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Failure of recovery from lead induced hepatoxicity and disruption of erythrocyte antioxidant defence system in Wistar rats

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ABSTRACT

Lead acetate (PbA) is one of the major environmental contaminants with grave toxicological consequences both in the developing and developed countries. The liver and erythrocyte antioxidant status and markers of oxidative were assessed. Exposure of rats to PbA led to significant decline ($p < 0.05$) in hepatic and erythrocyte glutathione peroxidase (GPx), glutathione S-transferase (GST), catalase (CAT), superoxide dismutase (SOD), and reduced glutathione (GSH) content. Similarly, malondialdehyde (MDA) and H_2O_2 concentrations were significantly ($p < 0.05$) elevated. Histopathology and immunohistology of liver of rats exposed to PbA showed focal areas of necrosis and COX-2 expression after 6 weeks of PbA withdrawal. Taken together, hepatic and erythrocytes antioxidant defence system failed to recover after withdrawal of the exposed PbA for the period of the study. In conclusion, experimental animals exposed to PbA did not recover from hepatotoxicity and disruption of erythrocyte antioxidant defence system via free radical generation and oxidative stress.

1. Introduction

The liver is known to be the highest depot of lead in soft tissues followed by the kidney (Mudipalli, 2007). Tissue distribution of lead in the livers, kidneys, heart, lung, spleen, muscles and bones has recently been reported (Kim and Oh, 2013; de Sousa et al., 2013). Documentation has shown that chronic ingestion of lead leads to a significant decrease in liver enzymic and non-enzymic antioxidant including reduced glutathione (GSH) levels, catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx) with concomitant increase in reactive oxygen species (ROS), Malondialdehyde (MDA) content, generation of superoxide anion ($O_2^{\bullet -}$), hydrogen peroxide (H_2O_2) content (Qiao et al., 2013; Mohammadi et al., 2013; Wang et al., 2013). Similarly, ROS have been reported to play a critical role in both physiological and pathological conditions with resultant increase in DNA damage and

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