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ORIGINAL ARTICLE

Association between subchronic and chronic lead exposure and levels of antioxidants and chemokines

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Abstract
Purpose This study aimed to compare the influence of lead on the non-enzymatic antioxidant defenses and the levels of chemokines in workers subchronically and chronically exposed to lead.
Methods The study population was divided into three groups. The first group consisted of male workers subchronically exposed to lead for 40 ± 3.2 days, while the second group included male workers chronically exposed to lead. The third group was a control group.
Results The levels of uric acid and bilirubin were significantly higher after a subchronic exposure to lead compared to the baseline by 22 and 35 %, respectively. Similarly, the values of total antioxidant capacity (TAC), total oxidant status (TOS), and oxidative stress index (OSI) increased by 15, 50, and 33 %, respectively. At the same time, the levels of thiol groups and albumin decreased by 5 and 8 %, respectively. Additionally, the levels of interleukin-8 (IL-8) and macrophage inflammatory protein-1β (MIP-1β) were significantly higher after a subchronic exposure to lead compared to the baseline by 34 and 20 %, respectively. Moreover, IL-8 level was significantly higher by 40 % in the group of workers chronically exposed to lead than in the control group, while the level of interferon gamma-induced protein-10 (IP-10) was significantly lower by 28 %.
Conclusions Similar to chronic lead exposure, subchronic exposure to lead is associated with elevated blood levels of uric acid and bilirubin in humans. This probably results in increased TAC value despite thiol depletion. However, the compensatory activation of non-enzymatic antioxidant defenses seems to be insufficient to protect against lead-induced oxidative stress, which may be additively enhanced by the pro-inflammatory action of chemokines, especially IL-8.

Keywords Subchronic exposure to lead · Chronic exposure to lead · Non-enzymatic antioxidants · Uric acid · Chemokines

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Introduction

Lead is a heavy metal, which has been widely used for decades in paint, gasoline, water pipes, storage batteries, and many other products (Lin et al. 2015). In the last decades, our view on lead toxicity has changed, giving more concern to exposures, previously considered safe. Lead can induce many adverse health effects on various body systems including the nervous, hematological, immune, and genitourinary systems. Nevertheless, due to its malleability, resistance to corrosion, and low melting point, lead is still widely used in many industries (Wang et al. 2012).

Ingestion and inhalation are the primary routes of lead entering the body. After absorption, lead is distributed through the bloodstream to various organs, such as brain, liver, and kidneys (Alya et al. 2015). It has been proposed that kidneys play an important role in the toxicokinetics of lead because they serve as the major organ responsible for its excretion. Therefore, kidneys are particularly exposed to lead toxicity. Lead primarily impairs the function and