PREFACE

Lead is one of the oldest established environmental toxicant and lead poisoning remains a major health problem, particularly in developing countries. Children living near facilities that process Pb, such as smelters, have been found to have unusually high blood Pb levels. Pb- exposure in India is a complex matter and needs to be understood in detail. The range of Pb (lead) exposure sources in India is extensive and as yet not well understood. Key suspected sources of Pb were considered to be water supplies, and tinned eating utensils, paints and auto-exhaust. Pb absorbed by the GIT comes from the intake of the Pb in food, beverages and soil or dust in case of older children and adults and in occupational exposure population mostly from atmospheric air. Pb is particularly toxic to the brain, kidneys, reproductive system, and cardiovascular system. Exposures can cause impairments in intellectual functioning, kidney damage, infertility, miscarriage, and hypertension. Several mechanisms have been proposed for lead induced abnormalities, but have yet been defined clearly. Disruption of variety of biochemical process rather than a single mechanism is responsible for the toxicity. As a result, research on the toxic effects of lead continues and the last decade has been particularly fruitful in providing new information on the manifold influences of this metal.

In adults, Pb exposure has been related to increased blood pressure and hypertension, conditions known to increase the risk of cardiovascular disease. People who have been exposed to higher concentrations of Pb may be at a higher risk for cardiac autonomic dysfunction. The experimental findings that have been reported suggest that Pb acts at multiple sites within the cardiovascular system. Chronic Pb exposure has been linked to serious, sometimes lethal disturbances in cardiac rhythmicity and contractile function.

Lead affects the blood forming system by interfering with several enzymatic steps in the heme synthesis pathway. Specifically, Pb decreases heme biosynthesis by inhibiting δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase activity. Ferrochelatase, which catalyzes the insertion of Fe
into protoporphyrin IX, is quite sensitive to Pb. Lead induces anemia by both interfering with heme biosynthesis and by diminishing red blood cell survival. Pb can reduce the life span of RBC.

Oxidative stress has been suggested to be one of the important mechanism(s) of toxic effects of Pb. Experimental studies have shown that Pb can generate reactive oxygen species (ROS) by depletion of glutathione and protein-bound sulfhydryl groups. Oxidative stress has been implicated in Pb associated tissue injury in the liver, kidneys, brain and other organs. In many organisms there are enzymes and small molecules with antioxidant capabilities that can protect against the adverse effects of ROS reactions.

Nutrient metals play important role in protecting the lead induced toxicity. Nutrient metals like calcium, iron and zinc protect the living organisms from the adverse effects of lead. These metals offer protection against lead by preventing its absorption in the gastrointestinal tract. Deficiency of dietary Ca, Fe and Zn helps in increasing the Pb absorption in gut. Therefore in the present study, an attempt was made to examine the effect of Ca, Fe and Zn nutrient metal mixture in reversing the altered lead sensitive parameters in blood and heart of lead exposed rats.

The author is aware of the fact that the more extensive and indepth studies are required for a thorough understanding of the Pb-toxicity. However, the present study provides additional information on Pb-induced perturbations in cardiovascular system of young and aged rats and the beneficial role of nutrients calcium, iron and zinc in countering lead toxicity.