Exposure to toxic metals is a widespread problem in most industrialized nations. Heavy metals generally interfere with a number of central nervous system (CNS), hematopoietic, hepatic and renal functions. Accumulated evidence shows that young children are more susceptible than adults to the deleterious effects of metals. Over the past few decades there has been growing awareness and concern that toxic biochemical and functional effects are occurring at lower levels of metal exposure than those that produce overt clinical and pathological signs and symptoms.

Pb is a neurotoxin that continues to be considered as a major global environmental health hazard. Pb is found in nature as a divalent cation, mainly forming stable complexes with sulfur. Pb is a heavy metal with no known biological function in humans. On the contrary, it can damage various systems of the body including the hematopoietic, renal and skeletal systems with the central nervous system being its primary target. The susceptibility to Pb toxicity is influenced by several factors such as environmental exposure, age and nutritional status. Human exposure to Pb occurs via food, water, air and soil. Food and water Pb sources include the use of Pb-containing ceramic dishware, metal plumbing, and food cans that contain Pb solder.

Toxic effects of Pb depend on both, the duration of exposure and the magnitude of the dose. Children are particularly sensitive to the deleterious effects of Pb. Pre and peri-natal exposure to Pb result in higher brain metal accumulation than later postnatal exposure due to an under-developed blood–brain barrier in early life.

Oxidative stress has been associated with Pb exposure in humans and in experimental animal models. In humans occupationally exposed to Pb, biomarkers of oxidative stress such as malondialdehyde, GSH status, glutathione peroxidase and catalase, exceeded the mean value of the control population.

Pb is recognized as a risk factor for neurologic and psychiatric disorders. In fact, Pb-induced brain damage occurs preferentially in the prefrontal cerebral cortex, cerebellum and hippocampus. Cognitive functions are localized in cerebral cortex, whereas the cerebellum regulates the execution of motor skills, and the hippocampus, which is the memory storage center, has also been related to behavior. Pb-induced effects on monoamine levels in different areas of the brain rely on various aspects, such as the
exposure level, the duration of exposure, the animal species used, and animal developmental stage at the onset of Pb exposure.

Nutritional status is another significant risk factor for Pb intoxication and its effects. Iron, zinc and calcium deficiencies increase the retention of ingested Pb, which can also increase Pb gastrointestinal absorption and affect the susceptibility to Pb neurotoxicity. The mechanisms underlying Pb neurotoxicity are still a matter of research. 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 the brain of lead exposed rats.

The author is aware of the fact that the more extensive and in-depth studies are required for a thorough understanding of the Pb-toxicity. However, the present study provides additional information on understanding the molecular mechanisms underlying the Pb-neurotoxicity and the relations between the Pb-induced neurochemical events and behavioral alterations in young, adult, aging rats and the beneficial role of nutrients calcium, iron and zinc in counteracting lead toxicity.