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Lead-induced oxidative stress and role of antioxidants in its management

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INTRODUCTION

Lead induced oxidative stress:

Lead is a persistent and common environmental contaminant. lead damages cellular material and alters cellular genetics. The mechanism all of these toxic metals have in common involves oxidative damage. Toxic metals increase production of free radicals and decrease availability of antioxidant reserves to respond to the resultant damage. Low-level exposures to lead may cause cognitive dysfunction, neurobehavioral disorders, neurological damage, hypertension, and renal impairment. The pathogenesis of lead toxicity is multifactorial, as lead directly interrupts enzyme activation, competitively inhibits trace mineral absorption, binds to sulfhydryl proteins (interrupting structural protein synthesis), alters calcium homeostasis, and lowers the level of available sulfhydryl antioxidant reserves in the body (Ercal *et al.*, 2001).

Mechanisms of lead toxicity: The effect of lead on oxidant/ antioxidant balance :

Lead toxicity leads to free radical damage via two separate, although related, pathways :

- The generation of reactive oxygen species (ROS), including hydroperoxides, singlet oxygen, and hydrogen peroxide and
- The direct depletion of antioxidant reserves (Ercal *et al.*, 2001).

In any biological system where ROS production increases, antioxidant reserves are depleted. In this situation, the negative effects on the human system's ability to deal with increased oxidant stress occur via independent pathways.

Effect of lead toxicity :

Lead binds to glutathione and sulfhydryl-containing enzymes:

One of the effects of lead exposure is on glutathione metabolism. Glutathione is a cysteine-based molecule produced in the interior compartment of the lymphocyte. More than 90 per cent of non-tissue sulphur in the human body is found in the tripeptide glutathione. In addition to acting as an important antioxidant for quenching free radicals, glutathione is a substrate responsible for the metabolism of specific drugs and toxins through glutathione conjugation in the liver (Meister *et al.*, 1983).

The sulfhydryl complex of glutathione also directly binds to toxic metals that have a high affinity for sulfhydryl groups. Lead effectively inactivate the glutathione molecule so it is unavailable as an antioxidant or as a substrate in liver metabolism (Christie and Costa, 1984). Concentrations of glutathione in the blood have been shown to be significantly lower than control levels both in animal studies of lead exposure and in lead-exposed children and adults (Ahamed *et al.*, 2005).

Lead also binds to enzymes that have functional sulfhydryl groups, rendering them non-functional and further contributing to impairment in oxidative balance. Levels of two specific sulfhydrylcontaining enzymes that are inhibited by lead – deltaaminolevulinic acid dehydrogenase (ALAD) and glutathione reductase (GR) – have been demonstrated to be depressed in both animal and human lead-exposure studies (Gurer-Orhan *et al.*, 2004).