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Toxicity of Creosote to Larval and Adult Lobsters and *Crangon* and Its Accumulation in Lobster Hepatopancreas

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The lethality of creosote to larval and adult lobsters (Homarus americanus) was investigated and creosote concentrations in the hepatopancreas of the exposed adults were measured. For comparison, the lethality to another crustacean, Crangon septemspinosa, was also investigated. The 96-h lethal thresholds were 0.02 mg/L for larval lobsters at 20°C, 1.76 mg/L for adults at 10°C and 0.13 and 0.11 mg/L for Crangon at 10° and 20°C respectively.

Some lobsters, particularly during commercial storage, may be exposed to creosoted lumber in wharves, breakwaters, and tidal storage pounds. MCLEESE (pers. comm.) showed that creosote at a high but undetermined concentration killed lobsters rapidly. Lack of suitable methods to measure creosote in water and animal tissues prevented further work at that time.

ZITKO (1975) found creosote concentration in periwinkles and whelks from the vicinity of the Biological Station wharf (creosoted) to be considerably higher than in those taken from Passamaquoddy Bay, an area with no nearby source of creosote. This indicates that some marine invertebrates are capable of accumulating and concentrating creosote.

EXPERIMENTAL

Lethality Tests

Adult lobsters (each weighing about 450 g) were tested in fibreglass tanks containing 30 L of sea water with 2 lobsters per test. The solutions were renewed at 48-h intervals. Temperature was 10°C, salinity was 30 o/oo and the solutions were aerated gently.

Lobster larvae were tested in 2-L glass beakers containing 1 L of sea water and one newly hatched larva, a total of 5 larvae being tested at each concentration. They were fed freshly hatched Artemia daily. Crangon of uniform size (about 1.3 g each) were tested in similar containers with 3 animals in each. The seawater solutions were aerated gently and were changed at 48-h intervals. Temperature was 20°C for lobster larvae and 10° and 20°C for Crangon.

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Time to 50% mortality (LT50) was estimated from a plot of percentage mortality against time on logarithmic probability paper. The lethal threshold (96 h LC50) was estimated as the geometric mean of the highest concentration without, and the lowest with mortality, the former being about 50% of the latter.

Analyses

The concentration of creosote in the 1- and 30-L containers, with a nominal concentration of 5 mg/L, was measured at 2, 4, 7, 24, and 48 h. Water samples of 100 mL were taken at each time and the creosote was partitioned into spectrograde hexane. Extracts were made up to 30 mL of hexane and the fluorescence emissions at 370 nm were measured after excitation at 310 nm. The concentration of creosote in the water samples was calculated by relating the fluorescence of the extracts to the fluorescence of a creosote standard in hexane of .5-mg/30 mL. A 5-g wet-weight sample of hepatopancreas was taken from adult lobsters at the time of death or at the termination of the test. These samples were extracted into spectrograde hexane for 1 h in a Soxhlet apparatus. The hepatopancreas extracts were analyzed for creosote by the method of ZITKO (1975).

RESULTS AND DISCUSSION

Concentration in Water

The concentration of creosote in water decreased exponentially with time according to the equation $C = ae^{-bt}$ (C = relative concentration, t = time in h). The a and b coefficients for creosote at 5 mg/L nominal concentration were 0.511 and -0.022 for the 1-L volume and were 0.841 and -0.067 for 30 L. It was assumed that these coefficients would apply approximately to the other test concentrations. Average concentrations during the tests were calculated according to the formula given in ZITKO et al. (1977), with exposure time taken as 48 h or as the LT50, if less than 48 h.

Lethality

Larval and adult lobsters and Crangon were tested at 5 or 6 concentrations of creosote ranging from 0.016 to 2.5 mg/L calculated average concentration.

Larval lobsters are considerably more sensitive to creosote than adults, the 96-h thresholds differing by about two orders of magnitude (Table 1). The gentle slope of the lethality line for larvae applies to test concentrations up to about 0.2 mg/L, where a sharp increase in slope occurs. At higher concentrations, the lethality line for lobster larvae is superimposed on that for Crangon at 20°C, indicating that larvae and Crangon have similar sensitivity to creosote except at the lowest concentrations tested. Consequently the 96-h threshold for larvae is lower than for Crangon.

TABLE 1

96-h thresholds and constants for the lethality lines (Log LT50 = A logC + B, LT50 in h, C in mg/L.

Test group	Threshold (mg/L)	Constants A	B
Adult lobsters, 10°C	1.76	-1.13	2.29
Larval lobsters, 20°C[a]	0.02	-0.22	1.17
Crangon, 10°C	0.13	-1.74	0.44
Crangon, 20°C	0.11	-2.44	-0.34

[a]Sharp increase in slope (A) -2.44 at about 0.2 mg/L, and (B) changing to -0.34.

The thresholds for Crangon at 10° and 20°C are intermediate between the thresholds for lobster larvae and adults. There is an indication of a slight temperature effect in that the threshold for Crangon at 10°C is slightly higher than at 20°C.

Judging from the results with Crangon, it is unlikely that temperature alone could account for the large difference in sensitivity to creosote between lobster larvae and adults. From the analyses, fluorescence spectra show that a slightly higher proportion of creosote constituents fluorescing at the lower wavelengths remained with time in the 1-L than in the 30-L tests. If these components are more toxic, they could contribute to the lower thresholds for larvae and Crangon. Either of these possible effects would be expected to produce relatively minor differences which would not account for the different sensitivities between larval and adult lobsters and between Crangon and adult lobsters.

Creosote in Hepatopancreas

Prior to the experiment, the adult lobsters had been held in the seawater supply of the Biological Station, the water intake being at the distal end of the wharf. The control lobster had a creosote concentration of 670 µg/g lipid (Table 2). Those exposed to creosote in the test had considerably higher values, the concentration increasing with test concentration. The data for 0.3 mg/L exposure suggest that the concentration in the hepatopancreas continues to increase with exposure time at least up to 120 h at a rate of about 85 µg/g lipid/h. The lobsters that died had concentrations of 47,500 and 23,700 µg/g lipid in the hepatopancreas. Those with 10,850 µg/g lipid or less were alive and apparently healthy when sampled.

TABLE 2

Creosote in lobster hepatopancreas.

Exposure concentration (mg/L)	Exposure time (h)	Fluorescence emission at 370 nm (Pyrene units/ µg/mL) x 10^6	Creosote concentration (µg/g lipid)	Lipid in hepato-pancreas (%)
2.5	100[a]	2800	47,500	12
1.3	165[a]	1400	23,700	36
0.3	120	640	10,850	18
0.3	100	420	7,120	28
0.3	25	190	3,220	25
Control	-	40	670	32

[a]Lobsters died within 2 h (2.5 mg/L) and 6 h (1.3 mg/L) of sampling

AIKEN and ZITKO (1977) found a lower concentration of polynuclear aromatic hydrocarbons (PAH's) in a commercial lobster than we did in our lab control lobster. This indicates the possibility of some uptake of PAH's from the laboratory water supply which originates at the end of the station wharf.

Further work is required to determine if death of adult lobsters is dependent on a particular concentration of creosote in the hepatopancreas or, more likely, whether death occurs at different concentrations, depending, in part, on the rate of accumulation. Also, further work is required to determine if the sensitivity of lobster larvae was affected by their prior exposure in the egg stage to the laboratory water supply.

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