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SUMMARY OF THE CADMIUM DETOXIFICATION/TOXIFICATION EXPERIMENT

The problem faced by investigators concerned with the impact of discharges on the environment is that most currently used indicators of the biological impact of contaminants may be as responsive to natural stresses as they are to contaminant stresses. In addition, these indicators do not distinguish between impacts of discharges caused by contaminants and those caused by the physical or nutritional characteristics of the effluent. Bioaccumulation of contaminants does not necessarily mean there will be toxic effects, because organisms can internally detoxify contaminants. The purpose of the research described here was to use this knowledge to develop a contaminant-specific assay of toxicity based upon a determination of the partitioning of contaminants between internal sites of detoxification and sites of toxic action. The cadmium detoxification/toxification experiment described in this and the following three papers is the first in a series funded by the Environmental Protection Agency to determine the efficacy of using detoxification/toxification assays for assessing the toxic impact of Cd, Cu, PCBs, and PCBols.

Previous studies have indicated that metal and organic contaminants will not cause severe toxic effects in organisms until these exceed the capacity of detoxification systems, including metallothionein (MT) and gluthathione (GSH), and spill over into sites of toxic action, including enzymes (ENZ) (Winge et al. 1973; Reid and Krishna 1973; Brown and Parsons 1978). This spillover of contaminants was proposed as being either rate/equilibrium or accumulation dependent (Brown et al. 1977; Brown and Parsons 1978; Brown and Chatel 1978). Most studies which related toxicity to spillover used acute or subacute exposures which resulted in severe effects, including growth reduction (Brown and Parsons 1978) and tissue necrosis (Reid and Krishna 1973). In the present experiment we chose to conduct low sublethal exposures so that we could 1) determine the binding capacity of detoxification systems under more realistic exposure conditions; 2) determine the efficiency of

detoxification systems; 3) determine if the partitioning of contaminants between the ENZ, MT, and GSH cytosolic pools is an accurate indicator of toxic effects; 4) employ more sensitive indicators of toxic effects than were used in previous detoxification/toxification studies; 5) determine the relative sensitivity of tissues to toxicity.

In this experiment scorpionfish were captured at Anacapa Island, transferred to holding tanks at SCCWRP, acclimated for 4 weeks, and then exposed to 0.1- and 1.0-mg Cd/L seawater with samples taken at 4, 8, and 16 weeks. Tissues examined included livers, kidneys, intestines, and gills. Parameters measured included partitioning of Cd, Cu, and Zn between the cytosolic ENZ, MT, and GSH pools and the mitochondrial/microsomal pellet; activities of the enzymes catalase, glutathione peroxidase, and alkaline phosphatase; histological changes; and blood chemistry.

Results indicated that, although the major portion of Cd accumulated in all tissues was bound to MT, this detoxification system was not 100% efficient. With increasing tissue Cd levels, the amount of Cd which was detoxified by MT increased, but the amount in the ENZ pool, although substantially smaller, also increased (Bay et al., this volume (a)). However, in no case did the amount of Cd in the ENZ pool approach levels found in our previous acute Cd exposure (Figure 1). In addition, although subtle toxic effects were sometimes noted (Bay et al., this volume (b)), they were not considered to be nearly so severe as those found in the acute Cd exposures, where severe tissue pathology was noted (Brown et al. 1984). Thus, in accordance with the hypothesis forming the basis for the present research, the severity of effects may increase as ENZ-Cd increases.

We conclude that exposure levels in this experiment were large enough to result in equilibrium-related increases of ENZ-Cd with increase of MT-Cd. However, the pattern of ENZ-Cd changes indicated that the rate of influx of Cd into tissues did not exceed the rate at which MT could be synthesized. Thus, although the exposure concentrations of 0.1 and 1.0 mg Cd/L were about 2500 and 25,000 times southern California coastal seawater concentrations, the detoxification capacity of scorpionfish was not exceeded. Interpretation of these data in terms of seawater Cd concentration must be made with caution, however, since Cd bioaccumulation appears to be determined by various factors in addition to exposure level. For example, scorpionfish collected from Cortes Bank (i.e., exposed to natural seawater Cd levels) had liver MT-Cd levels of 19.7 ± 8.9 mg/kg (mean ± 1 standard error; n = 6), almost twice the highest values found in the laboratory exposures (1.0 mg Cd/L, 8 weeks: MT-Cd = 10.8 ± 1.0, (mean ± 1 standard error; n = 7)) (data from Bay et al., this volume (a), and Brown et al., this volume). Most likely, the degree of spillover was not great enough to produce clearer effects. For instance, examination of Figure 1 reveals

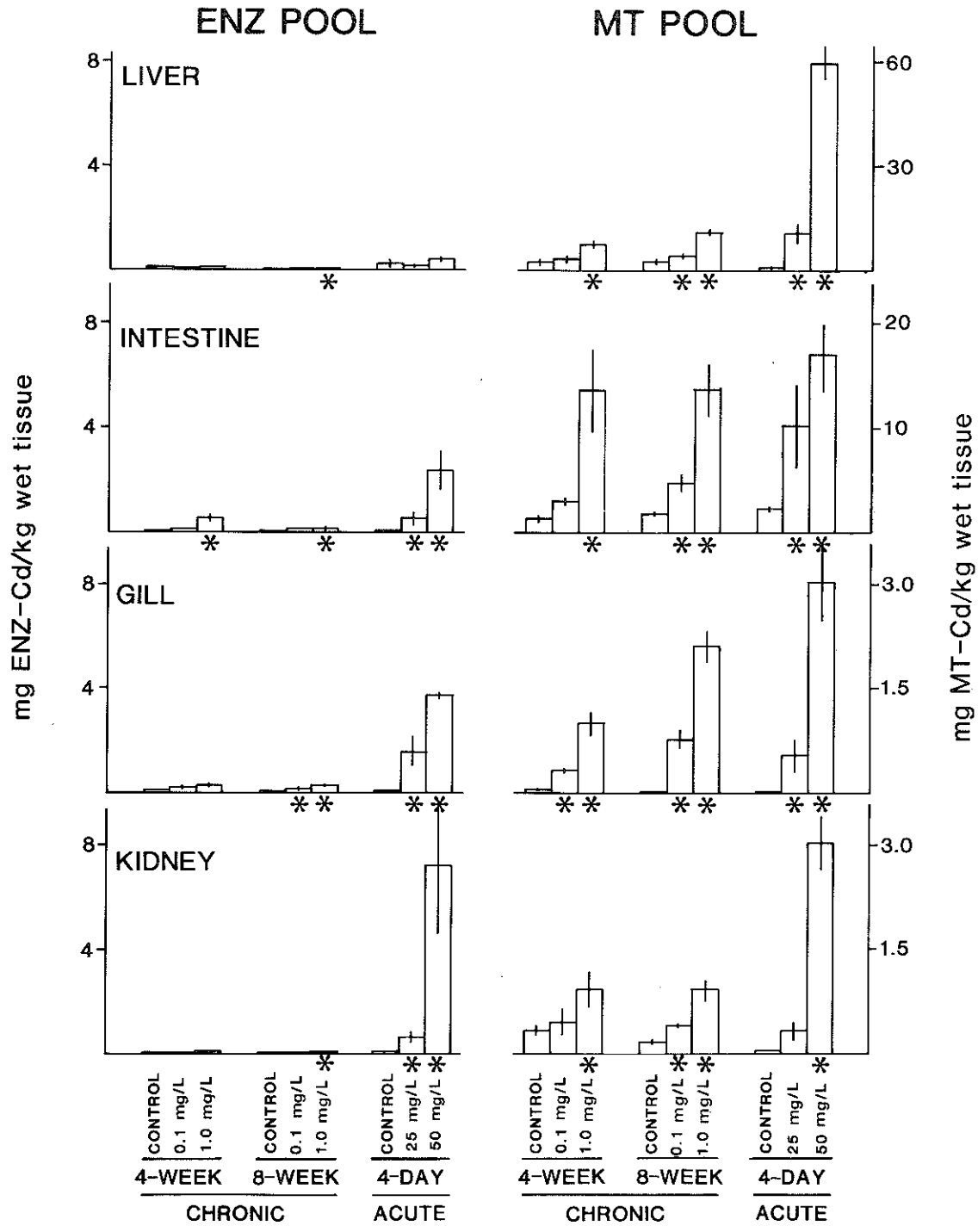

Figure 1. The amount of Cd that occurred in the ENZ pool as a result of the 4- and 8-week chronic exposures was much less than that resulting from the higher concentration 4-day acute exposures. Tissue-level pathology was evident only in the acute-exposure fish. Enzyme activity changes were observed in some chronically exposed fish. In the intestine and gills, the amount of MT-Cd following chronic exposure approached that resulting from acute exposure.

that, although the amount of spillover at the 1.0-mg Cd/L exposure level was significantly higher than control values, it was far less than that which produced severe pathology in the acute exposures (Brown et al. 1984). The main problem encountered in this experiment was that the occurrences of biological effects were so infrequent that correlations between spillover and biological effects could not be determined. Therefore, this experiment is being continued with a range of higher exposures, which should provide us with a more complete range of ENZ-Cd values and biological effects.

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