Manganese, though an essential trace element in animals, is known to produce harmful effects when its excessive amounts reach in the body tissues. Manganese poisoning mainly occurs in men employed in manganese mines and in factories where manganese is used in huge amounts. The clinical cases of manganese poisoning were reported for the first time by Couper (1837) among the workers engaged in the grinding of manganese dioxide in a factory manufacturing bleaching powder in Scotland. In minor cases, air pollution by manganese in certain limited areas around ferromanganese plants have raised concern in certain countries like USSR and Italy where instances of hazards by air borne manganese have been reported.

The problem of manganese poisoning is alarming and of great concern to all the countries of the world which produce manganese ore and those who use manganese compounds for various industrial purposes. In this context USSR, South America, India, South Africa, Ghana, Cuba and Morocco, which are among the chief countries producing manganese ores, face great risk of manganese poisoning to their workers in manganese mines and factories using manganese compounds.
The problem of manganese poisoning is also of great concern to all concerned with human welfare, public health and industrial hygiene.

The clinical manganese poisoning manifests itself in a chronic disabling condition involving mainly the neurological system. Manganese selectively acts on the nervous system though its concentration in brain was not been found to be higher than other organs of the body.

The experimental studies have shown that manganese produce biochemical lesions in the brain and other body organs. Despite these studies, there is paucity of knowledge on the mechanism and initial site of action, early diagnosis and chemotherapy of manganese poisoning.

The toxicity of manganese, therefore, needs further experimental studies to find out pathogenesis of manganese toxicity, methods for its early diagnosis and chemotherapy.

The present studies were, therefore, conducted with the above consideration to understand the pathogenesis of manganese toxicity and to evolve a possible diagnostic method for manganese poisoning. The studies conducted include histoenzymic and histomorphological studies on kidney and testis and biochemical estimations of serum calcium, inorganic phosphates and alkaline phosphatase together with histomorphology of related endocrine the parathyroid and bone to explain the possible mechanism underlying the biochemical changes observed.
The present studies elucidate the initial site of action and pathogenesis of manganese on the renal and testicular tissue. The mechanism of altered serum calcium, inorganic phosphates and alkaline phosphatase has also been discussed with their possible application in clinical diagnosis of early manganese poisoning.

The present studies will be of great help in future studies on the problems pertaining to toxicity studies on manganese.