Protective Action of Selenium Against Mercury in Northern Creek Chubs

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Large quantities of selenium as well as mercury were found in fishes, shell fishes, cats and humans in the Minamata Bay area of Japan in 1960 (IWATA et al. 1973). GANTHER et al. (1972) found that low-Hg tuna contained less selenium than high-Hg tuna. Despite high-Hg concentrations, tuna added to methyl mercury treated foods of Japanese quail and rats protected these animals. The investigators hypothesized that selenium in the tuna was antagonistic toward the damaging actions of methyl mercury. KOEMAN et al. (1973) found that marine mammals with high mercury burdens also contained high quantities of selenium. This suggested that the selenium protected the mammals against the toxic action of mercury compounds and that the two elements coaccumulate.

As early as 1967 PARIZEK and OSTADALOVA had demonstrated selenium's protective action against mercury in rats. Since that time several studies have been published concerning this interaction. IWATA et al. (1973) suggested that selenium might promote the elimination of methyl mercury from tissues. This is contrary to most other studies which indicate that selenium actually promotes the accumulation of mercury in the organisms. POTTER and MATRONE (1974) felt that adequate evidence was available to show that the protection afforded by selenium is not due to increased mercury excretion. The mechanism of this protection remains an enigma. While many laboratory studies have been published concerning this situation in birds and mammals, there is a paucity of information concerning mercury and selenium interactions in fish.

HUCKABEE (1974) reported that selenium and mercury mixtures were more toxic to carp eggs than mercury alone. Is selenium protective against mercury at this phylogenetic level? Does it promote mercury retention or lead to a decrease in tissue mercury? It was the purpose of this study to begin to investigate these questions.

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METHODS AND MATERIALS

Northern creek chubs (Semotilus atromaculatus) were collected from small streams around Vermillion, South Dakota. Fish between 3.5-5 cm were used. Each group of 20 was placed in approximately 14 liters of demineralized water (about 10,000 ohms) to which 50 ppm calcium carbonate was added. The temperature was maintained at 24-25°C. The aquaria were oxygenated and lined with polyethylene sheets.

The test substances were Hg given as mercuric chloride and selenium as selenium dioxide. Exposures to test substances were made for 48 hours; the fish were checked for mortality at eight hour intervals. Fish were not fed during pre-treatment or test periods. At the end of the test periods survivors were collected, rinsed, frozen and stored in plastic bags.

Later, they were taken to constant weight at 100°C. They were digested in mixtures of nitric and sulfuric acid with vanadium pentoxide as a catalyst (DEITZ et al. 1973). A Perkin-Elmer model 360 Atomic Absorption Spectrophotometer was used for the flameless analysis.

This method yielded excellent recovery from spiked samples. Typical recovery data from a composite fish sample spiked with one microgram of Hg was 100.09% $\pm$ 2.65% ($\bar{X} \pm$ S.D., N = 9).

RESULTS AND DISCUSSION

Creek chubs pretreated (48 hrs.) with Se (3 ppm) have lower mortality rates than those subjected to identical mercury concentrations for 48 hours without pretreatment (Fig. 1). All of the fish that died were dead by 32 hours of exposure. Consequently figure one represents the death rate for three time periods (32, 40 and 48 hours) of mercury exposure.

Selenium pretreatment at 3 ppm had no obvious effects upon the fish. In preliminary experiments we found that higher concentrations ($\geq$12 ppm) cause heavy mucous accumulations on the gills and death. This selenium pretreatment (3 ppm) while close to fatal levels, apparently protected the organisms from subsequent mercury exposure. Was this protection promoted because selenium prevents mercury accumulation?

When we compare the whole body mercury accumulations (dry weight) of the Se pretreated fish with

those not pretreated, we obtain a rather complicated pattern (Fig. 2).

At relatively low mercury concentrations (0.01, 0.04 and 0.07 ppm) Se pretreatment appears to favor the accumulation of Hg. At 0.07 micrograms/ml the mean mercury burden in the pretreated animals was 24.47 ppm, in the untreated only 18.60 ppm. However, none of these pretreated animals died despite their high mercury burdens while 30% (Fig. 1) of the untreated animals were dead. The protective action of selenium probably does not reside in its ability to prevent accumulation of total body mercury. We noted that slightly higher levels of Se pretreatment caused copious mucus secretion. Mercury is readily absorbed by mucus (USHA et al. 1975). Perhaps the higher levels of whole body mercury in the pretreated animals reflect the accumulation of that element in the skin, a comparatively harmless location. Of course, the co-accumulation of mercury and selenium is well known (GANTHER et al. 1972, KOEMAN et al. 1973, and KOSTA et al. 1975) in other organisms and our data is consistent with that of most other studies.

However, at the more lethal levels of mercury exposure (0.1, 0.13, and 0.16 ppm) a new trend is observed (Fig. 2). Selenium pretreated animals have lower body burdens than those not pretreated. IWATA et al. (1973) found that sodium selenite significantly decreased retention of methylmercury in various organs. We are unable to find published studies on inorganic mercury accumulations in fish with which to compare our results.

We chose inorganic mercury because mercurials other than methylmercury probably predominate in stream and lake water and sediments. It was known as early as 1967 (HANNERZ) that fish can concentrate inorganic mercury directly from water without the involvement of a food chain. In this study we plotted micrograms of Hg/gram of surviving fish (wet weight) against micrograms of Hg/ml of water and found a good correlation ($Y = 0.63 + 32 \cdot X$, $r = 0.87$. $P < 0.002$), indicating a concentration factor of about 32 times in the 48 hour test period.

Under environmental conditions Hg and Se and the complex products of their interactions would simultaneously assault a fish. The results might be very different from those observed in this study. In a study of that type, HUCKABEE and GRIFFITH (1974) found a synergistic effect of these two elements on

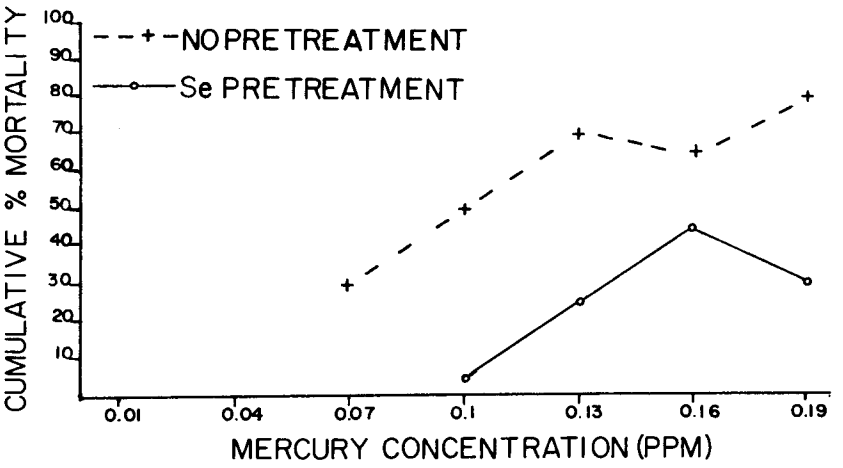

Fig. 1. The percent mortality of creek chubs subjected to various concentrations of $HgCl_2$ with and without Se pretreatment. Twenty animals were utilized in each of the two groups at each concentration.

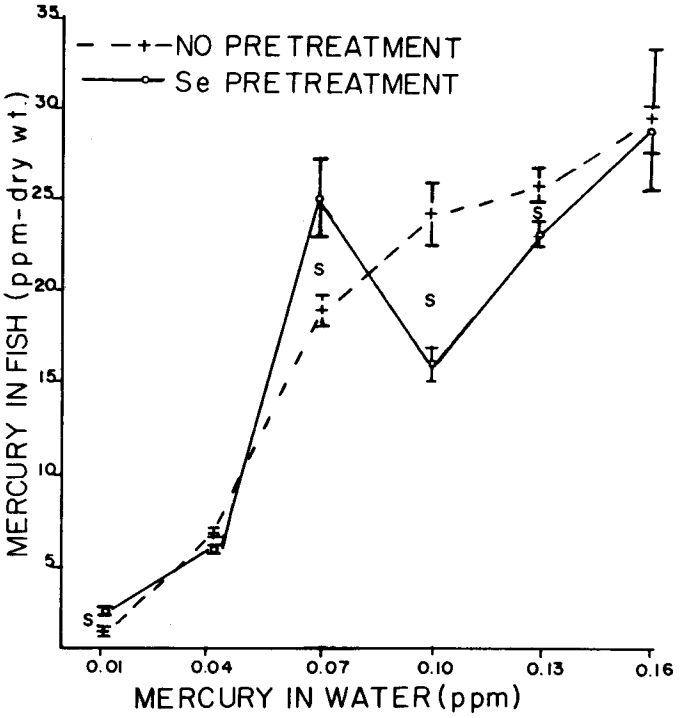

Fig. 2. Comparison of accumulation of Hg (dry weight basis) in creek chub survivors subjected to various concentrations of Hg with and without Se pretreatment. Points are means ± SE. An S indicates a significant difference between groups by Student's test ($P<0.05$).

carp eggs.

SUMMARY

We found selenium dioxide an effective antagonist to the toxic effects of mercuric chloride in the northern creek chub (Semotilus atromaculatus). Selenium pretreatment increased the whole body accumulation of mercury at lower environmental Hg concentrations. It decreased mercury accumulation in survivors at higher, acutely toxic concentrations.

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