Lead in Fish- Aquatic Life
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was the blood, which retained a relatively high and fairly constant concentration of mercury throughout the experimental period (4 weeks)."[11] The mercury in blood is apparently attached rather firmly to protein. It is possible that mercury, in *Maia*, exerts its poisoning effect by interfering with mechanisms for copper metabolism.

Copper

Copper is naturally present in sea water in various amounts depending on location, proximity to industrial effluents, currents, and unknown factors. Table 5-II summarizes the sources of the available data.[6, 7]

TABLE 5-II
AMOUNT OF COPPER IN SEA WATER

Location	*Copper Concentration (in mg/liter)*	*Reference No.*
Roscoff, France	1	7
Winfrith, England	2.9 – 1.0 mg/cu m	35
Woods Hole, Mass.	8 – 35	18
Long Island Sound	0 – 95	18
Bahamas	1 – 8	18
Gulf of Mexico	1 – 25	38
Friday Harbor, Wash.	1 – 2	8, 9
Japan	0.8 – 2.5	29, 30
Plymouth, England	1.5 – 25	2

Under conditions listed in Table 5-II, marine organisms thrive and carry on the usual life processes. It is evident that small amounts of copper are nontoxic to marine animals. In fact, small amounts of copper are essential for the production of certain respiratory pigments in animals.

There is a threshold of toxicity for copper. Experiments using *Nereis virens* as the test organism suggest that such a threshold for copper toxicity is about 0.1 mg copper per liter. Even at very high concentrations and for long exposure periods, a few individual animals resist the lethal action of the metal.

Nereis abstracts copper from the water surrounding it and concentrates the metal in the body wall and in the gut wall (Fig. 5.1). The rate of uptake is reported to be directly proportional to the concentration of copper in the water. For example, if *Nereis* is exposed to a 0.2-ppm solution of copper in sea water for four days, there is a steady increase in the copper content of body wall and gut; control worms kept in fresh sea water show no change in copper content of the gut but may lose copper from the body wall.[35] Copper in higher concentrations is lethal for *Nereis* (Fig. 5.2).

The shore crab, *Carcinus*, has a copper toxicity threshold of 1 or 2 ppm (11- to 12-day exposure time). It is known that 3 ppm of copper is a lethal concentration for oysters. Lower concentrations have been reported to facilitate the settling of larvae. The work has not been confirmed.[36, 6, 7]

The ninety-six-hour TL*m* for Japanese oysters exposed to copper is reported to be 1.9 ppm.[16] The American oyster is apparently more sensitive to copper.[36] Moreover, if oysters are exposed to a copper concentration as low as 0.13 ppm, they turn green in about twenty-one days.[17] Such oysters are unmarketable, although nontoxic to man. They represent a total economic loss to those depending on their sale for a livelihood.

Lead

Lead poisoning in experimental animals is known to be associated with cellular alterations of erythrocytes: swollen mitochondria, simple and compound vacuoles, clusters of ferritin in the cells. Alterations of the mitochondria have been ascribed to a blockage of heme synthesis.[34]

Detailed examination of the blood in fishes suspected of being exposed to lead poisoning may be of value. Hematological changes could very well be the first biological responses of fishes to threshold

levels of lead. Greatly lacking is detailed background information on blood changes in fishes as a result of stress from physical or chemical agents. Vigorous research to clarify these matters should be productive of much valuable information.

In higher animals and man, exposure to very small amounts of lead is accompanied by changes in blood enzymes.[50] For example, blood serum aldolase activity in human beings exposed to amounts of lead, so small as to elicit no signs or symptoms of poisoning, is increased.

Information on blood enzymes of fishes is scanty. It seems probable that the estimation of serum aldolase and of other enzymes in blood of fishes exposed accidentally and experimentally to lead could yield useful information. Such tests might be particularly revealing in circumstances involving low concentration of lead for prolonged periods of exposure.

In man, it is known that chronic lead poisoning shows a clinical picture much like that of multiple sclerosis.[15] Further, it has been suggested that there is a common pattern of causal mechanisms involved in multiple sclerosis and in the central nervous system damage due to lead poisoning.[15]

Reports of muscular dystrophy occurring naturally in fishes and amphibians are not uncommon.[46] Such reports lead one to speculate whether "naturally occurring" cases of multiple sclerosis and so forth in fishes and amphibians may be quite *unnatural* and are perhaps associated with some metal poisons (e.g., lead) in the environment. When cases of neuromuscular diseases are reported in fishes or amphibians, it would be well to examine the environment thoroughly for lead and other metals which in small amounts over long exposure periods might cause signs of disease aping the pattern of multiple sclerosis, muscular dystrophy, and related phenomena.

Mercury

Mercury in poisonous amounts can enter natural bodies of water from industrial effluents originating in factories which produce mercury-containing compounds or which use mercury in their processes. In addition, runoff from areas where mercurial formulations may have been used as fungicides is a source of pollution.

A number of studies indicate that salts of mercury alter markedly the epithelium of skin and gills in fishes.[30] Fishes immersed in solutions of mercuric chloride and of phenyl-mercuric acetate accumulate mercury in the body.

Curves of concentration and survival time of mercuric chloride for several species of fishes show that the relationship is linear on double logarithmic paper and that there is a break point for each species. Above the break point (higher concentration), the slope of the line is significantly less than below (lower concentration).

The equations for the lines above and below the break point all are of the following general form

$$\log T = a \log C + \log K$$

where T is survival time in minutes, a the slope of the line, C the concentration of mercury in mg per liter, and K a constant. In direct terms, the relationship has the form

$$T = K \cdot C^{a}$$

The exponent a will be different above and below the break point for each species. Below the break point (lower concentrations), a is remarkably constant for all species studied (Table 5-III).

Above the break point, it is also quite constant except for *Tilapia*. The break

TABLE 5-III

Values for slopes and break points for fishes exposed to $HgCl_2$. General equation is log T = a log C + log K.[5]

Species	Slope		Break Point (mg Hg/liter)
	Below Break	Above Break	
Tilapia	−.90	−.20	18
Eupomotis	−.94	−.43	30
Ambloplites	−1.29	−.42	30
Salmo	−1.13	−.38	10
Lebistes	−1.09	−.38	14
Gasterosteus	−1.11	−.40	10
Mean	−1.08	−.37	19

point falls between 10 and 30 mg Hg per liter.

Elevated temperature increases the toxicity of $HgCl_2$ to fishes. Larger fishes survive longer in a given concentration of $HgCl_2$ than do smaller ones. On double logarithmic paper, a plot of body weight against survival time gives a straight line with positive slope. The following equation describes the relationship

$$T = K \cdot W^{0.27}$$

where T is survival time, W is body weight, and K is a constant.

The following argument is used to support the view that the primary site of toxic action of $HgCl_2$ is the surface of the body and the gills in fishes.[5] The equation for body weight and survival time is rewritten as

$$\frac{1}{T} = K \cdot W^{-0.27}$$

The value of 1 divided by T is then the "rate of death." Respiratory metabolism in cold-blooded animals varies with the body weight taken to the 0.73 power.[20] Oxygen consumption per unit weight, therefore, decreases as

$$O_2 = K \cdot W^{-0.27}$$

It is clear that both the reciprocal of survival time (i.e. the rate of death) and the intensity of respiration decrease according to 0.27 power of the body weight. Both phenomena are consequently proportional to relative body surface $W^{0.73}/W$.

In sea water, mercuric chloride is not so toxic to fishes as in fresh water.

The addition of 10 μg per liter of $HgCl_2$ to water containing developing eggs of *Paracentrotus lividis* brings about a severe disturbance of development; a concentration of 5 μg per liter retards development markedly. The threshold for harmful effects of $HgCl_2$ on developing eggs of *Paracentrotus* is between 2 and 3 μg per liter.[41]

Minamata Disease

A large-scale poisoning occurred in Japan as a result of water polluted with mercury. An industrial plant on the shores of Minamata Bay, Kyushu, Japan, used mercury chloride as a catalyzer in the production of vinyl chloride. Effluent from the factory loaded with waste mercury salt was poured into Minamata Bay. Consumption of fishes caught in the bay was followed by severe illness and even death. (The fatal outcome of this so-called Minamata disease is very high.) The mercury content of organs from man and animals killed by eating fishes and shellfishes from the bay was extremely high. The mercury was especially concentrated in the brain. Fishes and shellfishes caught in the bay had very large amounts of mercury in their tissues.[47]

The effects on man of eating seafood from Minamata Bay were identical with the effects of mercury poisoning. Cats and rats that had been administered organic mercury compounds experimentally exhibited identical clinical signs and pathological responses as did animals that were fed on seafood from Minamata Bay. It is interesting to note that an inorganic mercury salt, originating in an industrial efflu-

ent, was in all probability transformed into an organic mercury compound during its passage through the marine food net. When seafood from the poisoned bay was eaten by man, signs and symptoms (as well as pathological findings) of *organic* mercury intoxication resulted. The process by which the inorganic form of mercury was changed to an organic form is unknown.

This serious epidemic indicates dramatically the havoc which can be wrought through poisoned waters. It also suggests that poisons, as they pass through the biological food net in a body of water, may be modified in such a way as to confuse invesigators who are attempting to identify the source and nature of the poison. It is important to realize that natural bodies of water are living dynamic systems—not dead or lifeless "sinks" into which any sort of waste may be discarded with impunity.

Metal wastes are particularly insidious because they can persist in solution for long periods of time.[51] Moreover, if precipitated from solution, through the action of organic sewage, heavy metals can form a poisonous blanket on the bottom of the body of water. Finally inorganic forms of metals may, in passing through the aquatic food web, be converted into organometal compounds or complexes with altered biological effects.

Silver

Silver is well known to be poisonous to aquatic animals. For example, a concentration of 400 μg per liter will kill 90 percent of tested adult *Balanus balanoides* in forty-eight hours.[10] Concentrations of $AgNO_3$ from 10 to 100 μg per liter cause abnormal or inhibited development of eggs of *Paracentrotus*. Even at a concentration of 2 μg of $AgNO_3$ per liter of water, there is a delay in development and deformation of the resulting plutei.[41] The threshold for effect is slightly below 0.25 μg of $AgNO_3$ per liter. The adverse effects of short exposure to silver in water persist for several days after the toxicant is removed from the medium.

The developing eggs of *Arbacia* are somewhat more resistant to silver in solution than are those of *Paracentrotus*. The threshold concentration for effect of $AgNO_3$ on development in *Arbacia* is in the vicinity of 0.5 μg per liter.

Copper also exerts an adverse effect on development of *Paracentrotus* eggs. With silver, copper acts additively on development, not synergistically.

Silver on a comparative basis is about eighty times as toxic as zinc, twenty times copper, and ten times mercury as evaluated with respect to effect on developing echinoderm eggs.[41] Because of its great toxicity, silver may play an important role in the ecology of plankton.

The fact that silver and mercury, in sublethal concentrations, exert profound effects on developing eggs is additional evidence of the importance of assessing such sublethal responses in water pollution studies. The direct killing action of a water contaminant is, of course, important. However, it is not all-important. Sublethal effects which involve disruption of normal embryology or of nutrition, for example, may be of even greater importance since the future of an aquatic species depends on its capacity to reproduce, to feed, to grow, to establish itself in a location. A toxicant which interferes with any of these processes is deleterious and not desirable in a body of water.

As knowledge of water pollution becomes more complete, it may develop that the sublethal effects of pollutants are more critical in the long view than are the acute lethal effects. This possibility should be kept in mind in the planning of future re-