Hepatic and neurobiological effects of foetal and breastfeeding and adulthood exposure to methylmercury in Wistar rats

Helen Tais da Rosa-Silva; Alana Castro Panzenhagen; Victória Schmidtt; Alexsander Alves Teixeira; Pedro Espitia-Pérez; Álvaro de Oliveira Franco; Moara Mingori; José F. Torres-Ávila; Carlos Eduardo Schnorr; Paolla Rissi Silva Hermann; Diogo Pompéu Moraes; Roberto Farina Almeida; José Cláudio Fonseca Moreira.

Abstract Methylmercury (MeHg) is an organic bioaccumulated mercury derivative that strongly affects the environment and represents a public health problem primarily to riparian communities in South America. Our objective was to investigate the hepatic and neurological effects of MeHg exposure during the phases foetal and breast-feeding and adult in Wistar rats. Wistar rats (n = 10) were divided into 3 groups. Control group received mineral oil; The simple exposure (SE) group was exposed only in adulthood (0.5 mg/kg/day); and double exposure (DE) was pre-exposed to MeHg 0.5 mg/kg/day during pregnancy and breastfeeding (±40 days) and re-exposed to MeHg for 45 days from day 100. After, we evaluated possible abnormalities. Behavioral and biochemical parameters in liver and occipital cortex (CO), markers of liver injury, redox and AKT/GSK3β/mTOR signaling pathway. Our results showed that both groups treated with MeHg presented significant alterations, such as decreased locomotion and exploration and impaired visuospatial perception. The rats exposed to MeHg showed severe liver damage and increased hepatic glycogen concentration. The MeHg groups showed significant impairment in redox balance and oxidative damage to liver macromolecules and CO. MeHg upregulated the AKT/GSK3β/mTOR pathway and the phosphorylated form of the Tau protein. In addition, we found a reduction in NeuN and GFAP immunocontent. These results represent the first approach to the hepatotoxic and neural effects of foetal and adult MeHg exposure.

Keywords

Methylmercury; Double exposure; Oxidative stress; Hepatotoxicity; Neurotoxicity; Akt/GSK3β/mTOR pathway