THE INFLUENCE OF TEMPERATURE AND SALINITY ON THE TOXICITY OF CADMIUM TO THE FIDDLER CRAB, *UCA PUGILATOR*

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ABSTRACT

The concentrations of cadmium lethal to the fiddler crab, *Uca pugilator*, were determined for various environmental regimes of temperature and salinity. Mortality was greatest in high temperatures and low salinities when tested for 240 hr. Concentrations of cadmium were greatest in green gland followed by gill, hepatopancreas, and muscle.

The waste discharge of electroplating plants, lead and zinc mines, and chemical plants frequently contains toxic cadmium salts which contribute to the widespread environmental pollution (McKee and Wolf, 1963), and the importance of this pollutant has been stressed by its relationship with the crippling "itai-itai" disease of Japan (Kobayashi, 1971). The effects of cadmium on aquatic organisms have been investigated for numerous freshwater organisms (Doudoroff and Katz, 1953; Ball, 1967; Mount and Stephan, 1967), and while the cadmium is normally flushed down to the estuarine and marine environments, only Gardner and Yevich (1970), Jackim, Hamlin, and Sonis (1970), and recently Eisler (1971) have examined the effects of cadmium on estuarine forms. Eisler alone has reported the effects of normal variations in salinity and temperature on the toxic effect of cadmium on mummichogs.

The present report is part of a program to examine the effects of chronic exposure of cadmium to fiddler crab, *Uca pugilator*. This study examines the synergistic role of salinity and thermal stress on the acute toxicity of cadmium to the crabs.

METHODS

Fifteen adult male ($\bar{x}$ = 2.2 g) and 10 adult female ($\bar{x}$ = 1.5 g) fiddler crabs were placed in 23 × 30 cm plastic boxes along with 250 ml of dilute filtered seawater. The containers were slightly tilted in the incubator so that the crabs could freely select total or partial immersion. No avoidance of the toxic solution was noted. Desired salinities were obtained by the addition of distilled water. The cadmium stock for all experiments was reagent grade $CdCl_2 \cdot 2\text{-}1/2\ H_2O$ made up to a stock solution of 1 mg Cd^{++} per ml water. Aliquots of this stock were added to each test chamber to bring the cadmium concentration to the desired levels of 1.0, 5.0, 10.0, 25.0, and 35.0 ppm Cd^{++}. All crabs were kept in constant temperature boxes on a 12-hr light-dark photoperiod for the 10-day duration of the experiment. The water was changed every third day to reduce the buildup of metabolic wastes and to keep the concentration of cadmium near the nominal level. Preliminary tests indicated no loss of cadmium from the test medium without organisms. Eisler (1971) showed less than 5% loss from similar concentrations of cadmium in dilute seawater. Dead organisms were removed every 24 hr during the tests.

To determine the synergistic effects of environmental stresses on the toxicity of cadmium, crabs were exposed to the different cadmium

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Manuscript accepted April 1972.
FISHERY BULLETIN: VOL. 71, NO. 1, 1973.

concentrations in water of 10, 20, and 30‰ salinity maintained at 10°, 20°, or 30°C. Each experimental group had a control maintained in uncontaminated water, but subjected to the salinity and temperature stresses.

Cadmium concentrations in the tissues of crabs exposed to lethal concentrations were determined by use of radioactive cadmium (^{109}Cd) using the following procedure. Fifteen male and 15 female crabs were placed in 300 ml of filtered seawater of 20‰ salinity at 30°C. Each of three test chambers received 2.3 μc ^{109}Cd and an aliquot of stock soultion to bring the cadmium level to 5, 15, or 25 ppm Cd^{++}. These thermosaline regimes and cadmium concentrations were chosen because the acute toxicity tests show that they cause relatively high mortality rates. Four active animals (two males, two females) were sacrificed from each chamber at 0, 12, 24, 36, 48, and 60 hr. The animals were frozen until dissection of the tissues could be accomplished. Four tissues were digested and analyzed: hepatopancreas, gill, green gland, and thoracic muscle. Individual crabs were analyzed; since results from males and females showed no measurable difference, the results were pooled. Concentrations of ^{109}Cd were determined by liquid scintillation on a Packard Tricarb Model 3320 counter.[2] Since each 2.3 μc represented 1.5, 4.5, or 7.5 mg of cadmium in the test water, a simple ratio of counts per minute to microgram of cadmium was determined from spiked samples and used to calculate the amount of cadmium in the tissues. Concentrations of cadmium are expressed as parts per million wet weight of tissue.

RESULTS

ACUTE TOXICITY

In general, the higher temperatures and lower salinities produced the greatest cadmium toxicity. The susceptibility of fiddler crabs to cadmium was most pronounced in the thermosaline regime of 30°C and 10‰. The concentration fatal to 50% of the organisms in 240 hr (TLm-240 hr) was calculated to be 2.9 ppm Cd^{++} (American Public Health Association, 1971). At higher cadmium concentrations, the time required to kill 50% of the crabs was considerably reduced.

TABLE 1.—Cadmium concentrations (Cd^{++} in ppm) lethal to 50% of test organisms (TLm) at different salinities, times, and temperatures.

Salinity	Time	Temperature		
		10°C	20°C	30°C
‰	hr	ppm	ppm	ppm
10	48	--	--	11.0
	96	--	32.2	6.8
	144	51.0	21.3	4.0
	192	28.5	18.0	3.0
	240	15.7	11.8	2.9
20	48	--	--	28.0
	96	--	46.6	10.4
	144	--	23.0	5.2
	192	52.0	16.5	3.7
	240	42.0	9.5	3.5
30	48	--	--	33.3
	96	--	37.0	23.3
	144	--	29.6	7.6
	192	--	21.0	6.5
	240	47.0	17.9	5.7

Table 1 shows the influence of temperature, salinity, and cadmium concentration on the level of toxicant which kills 50% of the crabs in different time periods. The effect of temperature was extremely pronounced and TLm values were generally more influenced by temperature changes than by salinity levels within a thermal regime. The influence of salinity on the TLm-240 hr was most pronounced at 10°C and 10‰ and at higher cadmium concentrations in shorter times. The combined role of temperature and salinity on cadmium toxicity indicates that temperature is less influential at higher salinities.

TISSUE ACCUMULATION

Gills

In the first 12 hr of exposure, gill tissue accumulated cadmium in proportion to the exposure concentration (Figure 1). Thus, gill tissue from crabs exposed to 25 ppm Cd^{++} contained 110 ppm; gill tissue from those exposed

[2] Reference to trade names in the publication does not imply endorsement of commercial products by the National Marine Fisheries Service, NOAA.

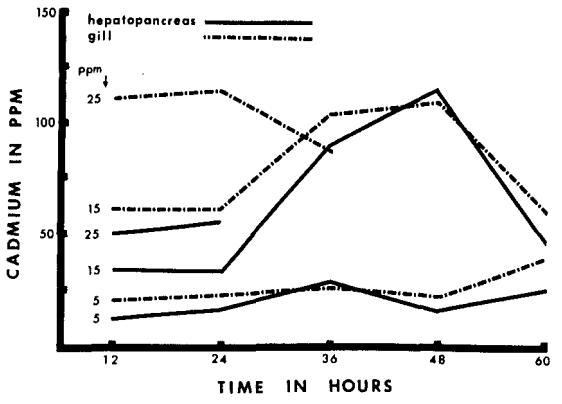

FIGURE 1.—Concentration of cadmium in gill and hepatopancreas of crabs in 5, 15 and 25 ppm Cd^{++} at 30°C, 20‰.

to 15 ppm Cd^{++} contained 59 ppm, while such tissue from those exposed to 5 ppm Cd^{++} contained 18 ppm. Each accumulation in gill tissue was about four times the concentration of cadmium in the surrounding water.

Gill tissues from crabs in 25 ppm Cd^{++} did not increase their cadmium concentration appreciably over 110 ppm in 24 hr and exhibited a decline in tissue concentration at 36 hr. The large mortality rate at 48 hr prevented reliable samples from being obtained. Gill tissue from crabs exposed to 15 ppm Cd^{++} showed an increase in cadmium content between 24 and 48 hr with a maximum accumulation of 109 ppm. The significance of the value around 110 ppm is unclear, but may represent a maximum tissue burden in terms of equilibrium with the external medium. The cadmium concentration in gill tissues from crabs sacrificed at 60 hr showed a marked reduction in cadmium content. Considering the large mortality of crabs in this concentration, the lower cadmium content in the tissues probably represents a reduced binding of the metal due to the destruction of tissue.

Crabs exposed to 5 ppm Cd^{++} continually concentrated cadmium in their gill tissue with a maximum of 39 ppm after 60 hr. No mortality occurred in this concentration, and only one animal died during this period in the acute toxicity tests. Significant mortality occurred only after 96 hr.

Hepatopancreas

The hepatopancreas from crabs in the different cadmium solutions concentrated cadmium about two times exposure level in 12 hr (25 ppm was concentrated to 50 ppm in tissue, 15 to 32 ppm in tissue, and 5 to 11 ppm in tissue). The hepatopancreas tissue in crabs exposed to the highest concentration was almost completely destroyed after 24 hr and precluded samples from these crabs (Figure 1). The hepatopancreas was changed from a firm glandular tissue to an amorphous and liquified condition. Crabs exposed to 15 ppm Cd^{++} showed an increase in cadmium levels to about 116 ppm in 48 hr, followed by a rapid decline. This decline might be associated with the destruction of the hepatopancreas tissue. Crabs exposed to water containing 5 ppm showed the same general increase in Cd^{++} concentration that was evident in gill tissue with a maximum of 24 ppm after 60 hr.

Green Gland

The bioaccumulation was highest in the green gland tissue (Figure 2) with maximum concentrations of 380 ppm in tissue from crabs exposed to 25 ppm, 171 ppm from crabs in 15 ppm, and 118 ppm from crabs in 5 ppm. These values are 12 to 20 times the exposure concentrations.

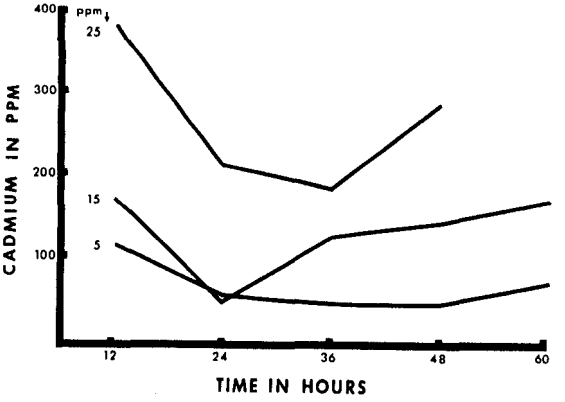

FIGURE 2.—Concentration of cadmium in green gland tissue of crabs in 5, 15 and 25 ppm Cd^{++} at 30°C, 20‰.

At all exposure levels the highest tissue accumulation occurred in the first 12 hr. At 24 hr, the concentrations in the green glands had shown a considerable decline and then increased steadily with values remaining over 10 times the exposure level. The 48-hr determination of 280 ppm is based on only two samples and needs verification.

Muscle

Muscle tissue remained almost constant over the entire time of the experiment, and tissue levels remained only slightly above the exposure levels with maximum concentrations of 29.3 ppm in crabs exposed to 25 ppm, 17.3 ppm from crabs in 15 ppm, and 8.9 ppm from crabs exposed to 5 ppm.

DISCUSSION

Cadmium toxicity is related to both temperature and salinity. The acute toxicity data for crabs maintained at different temperatures show a time delay in the onset of the lethal effect of cadmium. Whether this delay is due to differences in bioaccumulation rates or to differences in a temperature-dependent metabolic response to the metal remains to be examined. Fiddler crabs are often exposed to temperatures well in excess of 30°C, and higher temperatures would further accentuate the toxic effects of small amounts of cadmium.

There is a clear relationship of high susceptibility of fiddler crabs to cadmium in a low-salinity water. It has not been determined if this is due to interaction between the metal and the variety of salts in the seawater resulting in a nontoxic precipitate forming in proportion to the salinity (Bryan, 1971) or if the direction of the osmotic gradient in the higher salinities reduces the rate of entry of the metal.

The rapid accumulation of cadmium from the surrounding water results in considerable tissue destruction in the first 24 hr. High concentrations of cadmium were found in the gills and hepatopancreas of fiddler crabs. Similar results have been reported in crustacea exposed to zinc and mercury (Bryan, 1966; Vernberg and Vernberg, 1972) although high metal concentrations in the green glands were not reported for these metals. Gardner and Yevich (1970) reported gill tissue destruction in the mummichog beginning after 20 hr exposure to cadmium. The data presented here for Cd^{++} concentrations in fiddler crab gills indicate that 24 hr is the time when the cadmium content in crabs exposed to high Cd^{++} concentrations is reduced by tissue destruction. Yager and Harry (1966) showed a decrease in cadmium concentration in the liver of snails exposed to high concentrations of cadmium but attributed this decline to individual variation rather than to tissue destruction.

Mount and Stephan (1967) suggested that there is a threshold concentration of cadmium in the gill tissue of fishes and that death occurs when this concentration is exceeded. This threshold may be around 110 ppm for fiddler crabs.

The relationship between cadmium toxicity and temperature and salinity variation illustrates that physiological stresses, even within the usual ecological range experienced by the animals, lowers the tolerance of organisms to environmental pollutants.

ACKNOWLEDGMENTS

I wish to express thanks to Mrs. Barbara Caldwell and Mrs. Cary Clark for their technical help and to Dr. Winona B. Vernberg for her support and advice on the preparation of the manuscript. This study was supported by the Belle W. Baruch Coastal Research Institute, University of South Carolina.

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