

SELENIUM FROM OCEAN FISH PREVENTS METHYLMERCURY TOXICITY

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Supplemental selenium (Se) has been known to counteract mercury (Hg) toxicity since 1967, but the mechanisms of this protective effect have only recently become clear. Methylmercury (MeHg) is a highly specific irreversible inhibitor of Se-dependent enzymes (Se-enzymes). Supplemental dietary Se replenishes Se lost to MeHg binding, thereby maintaining normal Se-enzyme activities. Our prior animal studies have shown that the normal range of dietary Se (as sodium selenite) is effective in preventing and reversing MeHg toxicity. Since ocean fish are among the richest sources of dietary Se, we hypothesized that their Se contents would protect against the adverse effects otherwise associated with MeHg exposures from seafood consumption. In the current study, 120 weanling male Long Evans rats were fed diets containing either low or high MeHg (0.5 or 50 nmol MeHg/g). These diets were augmented with either sodium selenite (~0.1, 1.0, or 10 nmol Se/g) or Se from delipidated protein isolates from bigeye tuna, swordfish, or mako shark (3.5, 2.3, and 2.1 nmol/g respectively). Diets were prepared with torula yeast protein or Se from delipidated fish protein isolates added as 10% of the total diet in place of an equivalent amount of torula yeast protein; (2 Hg levels x 6 Se diets = 12 dietary treatments, 10 rats per group). Contributions of additional MeHg in the tuna, swordfish, and shark supplemented diets were (1.6, 2.3, and 3.6 nmol MeHg/g respectively). Rats were fed low Se torula yeast based diets for 5 weeks to deplete their tissue Se reserves. Rats were then switched to their assigned dietary treatments for the duration of the study. Rats fed high MeHg, low Se diets showed growth inhibition after 4 weeks, and hind limb crossing after 9 weeks. Rats fed all other dietary treatments grew normally and did not show symptoms of MeHg toxicity. MeHg from fish did not worsen MeHg toxicity as would typically have been expected. Instead, the Se from the fish was effective in preventing MeHg toxicity.