ANTAGONIC EFFECTS OF ETHANOL PLUS METHYLMERCURY IN RAT HEART AND STRIATED MUSCLE

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We have reported that methylmercury (MeHg) elicits central nervous system (CNS) oxidative damage in rats exposed to it in utero and during lactation. However, the combination of MeHg plus ethanol (EtOH) was able to reverse this damage. To further investigate this effect we measured the levels of lipid peroxidation (assayed as thiobarbituric acid reactive substances – TBA-RS) in several tissues from rats on the 60th postnatal day after in utero and lactational exposure to MeHg, EtOH, or their combination. We also investigated if an extract of *Cipura paludosa* Aubl. could protect the tissues from damage by MeHg and EtOH. Each group was treated with 1, 10 or 100 mg/mL of the *C. paludosa* extract or saline. Our results showed increased TBA-RS levels in the heart of rats treated with EtOH plus MeHg (8.130±1.127 versus 2.435±0.1563 µM/mg protein), and in the kidneys of animals treated with MeHg (1.463±0.1399 µM/mg protein) or EtOH 2.052±0.1968 µM/mg protein) when compared with the respective control group treated with saline (0.7377 ± 0.1793). Treatment with MeHg alone or in combination with EtOH caused a decrease in lipid peroxidation in skeletal muscle, when compared with the control group. The *C. paludosa* ethanolic extract presented a dose dependent protective effect against lipid peroxidation in most tissues. Our results indicate that MeHg in combination with EtOH causes oxidative stress in the heart while decreasing oxidative damage in skeletal muscle. These antagonic effects may be due to the different metabolic functions of these organs.

Keywords: *Cipura paludosa*, ethanol, methylmercury, oxidative stress.
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