Chesapeake Science Vol. 18, No. 4, p. 353-359 December, 1977

Physiological Response of Juvenile Striped Bass, *Morone saxatilis*, to Low Levels of Cadmium and Mercury

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ABSTRACT: Juvenile striped bass, *Morone saxatilis*, were exposed to 0.5, 2.5, and 5.0 parts per billion (ppb) cadmium as cadmium chloride for 30–90 days and to 1.0, 5.0, and 10.0 ppb mercury as mercuric chloride for 30–120 days. Following the longest exposure to each metal, the fish were allowed to recover for 30 days in running seawater. Gill-tissue respiration, glucose-6-phosphatase, malic enzyme, aspartate aminotransferase, and magnesium activation of AAT were measured. Animals exposed to either metal exhibited changes in gill-tissue respiration. There was no significant difference in enzyme activity during exposure to either metal; however, fish cleared for 30 days following exposure to cadmium exhibited a slight drop in liver AAT and G6PdH.

Introduction

In recent years, concern has increased over heavy metal pollution in the marine environment. Mercury and cadmium are among the heavy metals most toxic to marine organisms (Jackim et al. 1970; Conner, 1972; Calabrese et al. 1973; Waldichuk 1974) and have received the greatest attention in the scientific literature. This study was undertaken to determine the physiological effects on striped bass, *Morone saxatilis*, of chronic exposure to low levels of these two metals.

The striped bass is an important commercial and sport fish species, its use recorded back to colonial times (Raney 1952). On the East Coast, striped bass occur from northern Florida to Nova Scotia. Although the striped bass is capable of living in a wide range of salinities, it is generally an estuarine fish, migrating into freshwater to spawn (Talbot 1966). In many estuarine environments, it is exposed to considerable pollution, particularly in the highly industrialized Middle Atlantic region. Chittenden (1971) and Raney (1952) noted the absence of striped bass in the Delaware River, where it was once abundant, and attributed its absence to gross pollution. Raney (1952) made similar observations on declining numbers of this fish in the Roanoke and Connecticut Rivers as well. Despite such declines and despite the value of these fish, very little information is available about the effects of individual pollutants on them. Recent studies for the most part have concentrated on metal levels in striped bass caught in polluted waters (Tong et al. 1972; Alexander et al. 1973) and on lethal levels of various chemicals to larvae and fry in freshwater (Hughes 1973). Although the determination of lethal levels is important, sublethal concentrations of pollutants are more frequently encountered in the marine environment and do affect the physiology of a variety of animals (Brocksen and Bailey 1973; Waldichuck 1974; Calabrese et al. 1975; Thurberg et al. 1975). Research should include sublethal effects, since the

success of a valued marine species depends on its ability to function normally in its environment.

The variables examined in this study were gill-tissue respiration and enzyme activity. Respiratory changes are good indications of the general condition of an animal and have been related to stress from such factors as starvation (Beamish 1964), salinity (Thurberg et al. 1974), and pollutants (Brocksen and Bailey 1973; Collier et al. 1973). Gill-tissue respiration correlates well with whole animal respiration, particularly the standard or inactive rate of oxygen consumption (Vernberg 1956; Thurberg et al. 1975). Measurement of gill-tissue respiration has an advantage over whole animal respiration in that it eliminates changes caused by differences in activity.

Because the fish used were small, biochemical studies were necessarily limited in scope. Aspartate aminotransferase (E.C. 2.6.1.1, AAT) was monitored throughout the experimental series because it is a key enzyme of amino acid catabolism, an activity especially prominent in animals under stress (Gould et al. 1976). Also examined in the liver and skeletal muscle were two metal-activated oxidoreductases, the glycolytic shunt enzyme glucose-6-phosphate dehydrogenase (E.C. 1.1.1.49, G6PdH) and the malic enzyme (E.C. 1.1.1.40, ME), because enzymes whose activity is modulated by metal cations might show structural and functional perturbation in the presence of other metal cations.

Methods and Materials

Exposure

Juvenile striped bass were reared in freshwater at the Edenton National Fish Hatchery, U. S. Department of the Interior, Fish and Wildlife Service, Edenton, N. C. Upon arrival at the Milford laboratory, they were placed directly into flowing seawater and allowed to acclimate at least one month before exposure. The salinity throughout the acclimation and exposure periods was 24 ± 2 ‰. Temperatures ranged from 22°C, when the study began in July 1974, to 6°C, when it ended in December 1974. Throughout the acclimation and exposure periods the fish were fed a mixture of minced clams, *Spisula solidissima*, and Purina Trout Chow[1] daily. The fish were exposed in 80-liter glass aquaria filled to 60 liters with sand-filtered seawater by a proportional-dilution apparatus (Mount and Brungs 1967). The diluter controlled the intermittent delivery of toxicant-containing water and control water at a flow rate of 1 liter to each tank every 3 min throughout the test period. This provided a flow of 480 l/tank/day and an estimated 90% replacement time of 7 hr (Sprague 1969). Cadmium, as cadmium chloride ($CdCl_2 \cdot 2\text{-}1/2H_2O$), and mercury, as mercuric chloride ($HgCl_2$), were added at concentrations of 0.5, 2.5, and 5.0 ppb and 1.0, 5.0, and 10.0 ppb, respectively. Metal concentrations refer to calculated concentrations of the metal ion in solution not including background levels which were 0.5 ppb and less than 0.7 ppb for cadmium and mercury, respectively (Greig pers. commun.). These metal concentrations are realistic in terms of those present in polluted waters. Alexander et al. (1973) reported mercury concentrations up to 0.41 ppb in Block Island Sound, Rhode Island, and Tucker (pers. commun.) found cadmium concentrations up to 13.0 ppb in Raritan Bay, New Jersey.

Each test consisted of 60 fish per concentration, including controls (20 fish per aquarium). The fish averaged 4.2 g in weight and 6.3 cm in fork length at the beginning of the tests; at the end of the mercury test they averaged 14.3 g (range 8.5-20.5 g) and 10.7 cm (range 9.1-12.3 cm), while at the end of the cadmium test they averaged 10.1 g (range 5.0-16.5 g) and 9.7 cm (range 7.0-11.9 cm). Fish were exposed to mercury for 120 days and then allowed to recover in running seawater for 30 days. Cadmium exposure was 90 days followed by a 30-day recovery period in running seawater. Fish were removed at 30-day intervals for testing.

Oxygen Consumption Measurements

Gill tissue, including the arch, was excised from each fish and placed in a 15-ml War-

[1] Use of trade names is to facilitate description and does not imply endorsement by the National Marine Fisheries Service, NOAA.

burg-type flask containing 5 ml of seawater at the test metal concentration. No metal was added when fish were tested following the recovery period. Gill tissue oxygen consumption was monitored for 4 hr at 20°C in a Gilson Differential Respirometer. The flasks were shaken at 80 cycles/min. At the end of the 4-hr period, the gills were oven-dried at 100°C to a constant weight. Oxygen consumption was calculated as μlO_2/hr/mg dry weight of gill tissue, corrected to microliters of dry gas at standard temperature and pressure. The data were analyzed using the Student's "t" test. Nine control fish and nine test fish from each metal concentration were examined at each 30-day interval.

Biochemical Studies

Tissue Preparation. At the end of each 30-day exposure period, liver and skeletal muscle samples were excised from each fish. Because of their size, it was necessary to pool samples from three fish. The samples were placed in small plastic pouches, from which as much air as possible was excluded, then sealed and frozen-stored (−29°C) until testing. Because we have observed a slight and variable decrease in the AAT activity of teleost livers stored for over a month in this manner, all the liver specimens were examined within 1–4 weeks after tissue sampling. The variability was no greater than we have found in fresh liver samples.

For each testing series, aqueous homogenates were made in iced glass homogenizers containing 25-μ glass powder to facilitate grinding. The water was iced and doubly glass-distilled. For the liver samples, an initial 1:9 homogenate (19%, w/v) was made and centrifuged 40 min at 17,000 g and 4°C; the supernates were withdrawn carefully with a syringe, diluted 1:1.5 (v/w) with iced H_2O and recentrifuged. A small portion of each resulting 4% supernate was accurately diluted 1:1 with iced 50 m*M* $MgCl_2 \cdot 6H_2O$, and the major portion diluted 1:1 with iced water. Protein was determined by the biuret method (Gornal et al. 1949), using aliquots of the final aqueous 2% preparation. The skeletal muscle was minced and centrifuged for 1 hr at 17,000 g and 4°C, the resulting centrifuged tissue fluid serving as the enzyme preparation.

Assay Procedures. The water used in preparing all solutions was iced and doubly glass-distilled; solutions of coenzymes were made fresh daily; and malate and ketoglutarate substrates, neutralized with KOH, were prepared in 500-ml volumes and 5–7 ml amounts frozen-stored (−16°C) until use. Assays were performed at 340 nm on a double-beam, ratio recording spectrophotometer with a chamber temperature of 25°C, in an optical cuvette having a 10-mm pathlength. Final assay volume was 3.00 ml. Each assay was based on the oxidation or reduction of a pyridine dinucleotide coenzyme, the reduced form of which absorbs strongly at 340 nm. A linear-log recorder was used to follow reaction rates, which were read from the fastest portion of the recording.

The protocol for AAT was the same as that used for the cunner, *Tautogolabrus adspersus* (Gould and Karolus 1974). The keto acid (α-ketoglutarate) was the limiting substrate in liver AAT of this teleost, rather than the amino acid (aspartate), as has been observed in decapod crustaceans (Gould unpublished data).

Malic enzyme (ME) in the skeletal muscle of striped bass was driven optimally by magnesium and phosphate buffer, rather than the more usual manganese in combination with a Tris or glycylglycine buffer (Gould 1968). Mg was not an absolute requirement, but served as a positive modulator. ME was measured, therefore, both in the presence and absence of Mg, to determine whether there was any difference in sensitivity to Mg between control and metal-exposed animals, as has been observed in other teleosts and crustaceans (Gould et al. 1976). The assay medium with Mg contained 2.50 ml phosphate buffer, 0.10 *M*, pH 8.00; 0.10 ml nicotinamide adenine dinucleotide phosphate (NADP), 10 mg/ml H_2O; 0.06 ml $MgCl_2 \cdot 6H_2O$, 50 m*M*; 0.20 ml L-malic acid, 0.10 *M*, neutralized with KOH; 0.04 ml H_2O; and 0.10 ml enzyme preparation to start the reaction. For the assay without Mg, 0.10 ml H_2O was used.

For the G6PdH measurement, the cuvettes contained 2.55 ml Tris (hydroxymethyl) aminomethane buffer, 0.10 *M*, pH 8.00; 0.10 ml NADP, 8 mg/ml H_2O; 0.10

ml glucose-6-phosphate, sodium dihydrate, 15 m*M;* 0.15 ml $MgCl_2 \cdot 6H_2O$, 50 m*M;* and 0.10 ml enzyme preparation to start the reaction.

Results

Control fish exhibited normal variation in oxygen consumption rate, attributable to seasonal variation and growth of the fish; because of this, each exposure group's data were plotted as a percent of that group's control values (Table 1, Figs. 1, 2).

Fish exposed for 30 days to 0.5, 2.5, or 5.0 ppb cadmium consumed significantly less oxygen than did controls ($P < .01$). Fish exposed for 90 days and those allowed to recover for 30 days following a 90-day exposure respired at rates not significantly

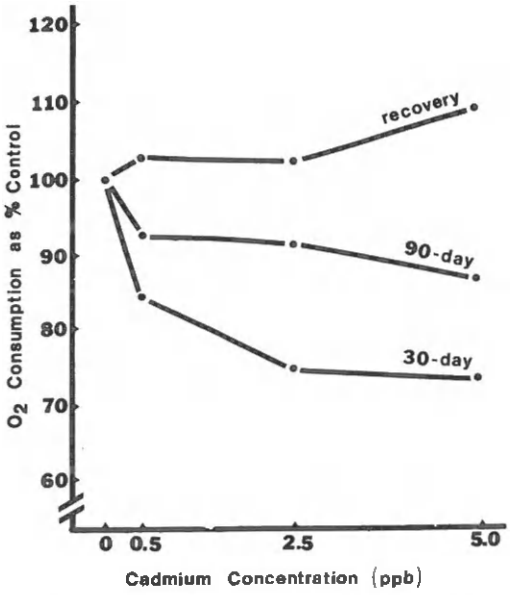

Fig. 1. Gill-tissue oxygen consumption of striped bass, *Morone saxatilis*, exposed to cadmium chloride. Each point represents mean respiration of nine fish. Recovery period was 30 days in running seawater following the 90-day exposure period.

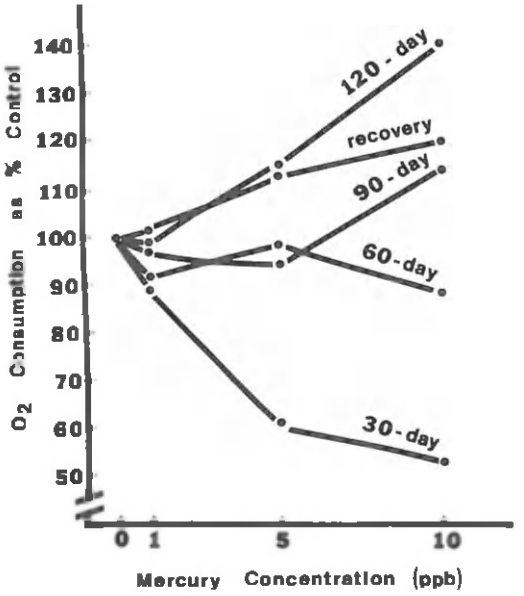

Fig. 2. Gill-tissue oxygen consumption of striped bass, *Morone saxatilis*, exposed to mercuric chloride. Each point represents mean respiration of nine fish. Recovery period was 30 days in running seawater following the 120-day exposure period.

TABLE 1. Gill-tissue oxygen consumption[1] of metal-exposed striped bass, *Morone saxatilis*.

	Cadmium concentration			
Length of exposure	Control	0.5 ppb	2.5 ppb	5.0 ppb
30 days	1.908 ± .064	1.580 ± .083[b]	1.410 ± .066[b]	1.380 ± .086[b]
90 days	.877 ± .076	.812 ± .022	.801 ± .033	.753 ± .027
30-day recovery	.813 ± .057	.829 ± .044	.830 ± .043	.871 ± .042
	Mercury concentration			
	Control	1 ppb	5 ppb	10 ppb
30 days	1.742 ± .239	1.550 ± .209	1.096 ± .025[a]	.971 ± .038[b]
60 days	.859 ± .037	.789 ± .042	.847 ± .042	.758 ± .038[a]
90 days	.769 ± .038	.747 ± .033	.727 ± .032	.887 ± .047
120 days	.679 ± .037	.673 ± .030	.785 ± .044	.946 ± .055[c]
30-day recovery	.822 ± .067	.842 ± .064	.927 ± .050	.986 ± .042

[1] Units are $\mu lO_2/hr/mg$ dry wt. ± standard error.
[a] Significantly different from control at .05 level.
[b] Significantly different from control at .01 level.
[c] Significantly different from control at .001 level.

different at the .05 level from those of controls.

The respiratory rate of animals exposed to 1 ppb mercury did not differ significantly from that of controls, regardless of exposure time. Fish exposed to 5 ppb mercury for 30 days respired at a rate significantly lower than that of controls ($P < .05$). After 60 days, respiration of exposed and control groups was approximately equal; absolute values for both groups had decreased from the 30-day levels. After longer exposure periods there was no significant difference between control animals and those exposed to 5 ppb mercury. Animals exposed to 10 ppb mercury exhibited decreased respiration at 30 days. This was followed by a gradual increase, compared to controls, at 60 and 90 days, reaching a rate significantly higher than that of controls at 120 days ($P < .001$). After 30 days in running seawater, respiration of these fish decreased from 139 percent of controls to 120 percent. In retrospect, a 60-day recovery period would have been valuable.

Neither AAT nor G6PdH activity changed significantly in the livers of fish during their exposure to cadmium or mercury. After 30 days in running seawater, however, fish that had been exposed to 5 ppb cadmium showed a highly significant decrease ($P < .001$) in both of these enzymes (Table 2). In liver preparations containing a high concentration of magnesium chloride (25 m*M*), a similar pattern emerged: there was no significant change in Mg activation in the livers of fish during exposure to the sublethal concentrations of either metal, but in fish that had been cleared for 30 days, there was a significantly higher Mg activation of liver AAT in fish that had been exposed to 2.5 and 5 ppb cadmium (Table 2). No significant changes were observed in skeletal muscle AAT or ME, or in Mg activation of ME.

Discussion

The lack of significant change in activity of the liver enzymes monitored during the 90- to 120-day period of exposure to the metal salts suggests a metabolism readily adaptable to environmental changes, not a surprising observation in an anadromous fish. It is also probable that the level of metal challenge was too low to elicit any detectable biochemical response in a fish whose metabolism, particularly in the juvenile stage, is geared to respond quickly to environmental changes.

The drop in both AAT and G6PdH activities in the liver of cleared fish previously exposed to 5 ppb cadmium for 3 months was the only significant change in these enzymes over the entire course of the experiment. We have no positive explanation for this phenomenon, but offer the following interpretation:

Certain metalloenzymes are known to be inhibited by cadmium, both *in vitro* and during short-term *in vivo* studies (Smith and Hill 1960; Hodgen et al. 1969). In recent work with an estuarine teleost (winter flounder, *Pseudopleuronectes americanus*) chronically exposed to sublethal amounts of cadmium, these enzymes increased in activity (Gould 1977). This increase was attributed to compensatory induction of the enzymes under cadmium attack. One might reasonably speculate, therefore, that in suc-

TABLE 2. Enzyme activity[1] in liver of striped bass, *Morone saxatilis*, held 30 days in running seawater after 90-day exposure to cadmium.

Prior Cd Exposure	Number sample pools	AAT Activity	Mg Activation of AAT (% normal activity)	G6PdH Activity
0	7	503 ± 38	120 ± 7	701 ± 42
0.5	4	516 ± 62	124 ± 3	576 ± 44
2.5	4	405 ± 58	147 ± 6[a]	583 ± 100
5.0	5	262 ± 29[c]	161 ± 10[b]	270 ± 38[c]

[1] Unit of activity is change in absorbance at 340 nm of 0.001/min/mg protein, under assay conditions ± standard error.

[a] Significantly different from control at .05 level.

[b] Significantly different from control at .01 level.

[c] Significantly different from control at .001 level.

cessfully adapting to chronic low-level cadmium challenge, the bass had been able to gear its metabolism to steady-state by an analogous compensatory production of cadmium-inhibited enzymes. When the metal challenge was removed and the demand for extra enzyme production shut off, the activity of enzymes involved in producing metabolites for energy mobilization (AAT) and biosynthesis (G6PdH) might be expected to drop briefly. This totally conjectural interpretation is offered as a possible area for future work.

Although the data for these two enzymes were based on 9–16 pools of three livers for each concentration of metal at each exposure interval, there was not enough material to explore metabolic pathways other than the glycolytic shunt and one area of nitrogen metabolism. On the whole, juvenile striped bass in an experiment of this scope do not yield enough samples of sufficient size to support a properly thorough biochemical study.

The oxygen consumption data support the results obtained on other cadmium- and mercury-exposed fish. Cunners, *T. adspersus*, subjected to short- or long-term cadmium exposure exhibited depressed gill-tissue oxygen consumption, while long-term mercury exposure elevated their respiration (Thurberg and Dawson 1974; MacInnes pers. commun.). Calabrese et al. (1975) reported that winter flounder, *P. americanus*, exposed to 5 and 10 ppb cadmium for 60 days showed depressed oxygen consumption, while those exposed to 10 ppb mercury had elevated levels. Oxygen consumption is a good general indicator of stress. In the case of striped bass it may be particularly valuable since there is some evidence that the limiting factor to striped bass in a grossly polluted natural environment is the oxygen level (Chittenden 1971; Talbot 1966).

A laboratory exposure to one contaminant cannot account for all natural conditions. Toxicity is influenced by any number of natural variables. In particular, MacLeod and Pessah (1973) have demonstrated increasing mercury toxicity with increasing temperature in the rainbow trout, *Salmo gairdneri;* Olson and Harrel (1973) demonstrated changes in mercury toxicity to the clam, *Rangia cuneata,* with changing salinity. Hence, the results of this study would likely vary somewhat under different experimental or environmental conditions. In spite of these limitations, a controlled exposure is a valuable tool and one of the few ways available of isolating the effects of a single pollutant.

Acknowledgment

The authors thank Miss Rita S. Riccio for her critical reading and typing of this manuscript.

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