# EFFECT OF ACUTE LEAD EXPOSURE ON THIOREDOXIN REDUCTASE-1 (TrxR1) AND ON OXIDATIVE STRESS PARAMETERS IN KIDNEYS OF RATS

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Oxidative stress is an important molecular mechanism of toxic effects of lead in kidney. TrxR1 is a selenoprotein involved in many celular redox processes. This study evaluated the effect of acute exposure (i.p.) to lead acetate on TrxR1 activity, d-aminolevulinate dehydratase (ALA-D) activity, thiobarbituric acid reactive species (TBARS) levels and antioxidant enzymes activities glutathione peroxidase (GSH-Px) and catalase (CAT) in kidneys. Adult male Wistar rats received a single intraperitoneal injection of 0, 25 or 50 mg/kg b.w. lead acetate and were killed 6, 24 or 48h later. TrxR1 activity increased 6, 24, and 48 h after a single exposure to 25 mg/kg lead acetate in comparison to control (13.8±2.3, 13.4±3.4, and 14.7±6.3 vs. 5.8±1.3, 3.6±0.2, and 9.1±1.5 nmol DTNB/min/mg protein, $p<0.05$, respectively). Exposure to 50 mg/kg lead acetate increased CAT activity after 48h when compared to control (140.1±3.9 vs. 69.4±11.4 k/g protein, $p<0.05$) and decreased ALA-D activity after 6, 24 and 48 h (0.8±0.2, 0.9±0.1, and 0.8±0.2 vs. 2.1±0.2, 2.0±0.2, and 2.3±0.5 nmol PBG/h/mg protein, $p<0.05$). Our results suggest that lead acute exposure increases CAT and TrxR1 activities as a defense response against lead-induced oxidative damage.

Keywords: lead; thioredoxin reductase-1; kidney.
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