Cadmium Toxicity and Bioconcentration in Laregmouth Bass and Bluegill

by

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Cadmium is one of the heavy metals of current interest in environmental contamination, primarily because of its highly toxic properties, its common occurrence in industrial discharges, and its existence in natural waters as a very high potential pollutant (BOWEN 1966). In addition to the direct toxicity of cadmium to fish, another and possibly more serious threat exists through the ability of these organisms to concentrate this metal. For example, an adult organism may accumulate a quantity of metal that does not cause death, but may be deleterious to some other stage of the life cycle (BRUNGS 1969).

The purpose of the present study was to detect and evaluate the effects of subacute exposure to cadmium in the largemouth bass and bluegill. Evaluation of toxicological effects was based on observations of behavioral effects, rate of growth, survival, and tissue and organ accumulation of cadmium.

Materials and Methods

A 6-month static bioassay, utilizing controlled artificial oxygenation of test solutions in laboratory aquaria, was conducted utilizing 0.85 ± 0.19, 0.08 ± 0.01, 0.008 ± 0.001, and 0.0005 ± 0.0002 (Control) mg/l of cadmium. Renewal of test solutions was employed to avoid a significant change in metal concentration of the test media, and to remove accumulated wastes. The laboratory, illuminated during daylight hours by flourescent ceiling fixtures fitted with cool white tubes, was thermostatically controlled for maintaining precribed test temperatures of 23.9 ± 0.6 C. Each of the experimental and holding tanks was continuously supplied with oil-free compressed air in quantities sufficient to maintain an average dissolved oxygen concentration of 6.5 ± 0.3 mg/l in each aquarium.

The holding tanks, used for "aging" the water, consisted of 55-gallon steel drums lined with polyethylene liners, which were replaced every 2 weeks. The test containers consisted of rectangular stainless steel tanks (23 x 14 x 8 inches) with polyethylene liners. New liners were installed in each tank every 7 days. Each container was supplied with 35 liters of experimental water; the

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depth of the water in each tank was never less than 7 inches. Tap water used for the study was supplied by the Oklahoma City Public Water Supply, and "aged" for 6 days prior to use.

The two species utilized for experimental purposes were bluegill (Lepomis macrochirus, Rafinesque), and the largemouth bass (Micropterus salmoides, Lacepede). Both species were obtained from the National Fish Hatchery, Farlington, Kansas. These young-of-the-year fish were acclimatized to laboratory conditions for 5 months prior to initiation of the test exposures. At time 0 the bass had an average weight (grams) of 10.45 $\pm$ 1.95 and a length (inches) of 9.93 $\pm$ 0.58; whereas, the bluegill had an average weight of 3.25 $\pm$ 0.83 and a length of 5.80 $\pm$ 0.49. In order to prevent any potential infections, the fish were treated with a 1:4000 dilution of formalin and 25 mg/l of tetracycline hydrochloride, 1 and 2 months prior to testing. The bass were fed a diet of Oregon Moist Fish Pellet (R.V. Moore Co., La Conner, Washington) every other day and the bluegill were fed New Age Fish Food (J.R. Clark Co., Salt Lake City, Utah) every other day, supplemented with chopped beef liver once a week.

Analytical procedures for the water analyses were conducted according to the procedures described in the 1971 edition of "Standard Methods" and/or the 1970 edition of Hach Water and Wastewater Analysis Procedures. Stock solutions of cadmium were prepared by dissolving an appropriate amount of $3CdSO4.H_2O$ in 0.2N nitric acid solution. Water samples (100 ml) were taken from each experimental tank on the first and 7th day of each week. Daily samples were taken every 4th week. The samples were acidified on the basis of 0.2 ml of concentrated nitric acid per 100 ml. The routine water analyses of dissolved oxygen, pH, alkalinity, chloride, and temperature were conducted on each test container for the complete exposure period. Additional water analyses (hardness, calcium, magnesium, sulfate, and phosphate) were performed on the "aged" water supply each day the test solutions were renewed. These analyses indicated the water to be moderately hard (180 mg/l) with about 70 per cent of the hardness present in the calcium carbonate and non-carbonate forms. Additionally the water had an average pH of 7.5 $\pm$ 0.2, an average alkalinity of 49.3 $\pm$ 13.7, an average chloride concentration of 193 $\pm$ 26, and an average sulfate concentration of 133 $\pm$ 17 mg/l. No other notable chemical characteristics were observed.

Each bass was divided into three samples for metal analysis: the gills (gill rakers, arches, and filaments); internal organs (liver, kidney, spleen, and digestive system); and the remainder of the total body. For the bluegill, the total body was utilized for metal analysis. The tissues were dried at 110 $\pm$ 3 C. for 24 hours, and then ashed in a Tracer-lab Model 600L Low Temperature Asher; the internal organs and gills of the bass and the whole bluegill were ashed for 48 hours; whereas; the body remainder of the bass was ashed for 72 hours. All samples were reconstituted with 10 ml of .04 N nitric acid solution.

The methylisobutyl ketone extraction method outlined in the 1969 edition of the FWPCA Methods for Chemical Analysis of Water and Wastes was utilized for extraction of all fish samples and tap water samples. A total volume of 10 ml was used for extraction of the fish samples; whereas, 100 ml were used for the tap water samples. Metal determinations for tissue and water samples were performed on a Jarrell-Ash Atomic Absorption Spectrophotometer Model 82-362. A Beckman Model 1005 10-inch recorder and Scale Expander (1 to 10X expansion) were used for the readout of percentage absorption. A Hetco burner, using air and hydrogen as the energy source, was used for the metal determinations. The flame conditions were optimized for maximum sensitivity.

Results and Discussion

Toxicity

Fifty per cent (8 of 16) of the bass were dead within 56 days at the 0.85 mg Cd/l exposure level, and 82 days for exposure of 0.08 mg Cd/l. The bass exposed to 0.008 mg Cd/l had only two deaths due to toxicity. Fifty per cent of the bluegill (8 of 16) exposed to 0.85 mg Cd/l were dead within 138 days; whereas, those exposed to 0.08 and 0.008 mg Cd/l survived for the entire 6-month exposure. The percentage survival for both species is depicted in Figure 1 and 2. The bass appeared to be more sensitive to cadmium than did the bluegill.

Growth

The rate of weight gains of the bass and bluegill exposed to cadmium were not statistically different from the controls; however, the rate of weight gains of the bluegill, but not the bass, tended to be lower as the concentration increased, especially during the last 2 months of exposure. The cadmium levels employed did not have a noticeable effect on growth of the bass.

Behavior

The first symptoms ("abnormal behavioral patterns") of a toxic reaction were observed during the 3rd week of exposure in a bass exposed to 0.85 mg Cd/l. The same symptoms (in bass) were observed at, 7 weeks in the 0.008 mg Cd/l level, and at 12 weeks at 0.008 mg Cd/l. These identical behavioral patterns were observed in the bluegill only in the 0.85 mg Cd/l exposure level; the symptoms first appeared during the 13th week of exposure.

The bass and bluegill, which died from cadmium toxicity, exhibited erratic, uncoordinated swimming movements, muscle spasms and convulsions, followed by loss of equilibrium, with periods of quiescence and paralysis. At death, the fins were fully spread, the branchiostegals and opercula were expanded, and the body was arched laterally in the area between the base of the pectoral fins and middle of the dorsal fin.

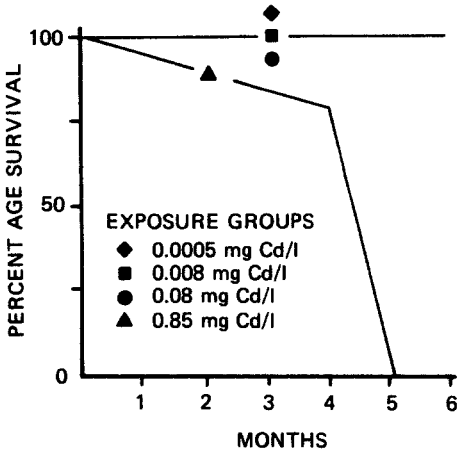

Figure 1. Survival of bluegill exposed to Cd. Each exposure group consisted of 16 fish at time 0.

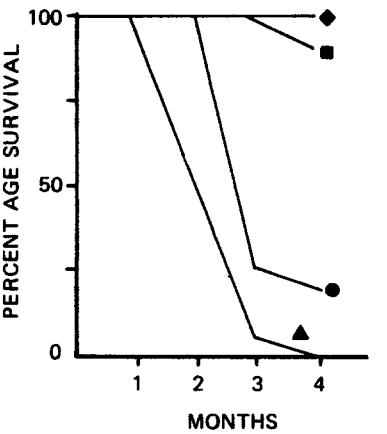

Figure 2. Survival of bass exposed to Cd. Each exposure group consisted of 16 fish at time 0.

The "abnormal behavior" by both the bass and bluegill suggested that the nervous system was the site of damage. The toxic manifestations would be consistent with the inhibition of the enzyme acetylcholinesterase, resulting in death by paralysis of the muscles of respiration and/or depression of the respiratory center.

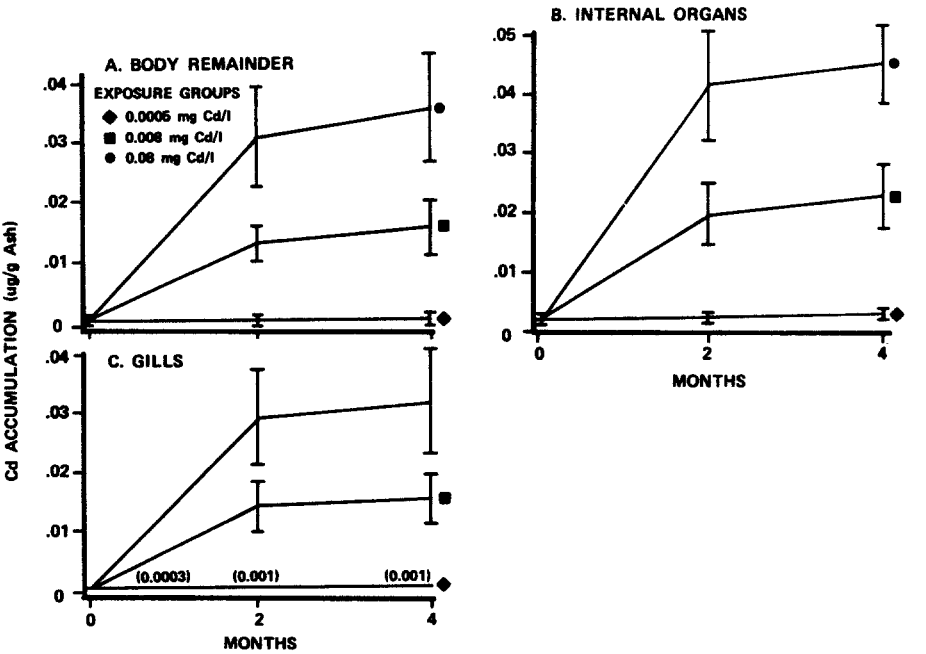

Figure 3. Cd accumulation in the body remainder (A), "pooled" internal organs (B), and gills (C) of largemouth bass. Number of fish per group at each time period was 5. Standard deviations are depicted in brackets or vertical lines.

Cadmium Accumulation

The bass and bluegill accumulated cadmium in concentrations greater than those of the exposure water. The quantity of metal accumulated increased as the exposure concentration increased (Figures 3 and 4). The maximum total body accumulation by the bass was 8-fold (0.008 mg Cd/l exposure) to 15-fold (0.08 mg Cd/l exposure) greater than the controls; whereas, the maximum accumulation by the bluegill was 6-fold (0.008 mg Cd/l exposure), 20-fold (0.08 mg Cd/l exposure), and 210 fold (0.85 mg Cd/l exposure) greater than the controls.

An equilibrium developed between the concentrations of the metal in the water and in the tissues. This was based on the absence of significant additional accumulation after the 2nd month of exposure (Figures 3 and 4).

These data indicate that the accumulation of cadmium was related to those mechanisms which affect uptake and elimination; therefore, the cessation of significant accumulation of this metal may be due to an effect produced by the metal concentrations on those mechanisms. It may be proposed that at first the uptake of cadmium exceeded its elimination, and accumulation occurred; however, at some point in time within the first 2 months of exposure, the mechanisms affecting elimination may have been stimulated so that the uptake rate approximated elimination. Those levels that are lethal to fish may be toxic due to the stimulation of the uptake mechanism, and/or inhibition of the elimination mechanism, to such an extent that the rapid rate of uptake and accumulation causes death.

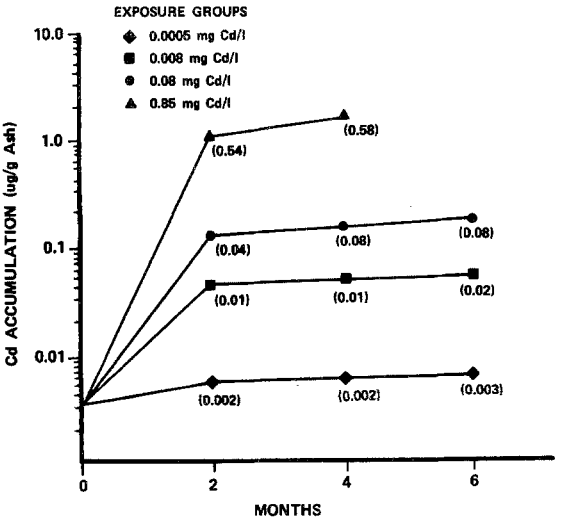

Figure 4. Accumulation in the bluegill. Number per group at each time period was 5. Standard deviations are depicted in brackets.

One mechanism that may possibly explain the accumulation of cadmium to only a certain level in the tissues is that of active transport, the system believed to be responsible for concentrating various elements (DUPRAW 1968). Inhibition of the enzymes controlling the carrier system of active transport may result in an enhancement of the transport of the element out of the cell and/or supression of the elemental transport into the cell. This enhancement and/or suppression may lead to an equilibrium so that additional metal accumulation is prevented or significantly reduced.

Cadmium had the greatest accumulation in the internal organs of the bass as compared to the gills and remainder of the body (Figure 3). This suggested that a relationship existed between the accumulation by these tissues and excretion. Cadmium had been reported to accumulate mainly in the kidney, liver, gut, gill, and to a lesser degree in the spleen (MOUNT 1967). All of these organs with significant accumulation, except the spleen, are capable of excretion. No significant accumulations have been reported in the bone or muscle tissues.

Summary

The exposure of juvenile largemouth and bluegill to cadmium (0.0005 to 0.85 mg/liter) resulted in accumulation of this metal in concentrations greater than those of the water. The quantity of metal accumulat d increased as the exposure concentration increased. An equilibrium developed between the concentrations of the metal in the water and in the tissues after approximately 2 months. Metal accumulations on the bass tissues were higher in the internal organs, followed by the gills and the remainder of the body.

The bass were more sensitive to cadmium than the bluegill. Abnormal behavior patterns observed in both species suggested that the nervous system was the site of damage.

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