Effects of Selenomethionine on Methylmercury-induced Toxicity in Mitochondria isolated from Liver Slices

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Methylmercury (MeHg) is an environmental toxicant and can cause cell injury by different cellular mechanisms, including calcium imbalance, free radical generation and thiol depletion. Selenium is an essential dietary nutrient with antioxidant properties and many selenium compounds have been used to reduce oxidative damage in cells. Thus, the objective of this study was to evaluate the effect of Selenomethionine on Hydrogen Peroxide generation and thiol oxidation in mitochondria isolated from rat liver slices after the exposure to MeHg or MeHg–Cys complex. Fifty slices were pre-incubated with Selenomethionine (SeMet, 25 µM) for 15 minutes at 37°C. After, MeHg (25 µM), Cysteine (Cys, 25µM) or MeHg-Cys complex (25 µM each) were added and the slices were incubated for more 30 minutes. The reduced sulphydryl groups were measured by Ellman method and Hydrogen Peroxide generation by Amplex Red and Horseradish Peroxidase. Our data show that MeHg caused a marked increase in thiol oxidation rate and that this effect was more pronounced in the mitochondria isolated from slices exposed to MeHg-Cys complex. These effects induced by both forms of mercury were avoided by SeMet pre-treatment. In the Hydrogen Peroxide generation, SeMet pre-treatment was effective in preventing formation caused by MeHg. Indeed, it was observed that the Hydrogen Peroxide production increased in all groups after calcium addition. In summary, our results demonstrate that SeMet provides partial protection against the toxic effects of MeHg and MeHg–Cys complex. However, additional studies to determine the precise mechanisms involved in this effect are needed.

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