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Niacin prevents mitochondrial oxidative stress caused by sub-chronic exposure to methylmercury.

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Abstract

Humans and animals can be exposed to different chemical forms of mercury (Hg) in the environment. For example, methylmercury (MeHg)-contaminated fish is part of the basic diet of the riparian population in the Brazilian Amazon Basin, which leads to high total blood and plasma Hg levels in people living therein. Hg induces toxic effects mainly through oxidative stress. Different compounds have been used to prevent the damage caused by MeHg-induced reactive oxygen species (ROS). This study aims to investigate the in vivo effects of sub-chronic exposure to low MeHg levels on the mitochondrial oxidative status and to evaluate the niacin protective effect against MeHg-induced oxidative stress. For this purpose, Male Wistar rats were divided into four groups: control group, treated with drinking water on a daily basis; group exposed to MeHg at a dose of 100 µg/kg/day; group that received niacin at a dose of 50 mg/kg/day in drinking water, with drinking water being administered by gavage; group that received niacin at a dose of 50 mg/kg/day in drinking water as well as MeHg at a dose of 100 µg/kg/day. After 12 weeks, the rats, which weighed 500-550 g, were sacrificed, and their liver mitochondria were isolated by standard differential centrifugation. Sub-chronic exposure to MeHg (100 µg/kg/day for 12 weeks) led to mitochondrial swelling ($p < 0.05$) and induced ROS overproduction as determined by increased DFCH oxidation ($p < 0.05$), increased gluthatione oxidation ($p < 0.05$), and reduced protein thiol content ($p < 0.05$). In contrast,

niacin supplementation inhibited oxidative stress, which counteracted and minimized the toxic MeHg effects on mitochondria.

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KEYWORDS: Methylmercury; mitochondria; niacin; protective effect; sub-chronic

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