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Effects of Cadmium, Mercury, and Silver on Marine Animals

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INTRODUCTION

This summary of recent work at the Milford Laboratory of the National Marine Fisheries Service's Middle Atlantic Coastal Fisheries Center is intended as a convenient reference for scientists investigating heavy-metal stress in marine animals. Increasingly in the past decade, attention has focused on this and related areas of study (for example, Waldichuk, 1973) because of widespread concern over extensive dumping of waste material and runoff of polluted waters into our estuarine, coastal, and oceanic ecosystems. Population growth and technological development are putting serious stress on these areas, and such stress fosters conditions that diminish the harvest of marine resources. The concern is not only for marine animals that are important to commercial and sport fisheries, but also for those animals whose presence indicates a healthy and stable environment.

Information on the nature and degree of man-induced damage to our living marine resources is either fragmentary or lacking. Yet such knowledge is essential for formulating baseline estimates of marine environmental quality, without which resource-oriented water-quality standards cannot be established or enforced by federal and state regulatory agencies.

To establish such standards, it is first necessary to determine how and to what degree pollutants, individually and in combination, affect various marine animals at different life stages. More important than death itself, perhaps, is the damage caused by sublethal concentrations of pollutants. The gradual elimination of an animal species by low levels of pollutants is no less serious than rapid death caused by high levels. Possibly it is even more serious, as low-level effects are less likely to be detected and traced to their source before irreparable damage has occurred.

Numerous studies have been published on the toxicity of heavy metals to aquatic animals, particularly finfish, but these have dealt primarily with freshwater rather than marine species. The National Marine Fisheries Service (NMFS), through the Middle Atlantic Coastal Fisheries Center (MACFC), Milford, Conn., has consequently undertaken research programs designed to generate the basic knowledge required for effective management of the marine environment and its living resources. The New York Bight, which is receiving international attention because of the large amount of waste material it receives, borders the most heavily populated and industrialized area in the United States. Because MACFC is located within this geographical area, it has initiated studies to determine the influence of pollutants on key marine animals within the Bight.

As part of this major effort, the MACFC laboratory has exposed important species of indigenous fishes, mollusks, and crustaceans to cadmium, mercury, and silver to study mortality rates and any physiological and biochemical changes caused by these heavy metals. Tissues of experimentally exposed animals have also been provided to MACFC laboratories at Oxford, Md., and Sandy Hook, N.J., for histopathological and biochemical examination, evaluation of immune response to various antigens, and measurement of metal uptake into various tissues and organs.

This report summarizes the data collected in the course of these studies. No attempt has been made to review the scientific literature, as such material has been covered in papers published previously and cited in this report.

METHODS

Test animals used in these studies were collected from Long Island Sound near Milford, Conn. Prior to heavy-metal exposure, they were acclimated in the laboratory for at least 2 weeks. Exposures were either short-term (hours to days) static tests or long-term (weeks to months) continuous-flow tests. Systems such as shown in Figure 1 were used for static tests, and the modified Mount

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Figure 1.—Exposure system for short-term static tests.

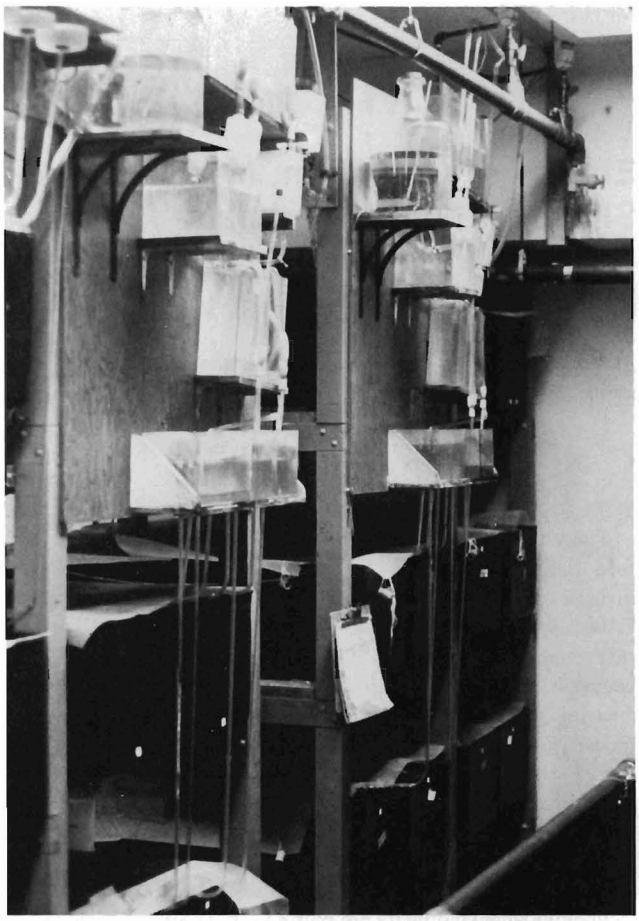

Figure 2.—Exposure system for long-term continuous-flow tests.

and Brungs (1967) proportional dilutor shown in Figure 2, for continuous-flow tests. At the end of each exposure period or at intervals during that time, animals were removed for evalutaion of metal stress.

For studies of inhibited development of bivalve eggs and larvae, modified techniques of Davis and Calabrese (1964) were used. Physiological and biochemical changes in juvenile and adult organisms were measured and evaluated as described by Thurberg et al. (1973), Calabrese et al. (1974, 1975), and Gould (in press). Except where otherwise noted, cadmium and mercuric chlorides and silver nitrate were used throughout.

OBSERVATIONS

Short-term tests were designed not only to measure mortality rates, but also to determine tolerance ranges and discover those metabolic systems most sensitive to metal stress. Long-term tests were designed primarily to study the physiological and biochemical effects of sublethal metal stress. The data are summarized in Tables 1-3 according to the metal used: cadmium, mercury, or silver. For ease of discussion, the following observations are arranged into three major groups: mollusks, crustaceans, and finfish.

Mollusks

Metal exposure tests on mollusks included evaluation of larval and juvenile mortality and oxygen consumption of adult animals. Both short- and long-term exposures were used, and salinity variables were examined.

Embryos of the American oyster, *Crassostrea virginica,* were exposed to mercury, silver, or cadmium and the concentrations that caused 50 percent mortality (LC_{50}) in 48 hours were determined. Mercury and silver were extremely toxic, with LC_{50} values of 5.6 and 5.8 parts per billion (ppb), respectively. Cadmium, on the other hand, had low toxicity with an LC_{50} of 3,800 ppb (Calabrese et al., 1973). Larvae of this same species were exposed to mercury and silver for 12 days; concentrations of these two metals causing 50 percent mortality were 12 and 25 ppb (unpublished data), respectively, indicating that embryos are more sensitive than larvae.

In similar tests, embryos and larvae of the hard clam, *Mercenaria mercenaria,* were exposed to mercury and silver. The LC_{50} values for embryos were 4.8 and 21.0 ppb, and for larvae 14.7 and 32.4 ppb, respectively (Calabrese and Nelson, 1974; unpublished data). A comparison of results with clam and oyster embryos and larvae indicates that clam embryos and larvae are as sensitive to mercury as oyster embryos and larvae, but less sensitive to silver.

Juvenile bay scallops, *Argopecten irradians,* were exposed to mercury, silver, or cadmium for 96 hours. Fifty percent mortality occurred at 33 ppb silver, 89 ppb mercury, and 1,480 ppb cadmium. Although in scallops the order of toxicity for mercury and silver was the reverse of that found for clam and oyster embryos and larvae, these two metals are still far more toxic than cadmium. Juvenile scallops exposed to the LC_5 and LC_{25} values of cadmium for 96 hours, however, had significantly higher oxygen-consumption rates than controls. Scallops exposed to silver at the LC_{25} level also had elevated respiration, whereas those at the LC_5 level respired at a slightly lower rate than controls (Nelson et al., 1976).

Mud snails, *Nassarius obsoletus,* exposed to sublethal concentrations of silver or cadmium, exhibited deviations from normal behavior and had altered oxygen-consumption rates. The oxygen-consumption rate of these snails after exposure to silver was depressed at concentrations greater than 500 ppb and was slightly elevated after exposure to 500-4,000 ppb cadmium (MacInnes and Thurberg, 1973).

A later study was designed to evaluate the sublethal effects of silver on American oyster, hard clam, blue mussel (*Mytilus edulis*), and soft-shell clam, *Mya arenaria,* held at various salinities for 96 hours. Each species respired at a significantly higher rate after exposure to levels of silver as low as 100 ppb. This sensitivity varied with salinity, and certain silver-salinity combinations were lethal (Thurberg et al., 1974). In long-term exposures (30-90 days) to 5 percent (10 ppb), the silver concentration used in the short-term study, these same species also had elevated oxygen-consumption rates. Rapid recovery after removal from the contaminated water is apparently not possible; bivalves placed in "clean" water for 30 days, following 30 days in silver-contaminated water, still had elevated respiration (unpublished data).

A subsequent study involved all three major life stages of the surf clam, *Spisula solidissima*. Larvae exposed to 50 ppb silver for 2-15 days respired at a rate higher than the controls. Juvenile clams exposed to silver for 96 hours had elevated respiration at 10 ppb, but not at 5 ppb. Adults exposed for 96 hours had higher respiration rates than controls at 50 and 100 ppb, but not at 10 ppb. Valve-movement studies conducted on adult clams showed more rapid rhythmic activity in exposed clams than in control animals. There was rapid accumulation of silver in gill and body tissues, with gills concentrating four times as much silver as other tissues (Thurberg et al., 1975).

Crustaceans

Different species of decapod crústaceans showed the same diversity of physiological response to metal exposure as did the mollusks. We looked at the salinity variable again, as well as different salts of mercury and cadmium.

Toxicity of cadmium during short-term (72 hours) exposure of mud crabs, *Eurypanopeus depressus,* was measured by mortality and oxygen-consumption rates (Collier et al., 1973): LC_{50} was 4,900 ppb and LC_{100}, 11,000 ppb. Gill-tissue oxygen consumption was depressed with increasing cadmium concentration.

Green crabs, *Carcinus maenas,* and rock crabs, *Cancer irroratus,* were exposed for 48 hours to various concentrations of cadmium chloride at five different salinities (Thurberg et al., 1973). Cadmium elevated blood-serum osmolality of green crabs, but not that of rock crabs, and oxygen consumption was depressed in both species. Concentrations of 2,000 ppb and higher were lethal to rock crabs, whereas green crabs survived at 4,000 ppb.

In further experimentation with rock crabs, effects of cadmium chloride and cadmium nitrate were compared. A difference in effect was found in short-term (96 hours) and long-term (30 days) exposure series, although these effects were strongest in the acute studies (Gould et al., 1976). A key enzyme of nitrogen metabolism, aspartate aminotransferase (AAT), also important in energy mobilization in invertebrates, was used as the biochemical index of stress. Throughout these studies, AAT was consistently elevated in crabs exposed to cadmium chloride, but not to cadmium nitrate. More mortalities occurred in the chloride groups and, in short-term studies, serum magnesium was elevated in crabs exposed to the chloride salt, but not in those exposed to the nitrate salt. In long-term studies, blood-serum osmolality was raised in the chloride, but not in the nitrate groups.

Sublethal effects of cadmium, mercury, and silver were evaluated in the American lobster, *Homarus americanus*. Cadmium induced greater physiological and biochemical changes than mercury, although uptake of mercury into various tissues was ten times greater than cadmium. Gill tissue from cadmium-exposed (3-6 ppb) lobsters had elevated rates of oxygen consumption and showed some signs of increased ATPase activity (Thurberg et al., in press). In antennal glands, ligand sensitivity was lost in a key regulatory enzyme of the pentose phosphate shunt, glucose-6-phosphate dehydrogenase (G6PdH). In the heart, magnesium sensitivity was lower in transaminase and activity increased, both in a metalloenzyme, lactic dehydrogenase (LdH), and in glycolytic enzymes involved in the mobilization of energy, glucose-phosphate isomerase (GPI) and pyruvate kinase (PK) (unpublished data). Chronic exposure to mercury (6 ppb) produced a lesser effect on ligand sensitivity of heart transaminase than cadmium exposure, and none at all on gill-tissue respiration. Neither cadmium nor mercury affected serum osmolality, and very little difference was found in any test when data from 30- and 60-day exposures were compared (Thurberg et al., in press).

In a comparison of mercuric chloride and mercuric nitrate, again for 30 days at sublethal concentrations (6 ppb), the chloride salt proved to be more toxic. Ligand sensitivity of G6PdH decreased in the antennal glands of chloride-exposed lobsters, but not in the nitrate group; in the heart muscle of the chloride group, activity increased, both in the metalloenzyme LdH and in the glycolytic enzymes GPI and PK, but not in the nitrate group; and in female gonads, glycolysis increased in the chloride, but not in the nitrate group. Neither gill-tissue oxygen consumption nor serum osmolality changed in lobsters exposed to either of the mercuric salts. In lobsters exposed under like conditions to silver nitrate, the biochemical pattern was similar to that observed in the mercuric chloride-exposed animals, but to a much lesser degree. Again, neither gill-tissue respiration nor serum osmolality changed. Heart transaminase was depressed in silver-exposed lobsters, however, as in cadmium-exposed rock crabs, but not in cadmium- or mercury-exposed lobsters. The order of metal toxicity for adult lobsters, therefore, as determined by these biochemical parameters, is $CdCl_2 > HgCl_2 > AgNO_3 > Hg(NO_3)_2$ (unpublished data).

Finfish

We have thus far examined three teleost species after exposure to metals: the cunner, *Tautogolabrus adspersus,* winter flounder, *Pseudopleuronectes americanus,* and striped bass, *Morone saxatilis;* and similar work with the coho salmon, *Oncorhynchus kisutch,* is presently under way.

In a short-term study (96 hours) of cadmium-exposed cunners, high levels (48,000 ppb) of this metal produced abnormally high serum osmolalities, and only 3,000 ppb Cd reduced the normal rate of gill-tissue oxygen consumption (Thurberg and Dawson, 1974). These indicators of stress are probably related to tissue damage as observed in concurrent histopathological and immunochemical studies on the same fish (Newman and MacLean, 1974; Robohm and Nitkowski, 1974). Cadmium uptake averaged 8.5 times higher in livers than in gills (Greig et al., 1974). Biochemical data for two liver enzymes showed significant changes in fish exposed to 24,000 ppb Cd. Transaminase activity was depressed, and a magnesium-linked enzyme required 10 times as much added magnesium to reach the same level of activity as in the control fish (Gould and Karolus, 1974). The latter observation was the first to point to metal-induced suppression of a specific metabolic control.

Another short-term study (96 hours) with cunners compared the effects of silver nitrate and silver acetate at 1,000 ppb Ag. Gill-tissue respiration was depressed by both salts, with no significant difference in their effects (Thurberg and Collier, in press; Gould and MacInnes, in press). Biochemical changes induced by exposure to silver, however, did differ significantly with the salt form. Liver transaminase was higher in fish exposed to the acetate salt than in those exposed to the nitrate salt, and shunt activity (G6PdH) in the liver was significantly depressed in the nitrate group, but not in the acetate group (Gould and MacInnes, in press).

In chronic exposures (30-60 days) of cunners to cadmium chloride, 100 ppb Cd depressed oxygen-consumption rates of gill tissue, as in the acute study, and changes were also observed in liver transaminase and G6PdH activities (MacInnes et al., 1977). Long-term exposure to mercury, on the other hand, elevated gill-tissue respiration (unpublished data).

Chronic exposures (30-90 days) of juvenile striped bass to very low levels of either cadmium (0.5-5 ppb) or mercuric (1-10 ppb) chloride depressed gill-tissue respiration, but induced no significant change in either of two liver enzymes monitored (Dawson et al., in press).

Chronic exposures (60 days) of winter flounder to cadmium chloride, to mercuric chloride, and to silver nitrate produced some contrasting data. At 10 ppb, cadmium depressed gill-tissue respiration, whereas 10 ppb Hg increased it and, although cadmium provoked no significant hematological response, mercury induced differences in plasma protein levels, osmolality, hematocrit, hemoglobin, and mean corpuscular hemoglobin (Calabrese et al., 1975). Silver caused no detectable change either in gill-tissue respiration or in hematological indices (unpublished data). There was no significant cadmium uptake in tissues of exposed fish, but considerable mercury accumulated in tissues of fish exposed to that metal (Calabrese et al., 1975). Biochemical data pointed to cadmium as the most toxic to adult flounder of the three metals tested, with mercury second and silver least effective. In the kidney and hematopoietic tissue of cadmium-exposed (10 ppb) fish, metalloenzyme activity increased and the regulatory shunt enzyme G6PdH lost sensitivity to the positive-modulating effect of magnesium (Gould, in press). Ligand sensitivity was also lowered in some heart enzymes; enzyme induction occurred in heart, gonad, and skeletal muscle and in the liver, glycolysis and shunt activity increased (unpublished data). In the mercury-exposed (10 ppb) flounder, the same phenomena were observed, but to a considerably lesser extent; and in silver-exposed fish, there was very little effect (unpublished data). Thus the order of metal toxicity for adult winter flounder appears to be the same as that observed for adult lobsters, $CdCl_2 > HgCl_2 > AgNO_3$.

DISCUSSION

Tables 1-3 capsulize the results of the various experimental series performed with cadmium, mercury, and silver, using marine animals selected as important to and representative of the New York Bight. The most obvious generalization is that both the order and degree of metal toxicity vary, not only with such parameters as salinity and metal salt form, but also with life stage and species. No other overall conclusion can be drawn at this point.

A few emerging patterns, however, may be seen in the data thus far. For example, early life stages appear to be more sensitive to mercury and silver than cadmium; this is true of the mollusks tested, and work with juvenile lobsters confirms this pattern (unpublished data). Other marine larval forms have been reported to have this same order of sensitivity (Waldichuk, 1974). Another consistent finding has been that mercury and silver are taken up very readily by tissues of juvenile and adult animals, whereas cadmium is taken up to a far lesser degree. The difference in uptake rates may account at least in part for the relative toxicities of these three metals in the rapidly metabolizing early life forms. The greater the body burden of metal, it seems, the greater the toxicity. Paradoxically, however, the order of toxicity in adult animals is reversed from that observed in juvenile stages, with cadmium producing more severe effects than either mercury or silver, despite the much lower rate of cadmium uptake. The adult animals examined had apparently acquired a relative tolerance for mercury and silver—perhaps a sequestering mechanism—that is lacking in juvenile forms and which enables their metabolism to function even with large body burdens of these metals.

One tentative observation arises from the work with finfish thus far. Striped bass, a marine species that moves to brackish or fresh water to spawn, adapted to long-term metal exposure with little sign of stress, in marked contrast to winter flounders and cunners. Current work with coho salmon, too, shows it to be remarkably stress-free after long-term cadmium exposure (60 days and 100 ppb). The relative metal tolerance of these two anadromous species points to a metabolism capable of adapting readily to environmental change.

Sprague (1971) has stated that "Understanding physiological action of a toxicant is the key to predicting important sublethal effects." Examination of tissues from metal-exposed crustaceans and finfish has revealed two general and basic effects of sublethal metal challenge. The first is the induction of enzymes that are either directly attacked (metalloenzymes) or involved in the mobilization of energy (glycolysis) or in

Table 1.—Cadmium—Effect on marine animals.

Organism	Exposure period	Concentration (ppb)	Oxygen consumption	Osmoregulation	Enzyme activity	Other
Crassostrea virginica American oyster (eggs)	48 hr					LC_{50} × 3,800 ppb (2)[1]
Argopecten irradians Bay scallop (juveniles)	96 hr	940	Elevated (16)			LC_{50} 1,480 ppb; significant Cd uptake (16)
Nassarius obsoletus Mud snail	72 hr	500	Elevated (13)			Distressed behavior (13)
Carcinus maenas Green crab	48 hr	500	Depressed (25)	Disruption (25)		
Cancer irroratus Rock crab	48 hr	120	Depressed (25)	No effect (25)		
	96 hr	1,000			Chloride salt increased transaminase, nitrate salt no effect (heart) (9)	Serum Mg unchanged (9)
	30 days	250	Depressed (u)		Chloride salt increased transaminase, nitrate salt no effect (heart) (u)	Serum Mg unchanged (u)[2]
Eurypanopeus depressus Mud crab	72 hr	4,000	Depressed (5)			LC_{50} = 4,900 ppb; LC_{100} = 11,000 ppb (5)
Homarus americanus American lobster	30 days	3-6	Elevated (22)	No effect (22)	Enzyme induction, lowered ligand sensitivity (heart, antennal gland) (22, u)	
	60 days	3-6	Elevated (22)	No effect (22)		Significant Cd uptake in gills (22)
Tautogolabrus adspersus Cunner	96 hr	3,000-48,000	Depressed (24)	Disruption (24)	Depressed transaminase, lowered ligand sensitivity (liver) (10)	Some histopathological effects (17); liver uptake 8.5 times greater than gills (12)
	30 days	50-100	Depressed (14)	No effect (14)	Depressed transaminase, higher shunt activity (liver) (14)	
	60 days	50	Depressed (14)	No effect (14)		
Morone saxatilis Striped bass	30 days	0.5-5.0	Depressed (7)		No effect (liver, skeletal muscle) (7)	
	90 days	5	No significant effect (7)		No effect (liver, skeletal muscle) (7)	90-day exposure and 30-day clearance; depressed transaminase and shunt activity (liver) (7)
Pseudopleuronectes americanus Winter flounder	60 days	5-10	Depressed (4)		Enzyme induction, lowered ligand sensitivity (heart, kidney, gonad, skeletal muscle) (8, u)	No detectable Cd uptake in blood or gills. No hematological or histopathological changes (4)
	150 days	10			Increased glycolysis and shunt activity, lowered ligand sensitivity (kidney, liver) (u)	

[1]Numbers in parentheses refer to citations in Literature Cited.
[2]u means unpublished.

the production of metabolites for biosynthesis (pentose shunt), and chronic demand for enzyme production is costly in terms of metabolic energy. The second effect, the loss of ligand sensitivity, by which enzyme reaction rates are regulated, is perhaps more serious. Even partial blocking of such physiological controls is a lessening of the metabolic flexibility necessary for an animal's adaptation and survival during environmental challenge, whether natural or anthropogenic.

Implications for recruitment to fish stocks should not be lightly dismissed. Heavy-metal pollution is greatest in our estuarine and inshore coastal waters. These highly polluted areas are the breeding grounds and nurseries for many marine species important to both commercial and sport fisheries, stocks whose contemplated management now engages our most earnest attention.

Marine scientists have only begun to explore the effects of this class of pollutant. It is necessary to proceed from the initial studies of individual metals to studies of synergistic effects, not only of metals combined with other metals and salt forms, but also of metals combined with other environmental challenges, whether issued by man or by nature. World-wide concern with marine pollution and its effects, manifested in international study groups and symposia, underscores the need for understanding the nature of such multiple stress.

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Table 2.—Mercury—Effect on marine animals.

Organism	Exposure period	Concentration (ppb)	Oxygen consumption	Osmoregulation	Enzyme activity	Other
Crassostrea virginica American oyster						
(eggs)	48 hr					LC_{50} = 5.6 ppb (2)[1]
(larvae)	12 days					LC_{50} = 12.0 ppb (u)[2]
Mercenaria mercenaria Hard clam						
(eggs)	48 hr					LC_{50} = 4.8 ppb (3)
(larvae)	10 days					LC_{50} = 14.7 ppb (u)
Argopecten irradians						
(juveniles) Bay scallop	96 hr					LC_{50} = 89.0 ppb; significant Hg uptake (16)
Homarus americanus American lobster	30 days	6	No effect (22)	No effect (22)	Some enzyme induction, some loss of ligand sensitivity with chloride salt; no effect with nitrate salt (heart, antennal gland, gonad) (22, u)	Significant Hg uptake in digestive gland, gills and tail muscle (22)
	60 days	6	No effect (22)	No effect (22)		
Tautogolabrus adspersus Cunner	30 days	5	Elevated (u)			
Morone saxatilis	30 days	5	Depressed (7)		No effect (liver) (7)	
Striped bass	60 days	10	Depressed (7)		No effect (liver) (7)	
	90 days	10	No significant effect (7)		No effect (liver) (7)	
Pseudopleuronectes americanus Winter flounder	60 days	5-10	Elevated (4)		Some enzyme induction, some loss of ligand sensitivity, increased shunt activity (kidney, liver, heart, gonad) (8)	Some blood changes; Hg uptake in blood and gills (4)

[1]Numbers in parentheses refer to citations in Literature Cited.
[2]u means unpublished.

Table 3.—Silver—Effect on marine animals.

Organism	Exposure period	Concentration (ppb)	Oxygen consumption	Osmoregulation	Enzyme activity	Other
Crassostrea virginica American oyster						
(eggs)	48 hr					LC_{50} = 5.8 ppb (2)[1]
(larvae)	12 days					LC_{50} = 25.0 ppb (u)[2]
	96 hr	100	Elevated (21)			Ag uptake by gills (21)
Mercenaria mercenaria Hard clam						
(eggs)	48 hr					LC_{50} = 21.0 ppb (3)
(larvae)	10 days					LC_{50} = 32.4 ppb (u)
	96 hr	100	Elevated (21)			Ag uptake by gills (21)
Argopecten irradians Bay scallop						
(juveniles)	96 hr	22	Elevated (16)			LC_{50} = 33.0 ppb; significant Ag uptake (16)
Spisula solidissima Surf clam						
(larvae)	2-15 days	50	Elevated (20)			
(juveniles)	96 hr	10	Elevated (20)			100 ppb lethal (20)
	96 hr	50	Elevated (20)			Increased valve movement; Ag uptake by gills (20)
Mytilus edulis Blue mussel	96 hr	100	Elevated (21)			Ag uptake by gills (21)
Mya arenaria Soft-shell clam	96 hr	100	Elevated (21)			Ag uptake by gills (21)
C. virginica, M. mercenaria, M. edulis, Placopecten magellanicus	30, 60, 90 days	10	Elevated (u)			
Nassarius obsoletus Mud snail	72 hr	500	Depressed (13)			Distressed behavior (13)
Homarus americanus American lobster	30 days	6	No effect (u)	No effect (u)	Depressed transaminase (heart), some loss of ligand sensitivity (antennal gland), enzyme induction (gonad); all effects small (u)	
Tautogolabrus adspersus Cunner	96 hr	120-500	Depressed (23)	No effect (23)	Depressed shunt activity (liver), changed ligand sensitivity (skeletal muscle) (11)	
Pseudopleuronectes americanus Winter flounder	60 days	10	No effect (u)		Depressed transaminase (liver), very little effect (kidney, heart, gonad) (u)	

[1]Numbers in parentheses refer to citations in Literature Cited.
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