Apocynin prevents oxidative damage caused by chronic exposition of mercury in rats.

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Chronic exposure to low-dose mercury induced vascular and endothelial dysfunction and increases contractile prostanoids production from cyclooxygenase-2. These effects might be attributable, at least partly, to increased oxidative stress. Antioxidant therapy has been shown to effectively prevent, reduce or reverse tissue damage caused by oxidative stress. The aim of this study was to investigate if apocynin (selective inhibitor of NADPH oxidase) treatment can prevent oxidative damage caused by mercury exposition. Four groups of rats were treated for 30 days with: a) Mercury group - HgCl₂ (treated with intramuscular injections of mercury chloride - 1st dose 4.6µg/kg, subsequent dose 0.07 µg/kg/day, i.m. to cover daily loss, according to the model described by Wiggers et al, 2008); b) Control group - Ct (im injection with saline); c) Apocynin group - Apo (treated with 1.5mM of apocynin in drink water) and d) ApoHg group - treated with HgCl₂ (im injection) plus apocynin (1.5mM in drink water). We analyzed plasma levels of lipid peroxidation (TBARS) and thiol groups (SH). Data were analyzed by Analysis of Variance (ANOVA) followed by a Bonferroni test. The HgCl₂-treated group showed increased levels of plasma malondialdehyde (MDA) compared with controls, indicating increased lipid peroxidation. Apocynin treatment (ApoHg) prevented the oxidative damage caused by mercury. The mercury exposition plus co-treatment with apocynin increased levels of SH groups. These results demonstrated for the first time that co-treatment with apocynin prevents oxidative stress caused by chronic exposition to small doses of mercury.

Keywords: Mercury, Chronic intoxication, Apocynin, Oxidative stress