INTRODUCTION

It has been known since ancient times that lead may cause poisoning in man. Exposure of lead can take place either through inhalation of dust, fumes, vapors or ingestion of contaminated foods or drinks. Because of its cumulative property it is capable of exerting toxic effects at any level of exposure. Toxic effect of lead on the body is known as Plumbism and it is now well recognized that inorganic lead produces not only clinically defined encephalopathies and neuropathies, but also various behavioral changes indicative of cerebral dysfunction. However, only within the past fifty years attention has been called to its effects in children who show the effects of lead poisoning.

ABSTRACT

It has been known since ancient times that lead is virtually toxic to every organ of body including central nervous system where it may manifest as encephalopathy and hypsomia yet the exact mechanism of these clinical manifestations remains inconclusive. The present study was aimed to see the microscopic changes in the olfactory bulb of mice induced by oral administration of a lead compound in adult albino mice. A total number of 20 adult albino mice of either sex were included in the present study consisting of equal numbers in both control and experimental groups. Experimental group received 4.5% and 5% lead nitrate and lead acetate trihydrate orally for a period of 3 weeks then animals of both groups were euthanized with overdose of general anesthesia and perfused with 10% formalin. Olfactory bulbs were dissected out and processed for paraffin embedding. Sections of 10µ thick were stained with HE and observed under light microscope. The results revealed generalized edema and petechial hemorrhages. Histopathology of the olfactory bulbs revealed edema and congestion with vacuoles of variable sizes almost throughout. Distortion of glomeruli, clumping of periglomerular cells and increasing number of pyknotic cells were also noticed. It was concluded that lead has toxic effects on the central nervous system including olfactory bulb in the form of edema, microscopic hemorrhages and neuronal loss which may explain the clinical manifestations of lead toxicity.

Keywords: Albino mice, Olfactory bulb, Lead nitrate and lead acetate trihydrate, Hypsomia.
Capillaries appeared dilated and congested. Distortion of glomerular contour was obvious. Periglomerular cells were hyperchromatic and showed clumping. Multiple vacuoles of variable sizes were noticed in the outer plexiform layer (Fig. 1 E, F, G and H). Granule cell layer showed loss of cells. Dark and pyknotic nuclei were also present. No such types of abnormalities were found in control group of mice.

![Image](https://example.com/image1.png)

**Figure 1:** Photomicrographs from olfactory bulb of control mice (A, B, C and D) showing typical laminar pattern without edema, congestion or vacuolation while those from experimental group (E, F, G and H) show edema, vacuolation, congestion, loss of glomerular contour and clumping of periglomerular cells. H &E stain, X100 (A &C), X400 (B&D).

**DISCUSSION**

The layers of the olfactory bulb which show damage mainly include the lamina glomerulosa, outer plexiform layer and the granule cell layer. Gross damage in the region of olfactory bulb was also seen in present study in the form of petechial haemorrhage which might have been due to capillary dilatation. These findings are in partial agreement with those reported by certain workers who exposed adult guinea pig to Lead carbonate and reported vascular changes in addition to encephalopathic effects of lead mediated directly at the neuronal level. Some other workers have demonstrated hypertrophy of vascular pericytes. Lead pellets implantation in the mice forebrain produced vascular changes in addition to parenchymal necrosis and spongiosis in the hypothalamus. Histological study of many parts of brain e.g. cerebral cortex, corpus striatum, choroid plexus and cerebellum after lead exposure revealed cerebellum to be most severely damaged. In addition to this study hemorraghes noticed along with damage to molecular and Purkinje cell layers and edema in the granule cell layer which correlated very well with the findings of the present study.

Histopathological findings of olfactory bulb on neuron and neuropil in the present study are to a great extent in agreement with those reporting degeneration of cells in the cerebral cortex and reduced number of Purkinje and granule cells of cerebellum and of hippocampal neurons on lead exposure as well as vacuolations after incubation of guinea pig hippocampus in a lead containing medium which was more pronounced in outer plexiform layer of olfactory bulb. The vascular changes observed in the present study are in agreement with those reported after exposure of lead in dogs which indicates that irrespective of animal species, olfactory bulb is vulnerable to lead acetate toxicity.

**CONCLUSION**

From the above study it was concluded that olfactory bulb is vulnerable to toxicity of lead similar to the other parts of brain and that histopathological changes mainly included edema, vacuolation and congestion, glomerular distortion and pyknotic periglomerular cells.

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**REFERENCES**


