed to protect lobster larvae would also protect adults, and may provide protection for other species at the same or different trophic levels (Johnson and Gentile 1979).

JUVENILES/ADULTS

Environmental factors affect growth, survival, and reproduction of the American lobster in a variety of ways. These factors can act singularly or in combination to cause sublethal stress that increases sensitivity to events that would normally be tolerated. Molt stage, season, social conditions, age, nutrition, and reproductive state are some factors that influence how lobsters are affected by an environmental event (Aiken and Waddy 1986).

Exposure of juvenile and adult lobsters to contaminants can alter physiological and behavioral mechanisms necessary for survival, and may affect reproductive success. Contamination of edible tissues can reach concentrations above those considered safe for human consumption, and, once accumulated, many contaminants are slow to depurate.

PHYSICAL ENVIRONMENT

TEMPERATURE

Eggs/Embryos

Embryo development is regulated by temperature (Perkins 1972; Templeman 1940; Aiken and Waddy 1985) and proceeds slowly below 6°C (Aiken and Waddy 1986). Embryos at different developmental stages can grow at varying rates under the same temperature conditions (Perkins 1972). The time from egg extrusion to hatch is 39 wk at a constant 10°C, and 16 wk at 20°C. Berried lobsters carry their brood from 10 to 11 mo (Cobb 1976). Duration of hatching varies with temperature; eggs will hatch completely within 2-3 days at 20°C, but may require as long as 2 wk at 15°C (Hughes and Matthiessen 1962).

Larvae/Postlarvae

Appearance of first-stage lobster larvae in the plankton varies regionally, and occurs within a narrow range of surface temperatures (Harding *et al.* 1982). In New England, hatching begins as seawater temperature approaches 15°C, with intensive hatching occurring during June and early July at 20°C (Hughes and Matthiessen 1962). Duration of the planktonic phase depends on seawater temperature. Lobsters pass through the larval phase to reach the fourth or postlarval stage more quickly as seawater temperatures increase (Charmantier *et al.* 1991). Lobsters reared at 15°C developed to postlarvae within 25 days, and at 19-20°C, in approximately 13.5 days (Templeman 1936b; Hughes and Matthiessen 1962). MacKenzie (1988) observed the best larval growth at 15 and 18°C. The highest temperatures at which Templeman (1933) was able to raise larvae to stage four ranged from 23.6 to 24.6°C. Gruffydd *et al.* (1975) determined temperature tolerance in larvae reared at 20°C, and subsequently exposed to high temperatures (30-36°C) for 24 hr. All larvae held at temperatures above 30°C died after 6 hr. The temperature tolerance of stage-two larvae is lower than at any other larval stage (Sastry and Vargo 1977).

Larval size may also be affected by temperature. Offshore areas, which are generally cooler, appear to produce larger first-stage larvae than inshore waters (Rogers *et al.* 1968), although this temperature-dependent size difference was not observed beyond the fourth stage.

Juveniles/Adults

Temperature affects lobster growth, survival, and reproduction more than any other environmental parameter. Juvenile and adult lobsters are found seasonally in waters ranging from 0 to approximately 25°C, and can tolerate abrupt temperature changes (Aiken and Waddy 1986).

McLeese (1956) and Cooper and Uzmann (1980) estimate that preadult lobsters tolerate temperatures from 1.8 to 30.5°C at optimal DO and salinity levels. Tolerance to high and low temperature depends on acclimation temperature. Acclimation temperature, salinity, and DO all affect the upper lethal temperature range which increases as acclimation temperature increases, and declines as salinity and oxygen levels decrease.

Stein *et al.* (1975) studied behavior of juvenile and adult lobsters held in two experimental tanks at ambient temperatures and under thermal stress. Diurnal activity, evoked by presence of food, showed little change from 5 to 28°C. Nocturnal activity, generally more spontaneous, was similar at temperatures of 5-18°C. Thereafter, activity in one tank increased with increasing temperature, leveling off at 28°C. In the other tank, activity did not increase above 20°C, and remained at a lower level. Patterns of residence and dominance changed with season, but were not

correlated with temperature. Frequency of aggressive behavior at 22-28°C was similar to the frequency at ambient temperatures. Supernormal temperatures, within a few degrees of the lethal temperature, did not alter lobster behavior in juveniles or adults in any clear or consistent pattern.

Juvenile lobsters reared at fluctuating temperatures between 15 and 22°C showed reduced growth, greater mortality, and less resistance to a stress temperature (31°C) than animals held at a constant 22°C over a 126-day period. These differences were not observed until after the initial 2-3 wk of exposure, suggesting that short-term exposure to temperature fluctuations may not be detrimental to young lobsters. Growth of juveniles cultured at constant temperatures was most rapid at 20 and 24°C, but depressed above 24°C (Ford *et al.* 1979).

SALINITY

Eggs/Embryos

Embryonic development can vary with salinity (Aiken and Waddy 1985). Embryos (intact eggs surrounded by two egg membranes) have higher osmotic pressure and require longer adaptation time to low salinities than hatchlings (*i.e.,* emergent from the outer egg membrane, but still surrounded by the inner egg membrane) or prelarvae (*i.e.,* inner egg membrane split and animal released). Regulation in dilute media is hyperosmotic in embryos, and hyperosmoconforming in hatchlings and prelarvae. This change in osmoregulation which accompanies hatch is due to rupture of the relatively impermeable outer egg membrane which may provide some measure of protection for the embryo against low salinity (Charmantier and Aiken 1987).

Larvae/Postlarvae

Newly hatched lobster larvae held in the laboratory avoided seawater salinity of 21.4 ppt (Scarratt and Raine 1967). Templeman (1936b) successfully cultured larvae to the fourth-stage at 18-35 ppt salinity, but not below 17 ppt. The best survival to stage four occurred at 30.0-31.8 ppt; the number of days to reach this stage remained constant at salinities above 21 ppt. Sastry and Vargo (1977) noted that lobsters completed development to postlarvae at salinities between 20 and 35 ppt at 15°C, and between 15 and 30 ppt at 20°C. The survival was higher at 35 ppt at 15°C, and at lower salinities at higher temperatures (*i.e.,* 20-30 ppt at 20°C).

Larval stages one through three are slightly hyperosmoconforming (Charmantier *et al.* 1984c). Charmantier *et al.* (1984b) found that adaptation to a dilute medium occurred within 2 hr in stage-three larvae, and within 6 hr in stage-four larvae. Rapid adjustment of hemolymph osmotic pressure to salinity change is important to planktonic lobster larvae which may be exposed to less saline waters following a heavy rainfall. Stage-four animals survived better than stage-three in all salinities tested, especially between 12.5 and 20 ppt. There were no stage-three or stage-four larvae that survived exposure to salinities less than 12.5 ppt. Hyperosmoconforming larvae rely on isosmotic intracellular regulation because as planktonic stages they may be exposed to low salinity surface waters, while hyperosmotic postlarvae are adapted to seek and settle on the bottom where seawater salinity is usually high (Charmantier *et al.* 1984b, 1988).

Charmantier *et al.* (1985) determined effects of increased salinity on larval and postlarval lobsters using "Instant Ocean" sea salts added to seawater. At 20°C, the limit to survival for stages one through four (*i.e.,* molt stage A) was measured at 40.8-44.2 ppt. Stage-four lobsters at molt stage C survived at higher salinities of 44.2-47.6 ppt. At 11°C, stages one through four survived at 40.8-44.2 ppt. A salinity of 39.6 ppt was the upper limit for larval molting. Development from hatch to stage four was possible up to 36.3 ppt. Larval stages one through three were less resistant to elevated salinity than postlarvae and juveniles.

Juveniles/Adults

Because freshwater tends to overlay more saline waters, benthic organisms such as juvenile and adult lobsters seldom encounter reduced salinities (Aiken and Waddy 1986). Still, lobsters have been observed dead in their burrows following abnormally low salinities caused by high winds and heavy spring runoff (Thomas and White 1969).

At metamorphosis, hemolymph osmoregulation changes from the larval to the juvenile and adult form (*i.e.,* slightly hyperosmotic in dilute medium), consistent with the larval ecology of lobsters (Charmantier *et al.* 1984b,c, 1988). Salinity tolerance is reduced prior to (*i.e.,* stage three, molt stage D) and following (*i.e,* stage four, molt stage A) metamorphosis (Charmantier *et al.* 1991).

Young lobsters held at 20°C for 24 hr experienced 50% mortality both when held at 17 ppt during metamorphosis, and when held at 10.5-12 ppt in postlarval stages four and five. A strong correlation was observed between increased ability to osmoregulate (*i.e.,* hyperregulate in low salinity) and improved salinity tolerance (Charmantier *et al.* 1988). Survival of juveniles was good at salinities above 10.2 ppt (Charmantier *et al.* 1984a).

Juvenile and adult lobsters tend to be osmoconformers, but hyperosmoregulation occurs toward the lower end of their salinity tolerance range (Charmantier *et al.* 1984a; Dall 1970). Juveniles require 24 hr (Charmantier *et al.* 1984a), and adults 75 hr, to adjust to low salinity (Dall 1970).

McLeese (1956) recorded lethal salinity of 6 ppt at 5°C, and 16.4 ppt at 25°C, for lobsters held under acclimating conditions of 30 ppt of salinity and 6.4 mg/l of DO. Low salinity is more stressful at higher seawater temperatures (Jury 1992). Salinities below 15 ppt are metabolically stressful and energetically costly to lobsters, and salinities below 10 ppt may result in mortality. Lobsters appear able to detect salinities above those producing an avoidance response (17 ppt), and prefer higher (25-20 ppt) over lower (15-10 ppt) salinities (Jury 1992).

According to Jury (1992), estuarine lobsters respond behaviorally or physiologically to low salinity differently by sex. Females deal with fluctuating salinity by moving to optimal habitats or avoiding suboptimal zones. Female lobsters and their eggs and/or larvae may be physiologically limited from moving too far up an estuary because of adverse effects of reduced salinity. Males seem better able to tolerate estuarine salinities; oxygen consumption at low salinities (10 ppt) was lower in males, although both sexes were able to hyperosmoconform at 10 ppt for up to 3 days. Lobsters rely on ventilatory pulsing, as well as increased oxygen consumption, heart rate, and scaphognathite rate, to adapt to decreasing salinity. Increased physiological processes require a greater energy output, and can indicate stress.

Physiological condition, sex, molt stage, and size affect lobster response and tolerance to low salinity, the effect of which depends greatly on the rate of change, duration, and severity of the freshet. Metabolic response of lobsters to salinity may affect their distribution, movements, and migratory patterns. Salinity combined with other environmental factors may determine the dynamics of estuarine lobster populations (Jury 1992).

DISSOLVED OXYGEN

Larvae/Postlarvae

Experiments to identify DO requirements for lobsters at various stages of development were performed by Poucher and Miller (1990), Miller, Poucher, and Coiro (1992), Miller, Poucher, Coiro, and Hayden (1992), and Poucher *et al.* (1992). In flow-through exposures to DO concentrations ranging from 0.7 to 4.5 mg/l, Poucher and Miller (1990) found similar patterns of low DO sensitivity in the first three planktonic stages. Complete mortality occurred in the most hypoxic treatment within 2 hr, and the effects at higher DO levels were associated with intermolt development. The median lethal concentration (LC_{50}) of DO ranged from 2.5 to 3.2 mg/l during 96-hr exposures, and weight and time-to-molt were affected. Nonmolting fourth-stage lobsters exposed for 96 hr were more tolerant, with an LC_{50} of 0.8 mg/l. A continuous 15-day exposure beginning with first-stage larvae documented pronounced effects on growth and molt rate.

Miller, Poucher, Coiro, and Hayden (1992) measured a 96-hr LC_{50} of 3.5 mg/l of DO at 19°C for stages one and two, 3.0 mg/l for stages two and three, and 2.4 mg/l for stages three and four. Time to 50% mortality of the experimental population (LT_{50}) for stage-one larvae held at DO concentrations of 1.7 mg/l was 1.8 hr. First-stage lobsters had an LC_{50} of 3.2 mg/l of DO, and an estimated LC_{10} of 4.5 mg/l. Postlarvae from offshore stock exposed to reduced DO for 21 days had significantly lower mean dry weights than control animals. Mean dry weights at day 21 measured 12, 36, and 62% below control weights for DO concentrations of 3.5, 2.2, and 1.6 mg/l, respectively. Molting time of fourth-stage lobsters at DO concentrations of 1.6 and 2.2 mg/l was significantly longer than in controls.

Larval lobsters appear more sensitive than juveniles and adults by at least a factor of two (Miller, Poucher, and Coiro 1992). Poucher *et al.* (1992) noted retarded growth and delayed molting at reduced oxygen concentrations near the levels of incipient mortality (LC_{10}s). Postlarvae and juveniles, while less sensitive than larvae, exhibited reduced growth at DO concentrations at least 1 mg/l higher than those affecting survival.

Upon release from the female, planktonic larvae are exposed briefly to subpycnocline conditions (Miller, Poucher, Coiro, and Hayden 1992). But because pelagic larvae occur chiefly above the pycnocline, while the later stages are benthic, Miller, Poucher, and Coiro (1992) have suggested that separate DO criteria be established for above and below the pycnocline.

Juveniles/Adults

Lobsters are surprisingly tolerant of low oxygen concentrations, regardless of the level of oxygen acclimation (Cooper and Uzmann 1980). A lobster kill off New Jersey was attributed to the only environmental anomaly observed: low DO near the bottom, combined with high seawater temperatures (Young 1973). Low levels of DO may be more detrimental to lobsters approaching molt, when oxygen consumption reaches its highest levels (Penkoff and Thurberg 1982).

McLeese (1956) measured the tolerance range of subadult lobsters for DO. Lower lethal limit was 0.20 mg/l of DO in lobsters acclimated at 5°C, 30 ppt of salinity, and 6.4 mg/l of DO; it was 1.72 mg/l of DO in lobsters acclimated at 25°C, 20 ppt of salinity, and 2.9 mg/l of DO. In a later study, McLeese (1964) found that oxygen consumption remained constant in lobsters held at ambient oxygen concentrations ranging from 1.0 to 8.5 mg/l. Quantity of oxygen consumed per gram of body weight declined with increasing body weight. Oxygen consumption increased as a response to feeding, crowding, increased activity, and rising water temperature. Oxygen consumption rate in lobsters increased as DO increased.

McLeese and Watson (1968) measured oxygen consumption in lobsters held at 5°C. At DO concentrations greater than or equal to 1.7 mg/l, mean oxygen consumption rate was 20.96 mg/kg per hour. In lobsters held at DO concentrations less than 1.7 mg/l, oxygen consumption rate declined with declining DO concentration.

McMahon and Wilkens (1975) determined that adults maintain oxygen consumption at moderately reduced levels of ambient DO by increased branchial waterflow, more effective uptake of oxygen by gills, and increased oxygen transport by the respiratory pigment hemocyanin. Branchial and cardiac pumping rates decrease and become irregular at oxygen tensions below 30-40 mmHg, and death results if exposure exceeds 1-2 hr. Recovery from short-term hypoxia is rapid once aerated water is reintroduced, providing no cellular damage has occurred. Oxygen consumption remains high for several hours following hypoxia, suggesting repayment of accumulated oxygen debt.

Miller, Poucher, Coiro, and Hayden (1992) measured significantly lower dry weights, as compared with controls, in

fifth-stage lobsters held at DO concentrations of 3.5 and 1.5 mg/l. No difference in mean days to molt occurred at DO concentrations of 3.5 mg/l, but a delay was observed at 1.5 mg/l. Molting occurred by day 14 in all control larvae and over 90% of postlarvae at DO concentrations of 3.5 mg/l. Molting occurred between day 16 and 26 at DO concentrations of 1.5 mg/l.

Postlarval, juvenile, and adult lobsters were much less sensitive to low DO concentrations than larvae (Miller, Poucher, and Coiro 1992). Juveniles experienced reduced growth at concentrations at least 1 mg/l higher than those affecting survival. Molt rate was intermediate between growth and survival in sensitivity to low DO concentrations (Poucher *et al.* 1992).

COMBINED EFFECTS OF TEMPERATURE, SALINITY, AND DISSOLVED OXYGEN

Effects of temperature, salinity, and DO on juvenile/adult lobsters are broadly interdependent (Phillips *et al.* 1980). Tolerance to extremes of any of these factors, either individually or combined, was significantly enhanced by prior acclimation or physiological conditioning at sublethal levels of the factors being tested. (McLeese 1956; Cooper and Uzmann 1980). Existing near the limit of tolerance for one factor will reduce a lobster's ability to survive drastic change in another (Phillips *et al.* 1980). In adult lobsters, lethal limits of salinity and DO varied with temperature, and molting animals proved less resistant to high levels of temperature and low levels of salinity and DO than hard-shelled animals (McLeese 1956).

NITROGEN

Larvae/Postlarvae

Ammonia is the chief excretory product of lobsters, and is toxic at low concentrations. Delistraty *et al.* (1977) determined an LC_{50} of 1.4 mg/l, and a safe ammonia concentration of 0.14 mg/l, of un-ionized ammonia-nitrogen (NH_3-N) for postlarvae. An increase in pH from 7.4 to 8.4, or in temperature from 21 to 25°C, increased toxicity, whereas a salinity increase from 31 to 36 ppt showed no effect. This value for lethal concentration falls within previously established NH_3-N LC_{50} values of 8.2 mg/l at 24 hr [Sastry, pers. comm., in Delistraty *et al.* (1977)] and 1.2 mg/l at 96 hr for stage-12 juveniles [L. Gleye, pers. comm., in Delistraty *et al.* (1977)].

Young-Lai *et al.* (1991) found that tolerance of American lobsters to ammonia increased with ontogenetic development; stage one being most sensitive, and stage four most tolerant. This study determined safe concentrations of total NH_3 at 20°C to be 5.8 mg/l at stage one, 8.7 mg/l at stage two, 12.5 mg/l at stage three, and 14.5 mg/l at stage four.

Juveniles/Adults

McLeese (1970) exposed lobsters to a 1000 µg/g solution of NH_4OH, and although the lobsters demonstrated no avoidance response, exposure caused sluggishness and, in some cases, immobility.

Cornick and Stewart (1977) determined a mean toxicity of 60 nmol/ml of un-ionized ammonia (NH_4Cl) for 50% of lobsters held at 5°C for 30 days. This was consistent with an earlier study where at 40 nmol/ml, 5% of lobsters died and 60% weakened after 24 days. The LT_{50} for the two highest concentrations of ammonia tested resulting in 100% mortality were 22 days at 5600 nmol/ml of NH_4Cl, and 12 days at 8000 nmol/l, indicating a rapid lethal effect of ammonia.

Young-Lai *et al.* (1991) found adult lobsters less affected by exposure to ammonia than earlier life stages, tolerating concentrations of 21.9 µg/g of total NH_3 at 20°C, and 37.7 µg/g at 5°C. Ability of lobsters to osmoregulate at low salinities decreased after exposure to ammonia.

LIGHT INTENSITY

Larvae/Postlarvae

Ambient light intensity influences vertical distribution of larvae (Fogarty 1983). Depth selection may be influenced by light intensity, since larvae demonstrate greater upward movement and less downward movement on dull days than on bright days (Ennis 1975). Larvae congregate at the surface in dull daylight and moonlight, but scatter to greater depths in darkness. Although a minimum intensity of light is required to attract larvae to the sea surface, early stage larvae will seek lower light levels in bright sunlight (Templeman 1933; Templeman and Tibbo 1945). Huntsman (1924) reported that laboratory-held larvae died after several days of direct exposure to light. Eagles *et al.* (1986) found that larvae survived better at low light intensity than at a higher intensity that was less than full sunlight. Larvae reared in continuous darkness were heavier (10.8 mg) at fourth stage than those (5.5 mg) reared at a regime of 12 hr light, then 12 hr dark. However, continuous darkness reduced overall survival. Templeman (1936b) observed that fourth-stage larvae reared in almost complete darkness survived better, had longer carapace lengths, and developed more quickly than those held in laboratory lighting.

Juveniles/Adults

In a field study of Long Island Sound lobsters, Weiss (1970) found light intensity to strongly affect burrow occupancy and foraging behavior. Lobsters usually stayed in their burrows whenever ambient light intensity exceeded 400×10^{-4} µW/cm^2. Lobsters first emerged from their burrows 25 min after sunset at an underwater light intensity of 2×10^{-4} µW/cm^2 from June

through November. During December and January, lobsters did not appear until 40 min after sunset when light intensity was less than that level.

WATER CURRENTS

Little is known about how wind and currents affect survival, growth, development rate, or dispersal of larval lobsters (Aiken and Waddy 1986). Field behavioral observations and plankton tows showed that larvae concentrate where currents come together and produce zones of strong vertical motion called downwelling (Cobb *et al.* 1983; Aiken and Waddy 1986), and zones of strong surface motion called windrows (Harding *et al.* 1982). It is unclear whether larvae congregate passively in windrows, actively seek out these zones, or upon encountering a windrow simply maintain position there (Harding *et al.* 1982). Vertical distribution studies indicating low concentrations of larvae in subsurface waters have led to speculation that wind-induced surface circulation patterns influence larval distribution. Prevailing southwesterly winds along much of the northeastern coast of the United States may transport larvae from offshore locations to coastal sites, providing some degree of larval recruitment to coastal populations (Fogarty 1983).

Larval lobsters occur in the water column from May through October in New England. In Southern New England, hatching occurs earlier, and larvae are present in greater numbers, than observed in the Gulf of Maine (Fogarty and Lawton 1983). Larvae are at peak abundance during July and August (Sherman and Lewis 1967; Lund and Stewart 1970), and are often patchy in distribution; the latter is not surprising for relatively short-term members of the plankton community which are released intermittently by mobile benthic adults (Harding *et al.* 1982).

Few larvae are caught near bottom. Stages one through three are confined to the upper mixed layer, and early stage-four postlarvae are restricted to near-surface waters (Harding *et al.* 1987; Charmantier *et al.* 1991). In laboratory studies, presence of a thermocline limits vertical distribution of larval stages to the upper portion of the water column (Boudreau *et al.* 1991). Stages three and four occur deeper in the water column than first- and second-stage larvae, whether or not a thermocline is present.

In a related laboratory study, Boudreau *et al.* (1992) observed that fewer postlarvae settled to the bottom in the presence of a thermocline compared with an unstratified control situation. The thermocline may act as a strong barrier to settlement of young lobsters in nature. Settlement of late-stage postlarvae may be greatest in shallow water areas, where the absence of a thermocline would allow settlement behavior to proceed unhindered.

Larval stages one through three have limited swimming ability horizontally (Ennis 1986), and their movements are controlled mainly by water currents. Early postlarvae are active swimmers capable of moving along bottom. These divergent patterns of movement are important in dispersal of young lobsters (Charmantier *et al.* 1991). Rapid, directional swimming by postlarvae, along with advective processes, affect patterns of horizontal distribution and recruitment to the benthic stage (Rooney and Cobb 1991). Although postlarval lobsters avoid taking refuge in floating weeds and sticks (Cobb *et al.* 1983), early-stage larvae have been found in association with drifting macroalgae (Grabe *et al.* 1983) and floating patches of seaweed (Harding *et al.* 1982).

SUBSTRATE/SHELTER

Larvae/Postlarvae

Larval stages one through three exist free-swimming in the water column, and do not burrow or take shelter in sediment until molting to stage four. Stage-four postlarvae, although efficient swimmers, are morphologically prepared for a benthic existence; settlement begins before the end of this stage (Botero and Atema 1982). Before initial burrowing, postlarval lobsters search for a suitable substrate. If a substrate is rejected, larvae return to the plankton briefly before resuming search behavior. Selection of substrate on which to settle must be completed before molting to the fifth-stage; a lobster that molts outside of a burrow is exposed to extreme predation pressure (Bryant-Rich and Barshaw 1988).

Substrate preference and selection in postlarval lobsters have been studied extensively. Botero and Atema (1982) found postlarvae to prefer macroalgal-covered rock substrates, followed by rock-on-sand, mud, and sand substrates. If not provided with a choice, animals settled most often on macroalgal-covered rocks, followed by rocks scattered on sand and mud. Two weeks after settling was completed in these substrates, postlarvae explored sand habitat further with dives to the bottom, but did not settle. Pottle and Elner (1982) found that small lobsters chose gravel substrates over silt-clay substrates. In field studies, Roach (1983) found hatchery-reared postlarvae to survive best on rock substrate, followed by vegetation, then mud. Barshaw *et al.* (1985) and Barshaw and Bryant-Rich (1988) observed postlarval lobsters to settle most quickly into eelgrass, followed by rocks with algae in sand, then by mud. In a later study, Barshaw *et al.* (unpublished) observed postlarvae to settle quickly around rocks and salt-marsh peat, but to delay settling into sand. Hudon (1987) found high densities of postlarvae on rocky substrate with or without algae. Johns and Mann (1987) found that fourth- and fifth-stage lobsters, when offered a choice, selected habitats with seaweed more frequently than those without.

Postlarval lobsters actively select and prefer shelter-providing substrate, and settle most readily into rock/gravel, macroalgal-covered rock, salt-marsh peat, eelgrass, and seaweed substrates (Cobb *et al.* 1983). These complex habitats reduce mortality (Johns and Mann 1987) by providing lobsters with some

protection from predation (Roach 1983; Barshaw *et al.*, unpublished) and from other density-dependent limiting factors, including food and shelter (Hudon 1987). Complex environments, such as eelgrass, provide a refuge for settling postlarvae that remain protected until reaching a less vulnerable size (Barshaw *et al.* 1985; Barshaw and Bryant-Rich 1988). Peat-reef habitats, where early-benthic-stage lobsters are common, may provide important nursery areas for small lobsters, and could play a critical role in ecology of inshore populations (Able *et al.* 1988).

Flat sandy or muddy habitats are less preferred by young lobsters. Postlarvae released near bottom resume swimming if a sandy or muddy substrate is encountered, but shelter and burrow upon finding suitable substrate. Absence of shelter in these areas may limit lobster distribution (Cobb 1971). Botero and Atema (1982) found that although mud was not the most preferred habitat, lobsters that chose or were offered only mud settled successfully, creating "U"-shaped burrows without the aid of rocks or shells as a starting point. Berrill and Stewart (1973) suggested that postlarval stages four through eight are well adapted for living on a mud bottom, but observed that lobsters require stones or shells scattered over firm mud under which to dig out their tunnels. Lobsters in mud spend more time on burrow maintenance and repair than those in eelgrass habitats or rock-with-algae-on-sand habitats (Barshaw *et al.* 1985). Once a lobster has created a burrow, it remains burrowbound until reaching a size between 20 and 40 mm carapace length (CL) (Bryant-Rich and Barshaw 1988).

Cues used by lobsters in selecting a microhabitat may vary over ontogenic development, since postlarvae and juveniles behave differently with respect to shelter size (Boudreau *et al.* 1990). Wahle (1992a) suggested that lobsters exhibit an ontogenic shift from a strong, apparently predator-reinforced association with sheltering habitats early in their life history to a more wide-ranging existence with increased size. Change from cryptic to roving behavior in young lobsters occurs when the energetic demands exceed the shelter-based food supply, and the perceived predation danger no longer deters individuals from leaving their shelter.

Juveniles/Adults

Juvenile lobsters have distinct substrate preferences, but are capable of adapting to various habitats. This flexibility increases opportunities for settlement and growth during early, vulnerable stages of life (Aiken and Waddy 1986). Wahle (1988) and Wahle and Steneck (1991) found early-benthic-phase (EBP) lobsters (5-40 mm CL) most commonly in cobble habitats, and rarely in featureless mud, sand, or bedrock substrates where larger lobsters were common. Bedrock surfaces without macroalgae or mussel growth were devoid of EBP lobsters, while kelp-and-mussel-colonized bedrock supported an abundant population.

The EBP may be the most habitat-restricted portion of the life cycle (Wahle 1988; Wahle and Steneck 1991, 1992); shelter-seeking behavior results in a strong preference for shelter-providing cobble habitat over mud, sand, or gravel. This association with shelter-providing habitats during the first years of benthic life decreases with small increments in body size as lobsters become better able to escape predation. Requirements for shelter-providing substrates may create a natural demographic bottleneck to benthic recruitment, when prime habitat is limited. The role that habitat plays in fundamentally limiting lobster populations is still unknown (Wahle and Steneck 1991).

Addison and Fogarty (1992) observed that although cobble substrate may be a preferred habitat for juvenile lobsters, the presence of juveniles in other substrates--including eelgrass, bedrock, and mud--make it difficult to accurately assess potential habitat. Defining what constitutes suitable lobster habitat is complex; shelter size, prey abundance and availability, distribution, composition and abundance of predators, all contribute to whether or not a habitat is attractive to lobsters. When predation pressure is low, risks associated with occupying suboptimal habitat decline, increasing range of habitat types available.

Shelter-seeking and shelter-occupancy behaviors in lobster depend on the size of the animal relative to the nature of the substrate it is on (Wahle 1992b). Although the size of rock substrate may limit what size lobster can shelter there, substrate manipulations by lobsters can make an otherwise uninhabitable crevice into a shelter. Substrate-related behaviors permit lobsters to be relatively opportunistic in shelter-use over the range of substrates they encounter.

Hudon (1987) found that in lobsters greater than 25 mm CL, carrying capacity of a habitat was determined largely by substrate coarseness and the quantity and size of available cover. These factors also influenced density, biomass, and size structure of lobsters found there. Lobsters greater than 50 mm CL were present in areas characterized by algae, stones, or large crevices. Low numbers of large lobsters were found walking on compacted sand and on mud consolidated by eelgrass. In areas with larger stone size and heavier algal cover, postlarvae, juveniles, and adults all coexisted at high abundance. Low densities of juveniles and adults were observed in areas of sand covered with eelgrass, while bare sandy bottoms supported no resident lobsters.

Distribution of lobsters seems to be governed by two factors: availability of suitable habitat and behavioral selection of favored areas within those habitats. Suitability of habitat is a function of the animal's physiological tolerances and the presence of food, substrate, predators, and competitors, and is influenced considerably by availability of appropriate shelter or suitability of substrate for modification into shelter (Cobb 1977).

Shelters are of prime importance to lobsters (Karnofsky *et al.* 1989), and are used as a retreat from predators and adverse environmental conditions (Cobb 1971). Barshaw and Lavalli (1988) observed more postlarvae with burrows in rock substrate than in eelgrass or mud, and greater success at avoiding predation by cunner (*Tautogolabrus adspersus*) in rock substrates. Fewer postlarvae had burrows in mud than in eelgrass, and both habitats experienced high predation by this nonburrowing fish. The Say mud crab (*Dyspanopeus sayi*) preyed heavily on lobsters in all three habitats. In an earlier study

comparing rocky and sandy substrates exposed to predation by cunner, better survival of postlarvae occurred on rocky bottom (Lavalli and Barshaw 1986). Neither substrate afforded adequate protection from the gulf grassflat crab (*D. texana*). [The works of Barshaw and Lavalli (1988) and Lavalli and Barshaw (1986) referred to two species of mud crabs, *Neopanope sayi* and *N. texani*, respectively. Recent systematic revision has changed the scientific names to *D. sayi* and *D. texana*, respectively.]

Finding a dark shelter may be the most important factor in shelter selection; postlarvae prefer opaque over transparent shelters, and selection does not appear to be influenced by presence or absence of colonizing microorganisms, width or height of shelter, or number of entrances (Cobb 1971). A lobster chooses or excavates a shelter that is of sufficient size to enable it to maintain contact with walls and roof (Cooper and Uzmann 1977). Selection of man-made objects to take shelter in or excavate beneath occurs commonly with juveniles and adults and is increasingly important on open and barren mud, shell or sand substrates (Cooper and Uzmann 1977). Lobsters were observed digging shelters under eelgrass, rocks, and boulders (Karnofsky *et al.* 1989). Although young juveniles are difficult to observe in the field, older animals with obvious burrows appear to spend their entire early existence within the burrow or tunnel system (Roach 1983), leaving their shelters only at night (Karnofsky *et al.* 1989).

FOOD REQUIREMENTS

Larvae/Postlarvae

Larvae are carnivorous, and if provided with a diet of live copepods, will complete larval development (Templeman 1936b; Phillips and Sastry 1980). Larvae are commonly cultured on live or frozen brine shrimp (*Artemia salina*) (Hughes *et al.* 1974). Gunn (1987) examined stomach contents of larvae from Long Island Sound and found crab larvae, copepods, and cladocerans were most commonly consumed. Other food items included insect remains, invertebrate eggs, nematodes, and diatoms.

Juinio and Cobb (1992) observed nine taxonomic prey groups in the gut contents of field-collected postlarval lobsters. Frequent occurrence of copepods, decapod crustacean larvae, fish eggs, and insect parts indicated a predominantly carnivorous feeding habit. Frequency of large copepods and decapod crustacean larvae in guts of postlarval lobsters relative to densities of these prey in plankton, suggests a preference for larger-sized prey, with feeding activity greatest at night.

Barshaw (1989) found no significant difference in survival between groups of laboratory-reared postlarvae fed plankton or brine shrimp. Postlarvae consumed sufficient plankton for a net energy gain; significant increases in carapace length and weight of plankton-fed animals were observed.

After settlement, postlarvae appear to remain in burrows during much of their early benthic life, necessitating that they feed in a different manner and on different prey items than older animals (Barshaw 1989). Burrowed postlarvae may feed on plankton in the water column in addition to preying upon benthic organisms (Lavalli and Barshaw 1989), and may survive exclusively on zooplankton for much of their first year (Lavalli 1988).

Juveniles/Adults

Adult lobsters are omnivorous. The bulk of their natural diet consists of bottom-dwelling invertebrates (mainly crabs, polychaetes, mussels, periwinkles, sea urchins, and sea stars), but include fishes and seaweeds as well (Ennis 1973; Carter and Steele 1982a,b). Weiss (1970) found that decapod crustaceans and gastropod and bivalve mollusks were the most common constituents of the diet. Other food items, in order of importance, included polychaetes, fishes, plants, hydroids, acidians, echinoderms, and bryozoans. The relative contribution of assorted prey varies considerably from one area to another. Lobster stomach contents reflect relative abundances of prey species in the habitat rather than changes in food preferences of the lobster itself (Weiss 1970; Miller *et al.* 1971; Ennis 1973).

FIELD OR LABORATORY EXPOSURE TO CONTAMINANTS

The information on contaminants presented below is summarized in 29 tables. Table 1 lists the abbreviations, acronyms, and codes used in Tables 2-29.

HEAVY METALS

The following information on heavy metal effects is summarized in Tables 2-5.

Arsenic

Arsenic metabolism in juvenile/adult lobsters was examined by Cooney and Benson (1980). Six lobsters were fed sodium alginate pellets containing ^{74}As-labeled *Dunaliella tertiolecta* (an alga), while three other lobsters were fed pellets with ^{74}As-arsenate. Hepatopancreas and muscle tissues served as primary storage sites for organic and inorganic arsenic. Lobsters that consumed the ^{74}As-labeled algae had high concentrations of ^{74}As-arsenate in the hepatopancreas (an average of 38% of the ^{74}As-arsenate measured in the animal), stomach (13%), and tail (12%). The animals fed ^{74}As-arsenate pellets showed a similar pattern; levels were highest in the hepatopancreas (30%), tail muscle (20%), and stomach (11%). Organo-arsenic compounds

were detected in algae-fed lobsters, but not in those fed arsenate. This suggests the presence of arsenic in crustaceans is a result of accumulation through the food chain, rather than synthesis from arsenate by the organism.

Cadmium

Larvae/Postlarvae

Johnson and Gentile (1979) used static bioassays at 20°C to determine toxicity of three heavy metals to first-stage lobster larvae. Cadmium [$Cd(NO_3)$] was less toxic than copper or mercury, with a 96-hr LC_{50} of 78 µg/l. Mortality after 96 hr measured 3% in lobsters exposed at 10 µg/l, and 10% in animals exposed at 33 µg/l. The highest concentration of cadmium tested (1000 µg/l) produced 50% mortality within 24 hr, and 100% mortality within 48 hr.

McLeese (1981) found cadmium to be quite lethal to first-stage lobster larvae, reporting a long-term lethal threshold (no specific time reported) of 30 µg/l.

Juveniles/Adults

Eisler, Gardner, *et al.* (1972) exposed juvenile lobsters to 10 µg/l of cadmium ($CdCl_2$ 2½ H_2O) for 21 days in flowing seawater. Treated lobsters contained more cadmium per unit wet weight than did control animals; concentrations were 41% greater in whole lobster, 25% in muscle, 49% in exoskeleton, and 78% in gill tissue. Cadmium values in experimental animals measured 0.72 µg/g wet weight in whole lobster, 0.25 in muscle, 0.88 in exoskeleton, and 0.87 in gill tissue.

Adult lobsters were exposed to sublethal concentrations (3-6 µg/l) of cadmium and mercury for 30-60 days by Thurberg *et al.* (1977). Thirty-day cadmium concentrations in gill tissue measured 1.8 µg/g at exposures of 3 µg/l, 3.4 µg/l at exposures of 6 µg/l, and 1.5 µg/g in control animals. Significant uptake of cadmium occurred in gills of animals held more than 60 days; concentrations measured 2.32 µg/g in the 3-µg/l treatment, 2.63 µg/g in the 6-µg/l treatment, and 1.63 µg/l in control animals. Gills showed elevated rates of oxygen consumption, and indications of increased ATPase activity. Enzyme induction and reduced ligand sensitivity in the heart and antennal gland also occurred.

Using a flow-through seawater system, Tucker (1979) exposed lobsters to 6 µg/l of cadmium for 30 days. Gill homogenates showed a 25% increase in ouabain-insensitive ATPase activity. No difference in Na+K+ATPase was observed between control and cadmium-exposed animals. The increased ATPase activity may have resulted indirectly from increased metabolic activity within gill tissue, rather than directly from any effect of cadmium on the enzyme.

Gould (1980) exposed lobsters to 6 µg/l of cadmium for 30 days, followed by a 7-day holding period in "clean" seawater at ambient or low salinity. Sublethal cadmium exposure elevated the activity of glycolytic enzymes in lobster tissues, resulting in increased glycolysis, an expenditure of energy reserves.

A lead smelting plant near Belledune Harbor, New Brunswick, contributed to heavy cadmium contamination of lobsters in and around the harbor. This prompted numerous field studies designed to monitor sublethal effects of cadmium (Uthe and Zitko 1980).

Haya *et al.* (1980) used metabolic measures to monitor sublethal stress in lobsters collected from Belledune Harbor. Although cadmium concentrations in lobsters varied widely between individuals, two distinct populations were found within the harbor: those significantly contaminated with cadmium, and those having concentrations similar to uncontaminated control lobsters from Stonehaven, New Brunswick. Hepatopancreas cadmium measured 483.2 µg/g dry weight in contaminated lobsters, 27.17 in the uncontaminated population, and 30.88 in control animals. Adenylate energy charge (AEC), a measure of metabolic state, was within the normal range for lobsters. Cadmium concentrations were not correlated with changes in AEC or adenine nucleotide pools, both indicators of sublethal stress. No significant difference in ATPase activity was observed between cadmium-contaminated and uncontaminated lobsters. Belledune Harbor animals, both those with high cadmium concentrations and those with low concentrations similar to controls, showed no difference in adenine nucleotides or AEC. By these measures, the Belledune Harbor lobsters were considered unaffected by cadmium contamination.

The hepatopancreas, gill, and green gland tissues of 118 lobsters from areas at or near Belledune Harbor were examined histologically using light microscopy (Odense and Annand 1980). No histopathological changes were observed in any of these tissues, regardless of the hepatopancreas-cadmium burden of the animal. Closer examination of cadmium contaminated tissues using ultrastructural studies might have revealed changes that were not obvious using light microscopy.

Uthe (1980) studied forced depuration of cadmium in lobsters collected from Belledune Harbor. Dietary administration of insoluble, indigestible cadmium-binding agents did not reduce cadmium in the hepatopancreas. Injection of divalent ions and chelators at high concentrations also failed to reduce hepatopancreas cadmium concentrations. These results suggest that hepatopancreas cadmium is tightly bound in lobsters, and is dynamically unavailable for depuration through the hemolymph or gut lumen.

Lobsters raised for 3 mo in secondary sewage effluent had cadmium concentrations in edible flesh of 0.5 µg/g dry weight as compared to control values of 0.09 (Furr *et al.* 1981).

McLeese (1981) reported a long-term lethal threshold (no specific time given) of 5000 µg/g cadmium for adult lobsters.

Lobsters captured near Belledune Harbor were held live for 8 mo in uncontaminated St. Andrews, New Brunswick, seawater (McLeese *et al.* 1981). Initial cadmium concentrations in tissues measured 519 µg/g dry weight in hepatopancreas, 218 in green gland, 51 in gills, 23 in pincher claw nonmuscle, and 15 in crusher claw nonmuscle. No decrease in tissue cadmium concentrations were observed over the 8-mo period.

In a 14-day study, Brouwer *et al.* (1986) found that cadmium concentrations in the hepatopancreas of lobsters fed cadmium-rich oysters were five times higher than controls, and four times higher than lobsters exposed to cadmium (100 µg/l) in seawater. Cadmium concentrations in gills of lobsters exposed to dissolved cadmium were significantly higher than controls, but not significantly different from lobsters fed contaminated oysters. Four lobsters held in uncontaminated seawater for 40 days after exposure showed no significant loss of cadmium; hepatopancreas concentrations remained higher than in control animals.

Addition of 45 µg/g cadmium to a crab-based diet did not significantly affect growth or survival of juvenile lobsters (Chou *et al.* 1987). Cadmium in the crab-based diet was taken up by the hepatopancreas more efficiently than when added to a casein-based diet. Hepatopancreas cadmium concentrations were linearly related to dietary cadmium intake.

Copper

Larvae/Postlarvae

Johnson and Gentile (1979) estimated a 96-hr LC_{50} of 48 µg/l for first-stage larvae exposed to copper at 20°C. Mortalities at 3.3 and 10 µg/l were not significantly different from the controls. At 33 and 100 µg/l, a more-graded mortality pattern emerged. No larvae survived exposure at 330 µg/l beyond 24 hr. Comparisons with studies performed by McLeese (1974a) indicate stage-one larvae are nearly twice as sensitive to copper as adults.

Juveniles/Adults

Wilder (1952) determined relative toxicity of certain heavy metals to adult lobsters by exposing animals to wooden tanks floored with metal sheets. Copper was most toxic; all lobsters died within 18 hr of exposure.

In laboratory studies, McLeese (1974a) determined a lethal threshold (LT_{50}) of 56 µg/l for copper ($CuSO_4$) at 5 and 13°C and 20-30 ppt. Copper was more rapidly lethal to lobsters at 13 than at 5°C. Lowering the temperature from 13 to 5°C increased LT_{50} by a factor of two. Toxicity of copper to lobsters was not influenced by salinities within the range of 20-30 ppt.

Chemosensory response of lobsters to herring muscle extract, applied for 3 min, was reduced by simultaneous presentation of copper estimated at 4-80 times the lethal threshold (McLeese 1975). The reduction resulted from an attempt to avoid or escape the copper. Exposure to copper at concentrations of 0.7-1.8 times the lethal threshold for up to 48 hr reduced chemosensory response of lobsters. Gradual recovery usually occurred after 48 hr in clean water.

Furr *et al.* (1981) raised lobsters in secondary sewage sludge for 3 mo, and reported copper concentrations in treated lobsters were higher (155 µg/g dry weight) than in control animals (136).

Iron

Wilder (1952) found that wooden tanks lined with sheets of iron proved nontoxic to juvenile/adult lobsters; 100% of the lobsters survived for a 2-mo period.

Martin and Odense (1974) reported iron concentration in gills of eight adult lobsters of 21.1 µg/g wet weight. Histological examination of gill branchial tubules found that although iron was present in scattered patches, it failed to form a coating around the tubules as occurs in other decapod crustaceans. Examination of the cuticle and respiratory membrane located iron in an external position with relation to the cuticle.

Lobsters cultivated in secondarily-treated sewage sludge contained 202 µg/g dry weight of iron (Furr *et al.* 1981). Concentration of iron in edible flesh of control animals measured 50 µg/g.

Lead

Juvenile/adult lobsters exposed to lead-lined wooden holding tanks experienced 100% mortality within 474 hr (Wilder 1952).

Furr *et al.* (1981) measured 1.5 µg/g dry weight of lead in the edible tissue of lobsters held for 3 mo in secondary sewage effluent. Control lobsters contained 0.2 µg/g of lead.

Salinity effects outnumbered those caused by lead in adult lobsters exposed to sublethal concentrations of lead ($PbNO_3$), followed by immersion in low-salinity seawater (17 ppt) (Gould and Greig 1983). Decreased pentose shunt activity (G6PDH) in the antennal gland was the only effect directly attributable to lead exposure (0.05 µg/g for 30 days). The antennal gland contained the highest lead concentrations among tissues sampled; 30-day concentrations measured 138 µg/g wet weight in exposed lobsters as compared to 5.6 in controls. Lead concentration in the antennal gland dropped by one-third, and G6PDH returned to control values, in lobsters removed for 2 days to clean seawater at either ambient or low salinity. A less direct effect of lead exposure was loss of several normal adjustments to low salinity observed in control animals; weakening of normal physiological mechanisms may indicate a decreased ability to handle environmental stress.

Mercury

Larvae/Postlarvae

Mercury (HgCl) was the most toxic of three heavy metals tested against first-stage lobster larvae (Johnson and Gentile 1979). Estimated 96-hr LC_{50} at 20°C measured 20 µg/l, and

was probably conservative due to the potential for mercury to volatilize. Survival after 96 hr of exposure at 3.3 and 10 µg/l was similar to that observed in controls. Within 24 hr of exposure, mortalities of 30, 97, and 100% were observed at 33, 100, and 330 µg/l.

Juveniles/Adults

Thurberg *et al.* (1977) performed 30- and 60-day laboratory exposures to determine sublethal effects of 0.003 and 0.006 µg/g of mercury (HgCl) on adult lobsters. Animals exposed to 0.006 µg/g of mercury for 30 days contained an average of 1 µg/g wet weight in tail muscle, 15 µg/g in hepatopancreas, and 85 µg/g in gill tissues. After 60 days exposure at 0.006 µg/g, the mean concentration of mercury in gill tissue measured 119 µg/g. There was no effect of mercury on gill tissue respiration.

Nickel

Nickel contamination measured 5.7 µg/g dry weight in edible tissue of juvenle/adult lobsters reared for 3 mo in secondary sewage effluent. Control concentrations measured 1.4 (Furr *et al.* 1981).

Selenium

Furr *et al.* (1981) held juvenile/adult lobsters in secondary sewage effluent for 3 mo. Selenium in edible tissue measured 3.1 µg/g dry weight as compared to 2.2 in control animals.

Silver

Calabrese *et al.* (1977) reported on a 30-day exposure of adult lobsters to 0.006 µg/g of silver from unpublished data. Treatment with silver produced a biochemical pattern similar to that seen in mercury-exposed lobsters, but to a lesser degree: depressed heart transaminase, some loss of ligand sensitivity in the antennal gland, and some enzyme induction in the gonad. All effects were minor.

Zinc

Wilder (1952) reported that juvenile/adult lobsters exposed to zinc-lined wooden tanks died within 210 hr of exposure.

In a laboratory study, McLeese (1970) tested lobster behavior in response to several toxic or malodorous substances. Introduction of 500 µg/g of zinc chloride resulted in sluggish and sometimes immobile behavior; no avoidance response was observed.

Furr *et al.* (1981) cultured lobsters in secondary sewage effluent for 3 mo. Edible tissue of exposed lobsters contained 207 µg/g dry weight of zinc as compared with 135 µg/g in control animals.

Adult lobsters were exposed to 60 µg/g of zinc in a flow-through system at 10°C for 96 hr, and were subsequently transferred to noncontaminated seawater for 168 hr (Haya *et al.* 1983). No difference was observed in AEC in tail muscle, hepatopancreas, or gill between exposed and control animals. Gill Na- and K-ATPase activity and residual ATPase activity were reduced after zinc exposure, and failed to return to control levels during the 168-hr recovery period. Gill concentrations of zinc increased from 126 µg/g dry weight to 670 µg/g after 6 hr of exposure, and to 2570 µg/g after 96 hr. Concentrations declined to 675 µg/g during depuration. Lobsters exposed to zinc for 48 hr responded behaviorally with vigorous movement of swimmeretts and walking legs, perhaps in an attempt to increase irrigation of gills.

Waiwood *et al.* (1987) exposed lobsters to 25 µg/g of zinc in a flow-through system for 4 days before transferring them to flowing clean seawater for 7 days. Zinc concentrations after exposure were greatest in gill tissue (2570 µg/g dry weight), followed by green gland (1032), hemolymph (753), hepatopancreas (734), crusher tissue (542), crusher muscle (465), pincher tissue (377), shell (318), testes (242), heart (207), pincher muscle (169), and tail muscle (126). Zinc concentrations in tissues remained elevated after depuration. Hepatopancreas zinc concentrations continued to increase during the 7 days in clean seawater.

ORGANICS

Polychlorinated Biphenyls

The following information on polychlorinated biphenyl (PCB) effects is summarized in Table 6.

Eggs/Embryos

Bend *et al.* (1976) injected female lobsters with PCB Aroclors 1242 and 1254, and found that egg masses accumulated the highest PCB residues of all tissues examined, with a mean value of 4.41 µg/g wet weight, and a range of 2.78-5.98 µg/g. James *et al.* (1977) determined a half-life of 40 days for PCBs in lobster egg masses.

Juveniles/Adults

Bend *et al.* (1972) injected ^{14}C-2,4,5,2',5'-pentachlorobiphenyl, a component of Aroclor 1254, into the pericardial sinus of adult lobsters. Specific activity of various organs was determined 1 day and 1 wk after dosing. Hepatopancreas tissue

showed the highest activity of any tissue at both assay times. No decrease in radioactivity was detected in hepatopancreas, stomach, gills, or egg masses. Green gland, heart, tail, and claw muscle showed a statistically significant decrease, suggesting a redistribution or excretion of 2,4,5,2',5'-pentachlorobiphenyl or its metabolites over a 1-wk period.

In followup studies, Bend *et al.* (1973, 1974, 1976) examined distribution of ^{14}C-2,4,5,2',5'-pentachlorobiphenyl after a single pericardial sinus injection over a 2-, 4-, and 8-wk periods. Hepatopancreas tissue contained the highest radioactivity and the longest half-life (45 days), and contained more than 90% of the recovered radioactivity. Virtually all hepatopancreas PCB was unaltered. ^{14}C-PCB persisted in egg masses (39 days), stomach (28), gill (24), and intestine (23). The following tissues lost their radioactivity quickly: plasma (5 days), green gland (4.5), heart (3.5), male gonad (3.5), tail (3.5), and claw tissue (3.5). The hepatopancreas may be the principal storage site for PCBs, and the feces the major means of elimination. Bend *et al.* (1976) measured ambient concentrations of PCBs in lobster hepatopancreas ranging from 1.13 to 2.27 µg/g wet weight, with an average of 1.57 µg/g. Muscle concentrations were lower at 0.07-0.13 µg/g, with a mean value of 0.097.

James *et al.* (1977) injected 2,4,5,2',5',-pentachlorobiphenyl, a component of Aroclor 1254, into lobster hemolymph. Ninety percent of the dosage was taken up by the hepatopancreas; PCB was also absorbed by gonads. Lobsters eliminated PCBs slowly, with a half-life of 40 days in the hepatopancreas and 7 days in male gonads.

Connolly (1991) developed a food chain model for PCB contamination of lobsters from New Bedford Harbor. Contaminated food contributed 55-80% of the PCB measured. Sediment accounted for 35% of the trichlorobiphenyl, and 65% of the hexachlorobiphenyl, found in lobsters. Lobsters derive PCB from the water column through direct uptake across the gill, and by feeding on animals that accumulate PCB through the water column or sediment. The amount of PCB that accumulates is determined by how efficiently the ingested material is assimilated.

Contaminant concentrations in lobsters from Quincy Bay, Massachusetts, were evaluated for potential carcinogenic risk through human consumption (Cooper *et al.* 1991). This study estimated an increased cancer risk for any long-term consumer of even small quantities of Quincy Bay lobster tomalley (hepatopancreas), attributable to mean PCB concentrations of 30 µg/g in this organ.

Halogenated Hydrocarbons

The following information on halogenated hydrocarbon effects is summarized in Tables 7 and 8.

Eggs/Embryos

Guarino *et al.* (1974) found that when 100 µg/l of ^{14}C-DDT was administered to lobsters intravascularly, egg masses retained more than 1% of the dose for 1 mo after treatment. Residue concentrations of total DDT metabolites in untreated lobsters were highest in egg masses (1 µg/g).

High concentrations of pesticides in lobster eggs could alter reproductive performance. Lobsters may be able to withstand acute sublethal exposure to DDT by depositing the pesticide into fatty tissues, including the egg masses. High DDT concentrations in lobster egg masses may compromise survival of newly hatched larvae.

Larvae/Postlarvae

McLeese (1974b) studied effects of the organophosphate insecticide fenitrothion on lobsters. The lethal threshold of fenitrothion was 0.015 µg/l for larvae. Larval and adult lobsters both had a 96-hr LC_{50} of about 1 µg/l. Larvae were normal up to a concentration of 0.01 µg/l; above this concentration, larvae turned red because of chromatophore expansion. At concentrations of 0.1 and 1.0 µg/l of fenitrothion, larvae were unable to swim normally and sank to the bottom in a moribund condition, in about to ½ of the LT_{50}.

Stage-one larvae were more susceptible to chloramine (NH_4OH and NaOCl) than to similar concentrations of applied free chlorine (NaOCl) in a continuous-flow bioassay (Capuzzo *et al.* 1976). Estimated LC_{50} values were 16,300 µg/l for free chlorine, and 2020 µg/l for chloramine at 25°C; a synergistic effect with temperature was demonstrated. Exposure to free chlorine at 20°C resulted in no significant larval mortality, whereas exposure at 30°C resulted in an estimated LC_{50} value of 2500 µg/l. Chloramine was considerably more toxic, with an estimated LC_{50} value of 4080 µg/l at 20°C, and 560 µg/l at 30°C. Respiration rates changed during and after exposure to both contaminants; initial respiratory distress was detected at 50 µg/l of applied chloramine, and 5000 µg/l of applied free chlorine. Stress to lobster larvae induced by chlorine and chloramine may be irreversible; presence of these contaminants in the water column could significantly affect natural populations of this species.

In a followup study, Capuzzo (1977) exposed stage-one larvae to either 1000 µg/l of applied free chlorine or 1000 µg/l of applied chloramine in a continuous flow-through bioassay at 25°C for 60 min, and monitored their progress over a 19-day period. Exposed lobsters exhibited significant reductions in standard respiration rate and in growth as expressed by dry weight; the greatest difference was observed among chloramine-exposed animals. Acute exposure to either free chlorine or chloramine subsequently reduced growth and metabolic activity of larvae.

Juveniles/Adults

Guarino *et al.* (1972, 1974) administered ^{14}C-DDT to lobsters either intravascularly, orally, or by exposure through ambient seawater. When lobsters received intravascular injections, removal of pesticide from the plasma was rapid, followed

by an increase in hepatopancreas radioactivity. Within 7 days of injection, 90% of the administered dose had concentrated in the hepatopancreas. One month after treatment with 100 ng/g of ^{14}C-DDT, only egg masses, muscle, and hepatopancreas contained more than 1% of the administered dose. When introduced through ambient seawater or in food, 90% of the dose was measured in the hepatopancreas at 1 wk after treatment. Residue concentrations of total DDT metabolites in untreated lobsters were highest in the egg masses (1000 ng/g wet weight), followed by the hepatopancreas (400 ng/g) and muscle (100 ng/g). Total DDT metabolites in whole lobsters ranged from 140 to 260 ng/g wet weight.

Adult lobsters were exposed to fenitrothion, an organophosphate insecticide, by McLeese (1974b). Olfactory response to cod muscle was not affected by simultaneous presentation of fenitrothion at concentrations ranging from 0.05 to 25.4 µg/l. Larval and adult lobsters showed a similar sensitivity to this insecticide, having a 96-hr LC_{50} at 15°C of about 1 µg/l. The 24- and 48-hr LC_{50}s for adult lobsters were estimated between 10 and 100 µg/l, and the lethal threshold measured 0.3 µg/l or lower.

McLeese (1974c) performed a similar, related study with adult lobsters, using the organophosphate insecticide phosphamidon, in static and flowing seawater tests. The 96-hr LC_{50} value was 180 µg/l at 4°C, and the lethal threshold value was 18 µg/l. The LC_{50} measured 50 µg/l, and the lethal threshold value was 28 µg/l at 12°C. Reduced temperature resulted in longer survival; the LT_{50} for animals at 4°C was four times as long as those at 12°C. Phosphamidon was 50-60 times less toxic to lobsters than fenitrothion.

Lobsters exposed to 2.5-100 times the lethal threshold concentration of phosphamidon and an extract of herring muscle simultaneously experienced no reduction in chemosensory response. Exposure to phosphamidon for 48 hr at 0.7-1.8 times the lethal threshold also had no measurable effect (McLeese 1975).

In static exposures at 20°C, the insecticide methidathion proved more toxic to adult lobsters than phosphamidon (McLeese and Metcalfe 1979b). Lethal thresholds were 10 and 18 µg/l, respectively. The 48-hr LC_{50} was estimated at 107 µg/l for phosphamidon, and 14 µg/l for methidathion. Mixtures of these two insecticides produced greater than additive toxicity to lobsters.

McLeese *et al.* (1980a) observed rates of dietary accumulation and clearance of two chlorobiphenyls: 2,2',4,5' tetrachlorobiphenyl (TPCB) and 2,2',4,4',5,5' hexachlorobiphenyl (HPCB), and of endrin in lobsters fed tainted mussels. Concentrations of TPCB and HPCB in food were 600 and 550 µg/l wet weight at the low dosage, and 4000 and 4900 µg/l at the high dosage, respectively. Maximum concentration factors (concentration in hepatopancreas to concentration in food) for TPCB and HPCB measured 5.1 and 7.3 at the low dosage, and 1.0 and 2.9 at the high dosage, respectively. Chlorobiphenyls accumulated to a greater degree in the hepatopancreas than in the tail or muscle, perhaps due to the high lipid concentration of the hepatopancreas. In all cases, chlorobiphenyls in the hepatopancreas decreased during a 6-wk clearance period. Concentrations in the low dosage approached that of the controls, while at the high dosage tests, TPCB and HPCB declined by 60 and 40%, respectively. During the endrin test, mean concentration of spiked mussels measured 4.7 µg/g. Maximum concentrations of endrin 2 wk after feeding measured 1.95 µg/g wet weight in the hepatopancreas, and 0.0047 in tail meat. After a 4-wk excretion period, endrin in tail muscle had decreased by 35%.

McLeese *et al.* (1980b) determined lethality of the insecticides permethrin, cypermethrin, and fenvalerate to adult lobsters using static tests. Lethal threshold (µg/l) for these contaminants at 10°C measured 0.68, 0.04, and 0.08, respectively, with 96-hr LC_{50}s (µg/l) being 0.73 for permethrin, 0.04 for cypermethrin, and 0.14 for fenvalerate.

Lake *et al.* (1981), cited in Capuzzo *et al.* (1987), measured polychlorinated dibenzofurans and related compounds in lobsters from Narragansett Bay. Although concentrations in biological resources decreased with increased distance from an urbanized area (Providence River), the compounds were still detectable in samples from less polluted areas (lower Narragansett Bay). The high toxicity of these and related compounds warrant further documentation of the potential transfer of industrial contaminants to humans through consumption of marine products.

Petroleum Hydrocarbons

The following information on petroleum hydrocarbon effects is summarized in Tables 9 and 10.

Larvae/Postlarvae

Toxicity of two nonionic, oil-dispersing agents, Aquaclene-100 and Colloid-88, was determined for fourth-stage lobster larvae at 20°C (Engel and Neat 1971). Median tolerance limit (TL_m) to Aquaclene-100 was 54 µg/g at 24 hr, and 46 µg/g at 48 and 72 hr. Exposure to Colloid-88 resulted in a TL_m of 50 µg/g at 24 hr, and 46 µg/g at 48, 72, and 98 hr. Larvae seemed particularly sensitive to these dispersants immediately after molting. The 24-hr toxicity observed for larvae suggests that toxicity of oil dispersants is due to the relatively volatile solvent fraction.

Wells (1972) found emulsified Venezuelan crude oil lethal to lobster larvae at 100 µg/g within 24 hr at 20-21°C. Larvae at 10 µg/g survived poorly and were unable to complete metamorphosis. At these concentrations, larvae experienced changes in color and behavior. Larvae in 0.1 and 1.0 µg/g of oil survived and developed to fourth stage in a manner similar to controls. Ninety-six-hour LC_{50}s ranged from 2 to 30 µg/g. Some larvae at 6 µg/g did not develop beyond third stage, even after 30 days, and those that did were intermediate between third and fourth stage.

Wells and Sprague (1976) exposed larval and postlarval lobsters to Venezuelan crude oil for up to 8 wk at 20°C. Stage-one larvae were more sensitive to oil than later larval stages,

having a 96-hr LC_{50} of 0.86 µg/g. The 96-hr LC_{50} was 4.9 µg/g for third- and fourth-stage lobsters. Larvae beginning the test at stage one had a 30-day LC_{50} value of 0.14 µg/g; a slowdown of larval development was observed at this concentration. Decreased food consumption was observed at 0.19 µg/g; this may have resulted from larval weakening in oil, interference with chemoreception and food detection, and/or reduced palatability of oiled food. "Intermediate" larvae, those which retain the characteristics of previous stages, were observed in 11.8% of oil-exposed larvae and in 1.5% of control larvae. Postlarval lobsters did not avoid oiled substrate, but dug significantly more burrows. Neither growth nor survival of postlarvae was affected in or by substrates containing up to 1740 µg/g of oil. The harmful concentrations stated for oil may have been conservatively high because they were based on initial concentrations that declined by 59% during the experiment. The LC_{50} of oil particulates was 1.8 µg/g, one-third the toxicity of soluble components.

Forns (1977) exposed larvae to 0.1 and 1.0 µg/g of South Louisiana crude oil in a flow-through system at ambient seawater temperatures (19.2-23.8°C). Animals exposed to 0.1 µg/g of crude oil were active feeders, had consistent locomotive behavior, and were aggressive, whereas larvae at 1.0 µg/g were lethargic, displayed minimal mobility and depressed feeding, and appeared nearly dead. Control and 0.1 µg/g-exposed larvae molted to the fourth stage within 12 days of hatching, while animals in the 1.0 µg/g treatment required at least 15 days to reach metamorphosis. Total survival of lobsters at 1.0 µg/g of crude oil was 50% below that of the 0.1 µg/g and control animals. Additionally, larvae held at 1.0 µg/g experienced an alteration of color from the normal, almost transparent, pale blue to bright red, due to chromatophore expansion. Such a coloration change could threaten larval survival by making them more vulnerable as prey.

Larval lobsters were exposed to 0.25 µg/g of South Louisiana crude oil naturally or chemically dispersed in seawater for 96 hr at 20°C in flow-through bioassays, and/or were fed oil-contaminated *Artemia* (Capuzzo and Lancaster 1981, 1982; Capuzzo *et al.* 1984). Oil-exposed larvae experienced a disruption in energetics, significant molting delays, respiration rate reductions, slowed growth rates, and oxygen:nitrogen ratio changes; all of which occurred within 24 hr in stage-one animals, and within 72 hr in later stages. Impairment of molting and growth due to oil exposure may be related to interference with normal energetic pathways. Disrupted patterns of lipid storage, utilization, and biosynthesis occurred during larval development and metamorphosis. Significant differences in protein and lipid content and in protein:lipid ratio were observed between control and oil-exposed lobsters. Among oil-exposed animals, significantly higher protein content and significantly lower lipid content were detected. Exposure to naturally and chemically dispersed oil resulted in similar toxic effects for larvae; therefore, toxicity was not enhanced by use of a chemical dispersant. Larvae fed oil-contaminated *Artemia* showed earlier signs of stress than larvae exposed to oil-seawater dispersions; the later larval stages showed some restoration of energetic patterns during exposure. Reduced survival rates were observed in third- and fourth-stage larvae fed the spiked *Artemia*. Hydrocarbon turnover appeared to be rapid, and little bioaccumulation of petroleum-derived hydrocarbons was observed.

A change in energetics due to oil exposure may be crucial as larvae approach metamorphosis and begin to shift habitat preference from pelagic to benthic, and/or to adopt new behavior. Such changes may affect both larval recruitment and postlarval survival because energy reserves may not be sufficient during this transition period (Capuzzo *et al.* 1984).

Juveniles/Adults

The wreck of the vessel *Arrow*, which released approximately 72,000 barrels of Bunker C fuel oil into Chedabucto Bay, Canada, prompted studies on acute toxicity of oil and oil dispersants on juvenile lobsters (Sprague and Carson 1970), as well as an assessment of oil accumulation by benthic organisms in the area (Scarratt and Zitko 1972). Sprague and Carson (1970) exposed lobsters to oil in static tests at 5°C for 7 days. A 96-hr median LC_{50} was determined for each toxicant: Bunker C fuel oil at greater than 10,000 µg/g, the dispersant Corexit 8666 at greater than 10,000 µg/g, and the dispersant Dispersol SD at greater than 1000 µg/g, as well as a combination of oil and Corexit at greater than 10,000 µg/g. Lobsters were extremely resistant in all tests. Scarratt and Zitko (1972) found the following concentrations of Bunker C fuel oil in lobster tissues 3 mo after the spill: abdominal muscle--1 µg/g wet weight, claw muscle--2, stomach--15, and intestine--103. Within 6 mo, concentrations had increased to 4, 3, 230, and 130 µg/g wet weight, respectively. Control lobsters collected from Pictou, Nova Scotia, contained some fuel oil residue: abdominal muscle--5 µg/g, claw muscle--4, stomach--19, and intestine--57. A laboratory study exposing lobsters to 10,000 µg/g of Bunker C oil at 4°C for 6.5 days, followed by 7 days in flowing seawater, resulted in high body burdens. Abdominal and claw muscle contained 137 and 35 µg/g wet weight, respectively. Stomach and intestinal concentrations were recorded at 2840 and 1810 µg/g.

Atema and Stein (1972, 1974) and Blumer *et al.* (1973) observed changes in behavior and feeding of adult lobsters when small quantities of crude oil (10 µg/g) were added directly to test aquaria. Crude-oil exposure altered water-chemistry-sensing movements. Timing of feeding behavior showed that the delay period between noticing food and going after it doubled when oil was added. The water-soluble fraction of crude oil alone (in the 0.05 µg/g range) had no noticeable effect on behavior or feeding. Morphological changes in odor receptors after oil exposure were not detected by light or electron microscopy. Small quantities of oil mixed into seawater create a noxious smell in the lobster's environment, depressing the animal's appetite and chemical excitability.

Atema *et al.* (1973) and Atema (1976, 1977) found a significant change in lobster feeding behavior after exposure to kerosene and kerosene fractions. Lobster communities were monitored after a 30-min exposure to two asbestos strips treated with 0.02 ml of test material. Exposure to whole kerosene and

its branched-chain fractions induced searching and feeding behavior, leading to ingestion of the strips. Whole kerosene was less attractive than the branched-chain cyclic fraction due to the polar-aromatic fraction contained in whole kerosene. This polar aromatic fraction, although inducing a searching behavior at a distance, repulsed lobsters at close range. The straight-chain fraction did not influence lobster feeding behavior. Stimulation of feeding behavior was traced to the branched-chain, cyclic, saturated hydrocarbons present in kerosene.

Atema (1976, 1977) exposed lobsters to kerosene fractions (in the thousandths of a milliliter range) soaked into pieces of brick, and placed upstream of their shelters. Whole kerosene fractions depressed activity, followed by some attraction and feeding attempts. Afterward, food ingestion ceased for 3-7 days. Lobsters were ambivalent when presented with a branched-cyclic fraction, demonstrating aggressive behavior toward the brick, and simultaneous feeding by the maxillipeds and rejection of food by the periopods. The polar-aromatic fractions increased lobster activity including spastic behavior, fast approach and rejection of the brick, and accelerated food ingestion. The straight-chain fraction depressed lobster activity, prolonging attempts at feeding. Food ingestion ceased for 3-4 days after feeding attempts on this fraction. Sublethal exposure to various hydrocarbon fractions resulted in both feeding attraction and repulsion.

Atema (1976, 1977) examined the effects of 10 µg/g of La Rosa crude oil on lobsters. Behavior was monitored for 5 days before and after oil was introduced. Movement of chemosensory-related appendages changed significantly after exposure, and lobsters required twice as much time to locate food. Oil exposure decreased chemical stimulation and increased tactile behavior. Animals subjected to only the soluble fraction of La Rosa crude oil (0.01 µg/g) showed no measurable effect.

Lobsters were exposed to predetermined concentrations of the water-accommodated fraction of No. 2 fuel oil in a flow-through dosing system over a 5-day period (Atema 1977; Atema *et al.* 1979; Atema, Karnofsky, *et al.* 1982). At 0.8 and 0.15 µg/g, effects were minor, and recovery immediate, after removal from oil. Exposure to 0.3 µg/g caused consistent sublethal interference with feeding behavior which continued for 1 day beyond treatment. After 30 hr at 1.5 µg/g, feeding ceased for up to 6 days. Severe neuromuscular abnormalities appeared in the 1-2 µg/g treatments, leading to cramped postures, spastic behavior, unresponsiveness, and death. The chemoreceptors of lobsters showed modified responses to food odors at 3 µg/g, suggesting that this concentration of oil interfered with their ability to detect food. Oil may alter feeding behavior directly by affecting chemoreception, or indirectly by affecting feeding motivation such as hunger or fear.

Lobsters held in floating live cars were exposed to a spill of light Iranian crude oil off the New Brunswick coast (Aiken and Zitko 1977). Within 24 hr of exposure, lobsters in the top layers of the crates were dead or dying from osmoregulatory imbalance. Removal of lobsters to clean seawater reversed the symptoms in many animals. Spill conditions were simulated using laboratory exposures of lobsters to Iranian crude oil as a light slick (40 ml) under turbulent conditions or as a heavy slick (280 ml) under calm conditions. In both cases, healthy animals survived exposure without suffering osmoregulatory impairment, behavioral problems, or obvious histological damage. These laboratory results suggest the osmoregulatory symptoms displayed by lobsters held in commercial crates probably resulted from poor storage conditions rather than contact with crude oil.

Adult lobsters were dosed weekly with 100 ml of Venezuelan crude oil in flowing seawater for a 14-wk period (Payne *et al.* 1983). Concentrations in the experimental tank ranged from 0.3-0.5 µg/g on day 1 to 0.04-0.06 µg/g by day 6. This exposure elevated serum calcium concentrations, decreased serum freezing point depression, and altered values of plasma proteins, lipids, and essential amino acids. Lipid values in blood hemocytes were reduced by half. Gill browning, a sublethal effect, increased dramatically in oil-exposed animals, indicating possible gill-tissue damage. Since gill browning is pathological in nature, this effect could be used for biologically monitoring point sources of petroleum pollution in lobster habitats.

Polycyclic Aromatic Hydrocarbons

The following information on polycyclic aromatic hydrocarbon (PAH) effects is summarized in Table 11.

Larvae/Postlarvae

McLeese and Metcalfe (1979a) determined a 96-hr lethal threshold of 0.02 µg/g of creosote for lobster larvae at 20°C. Larvae were much more sensitive to creosote than adults; 96-hr thresholds differed by two orders of magnitude.

Juveniles/Adults

PAH concentrations in lobsters were determined before and after a 3-mo storage in a commercial tidal pound constructed of creosoted lumber (Dunn and Fee 1979). Initially, mean PAH values in tail muscle measured less than 1.0 ng/g. After impoundment, concentrations averaged 79 ng/g, with the most contaminated lobster containing 281 ng/g.

McLeese and Metcalfe (1979a) determined a 96-hr lethal threshold of 1.76 µg/g of creosote for adult lobsters at 10°C. Hepatopancreas creosote concentrations increased with increasing exposure concentration. Control animals had a concentration of 670 µg/g of creosote per gram of lipid, as compared to 3220 µg at a treatment of 0.3 µg/g (*i.e.*, the lowest exposure concentration), and 47,500 µg at a treatment of 2.5 µg/g (*i.e.*, the highest exposure concentration).

Lobster mortality was linked to PAH contamination in a culture facility (Zitko 1981). Five dead lobsters showed high hepatopancreas concentrations of fluoranthene and pyrene that may have leached into the facility seawater from creosote-treated pilings at a nearby wharf. Dimethylnaphthalenes in the lobsters may have been a product of creosote leaching or spilled fuel oil. BHT (2,6-di-*tert*-butyl-4-methylphenol) detected in hepatopan-

creas tissues probably originated from materials present in the culture facility, including plastic pipes, paint, sealants, *etc.* Lobsters in holding facilities may not be free from contaminants, and may contain higher concentrations than those in the wild.

McLeese (1983) surveyed commercial lobstermen in Canada, and determined that 48% of their lobsters were stored for periods of 2 hr to 2 wk in crates, cars, or tanks where there was the possibility of creosote exposure. PAH contamination of lobsters during these short-term exposures was not severe. About 6.7% of the lobsters were stored for over 2 mo in tidal pounds where creosoted materials were used. These animals contained carcinogenic PAH measurements above control values. Contamination of lobsters with PAH was greater during summer than winter, although most lobster storage took place during winter.

Contamination of lobsters with PAH compounds increased after 3-mo of storage in a commercial tidal pound constructed of creosoted lumber (Uthe *et al.* 1984). More PAH accumulated at warmer than cooler temperatures. Concentrations continued to rise in tail muscle after transfer to uncontaminated water, presumably as PAH was transferred from the hepatopancreas to other tissues. Holding contaminated animals in clean water resulted in reduced PAH concentrations; this reduction was most rapid during summer.

Williams *et al.* (1985) compared flavor of cooked meat from lobsters exposed to a diesel spill for 10 hr in Arnold's Cove, Newfoundland, to uncontaminated animals from Fairhaven, Newfoundland. Although exposure to oil did not make the meat unpalatable, the tainted meat did have a slightly different flavor.

Organic Metals

The following information on organic metal effects is summarized in Table 11.

Larvae/Postlarvae

Lobster larvae are very sensitive to tributyltin oxide (Laughlin and French 1980). At 0.02 µg/g, 100% mortality occurred within 24 hr. Concentrations between 0.005 and 0.015 µg/g were acutely toxic within 6 days. The one larva held at 0.001 µg/g that survived to fourth-stage weighed significantly less (*i.e.*, 1.132 mg dry weight) than the six surviving control animals (range of 2.436-3.775 mg).

Juveniles/Adults

Guarino *et al.* (1972) looked at organ distribution of ^{14}C-methyl mercury in lobsters which had received the pollutant intravascularly, orally, or by uptake from ambient seawater. Twenty-four hours after intravascular injection, high concentrations were observed in heart, hepatopancreas, and gonads. Large amounts of radioactivity were also present in intestine, brain, and gill. Hepatopancreas concentrations decayed with a half-life of about 14 days, with a corresponding redistribution of metal into tail muscle which doubled by 33 days after injection.

Six days after methyl mercury was administered via food, 68% of the absorbed dose was measured in hepatopancreas, and 5% in tail muscle. When introduced through ambient seawater, the hepatopancreas contained 23% methyl mercury and the tail muscle 60%.

Other Organic Chemicals

The following information on other organic chemical effects is summarized in Table 11.

McLeese (1970) exposed juvenile/adult lobsters to a 1000 µg/g concentration of several organic chemicals, including glacial acetic acid, formalin, and pyridine. No avoidance response was observed; however, responses to formalin and pyridine exposure included sluggishness and, in some cases, immobility.

INDUSTRIAL WASTES

Yellow Phosphorus

The following information on yellow phosphorous effects on juvenile/adult lobsters is summarized in Table 12.

An incipient lethal concentration of 40 µg/l was determined for yellow phosphorus exposure in adult lobsters (Zitko *et al.* 1970). Toxic effects of phosphorus appear to be irreversible; lobsters exposed to contaminated mud and then moved to clean water died simultaneously with those remaining in continuous contaminant contact. LT_{50}s in both instances were 265 hr. Time-concentration LT_{50}s for lobsters were determined for 10-120 µg/l. Lobsters exposed to lethal concentrations of yellow phosphorus experienced muscle tone loss, lethargy, jerky uncoordinated pereiopod movement, and loss of neuromuscular response. Primary cause of mortality was asphyxiation; at time of death, hemolymph in the immediate vicinity of the heart and stomach had gelled, and was unable to circulate freely through the gills.

Aiken and Byard (1972) exposed adult lobsters to phosphorus-contaminated mud from a polluted site in Long Harbor, Newfoundland. An industrial discharge of yellow phosphorus in this area during 1969 resulted in crustacean mortality which was attributed to hemolymph coagulation and asphyxiation. Laboratory exposure to yellow phosphorus caused degenerative changes in all four cell types in the hepatopancreas. Mortality appeared to be the result of hemolymph coagulation and asphyxiation. Hemolymph clotting may be triggered by damage to cells of the antennal gland and hepatopancreas, rather than by the phosphorus directly. Toxic effects of yellow phosphorus

on lobsters are cumulative, and are not rapidly reversed by transfer to clean water.

Potash Brine

The following information on potash brine effects is summarized in Table 13.

Larvae/Postlarvae

Charmantier *et al.*(1985) conducted bioassays using larval and postlarval lobsters to determine dilution criteria for potash brine in seawater. Concentrations in excess of 3 g/l caused larvae to change from their natural blue-green color to red-green. Contact with brine resulted in several seconds of avoidance response, followed by a quiescent period when larvae and postlarvae were lying on their sides rapidly moving their appendages. The 96-hr LC_{50} ranged from 1 to 2 g/l of potash ore in larval stages one through three at 20°C. Development from hatch to stage four was only possible when concentrations of potash brine were under 1.5 g of ore per liter of seawater.

Juveniles/Adults

Charmantier *et al.* (1985) found that juvenile lobsters were more resistant to brine than larvae: LC_{50}s measured 2.25-3 g/l of potash ore at 20°C, and 1.5-2.25 at 11°C. Juveniles did not display the same color changes as larval or postlarval lobsters. Lethal effects of brine were not caused by an increase in osmotic pressure or by metal toxicity, but instead by high concentrations of potassium which affect muscular contractions, especially in cardiac muscle.

Bleached Kraft Pulp Mill Effluent

The following information on bleached kraft pulp mill effluent effects is summarized in Table 14.

Larvae/Postlarvae

Newly hatched lobster larvae were exposed to bleached kraft pulp mill effluent (BKME) in 1-wk to 3-wk bioassays at 17°C (Sprague and McLeese 1968b,c). Mortality occurred within 2 days at 32% BKME, and within 5-10 hr at 100% BKME. Larvae did not appear particularly susceptible to BKME, and concentrations below 10% should have little effect on survival. Generally, lobster larvae would avoid higher harmful concentrations because of associated low salinity.

BKME seems to contain at least two different toxic mechanisms or materials that affect aquatic animals. Simulated BKME was stored in the laboratory in clean tanks and biologically oxidized to determine whether toxicity to larvae would be reduced by either method. BKME that was stored under clean and quiescent conditions only slightly reduced toxicity over a 2-wk period. Bio-oxidation of BKME for 1 wk resulted in almost no change in toxicity to larval lobsters. Two weeks of biological treatment reduced the toxic affect of BKME by 5-6 times, but did not eliminate it (Sprague and McLeese 1968a,b).

Scarratt (1969) surveyed the larval lobster population 1 yr after opening of a bleached kraft mill near Pictou, Nova Scotia, and found no immediate or direct effect of BKME on distribution or abundance of larvae.

Juvenile/Adults

Exposure of adult lobsters to high levels of BKME gave variable results (Sprague and McLeese 1968b,c). Each of 19 bioassays was consistent within itself, but presented no overall pattern. For example, most lobsters died in 56% BKME, but more than half survived exposure at 100%. At concentrations of 32% BKME, three tests showed no mortality, and two tests produced survival longer than 6 days. In two tests at 24% BKME, only one of ten lobsters died. This generally good survival at 24 and 32% (*i.e.*, high levels) of BKME suggests that adult lobsters are relatively resistant.

In laboratory tests, McLeese (1970) found that adult lobsters did not avoid BKME at concentrations up to 20%. Introduction of BKME 50 cm upstream of resting lobsters caused no behavioral response, suggesting that exposure to dilute solutions of BKME in nature would have no immediate or direct effect on lobster movement.

Effects of BKME on ability of adult lobsters to detect and respond to odors was studied by McLeese (1973), who found that feeding response was not affected by exposure to BKME at low concentrations (0.01-2.0%) for short periods.

DRILLING MUDS

The following information on drilling mud (DM) effects is summarized in Table 15.

Larvae/Postlarvae

Larval stages one through four were exposed in a continuous-flow bioassay to five drilling fluids for 96 hr at concentrations ranging from 1 to 500 mg/l. Biochemical effects, including reduced respiration rates, reduced oxygen-to-nitrogen ratios, and increased protein-to-lipid ratios, were observed at sublethal concentrations as low as 10-50 mg/l.

Growth and development of larvae were seriously impaired by exposure to three of the five drilling fluids at concentrations of 50 and 100 mg/l. Feeding rates were significantly reduced after 24 hr at 50 mg/l. Chemical components in drilling fluids, rather than physical properties, were the suspected cause of detrimental effects (Derby and Capuzzo 1984).

Juveniles/Adults

Derby and Atema (1981) investigated effects of whole DM on normal activity of primary chemosensory neurons in walking legs of adult lobsters. Exposure of legs for 3-5 min in 10 mg/l of DM suspended in seawater altered responses to food odors in 29% of chemoreceptors examined. Similar exposure to 100 mg/l resulted in interference with 44% of the receptors inspected. In 4- to 8-day treatments, lobsters exposed to 10 and 100 mg/l of DM experienced inhibition in 25 and 37.5 % of leg chemoreceptors, respectively. It is uncertain whether the particulate or soluble fraction of the mud is more toxic. Quantity of DM coating the substrate may be the most important factor for benthic lobsters, whose leg chemoreceptors would be chronically and directly exposed.

Atema, Leavitt, *et al.* (1982) identified effects of DM on behaviors that influence field survival in postlarval, juvenile, and adult lobsters. DM interfered with normal behavior by chemical toxicity in the water column, and by physically covering the substrate. Water column toxicity was characterized by feeding and molting delays, severe delays in shelter construction, increased walking and swimming, unprovoked tail flipping, and lethargy. Physical effects of substrate covered with DM included severe delays in shelter construction and in quality of burrows constructed. Substrate covered with small amounts (*i.e.,* 1-4 mm) of DM may increase exposure of lobsters to predators and currents, and may inhibit settling and survival of postlarvae.

Barshaw and Atema (1984) observed effects of J-5 DM on juvenile lobsters. In a long-term study, lobsters were held in tanks containing either a 1-mm surface layer of DM, a similar layer of barite-bentonite (BB)(the major constituent of DM, which lacks the toxic additives but has it's physical properties), or a mud control. Lobsters in DM settled later, and after 6 mo suffered higher mortality than in the other two treatments. The physical properties of DM alone seem to hinder burrow construction in lobsters; both DM- and BB-exposed lobsters burrowed less than mud control animals.

OCEAN-DISPOSAL WASTES

Studies of effects of waste disposal in the ocean--typified by the following studies--have focused on two categories of wastes: dredge spoils and sewage sludge. American and Canadian studies of effects of dredge spoil and sewage sludge disposal on juvenile/adult lobsters have further focused on two heavily developed coastal areas: New York Bight and Halifax Harbor.

The New York Bight is polluted with raw sewage, sewage sludge, and contaminated dredge spoils. The receiving sediments are high in heavy metals, petrochemicals, and organics. Migrating lobsters collected in this area were debilitated and diseased (Pearce 1970). Laboratory studies duplicating these effects determined that disease results from fouling of lobster gills and branchial cavities, followed by development of lesions on the outer exoskeleton and thin cuticles covering the gills.

Pottle and Elner (1982) documented a preference by juvenile lobsters for gravel over silt-clay habitats, suggesting possible detrimental effects of ocean dumping on lobster "nursery" areas. Gravel substrate smothered by dumping of dredge spoils or settling of suspended material may increase competition among young lobsters for shelter in available gravel habitat. This could increase mortality from predation, and reduce growth due to crowding. Reduced prey availability resulting from destruction of habitat might increase inter- and intraspecific competition. Effects of dumping dredge spoils would be magnified if those spoils were redeposited by currents into neighboring habitats where animals have taken refuge.

Two-hundred cubic meters of noncontaminated sand-silt-clay sediment were dumped on a presurveyed juvenile lobster habitat in Halifax Harbor, Nova Scotia. Lobsters were surveyed on the dumpsite, the adjacent hard bottom, and a control site prior to dumping and over a 12-mo period after dumping. Lobster densities at the dumpsite remained low relative to those observed on adjacent hard bottom and control sites. Elimination of suitable shelter by soft sediment may have accounted for the low densities. Lobsters will shelter in soft bottom, but usually require solid objects to burrow beneath. Although suitable shelter was available on adjacent hard bottom, no lobsters were observed during the initial two surveys after the dumping. This suggests that dumping affected not only those lobsters on the actual bottom where dumping occurred, but also those on adjacent bottom. Lobsters that survived dumping may have moved away as shelter was destroyed and prey availability declined. Dumping could result in increased predation risk to lobsters, and in increased interspecific and intraspecific competition (Elner and Hamet 1984).

Analysis of hepatopancreas tissue from adult lobsters collected from the "12-Mile" Sewage Sludge Dumpsite in the New York Bight apex revealed significantly higher concentrations of PCBs in lobsters near the dumpsite than in a reference area farther away. Lowest PCB concentrations in lobsters were reported during the last sampling, November 1988, which was 11 mo after dumping ceased (Draxler *et al.* 1991).

MISCELLANEOUS

Wood

Stewart and Cornick (1964) used sawdust extracts to determine sensitivity of juvenile/adult lobsters to holding tanks constructed of various types of wood. Western red cedar was the only wood to contain a seawater extractable material that resulted in lobster mortality; death occurred within 56 hr of exposure. Redwood and Tennessee cedar extracts did not harm animals, although they were highly colored; the redwood stained

the carapace and persisted through cooking. Black spruce, Douglas fir, and balsam fir were nontoxic.

Artificial Seawater

Exposure to fluoride at concentrations five times those contained in municipal water supplies was not toxic to juvenile/adult lobsters, indicating that fluorinated freshwater may be usable in preparation of artificial seawater for holding lobsters. Lobsters also proved tolerant to tris-buffer which may be useful for regulating acidity in lobster holding tanks (Stewart and Cornick 1964).

Surfactants

Wells and Sprague (1976) exposed stage-one lobster larvae to dodecyl sodium sulphate (DSS) as a reference toxicant for crude oil using the same procedures as in an oil exposure. Larvae were sensitive to DSS, having a 2-day LC_{50} of 3.5 µg/g, and a 4-day LC_{50} of 0.72 µg/g.

Quicklime

Shumway *et al.* (1988) exposed juvenile lobsters to quicklime (calcium oxide), a strong alkali, which is used as a deterrent to starfish. Quicklime was introduced at a concentration of 3.3 kg per 1.2x0.9-m tank. Although quicklime eliminated starfish effectively, no mortality or gill damage was observed in lobsters.

Detergents

Juvenile lobsters were exposed to sodium nitrilotriacetic acid $(CH_2COONa)_3N\ H_2O$ (NTA) and two NTA-containing commercial synthetic detergents (syndets Ch and Ga) in a static toxicity test at 20°C and 20 ppt (Eisler, Zaroogian, *et al.* 1972). The LC_{50} to NTA measured 7100 µg/g at 24 hr, 3800 µg/g at 96 hr, and 3150 µg/g at 168 hr. Lobsters were highly resistant to syndet Ch and Ga. No pathologic conditions were observed in lobsters surviving exposure to 1000 µg/g of NTA for 168 hr.

Suspended Solids

Cobb (1972) studied effects of two suspended solids, quartz "flour" and kaolin, on survival of lobster larvae. Larvae appeared particularly sensitive to quartz particles of 30-55 microns maximum width. Dead larvae contained extensive particle deposits around the gills beneath the carapace. Direct blockage of water movement around gills, rather than abrasion, appeared to cause death. Larvae survived normally at high concentrations of kaolin for several days with no accumulation of material around gills. Kaolin was associated with significantly high mortality only at concentrations above 0.01 by weight; at this concentration, some cleaning mechanism that keeps the gills clear of kaolin may have become fatigued.

CONTAMINANT CONCENTRATIONS IN TISSUES OF FIELD-COLLECTED LOBSTERS

HEAVY METALS

Field surveys have documented a variety of heavy metals in lobster tissues, including arsenic, cadmium, copper, chromium, iron, lead, mercury, nickel, selenium, silver, and zinc. The hepatopancreas and egg masses usually contain the highest body burdens. Tables 16-26 list heavy metal concentrations in lobster tissues collected along the coast of the United States and Canada; when necessary, values have been converted to µg/g for consistency.

Cadmium has been the most studied of the heavy metals because of contamination of Belledune Harbor, New Brunswick, by a lead smelting plant. Discovery of high cadmium concentrations in lobsters from this area prompted numerous field surveys designed to determine the extent of contamination (Uthe and Zitko 1980). Despite significantly elevated cadmium burdens, Belledune Harbor lobsters demonstrated no deleterious effects of exposure.

ORGANICS

Polychlorinated Biphenyls

Crustaceans accumulate PCBs in their tissues by ingesting contaminated food and sediment particles, and by absorbing them through the water. These compounds are strongly lipophilic and are accumulated in fatty tissues (Metcalf and Eddy, Inc. 1981).

PCB contamination of wild lobsters has been well documented. In Massachusetts, widespread contamination of the Acushnet River estuary near New Bedford Harbor, and of the surrounding environment, has resulted in accumulation of PCBs in lobsters and in a ban on fishing for lobsters in parts of the estuary (Weaver 1984; Prince 1986). Sediment PCB concentrations exceeded 50 µg/g dry weight in some locations, and concentrations as high as 19% by weight were found in more contaminated parts of the estuary. PCB contamination in edible portions of lobsters exceeded the action level of 2 µg/g wet

weight established by the U.S. Food and Drug Administration (Prince 1986). Tissue concentrations of PCB in lobsters are listed in Table 27; values have been to converted to μg/g for consistency.

Halogenated Hydrocarbons

Many pesticides and insecticides are toxic not only to insects--the target organisms--but to lobsters as well (Cobb 1976). Organophosphate insecticides can potentially interfere with olfactory behavior of lobsters by masking odors, blocking olfactory receptors, or disrupting nerve function (McLeese 1974b). Field surveys have documented at least 20 different halogenated hydrocarbons in lobster tissues as shown in Table 28; when necessary, values have been converted to ng/g.

Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons have been measured in tissues of lobsters collected near coal-coking plants (Uthe and Musial 1986), collected in polluted harbors (Gardner and Pruell 1987), or exposed to creosote-impregnated lumber in tidal pounds (Dunn and Fee 1979; Uthe *et al.* 1984). Studies have measured over 20 individual compounds in lobster tissues as listed in Table 29; PAH values have been converted to ng/g when necessary.

SUMMARY

Extensive information is available on effects of environmental factors and contaminants on various life stages of the American lobster. Although this report cites over 200 references, there doubtlessly remain additional data that we were not able to access.

Based on this literature review, much remains to be learned concerning effects of unfavorable environmental conditions on developing lobster embryos. Although field studies have documented elevated contaminant concentrations in egg masses of adult lobsters, effects of this contamination can only be speculative. Benthic-dwelling, berried female lobsters may expose their broods to high levels of sediment contamination during the lengthy developmental period. The survival of eggs to hatching and the viability of larvae could potentially be compromised by adverse conditions, an area deserving further study.

Laboratory studies have shown that larval stages one through three are highly sensitive to environmental changes and contaminant exposure. In the water column, larvae may come in contact with low salinities, elevated temperatures, oil slicks, or other toxicants as they are carried about in surface currents. Dissolved oxygen and ammonia sensitivity were highest in the first three planktonic stages. Low concentrations of cadmium, copper, and mercury were lethal to larvae, but other heavy metals may be toxic as well. Generally, exposure to low concentrations of organic contaminants proved lethal to larvae. Insecticides, crude oil, oil-dispersing agents, and creosote were quite toxic to planktonic larvae. Sublethal effects included respiratory distress, altered coloration, behavioral changes, and reduced growth, survival, and metabolic activity. Other substances, including tributyltin oxide, potash brine, drilling mud, and surfactants, resulted in similar toxic effects. Long-term exposure to suspended solids fatigued normal gill functions. Larvae were somewhat resistant to low levels of bleached kraft pulp mill effluent and some synthetic detergents.

Metamorphosis to the postlarval stage seemed to equip lobsters better for dealing with fluctuations in environmental conditions or tolerating contaminant stress. Fourth-stage lobsters were more tolerant to salinity, DO, ammonia, and crude oil than the first three planktonic stages. Pelagic postlarvae are excellent swimmers and could potentially avoid adverse conditions when exposed in the water column. This potential avoidance capability changes after the young lobster settles to the bottom where it spends most of its first year in a burrow. This burrowing behavior could place postlarval and juvenile lobsters at risk for long-term environmental or contaminant stress.

Adult lobsters are relatively hardy and can survive a range of environmental conditions. Studies have found them more resistant to fluctuations and extremes in temperature, salinity, and DO than earlier life stages. Adults have a greater tolerance to contaminants than larval or postlarval lobsters. Exposure of lobsters to heavy metals in the laboratory produced sublethal effects including impaired chemoreception and biochemical changes. Field surveys indicated high concentrations of contaminants in lobsters without any apparent loss of well-being. PCBs, pesticides, and PAHs accumulate quickly in lobster tissues, especially in the hepatopancreas, and can be slow to depurate. Organic chemical exposure interfered with normal behavioral, chemosensory, and physiological processes. Industrial waste pollution resulted in lobster mortality by causing asphyxiation or by affecting cardiac function. Addition of drilling muds to the environment altered normal lobster behavior and impaired chemoreception, while dumping of dredged materials eliminated habitat and increased incidence of disease.

Understanding how changes in the physical and chemical environment affect the biology of the American lobster may be an important tool in managing the fisheries. This review consolidates the available information on habitat requirements, contaminant effects, and contaminant body burdens for this commercially important species. The information presented here may prove useful in identifying areas for future study.

ACKNOWLEDGMENTS

The authors thank Dr. Anthony Calabrese, Jon Gibson, Dr. Frederick Thurberg, and Edith Gould for editorial assistance; Judy Berrien for library assistance; and Mimi Johnson and David Hansen for use of their library collections.

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Table 1. Key to abbreviations, acronyms, and codes used in tables

Tissues

ANTENGL =	antennal gland
BODY =	whole body
CLWMUS =	claw muscle
CLW/TL =	claw/tail
COOKMT =	cooked meat
CRUCLAW =	crusher claw
CRUMUS =	crusher claw muscle
EDTISS =	edible tissue
EGG/OV =	eggs/ovary
EXOSKEL =	exoskeleton
FLESH =	edible flesh
GREENGL =	green gland
HEMOL =	hemolymph
HEPAT =	hepatopancreas
JUVENILE =	whole body
MGONAD =	male gonad
PINCLAW =	pincher claw
PINMUS =	pincher claw muscle
STAGE1 =	1 g pooled
STAGE4 =	1 g pooled
STOM =	stomach

Heavy Metals

AG =	silver
CD =	cadmium
CU =	copper
FE =	iron
HG =	mercury
NI =	nickel
PB =	lead
SE =	selenium
SN =	tin
ZN =	zinc

Halogenated Hydrocarbons

DDT =	1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane
DDE =	1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene
DDD =	1,1-dichloro-2,2-bis(p-chlorophenyl)ethane
PCDD =	polychlorinated dibenzo-p-dioxin
PCDF =	polychlorinated dibenzofuran
HCB =	hexachlorobenzene
A-HCH =	alpha-hexachloro-cyclohexane
G-HCH =	gamma-hexachloro-cyclohexane
A-CHLOR =	alpha-chlordane
G-CHLOR =	gamma-chlordane
tetraCDDs =	tetrachlorodibenzo-p-dioxins
tetraCDFs =	tetrachlorodibenzofurans
pentaCDDs =	pentachlorodibenzo-p-dioxins
pentaCDFs =	pentachlorodibenzofurans
hexaCDDs =	hexachlorodibenzo-p-dioxins
hexaCDFs =	hexachlorodibenzofurans
H/O CDDs/CDFs =	hepta- and octa-chlorinated dibenzo-p-dioxins and dibenzofurans
FEN =	fenitrothion
PHOS =	phosphamidon
METH =	methidathion
TPCB =	2,2',4,5' tetrachlorobiphenyl
HPCB =	2,2',4,4',5,5' hexachlorobiphenyl
END =	endrin
CHLORA =	chloramine
CHLORI =	chlorine
PERM =	permethrin
CYPER =	cypermethrin
FENVAL =	fenvalerate

Organic Metals

TBTO =	tributyltin oxide
MM =	methyl mercury
^{14}C-METHM =	^{14}C-methyl mercury

Polycyclic Aromatic Hydrocarbons

CREO =	creosote
PHEN =	phenanthrene
FLUOR =	fluoranthene
PYR =	pyrene
TRIP =	triphenylene
CHRY =	chrysene
B(a)A =	benz(a)anthracene
B(e)P =	benzo(e)pyrene
B(b)F =	benzo(b)fluoranthene
B(k)F =	benzo(k)fluoranthene
B(a)P =	benzo(a)pyrene
B(ghi)P =	benzo(ghi)perylene
DB(ah)A =	dibenz(ah)anthracene
INDENO =	indeno(1,2,3,-cd)-pyrene
BEN =	benzene
NAP =	naphthalene
ANTH =	anthracene
FLUORE =	fluorene
BFA =	benzofluoranthenes
PERY =	perylene
COR =	coronene

Industrial Wastes

DM =	drilling mud
BB =	barite bentonite
BKME =	bleached pulp kraft mill effluent

Other Organic Chemicals

GLAA =	glacial acetic acid
FORM =	formalin
PYR =	pyridine

Codes

ND =	not detectable
NG =	not given
T =	trace

Table 2. Sublethal effects of cadmium on juvenile and adult American lobsters in laboratory exposures

Dose (µg/l)	Time (days)	Medium	Tissue	Concentration (µg/g)	Effects/Results	Reference
10	21	seawater	BODY	0.72	concentrations 25-78% greater than controls (concentrations expressed in wet weight)	Eisler, Zaroogian, et al. 1972
			MUSCLE	0.25		
			EXOSKEL	0.88		
			GILL	0.87		
3	30	seawater	GILL	1.8	elevated O_2 consumption	Thurberg et al. 1977
6				3.4		
control				1.5		
3	60	seawater	GILL	2.32		
6				2.63		
control				1.63		
			HEART		reduced ligand sensitivity	
			ANTENGL			
6	30	seawater	GILL		25% increase in gill homogenate ouabain-insensitive ATPase activity	Tucker 1979
6	30	seawater	HEART		elevated enzyme activity	Gould 1980
			ANTENGL			
			MUSCLE			
100	14	food	HEPAT	41.0	no cadmium loss after 40 days depuration	Brouwer et al. 1986
10	60	seawater	HEPAT	10.0		
control			HEPAT	7.5		

Table 3. Sublethal effects of cadmium in field exposures of adult American lobsters from Belledune Harbor, New Brunswick

Tissue	Effects/Results	Reference
HEPAT GILL MUSCLE	elevated tissue levels; did not affect lobster metabolic energy state: adenylate energy charge, adenine nucleotide pools, or ATPase activity	Haya et al. 1980
HEPAT GILL GREENGL	no histological changes in these tissues regardless of hepatopancreas burden	Odense and Annand 1980
HEPAT	no reduction in hepatopancreas cadmium levels could be induced by dietary administration of cadmium-binding agents or by injection of divalent ions and chelators at high levels	Uthe 1980
HEPAT GREENGL GILL CLAW	removal from the harbor to uncontaminated seawater for 8 mo resulted in no significant decrease in cadmium levels	McLeese et al. 1981

Table 4. Lethal effects of various heavy metals on several life stages of American lobsters in the laboratory

Metal	Dose (µg/l)	Mortality (%)	Time (hr)	Life Stage	Comments	Reference
CU		100	18	adult	tank lined with metal sheets	Wilder 1952
PB		100	474	adult		
ZN		100	210	adult		
CU	1000	50	22-33	adult	30‰ salinity, 13°C	McLeese 1974a
	560	50	36-52	adult		
	100	50	72-140	adult		
	1000	50	52-58	adult	30‰ salinity, 5°C	
	560	50	70-72	adult		
	100	50	172-270	adult		
CD	10	3	96	stage 1	20°C	Johnson and Gentile 1979
	33	10	96	stage 1		
	78	50	96	stage 1		
	1000	50	24	stage 1		
	1000	100	48	stage 1		
CU	48	50	96	stage 1		
	330	100	24	stage 1		
HG	20	50	96	stage 1		
	33	30	24	stage 1		
	33	73	96	stage 1		
	100	97	24	stage 1		
	330	100	24	stage 1		
CD	30	NG	NG	stage 1		McLeese 1981
	5000	NG	NG	adult		

Table 5. Sublethal effects of various heavy metals in seawater on juvenile and adult American lobsters in laboratory exposures

Metal	Dose (µg/g)	Time (days)	Tissue	Concentration (µg/g)	Effects/Results	Reference
ZN	500				sluggish and immobile behavior; no avoidance response	McLeese 1970
CU	0.225-4.5	2.5 min			chemosensory response to herring reduced by simultaneous addition of copper at 4-80 times the lethal threshold	McLeese 1975
CU	0.04-0.1	2			copper at 0.7-1.8 times the lethal threshold causes a gradual decline in chemosensory response	

Table 5. Cont.

Metal	Dose (µg/g)	Time (days)	Tissue	Concentration (µg/g)	Effects/Results	Reference
HG	0.006	30	TAIL	1.0	concentrations expressed in wet weight	Thurberg et al. 1977
			HEPAT	15.0		
			GILL	85.0		
		60	GILL	119.0		
AG	0.006	30	HEART		depressed transaminase	Calabrese et al. 1977
			ANTENGL		loss of ligand sensitivity	
			GONAD		enzyme induction	
CU	NG	90	FLESH	155.0	cultured in secondary sewage from plant in Wareham, MA, which contained undetermined metal concentrations (tissue concentrations expressed in dry weight)	Furr et al. 1981
control				136.0		
FE		90	FLESH	202.0		
control				50.0		
PB		90	FLESH	1.5		
control				0.2		
NI		90	FLESH	5.7		
control				1.4		
SE		90	FLESH	3.1		
control				2.2		
CD		90	FLESH	0.5		
control				0.09		
ZN		90	FLESH	207.0		
control				135.0		
PB	0.05	30	ANTENGL	138.0	decreased pentose shunt activity; loss of adjustments to low salinity levels returned to normal after 2 days in clean seawater (concentrations expressed in wet weight)	Gould and Greig 1983
control			ANTENGL	5.6		
ZN	60	4	GILL	2570	inital dose, decreased to 25 µg/g by 12 hr; Na-, K-ATPase and residual ATPase activity were reduced;	Haya et al. 1983
		2	GILL		attempted to increase irrigation by swimmerett and walking leg movements (concentrations expressed in dry weight)	
ZN	25	4	GILL	2570	zinc remained elevated after 7-day depuration period (concentrations expressed in dry weight)	Waiwood et al. 1987
			GREENGL	1032		
			HEMOL	753		
			HEPAT	734		
			CRUMUS	465		
			PINMUS	169		
			TAIL	126		

Table 6. Sublethal effects of ^{14}C-polychlorinated biphenyls pericardially injected into adult American lobsters in the laboratory

Tissue	Effects/Results	Reference
HEPAT	highest specific activity of all tissues	Bend et al. 1972
HEPAT	no decrease in radioactivity within 1 wk	
STOM		
GILL		
EGG		
GREENGL	significant decrease in PCB after 1 wk through redistribution or excretion	
HEART		
TAIL		
CLWMUS		
HEPAT	contained 90% of recovered PCB; unaltered half-life of 45 days	Bend et al. 1973, 1974, 1976
EGG	radioactivity persisted for 39 days	
STOM	half-life of 28 days	
GILL	half-life of 24 days	
HEMOL	radioactivity was lost quickly--5 days	
GREENGL	half-life of 4.5 days	
HEART	half-life of 3.5 days	
HEPAT	contained 90% of PCB; half-life of 40 days	James et al. 1977
MGONAD	half-life of 7 days	

Table 7. Sublethal effects of various halogenated hydrocarbons on American lobsters in laboratory exposures

Contaminant	Dose (µg/l)	Time (days)	Tissue	Concentration (µg/g wet weight)	Effects/Results	Reference
^{14}C-DDT	100	30	EGG		retained more than 1% of the intravascular dose	Guarino et al. 1972, 1974
			HEPAT			
			MUSCLE			
			HEMOL		removal of pesticide rapid	
		7	HEPAT		contained 90% of the dose delivered intravascularly, by seawater or food	
FEN	0.05-25.4				did not affect olfactory response to simultaneously presented cod muscle	McLeese 1974b
	0.015				lethal threshold for larvae	
	0.01				larvae turned red--chromatophore expansion	
	0.1-1.0				larvae sank to the bottom	
	<0.3				lethal threshold for adults	
TPCB	600	42	HEPAT		accumulated more chlorobiphenyls through diet than tail or muscle; concentrations decreased during clearance	McLeese, Metcalfe, and Pezzack 1980
	4000					
HPCB	550					
	4900					
END		14	HEPAT	1.95		
			TAIL	0.0047	decreased by 35%	

Table 8. Effects of halogenated hydrocarbons on several life stages of American lobsters in laboratory exposures

Contaminant	Dose (µg/l)	Mortality (%)	Time (hr)	Life Stage	Comments	Reference
FEN	1.0	50	96	larva/adult	15°C	McLeese 1974b
	10.0	50	24	adult		
	100.0	50	48	adult		
PHOS	180.0	50	96	adult	4°C	McLeese 1974c
	50.0	50	96	adult	12°C	
PHOS	45-1820		2.5 min	adult	2.5-100 times the lethal threshold; simultaneous exposure with herring muscle demonstrated no reduction in chemoreception	McLeese 1975
PHOS	12.7-32.5		48	adult	no measurable effect	
CHLORI	16300	50	96	stage I	25°C	Capuzzo et al. 1976
	2500	50	96	stage I	30°C	
CHLORA	4080	50	96	stage I	20°C	
	2020	50	96	stage I	25°C	
	560	50	96	stage I	30°C	
CHLORI	5000			stage I	initial respiratory distress	
CHLORA	50			stage I	begins	
CHLORI	1000		1	stage I	25°C; reduced respiration,	Capuzzo 1977
CHLORA	1000		1	stage I	growth, and metabolic activity	
PHOS	107	50	48	adult	20°C; mixture of both results in greater than additive toxicity	McLeese and Metcalfe 1979a
METH	14	50	48	adult		
PHOS	18	50	48	adult	20°C; lethal threshold	
METH	10	50	48	adult	lethal threshold	
PERM	0.73	50	96	adult	10°C; 96-hr LC_{50}	McLeese, Metcalfe, and Zitko 1980
CYPER	0.04	50	96	adult		
FENVAL	0.14	50	96	adult		
PERM	0.68	50	96	adult	10°C; lethal threshold	
CYPER	0.04	50	96	adult	lethal threshold	
FENVAL	0.08	50	96	adult	lethal threshold	

Table 9. Sublethal effects of oil and oil-dispersing agents in seawater on juvenile and adult American lobsters in laboratory and field exposures

Contaminant	Dose	Unit	Time (days)	Effects/Results/Comments	Reference
Bunker C	>10,000	µg/g	4	lobsters were highly resistant	Sprague and Carson 1970
Corexit	>10,000		4	in all tests	
Dispersol	>1,000		4		

Table 9. Cont.

Contaminant	Dose	Unit	Time (days)	Effects/Results/Comments	Reference
oil/Corexit	>10,000		4	mixture of oil and dispersant; dose of each component	
oil	10	µg/g		altered water-chemistry-sensing movements; period between noticing and approaching food doubled	Atema and Stein 1972, 1974; Blumer et al. 1973
H_2O-soluble fraction	0.05	µg/g		no effect on behavior or feeding	
whole kerosene and branched-chain fraction	20	µl	30 min	initiated searching and feeding behavior; ingested contaminant-soaked asbestos strips; polar aromatic fraction present in whole kerosene made it less attractive than the branched-chain fraction by itself	Atema et al. 1973; Atema 1977
polar aromatic	20	µl	30 min	at a distance, induced searching behavior; repulsed lobsters at close range	
straight chain				no influence on feeding	
branched chain				stimulated feeding	
whole kerosene		µl range		soaked into bricks; some attraction and feeding, afterwards food ingestion ceased for 3-7 days	Atema 1976, 1977
branched-chain polar aromatic				ambivalent response: simultaneous feeding by maxillipeds and rejection by periopods; increased activity; spastic behavior accelerated food ingestion	
straight chain				depressed activity; prolonged feeding attempts afterwards; food ingestion ceased for 3-4 days	
la Rosa	10	µg/g		movement of chemosensory-related appendages changed; decreased chemical stimulation and increased tactile behavior; twice as much time required to locate food	Atema 1976, 1977
soluble fraction	0.01	µg/g		no measurable effect	
#2 fuel	0.8-0.15	µg/g	5	minor effects; recovery immediate after removal	Atema 1977; Atema et al. 1979; Atema, Karnofsky, et al. 1982
	0.3		5	sublethal interference with feeding, continued 1 day beyond dosing	
	1.5		1.4	feeding ceased for up to 6 days	
	1-2		5	severe neuromuscular abnormalities: cramped postures, spastic behavior, unresponsiveness, and death	

Table 9. Cont.

Contaminant	Dose	Unit	Time (days)	Effects/Results/Comments	Reference
	3		5	modified response of chemoreceptors to food	
Iranian spill			1	lobsters in top layers of floating crates dead or dying from osmoregulatory imbalance; removal to clean water reversed symptoms	Aiken and Zitko 1977
Iranian	.04	µl		simulated spill: light slick in turbulent conditions; healthy animals showed no osmoregulatory or histological damage	
Iranian	0.28	µl		heavy slick in calm conditions; healthy animals showed no osmoregulatory or histological damage	
Venezuelan	0.3-0.5	µg/g	98	elevated serum calcium; decreased serum freezing point; altered plasma protein, lipid, and amino acids; lipid values in blood hemocyctes decreased by half; gill browning	Payne et al. 1983

Table 10. Effects of crude oil and oil-dispersing agents on larval American lobsters in laboratory exposures

Contaminant	Dose (µg/g)	Mortality (%)	Time (hr)	Larval Stage	Comments	Reference
Aquaclene-100	54		24	4	20°C; most sensitive to oil-dispersing agents after molt	Engel and Neat 1971
	46		48-72	4		
Colloid-88 dispersants	50		24	4	toxicity of dispersents due to volatile solvent fraction	
	46		48-98	4		
South Louisiana oil	0.1			1	active feeding; aggressive; molted to stage 4 within 12 days of hatching--same as controls	Forns 1977
	1.0			1	lethargic; limited mobility; depressed feeding; 15 days to stage 4; survival 50% below 0.1-µg/g treatment and controls; altered coloration	
Venezuelan oil	100	100	24	1-4	20-21°C	Wells 1972
	10				survived poorly; unable to metamorphose; color and behavior changes; stages 1-3 more sensitive than stage 4	
	0.01-1.0				survived to metamorphosis; similar to controls	

Table 10. Cont.

Contaminant	Dose (µg/g)	Mortality (%)	Time (hr)	Larval Stage	Comments	Reference
	2-30	50	96			
	6			3	stunted at this stage; even after 30 days, some intermediate larvae	
Venezuelan Tia Juana oil	0.86	50	96	1	20°C	Wells and Sprague 1976
	4.9	50	96	3-4		
	0.14	50	720	1	slowed larval development	
	0.19			1	decreased food consumption	
				4	11.8% of oil-exposed larvae were intermediate between stages 3 and 4	
				4	did not avoid oiled substrate; burrowed more than controls	
	1740			4	no effect on growth or survival	
DSS	3.5	50	48	1	reference toxicant	
Surfactant	0.72	50	96	1		
South Louisiana oil	0.25	24		1	reduced respiration rate	Capuzzo and Lancaster 1981, 1982; Capuzzo et al. 1984
	0.25	96		1-4	reduced respiration and O:N ratio; higher protein and lower lipid contents; molt delays; disrupted energetics; 20°C	

Table 11. Sublethal effects of polycyclic aromatic hydrocarbons, organic metals, and other organic chemicals in seawater on juvenile and adult American lobsters in laboratory exposures

Contaminant	Dose (µg/g)	Time (days)	Tissue	Effects/Results	Reference
CREO	0.02	4		lethal threshold for larvae at 20°C	McLeese and Metcalfe 1979a
	1.76	4		lethal threshold for adults at 10°C	
			HEPAT	increased with increasing dose	
TBTO	0.005-0.015	6		100% mortality	Laughlin and French 1980
	0.02	1		100% mortality	
^{14}C-METHM	NG	1	HEART HEPAT GONAD INTEST BRAIN GILL	delivered by intravascular injection, through food, or in seawater, levels high in these tissues	Guarino et al. 1972
			HEPAT	by injection, half-life of 14 days	
		6	HEPAT	by food; contained 68% of METHM	
		6	HEPAT	by seawater; contained 23% of METHM	
		33	TAIL	redistributed into tail, doubled post-injection	
		6		by food; contained 5% of METHM	
		6		by seawater; contained 60% of METHM	

Table 11. Cont.

Contaminant	Dose (µg/g)	Time (days)	Tissue	Effects/Results	Reference
GLAA	1000			No avoidance response	McLeese 1970
FORM	1000			No avoidance response, but animals sluggish and immobile	
PYR	1000				

Table 12. Effects of yellow phosphorus on juvenile and adult American lobsters in laboratory exposures

Dose (µg/l)	Mortality (%)	Time (hr)	Effects/Results	Reference
120	50	149	loss of muscle tone; lethargy; jerky, uncoordinated pereiopod movement; loss of neuromuscular response; toxic effects irreversible; mortality caused by asphyxiation and hemolymph gelling	Zitko et al. 1970
80	50	205		
60	50	215		
50	50	239		
40	50	240		
			degenerative changes in all four cell types of the hepatopancreas; mortality caused by hemolymph clotting and asphyxiation	Aiken and Byard 1972

Table 13. Effects of potash brine on larval, postlarval, and juvenile American lobsters in laboratory exposures

Dose (g/l)	Mortality (%)	Time (hr)	Life Stage	Effects/Results	Reference
1-2	50	96	1-3	20°C; larvae changed color from blue-green to red; avoidance was followed by a quiescent period with larvae lying sideways rapidly moving their appendages	Charmantier et al. 1985
2.25-3.0	50	96	4	20°C	
1.5-2.25	50	96	4	11°C	
<1.5				required to complete larval stages 1-4	
2.25-3.0	50	96	juvenile	20°C; postlarval and juvenile lobsters did not experience a color change when exposed to the brine	

Table 14. Effects of bleached kraft pulp mill effluent on several life stages of American lobsters in laboratory exposures

Dose (%)	Mortality (%)	Time (hr)	Life Stage	Comments	Reference
32	100	48	stage 1		Sprague and McLeese 1968a,b
100	100	5-10	stage 1		
24	20	NG	adult	each bioassay was consistent within itself, but showed no overall pattern; results show that adult lobsters are relatively resistent to BKME	
32	100	150	adult		
32	60	250	adult		
56	100	35	adult		
56	80	77	adult		
75	100	75	adult		
100	40	NG	adult		
20			adult	no avoidance or behavioral response	McLeese 1970
0.01-2.0			adult	no effect on feeding	McLeese 1973

Table 15. Effects of drilling muds on several life stages of American lobsters in laboratory exposures

Dose (mg/l)	Mortality (%)	Time	Life Stage	Comments	Reference
10-50			stage 1	reduced respiration rates; reduced oxygen: nitrogen ratios; increased protein:lipid ratios	Derby and Capuzzo 1984
50		1 day	stage 1		
50	25	4 days	stage 1	impaired growth and development	
100	20	4 days	stage 1		
250	35	4 days	stage 1	all exposures used J-M drilling muds	
500	40	4 days	stage 1		
10		3-5 min	adult	altered responses to food odors in 29% of the chemoreceptors examined; used whole DM	Derby and Atema 1981
100		3-5 min	adult	interfered with 44% of receptors	
10		4-8 days	adult	interfered with 25% of leg receptors	
100		4-8 days	adult	interfered with 37.5% of leg receptors	
7		36 days	stage 4	delays in molting, feeding, and shelter construction; increased walking and swimming; tail flipping; lethargy	Atema, Leavitt, et al. 1982
			stage 4 and 5	lobsters in substrate covered with a 4-mm layer of drilling mud showed delays in shelter construction and a decrease in shelter quality	
			juvenile	lobsters on substrate coated with a 1-mm surface layer of J-5 DM or BB burrowed less than in a mud control; lobsters in DM settled later and suffered higher mortality than the BB or mud control	Barshaw and Atema 1984

Table 16. Arsenic concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
NG	NG		3-26	wet	Chapman 1926 and Cheftel 1959 (cited in Lunde 1977)
COOKMT	Belledune Harbor - West	3.92		wet	Uthe and Chou 1985
COOKMT	Heron Island, NB	2.30		wet	
HEPAT	Long Island Sound		0.00-0.22	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound		1.8-15.0	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 17. Cadmium concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
HEPAT	North Lake, PE	12.90	5.48-38.9	wet	Chou and Uthe 1978
HEPAT	Shediac, NB	12.70	4.37-47.4	wet	
HEPAT	Halifax, NS	8.59	4.64-15.1	wet	
HEPAT	Victoria Beach, NS	7.02	3.04-16.2	wet	
HEPAT	Petit Roucher, NB	3.67	1.69-9.08	wet	
HEPAT	Belledune Harbor, NB	483.20	106.3-1103	dry	Haya et al. 1980
HEPAT	Belledune Harbor, NB	27.17	7.53-65.46	dry	
HEPAT	Stonehaven, NB	30.88	9.48-70.11	dry	
HEPAT	Belledune Harbor - West	203.70		wet	Uthe et al. 1980
HEPAT	Belledune Harbor - East	88.80		wet	
HEPAT	Belledune Harbor - 0.8 km outside	80.30		wet	
HEPAT	Belledune Harbor - outside, opposite dock	32.80		wet	
HEPAT	Belledune Harbor - outside, breakwater	30.40		wet	
HEPAT	Belledune Harbor - 1.6 km east of harbor	34.90		wet	
HEPAT	Heron Island, NB	3.85		wet	
HEPAT	Comfort Cove, NF	2.82	1.72-6.43	wet	Uthe and Freeman 1980
HEPAT	Beach Point, PE	17.22	6.07-52	wet	
HEPAT	Tignish, PE	14.48		wet	
HEPAT	French River, PE	11.88		wet	
HEPAT	North Lake, PE	14.65		wet	
HEPAT	Meat Cove, NS	6.32		wet	
HEPAT	Belledune Harbor, NB	519.00		dry	McLeese et al. 1981
GREENGL	Belledune Harbor, NB	218.00		dry	
GILL	Belledune Harbor, NB	51.00		dry	
PINCLAW	Belledune Harbor, NB	23.00		dry	

Table 17. Cont.

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
CRUCLAW	Belledune Harbor, NB	15.00		dry	
EGG/OV	Belledune Harbor, NB	0.85		dry	
HEPAT	Belledune Harbor, NB	400.00	27-1600	dry	Ray et al. 1981
HEPAT	Stonehaven, NB	31.00	9.6-70	dry	
HEPAT	Maine	22.00	3.8-68	dry	
EGG/OV	Belledune Harbor, NB	5.90		dry	
EGG/OV	Stonehaven, NB	0.20		dry	
MUSCLE	New York Bight		<0.05-0.15	wet	Reid et al. 1982
MUSCLE	Long Island Sound		0.08-0.12	wet	
CLW/TL	Boston Harbor, MA		<0.005-0.044	wet	Metcalf & Eddy 1984
HEPAT	Belledune Harbor - West	174.00	47.6-372	wet	Uthe and Chou 1985; Uthe et al. 1986
HEPAT	Belledune Harbor - East	62.20	13.7-263	wet	
HEPAT	Heron Island, NB	3.85	2.19-8.26	wet	
COOKMT	Belledune Harbor - West	0.89	0.42-3.06	wet	
COOKMT	Belledune Harbor - East	0.35	0.07-1.28	wet	
COOKMT	Heron Island, NB	0.04	0.03-0.09	wet	
CLW/TL	Long Island Sound	0.05	<0.04-0.07	NG	Chytalo 1986
HEPAT	Long Island Sound - Eastern Basin	7.08	3.33-11.7	NG	
HEPAT	Long Island Sound - Central Basin	6.96	2.64-13.9	NG	
HEPAT	Long Island Sound - Western Basin	5.20	2.25-7.84	NG	
HEPAT	Long Island Sound - Narrows	5.12	1.46-18.8	NG	
CLAW	Lydonia Canyon		0.01-0.18	wet	Cooper et al. 1987; Pecci 1987
TAIL	Lydonia Canyon		0.04-0.16	wet	
HEPAT	Lydonia Canyon	19.50		wet	
EGG	Lydonia Canyon		0.13-0.20	wet	
CLW/TL	Long Island Sound	0.05	0.04-0.06	wet	Connecticut Department of Environmental Protection 1987
HEPAT	Long Island Sound	8.8	3.0-18.0	wet	
HEPAT	Boston Harbor, MA		1.21-6.63	dry	Gardner and Pruell 1987
TAIL	Boston Harbor, MA		0.003-0.029	dry	
EGG	Long Island Sound		0.12-0.36	dry	Greig 1988[a]
STAGE1	Long Island Sound		0.08-0.79	dry	
JUVENILE	Long Island Sound		0.4-0.63		
CLW/TL	Boston Harbor - Deer Island, MA	0.0043	0.0014-0.0108	wet	Wallace et al. 1988
CLW/TL	Willows Pier, MA	0.0040	0.0004-0.0126	wet	
CLW/TL	Salem Harbor, MA	0.0052	0.0005-0.0190	wet	
EGG	Long Island Sound		0.23-0.49	dry	Greig 1989[a]
STAGE1	Long Island Sound		0.29-0.89	dry	
HEPAT	Long Island Sound		1.8-17.0	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound		2.5-11.0	wet	
HEPAT	Long Island Sound - New Haven, CT		8.8-10.7	wet	Greig and Pereira 1993
HEPAT	Long Island Sound - New London, CT		3.1-3.5	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 18. Copper concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
HEPAT	Halifax, NS	120.0		wet	Chou and Uthe 1978
HEPAT	Shediac, NB	41.3		wet	
HEPAT	Petit Roucher, NB	24.4		wet	
HEPAT	Victoria Beach, NS	19.1		wet	
HEPAT	North Lake, PE	12.9		wet	
MUSCLE	New York Bight		2.27-9.46	wet	Reid et al. 1982
MUSCLE	Long Island Sound		7.47-15.48	wet	
CLW/TL	Boston Harbor, MA		3.6-18.7	wet	Metcalf & Eddy 1984
COOKMT	Belledune Harbor, NB	22.77		wet	Uthe and Chou 1985
COOKMT	Heron Island, NB	19.6		wet	
CLAW	Lydonia Canyon		5.02-22.82	wet	Cooper et al. 1987; Pecci 1987
TAIL	Lydonia Canyon		7.88-13.68	wet	
HEPAT	Lydonia Canyon	18.3		wet	
EGG	Lydonia Canyon		43.9-62.0	wet	
HEPAT	Long Island Sound	393.0	39-630	wet	Connecticut Department of Environmental Protection 1987
CLW/TL	Long Island Sound	2.32	0.22-4.5	wet	
TAIL	Boston Harbor, MA		10.1-24.6	dry	Gardner and Pruell 1987
HEPAT	Boston Harbor, MA		37.1-644	dry	
EGG[a]	Long Island Sound - Bridgeport, CT	220	164-257	dry	Mercaldo-Allen et al. 1994
STAGE1[a]	Long Island Sound - Bridgeport, CT	148	93.5-201	dry	
JUVENILE[a]	Long Island Sound - Bridgeport, CT	24.9	22.2-27.1	dry	
EGG[a]	Long Island Sound - Milford, CT	227	153-353	dry	
STAGE1[a]	Long Island Sound - Milford, CT	180	120-244	dry	
JUVENILE[a]	Long Island Sound - Milford, CT	27.9	24.3-30.7	dry	
EGG[a]	Long Island Sound - New Haven, CT	200	151-239	dry	
STAGE1[a]	Long Island Sound - New Haven, CT	160	120-204	dry	
JUVENILE[a]	Long Island Sound - New Haven, CT	25.8	25.2-26.6	dry	
EGG[b]	Long Island Sound - Milford, CT	203	139-384	dry	
STAGE1[b]	Long Island Sound - Milford, CT	132	89.3-154	dry	
EGG[b]	Long Island Sound - New Haven, CT	172	132-229	dry	
STAGE1[b]	Long Island Sound - New Haven, CT	110	90.5-130	dry	
EGG[b]	Long Island Sound - Rye, NY	175	105-309	dry	
STAGE1[b]	Long Island Sound - Rye, NY	127	107-147	dry	
CLW/TL	Boston Harbor - Deer Island, MA	14.4		wet	Wallace et al. 1988
CLW/TL	Willows Pier, MA	8.2		wet	
CLW/TL	Salem Harbor, MA	9.9		wet	
HEPAT	Long Island Sound		61-1000	wet	Olsen 1989[c], 1990[c]
HEPAT			60-1300	wet	
HEPAT	Long Island Sound - New Haven, CT		363.5-1489.9	wet	Greig and Pereira 1993
HEPAT	Long Island Sound - New London, CT		150.1-558.4	wet	

[a] 1988 data.
[b] 1989 data.
[c] Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 19. Chromium concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
MUSCLE	New York Bight		<0.10-0.52	wet	Reid et al. 1982
MUSCLE	Long Island Sound		0.26-0.50	wet	
CLAW	Lydonia Canyon	0.05	ND-0.07	wet	Cooper et al. 1987; Pecci 1987
TAIL	Lydonia Canyon		ND-0.17	wet	
HEPAT	Lydonia Canyon	0.07		wet	
EGG	Lydonia Canyon		0.24-0.33	wet	
CLW/TL	Long Island Sound	0.05	0.00-0.10	wet	Connecticut Department of
HEPAT	Long Island Sound	0.11	0.00-0.30	wet	Environmental Protection 1987
HEPAT	Boston Harbor, MA		<0.18-7.85	dry	Gardner and Pruell 1987
TAIL	Boston Harbor, MA		<0.02-1.13	dry	
EGG	Long Island Sound		0.49-2.5	dry	Greig 1988[a]
STAGE1	Long Island Sound		0.45-13.6	dry	
JUVENILE	Long Island Sound		0.4-2.0	dry	
CLW/TL	Boston Harbor - Deer Island, MA	0.048		wet	Wallace et al. 1988
CLW/TL	Salem Harbor, MA	<0.048		wet	
CLW/TL	Willows Pier, MA	<0.054		wet	
EGG	Long Island Sound		0.53-5.0	dry	Greig 1989[a]
STAGE1	Long Island Sound		0.62-14.7	dry	
HEPAT	Long Island Sound		0.00-0.25	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound	0.5		wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 20. Iron concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
GILL		21.1		wet	Martin and Odense 1974
HEPAT	Long Island Sound		11-29	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound		15-62	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 21. Lead concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
MUSCLE	New York Bight		<0.4-0.6	wet	Reid et al. 1982
MUSCLE	Long Island Sound		<0.5-0.3	wet	
CLW/TL	Boston Harbor, MA		0.02-0.08	wet	Metcalf and Eddy 1984
COOKMT	Belledune Harbor - West	1.0		wet	Uthe and Chou 1985
COOKMT	Belledune Harbor - East	0.98		wet	
COOKMT	Heron Island, NB	0.051		wet	
CLW/TL	Long Island Sound		<0.05-0.12	wet	Chytalo 1986
HEPAT	Long Island Sound - Eastern Basin	0.09		wet	
HEPAT	Long Island Sound - Western Basin	0.12		wet	
HEPAT	Long Island Sound - Narrows	0.15		wet	
HEPAT	Long Island Sound - Central Basin	0.66		wet	
CLAW	Lydonia Canyon		0.40-0.15	wet	Cooper et al. 1987; Pecci 1987
TAIL	Lydonia Canyon		0.07-0.69	wet	
HEPAT	Lydonia Canyon	0.62		wet	
EGG	Lydonia Canyon		ND-0.07	wet	
HEPAT	Long Island Sound	0.43	0.00-1.30	wet	Connecticut Department of Environmental
CLW/TL	Long Island Sound	0.39	0.25-0.64	wet	Protection 1987
HEPAT	Boston Harbor, MA		<0.24-<0.41	dry	Gardner and Pruell 1987
TAIL	Boston Harbor, MA		0.51-0.85	dry	
EGG	Long Island Sound		0.64-18.8	dry	Greig 1988[a]
STAGE1	Long Island Sound		0.06-14.5	dry	
JUVENILE	Long Island Sound		0.4-2.0	dry	
CLW/TL	Boston Harbor - Deer Island, MA	<0.017		wet	Wallace et al. 1988
CLW/TL	Salem Harbor, MA	<0.024		wet	
CLW/TL	Willows Pier, MA	<0.064		wet	
EGG	Long Island Sound		1.1-4.5	dry	Greig 1989[a]
STAGE1	Long Island Sound		2.0-5.5	dry	
HEPAT	Long Island Sound		0.40-0.88	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound		1.0-5.0	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 22. Mercury concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
TAIL	Cape May, NJ	0.31		wet	Greig et al. 1975
HEPAT	Cape May, NJ	0.60		wet	
MUSCLE	New York Bight		0.04-0.15	wet	Reid et al. 1982
MUSCLE	Long Island Sound		0.06-0.07	wet	
CLW/TL	Boston Harbor, MA		0.002-0.130	wet	Metcalf and Eddy 1984
CLW/TL	Long Island Sound		0.07-0.11	wet	Chytalo 1986
HEPAT	Long Island Sound		0.06-0.09	wet	
CLAW	Lydonia Canyon	0.218	0.15-0.60	wet	Cooper et al. 1987; Pecci 1987
TAIL	Lydonia Canyon		0.27-0.97	wet	
HEPAT	Lydonia Canyon	0.087		wet	
EGG	Lydonia Canyon		0.035-0.088	wet	
CLW/TL	Long Island Sound	0.09	0.05-0.15	wet	Connecticut Department of Environmental Protection 1987
HEPAT	Long Island Sound	0.07	0.06-0.08	wet	
HEPAT	Boston Harbor, MA		0.051-0.123	dry	Gardner and Pruell 1987
TAIL	Boston Harbor, MA		<0.088-0.746	dry	
CLW/TL	Boston Harbor - Deer Island, MA	0.141		wet	Wallace et al. 1988
CLW/TL	Salem Harbor, MA	0.148		wet	
CLW/TL	Willows Pier, MA	0.176		wet	
HEPAT	Long Island Sound		0.00-0.06	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound		0.02-0.35	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 23. Nickel concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
MUSCLE	New York Bight		0.08-0.46	wet	Reid et al. 1982
MUSCLE	Long Island Sound		0.25-0.27	wet	
HEPAT	Long Island Sound	0.87	0.40-1.8	wet	Connecticut Department of Environmental Protection 1987
CLW/TL	Long Island Sound	0.36	0.14-1.10	wet	
CLW/TL	Boston Harbor - Deer Island, MA	<0.030		wet	Wallace et al. 1988
CLW/TL	Salem Harbor, MA	<0.032		wet	
CLW/TL	Willows Pier, MA	<0.030		wet	
HEPAT	Long Island Sound		0.30-1.20	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound		0.0-2.0	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 24. Selenium concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
HEPAT	Halifax, NS	1.07		wet	Chou and Uthe 1978
HEPAT	Shediac, NB	1.33		wet	
HEPAT	Petit Roucher, NB	0.955		wet	
HEPAT	Victoria Beach, NS	1.22		wet	
HEPAT	North Lake, PE	1.19		wet	
HEPAT	Long Island Sound		0.00-1.10	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound		0.3-5.0	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 25. Silver concentrations in tissues of field-collected adult American lobsters

Tissue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
HEPAT	Halifax, NS	2.22		wet	Chou and Uthe 1978
HEPAT	Shediac, NB	1.45		wet	
HEPAT	North Lake, PE	0.98		wet	
HEPAT	Petit Roucher, NB	0.688		wet	
HEPAT	Victoria Beach, NS	0.438		wet	
MUSCLE	New York Bight		0.10-0.73	wet	Reid et al. 1982
MUSCLE	Long Island Sound		0.50-0.61	wet	
CLW/TL	Boston Harbor, MA		0.14-0.47	wet	Metcalf & Eddy 1984
COOKMT	Belledune Harbor, NB	0.493		wet	Chou and Uthe 1985
COOKMT	Heron Island, NB	0.407		wet	
HEPAT	Long Island Sound - New Haven, CT		4.8-23.4	wet	Greig and Pereira 1993
HEPAT	Long Island Sound - New London, CT		2.3-8.8	wet	

able 26. Zinc concentrations in tissues of field-collected adult American lobsters

issue	Location	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
EPAT	North Lake, PE	33.6		wet	Chou and Uthe 1978
EPAT	Petit Roucher, NB	30.6		wet	
EPAT	Victoria Beach, NS	28.4		wet	
EPAT	Halifax, NS	27.7		wet	
EPAT	Shediac, NB	22.5		wet	
IUSCLE	Long Island Sound		14.44-19.25	wet	Reid et al. 1982
IUSCLE	New York Bight		5.75-18.03	wet	
:LAW	Lydonia Canyon	40.6	18.7-37.0	wet	Cooper et al. 1987; Pecci 1987
TAIL	Lydonia Canyon		25.9-33.3	wet	
IEPAT	Lydonia Canyon	37.3		wet	
:GG	Lydonia Canyon		39.8-41.5	wet	
CLW/TL	Long Island Sound	20.4	5.4-34	wet	Connecticut Department of
HEPAT	Long Island Sound	62.0	28-130	wet	Environmental Protection 1987
CLW/TL	Boston Harbor - Deer Island, MA	17.6		wet	Wallace et al. 1988
CLW/TL	Salem Harbor, MA	21.3		wet	
CLW/TL	Willows Pier, MA	20.1		wet	
HEPAT	Long Island Sound		14-200	wet	Olsen 1989[a], 1990[a]
HEPAT	Long Island Sound		16-150	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 27. Polychlorinated biphenyl concentrations in tissues of laboratory-exposed and field-collected adult American lobsters

Location/ Source	PCB Aroclor	Tissue	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
pericardial injection of ^{14}C	1242 & 1254	EGG	4.41	2.78-5.98	wet	Bend et al. 1976
		HEPAT	1.57	1.13-2.27	wet	
		MUSCLE	0.097	0.07-0.13	wet	
Acushnet Estuary, MA	1248	EDTISS	11.0	2.0-21.0	wet	Metcalf & Eddy 1981
	1254	EDTISS	12.0	0.6-84.0	wet	
	1260	EDTISS	2.0	1.0-3.1	wet	
Boston Harbor, MA	1254	CLW/TL	NG	0.01-0.16	wet	Metcalf & Eddy 1984
New Bedford Harbor, MA - areas 1-3		FLESH	8.7	0.1-84	wet	Weaver 1984
New Bedford Harbor, MA - area 3		FLESH	4.4	0.7-8.8	wet	
New Bedford Harbor, MA[a]		EGG	11.2	2.1-29.3	wet	Mercaldo-Allen et al. 1994
		STAGE1	3.93	0.54-11.4	wet	
		STAGE4	<0.16	<0.13-0.30	wet	
Long Island Sound - Milford, CT[a]		EGG	1.56	0.5-2.8	wet	
		STAGE1	0.54	<0.03-1.3	wet	
		STAGE4	<0.10	<0.09-0.02	wet	

Table 27. Cont.

Location/ Source	PCB Aroclor	Tissue	Mean (µg/g)	Range (µg/g)	Wet/Dry Weight	Reference
New Bedford Harbor, MA[b]		EGG	9.68	3.9-16.6	wet	
		STAGE1	3.66	2.6-4.9	wet	
		JUVENILE	<0.03	<0.02-0.09	wet	
Long Island Sound - Milford, CT[b]		EGG	1.36	0.68-1.9	wet	
		STAGE1	0.40	0.30-0.50	wet	
		JUVENILES	0.03	0.02-0.05	wet	
Long Island Sound		CLW/TL	<0.1	NG	wet	Chytalo 1986, 1989
Long Island Sound - Western Basin		HEPAT	3.7	2.5-4.9	wet	
Long Island Sound - Narrows		HEPAT	3.63	1.7-6.0	wet	
Long Island Sound - Central Basin		HEPAT	3.02	0.66-13.9	wet	
Long Island Sound - Eastern Basin		HEPAT	2.38	0.93-3.7	wet	
Long Island Sound		CLW/TL	NG	0.0-0.04	wet	Connecticut Department of Environmental Protection 1987
		HEPAT	NG	0.11-12.0	wet	
Boston Harbor, MA	1242	HEPAT	NG	1.36-3.84	dry	Gardner and Pruell 1987
	1254	HEPAT	NG	46.2-109	dry	
	TOTAL	HEPAT	NG	47.6-113	dry	
	1254	TAIL	NG	0.643-1.89	dry	
Long Island Sound - Bridgeport, CT[c]		EGG	2.42	1.10-4.40	wet	Mercaldo-Allen et al. 1994
		STAGE1	0.34	0.22-0.46	wet	
		JUVENILE	0.05	0.03-0.05	wet	
Long Island Sound - Milford, CT[c]		EGG	1.59	0.57-2.60	wet	
		STAGE1	0.35	0.25-0.46	wet	
		JUVENILE	0.04	0.03-0.06	wet	
Long Island Sound - New Haven, CT[c]		EGG	1.74	1.10-2.4	wet	
		STAGE1	0.51	0.09-1.69	wet	
		JUVENILE	0.04	0.03-0.05	wet	
Boston Harbor - Deer Island, MA		CLW/TL	0.0339	NG	wet	Wallace et al. 1988
Salem Harbor, MA		CLW/TL	0.0179	NG	wet	
Long Island Sound - Milford, CT[d]		EGG	3.58	1.09-9.12	wet	Mercaldo-Allen et al. 1994
		STAGE1	3.29	0.07-10.7	wet	
Long Island Sound - New Haven, CT[d]		EGG	1.08	0.12-2.21	wet	
		STAGE1	5.16	1.8-9.0	wet	
Long Island Sound - Rye, NY[d]		EGG	1.43	0.28-3.46	wet	
		STAGE1	0.80	0.10-2.6	wet	
Long Island Sound		HEPAT	NG	0.11-7.62	wet	Olsen 1989[e], 1990[e]
		HEPAT	NG	0.88-4.53	wet	
12-Mile Dumpsite during dumping		HEPAT	NG	1.4-2.5	wet	Draxler et al. 1991
12-Mile Dumpsite 11 mo after dumping		HEPAT	NG	0.7-1.1	wet	
12-Mile Dumpsite control area during dumping		HEPAT	NG	1.5-1.6	wet	
12-Mile Dumpsite control area 11 mo after dumping		HEPAT	NG	0.4-1.1	wet	
Boston Harbor, MA	1254	EDTISS	1.10	NG	wet	Schwartz et al. 1991
Salem Harbor, MA	1254	EDTISS	0.61	NG	wet	
Coastal Massachusetts	1254	EDTISS	0.44	NG	wet	

[a] 1985 data.
[b] 1986 data.
[c] 1988 data.
[d] 1989 data.
[e] Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

able 28. Halogenated hydrocarbon concentrations in tissues of field-collected adult American lobsters

ocation	Contaminant	Tissue	Mean (ng/g)	Range (ng/g)	Wet/Dry Weight	Reference
ew Brunswick and Prince Edward Island	total DDT	EGG	360	70-94	wet	Sprague and Duffy 1971
	DDE	EGG	310	70-94	wet	
	DDD	EGG	10	<10-40	wet	
	DDT	EGG	40	<10-90	wet	
	total DDT	MUSCLE	40	20-80	wet	
	DDE	MUSCLE	30	20-40	wet	
	DDT	MUSCLE	10	<10-20	wet	
	DDD	MUSCLE	10	<10-20	wet	
ong Island Sound	dieldrin	NG	12		wet	Foehrenbach 1972
	total DDT	NG	216		wet	
renton, ME	total DDT	EGG	1000		wet	Guarino et al. 1972, 1974
	total DDT	HEPAT	400		wet	
	total DDT	MUSCLE	100		wet	
	total DDT	BODY	200	140-260	wet	
iramichi Bay, NB	PCDD	HEPAT		0.006-0.044	wet	Clement et al. 1987
	PCDF	HEPAT		0.3-0.45	wet	
Limestone Point, NB	PCDD	HEPAT		0.025-0.035	wet	
	PCDF	HEPAT		0.38-0.45	wet	
South Arm, Sydney Harbor, NS	PCDD	HEPAT		0.008-0.013	wet	
	PCDF	HEPAT		0.34-0.64	wet	
Mouth, Sydney Harbor, NS	PCDD	HEPAT		ND		
	PCDF	HEPAT		0.03-0.066	wet	
Port Morien, NS	PCDD	HEPAT		ND		
	PCDF	HEPAT		0.004-0.009	wet	
Boston Harbor, MA	HCB	TAIL		0.47-1.06	dry	Gardner and Pruell 1987
	HCB	HEPAT		18-36.1	dry	
	A-HCH	HEPAT		9.38-68.3	dry	
	G-HCH	TAIL		0.0-0.46	dry	
	G-HCH	HEPAT		0.00-6.6	dry	
	A-CHLOR	TAIL		0.00-1.25	dry	
	A-CHLOR	HEPAT		14.9-148	dry	
	G-CHLOR	TAIL		0.0-2.75	dry	
	G-CHLOR	HEPAT		31.3-256	dry	
	DDT	HEPAT		9.04-124	dry	
	DDE	TAIL		12.7-37	dry	
	DDE	HEPAT		1370-3200	dry	
	DDD	TAIL		0.54-0.67	dry	
	DDD	HEPAT		38-527	dry	
Long Island Sound - Eastern Basin	CHLOR	HEPAT	50		wet	Chytalo 1986[a]
Long Island Sound - Central Basin	CHLOR	HEPAT	60		wet	
Long Island Sound - Western Basin	CHLOR	HEPAT	110		wet	
Long Island Sound - Narrows	CHLOR	HEPAT	130		wet	
New York Bight and New Jersey	tetraCDDs	HEPAT		0.4777-0.8895	wet	Rappe et al. 1991
	tetraCDDs	MEAT		0.0066-0.0083	wet	
	tetraCDFs	HEPAT		1.4607-1.6764	wet	
	tetraCDFs	MEAT		0.023-0.0312	wet	
	pentaCDDs	HEPAT		0.4536-0.4767	wet	
	pentaCDDs	MEAT		0.01-0.011	wet	
	pentaCDFs	HEPAT		0.9612-1.0555	wet	
	pentaCDFs	MEAT		0.0298-0.0373	wet	

Table 28. Cont.

Location	Contaminant	Tissue	Mean (ng/g)	Range (ng/g)	Wet/Dry Weight	Reference
	hexaCDDs	HEPAT		0.456-0.6554	wet	
	hexaCDDs	MEAT		0.003-0.0034	wet	
	hexaCDFs	HEPAT		0.1664-0.1777	wet	
	hexaCDFs	MEAT		0.0077-0.0079	wet	
	H/O CDDs/CDFs	HEPAT		0.3137-0.4118	wet	
	H/O CDDs/CDFs	MEAT		0.0-0.0085	wet	

[a]Unpublished data. Available from: R. Mercaldo-Allen, National Marine Fisheries Service, 212 Rogers Ave., Milford, CT 06460.

Table 29. Polycyclic aromatic hydrocarbon concentrations in tissues of field-collected adult American lobsters

PAH	Tissue	Location/Comments	Mean (ng/g)	Range (ng/g)	Wet/Dry Weight	Reference
PHEN	TAIL	exposed to creosote-impregnated	100		wet	Dunn and Fee 1979
FLUOR	TAIL	timber for 4 mo in a commercial	1815		wet	
PYR	TAIL	tidal pound	537		wet	
TRIP	TAIL		627		wet	
CHRY	TAIL		303		wet	
B(a)A	TAIL		222		wet	
B(e)P	TAIL		277		wet	
B(b)F	TAIL		261		wet	
B(k)F	TAIL		169		wet	
B(a)P	TAIL		281		wet	
B(ghi)P	TAIL		51		wet	
DB(ah)A	TAIL		153		wet	
INDENO	TAIL		137		wet	
PHEN	HEPAT	Maritime provinces, Canada		T-1450	wet	McLeese 1983
FLUOR	HEPAT			19-500	wet	
PYR	HEPAT			ND-245	wet	
TRIP	HEPAT			ND-1720	wet	
B(a)A	HEPAT			3-720	wet	
CHRY	HEPAT			T-1000	wet	
B(a)P	HEPAT			5.6-577	wet	
B(b)F	HEPAT			2-78	wet	
B(k)F	HEPAT			T-18	wet	
B(a)P	HEPAT			T-30	wet	
B(ghi)A	HEPAT			1.3-113	wet	
INDENO	HEPAT			0.7-74	wet	
PHEN	TAIL			ND-130	wet	
FLUOR	TAIL			1.2-102	wet	
PYR	TAIL			ND-103	wet	
TRIP	TAIL			ND-100	wet	
B(a)A	TAIL			ND-140	wet	
CHRY	TAIL			ND-16	wet	
B(A)P	TAIL			ND-30	wet	
B(b)F	TAIL			ND-9	wet	
B(k)F	TAIL			T-2.6	wet	
B(a)P	TAIL			ND-2.7	wet	
B(ghi)P	TAIL			ND-3	wet	
INDENO	TAIL			ND-4	wet	
PHEN	HEPAT	South Arm, Cape Breton, NS --		2900-3470	wet	Sirota et al. 1983

Table 29. Cont.

PAH	Tissue	Location/Comments	Mean (ng/g)	Range (ng/g)	Wet/Dry Weight	Reference
FLUOR	HEPAT	near coal-coking plant		10700-12400	wet	
PYR	HEPAT			2940-6710	wet	
TRIP	HEPAT			14900-23100	wet	
B(a)A	HEPAT			13400-23400	wet	
CHRY	HEPAT			2820-5050	wet	
B(e)P	HEPAT			5060-9330	wet	
B(b)F	HEPAT			1640-2350	wet	
B(k)F	HEPAT			392-588	wet	
B(a)P	HEPAT			637-1000	wet	
B(ghi)P	HEPAT			463-493	wet	
INDENO	HEPAT			787-855	wet	
PHEN	HEPAT	Morien and Mira Bays, NS --		20-345	wet	
FLUOR	HEPAT	controls		46-407	wet	
PYR	HEPAT			ND-197	wet	
TRIP	HEPAT			ND-141	wet	
B(a)A	HEPAT			9-38	wet	
CHRY	HEPAT			2.5-12	wet	
B(e)P	HEPAT			12-23	wet	
B(b)F	HEPAT			3-6.5	wet	
B(k)F	HEPAT			0.8-1.9	wet	
B(a)P	HEPAT			0.4-2.1	wet	
B(ghi)P	HEPAT			1.4-6.8	wet	
INDENO	HEPAT			2.1-5.0	wet	
BEN	HEPAT	exposed to a diesel spill for		17-61	NG	Williams et al. 1985
NAP	HEPAT	10 hr in Arnold's Cove, NF		1304-2985	NG	
FLUOR	HEPAT			289-679	NG	
PHEN	HEPAT			451-1321	NG	
PYR	HEPAT			151-376	NG	
BEN	HEPAT	Fairhaven, NF -- control		16-77	NG	
NAP	HEPAT			1646-2256	NG	
FLUOR	HEPAT			296-1380	NG	
PHEN	HEPAT			94-1202	NG	
PHEN	HEPAT	exposed to creosote-impregnated	1799		wet	Uthe et al. 1984
FLUOR	HEPAT	lumber in a commercial tidal	6240		wet	
PYR	HEPAT	pound for 3 mo during summer	2322		wet	
TRIP	HEPAT		16870		wet	
B(e)A	HEPAT		9406		wet	
CHRY	HEPAT		9480		wet	
B(a)P	HEPAT		870		wet	
B(b)F	HEPAT		1127		wet	
B(k)F	HEPAT		286		wet	
B(a)P	HEPAT		413		wet	
B(ghi)P	HEPAT		760		wet	
IDENO	HEPAT		1924		wet	
PHEN	TAIL		237		wet	
FLUOR	TAIL		159		wet	
PYR	TAIL		48		wet	
TRIP	TAIL		431		wet	
B(a)A	TAIL		292		wet	
CHRY	TAIL		210		wet	
B(e)P	TAIL		25		wet	
B(b)F	TAIL		34		wet	
B(k)F	TAIL		9		wet	
B(a)P	TAIL		14		wet	
B(ghi)P	TAIL		24		wet	
INDENO	TAIL		62		wet	
PHEN	HEPAT	freshly caught from four locations		14-100	wet	
FLUOR	HEPAT	off Nova Scotia		19-400	wet	
PYR	HEPAT			T-140	wet	
TRIP	HEPAT			ND-150	wet	

Table 29. Cont.

PAH	Tissue	Location/Comments	Mean (ng/g)	Range (ng/g)	Wet/Dry Weight	Reference
B(a)A	HEPAT			8.5-600	wet	
CHRY	HEPAT			4-240	wet	
B(e)P	HEPAT			12-340	wet	
B(b)F	HEPAT			6.5-47	wet	
B(k)F	HEPAT			1.0-13	wet	
B(a)P	HEPAT			0.8-22	wet	
B(ghi)P	HEPAT			2.5-21	wet	
INDENO	HEPAT			4.2-47	wet	
FLUOR	HEPAT	South Arm, Cape Breton, NS --		4220-15200	wet	Uthe and Musial 1986
PYR	HEPAT	near coal-coking plant		2910-13100	wet	
B(a)A	HEPAT			762-32700	wet	
CHRY	HEPAT			252-1240	wet	
B(e)P	HEPAT			1550-3600	wet	
B(b)F	HEPAT			1020-3820	wet	
B(k)F	HEPAT			502-955	wet	
B(a)P	HEPAT			711-1430	wet	
B(ghi)P	HEPAT			232-769	wet	
INDENO	HEPAT			486-931	wet	
FLUOR	HEPAT	Port Morien, NS -- control		90-162	wet	
PYR	HEPAT			35-46	wet	
B(a)A	HEPAT			6-79	wet	
CHRY	HEPAT			2-43	wet	
B(e)P	HEPAT			15-29	wet	
B(b)F	HEPAT			7-16	wet	
B(k)F	HEPAT			1.9-8	wet	
B(a)P	HEPAT			1.6-8	wet	
B(ghi)P	HEPAT			2.4-10	wet	
INDENO	HEPAT			T-5	wet	
FLUORE	HEPAT	Boston Harbor, MA		48-82.7	dry	Gardner and Pruell 1987
PHEN	HEPAT			228-404	dry	
ANTH	HEPAT			11.5-30.5	dry	
FLUOR	HEPAT			1060-1440	dry	
PYR	HEPAT			755-999	dry	
CHRY	HEPAT			221-541	dry	
BFA	HEPAT			104-364	dry	
PERY	HEPAT			0.0-25.1	dry	
INDENO	HEPAT			0.0-138	dry	
COR	HEPAT			0.0-22.5	dry	
B(a)A	HEPAT			17.9-169	dry	
B(e)P	HEPAT			40.1-182	dry	
B(a)P	HEPAT			13.9-117	dry	
FLUORE	TAIL			2.13-3.61	dry	
PHEN	TAIL			10.9-18.5	dry	
ANTH	TAIL			0.42-2.58	dry	
FLUOR	TAIL			31.6-64.5	dry	
PYR	TAIL			20.6-48.6	dry	
CHRY	TAIL			10.9-33.2	dry	
BFA	TAIL			5.71-20.9	dry	
PERY	TAIL			0.0-1.86	dry	
INDENO	TAIL			0.0-13.9	dry	
COR	TAIL			0.0-2.95	dry	
B(a)A	TAIL			1.86-6.64	dry	
B(e)P	TAIL			3.96-11.2	dry	
B(a)P	TAIL			1.76-8.92	dry	