

Effect of Lead Nitrate on Thyroid Function of the Indian Palm Squirrel, *Funambulus pennanti* (Wroughton)

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Some of the known toxic effects of lead in mammals including man are, impaired heme synthesis, anemia, hepatopathy, nephropathy, behavioral disorders and neuropathy (Goyer and Rhyne 1973; Vallee and Ulmer 1972; Silbergeld and Goldberg 1974; Silbergeld 1983; Goyer 1981). However, very little is known about the effect of lead on endocrine physiology. Some data are available on lead induced impairment of thyroid function in occupationally exposed men and experimental rats (Zel'tser 1962; Sandstead 1967; Sandstead et al.1969; Robins et al.1983).

As lead nitrate is largely consumed through water and food, in this study the wild rodent <u>Funambulus pennanti</u> were administered lead through their drinking water and their thyroid structure, radioiodine 131-I percentage uptake and protein bound iodine (PBI) level were assessed.

MATERIALS AND METHODS

Twenty F. pennanti collected from the Banaras Hindu University campus in Varanasi were divided into two equal groups and acclimated to the laboratory conditions for ten days prior to initiation of the experiment. They were fed on water soaked bengal gram, ad-libitum. The experimental group was provided with 500 ppm of lead nitrate in drinking water for ten days. The control group was given chlorine free drinking water. Twenty four hours before sacrificing them on the tenth day, five squirrels from each group were given 5 uCi tracer dose of 131-I intraperitoneally, Percentage uptake of 131-I was assessed. Serum of another five animals from each group was used to record the protein bound iodine (PBI). For histological evaluation, thyroid was fixed in Bouin's fluid for 48 hours, 5 micra paraffin sections were cut and stained in Ehrlich hematoxylin and eosin (Ehrlich 1886).

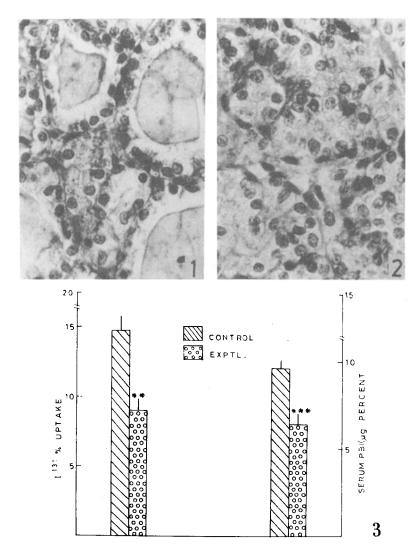


Fig. 1. Histological photograph of control thyroid with low epithelium and colloid in the lumen. X 650.

- Fig. 2. Histological photograph of hypertrophied thyroid of lead nitrate treated animal with no colloid. X 650.
- Fig. 3. Percentage uptake of 131-I in thyroid and serum protein bound iodine (PBI (µg per cent) in the normal and lead exposed F. pennanti. Each column represents the mean of five animals + SEM. ** = P 0.05, *** = P 0.01.

RESULTS AND DISCUSSION

In the control F. pennanti, the follicular epithelium is low and the lumen is filled with colloid (Fig. 1). Exposure to lead nitrate reduced colloid greatly and obliterated lumen. The epithelial cells were hypertrophied and they lost their follicular arrangement (Fig. 2). In experimental animals percentage uptake of 131-I (P<0.05) and PBI (P<0.01) level were significantly reduced (Fig. 3). These data suggest that lead nitrate inhibits thyroid function in F. pennanti.

In several mammals, including man, lead toxicity is shown to reduce thyroidal 131-I untake (Slingerland 1955; Sandstead 1967; Robins et al. 1983). In the present study the decreased 131-I uptake suggests reduction in serum thyroxine level which might have stimulated TSH secretion resulting in the hypertrophy of the thyroid epithelium, Sandstead et al. (1969) have observed decrease in the TSH reserves of lead intoxicated patients which could not be brought back to normal level by TSH administration. Robins et al. (1983) also suggested depression of hypothalamic pituitary axis in lead intoxicated patients. Lead is suggested to interfere with the reactions between iodine and sulfur, probably by combining with the latter which may be responsible for the lowering of 131-I uptake (Sandstead et al.1969). Various enzyme systems involved in this reaction are susceptible to lead toxicity as lead inhibits the activities of enzymes by combining with sulfhydryl (SH) groups (Bair et al. 1956; Vallee and Ulmer 1972). În F. pennanti 500 ppm of lead, administered through drinking water for ten days, is capable of impairing thyroid function.

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REFERENCES

- Bair H, bassler KH, Lang K (1956) Uber wirkungen vor Blei im intermediarstaf wechoel. Arch Exp Path Pharmacol 229:495-504.
- Ehrlich P (1886) Fragekasten Zeitschrift fur Wissenschaflliche Mikroskopic und fur mikroskopische Technik. 3:150.
- Goyer RA, Rhyne BC (1973) Pathological effects of lead. In: Richter GW and Epstein MA·Int Rev Exptl Pathol 12:1-77.
- Goyer RA (1981) Lead. In: Disorders of mineral metabolism. Vol. I Acad. Press. New York pp 159-199.

Robins JM, Cullen MR, Connors BE, Kayne RD (1983)
Depressed thyroid indexes associated with occupational
exposure to inorganic lead. Arch Intern Med 143:220-224
Sandstead HH (1967) Effect of chronic lead intoxication
on in vivo 131-I uptake by the rat thyroid. Proc Soc

Exptl Biol Med 124:18-20.

- Sandstead HH, Stant EG, Brill AB, Arias LI and Terry RT (1969) Lead intoxication and the thyroid. Arch Intern Med 123:632-635.
- Silbergeld EK, Goldberg AM (1974) Lead induced behavioral dysfunction: An animal model of hyperactivity. Exp Neurol 43:146-157.
- Silbergeld EK (1983) Experimental studies of lead neurotoxicity: Implications for mechanisms, dose response and reversibility. In: Rutter M and Russell JR (ed) Lead versus Health. John Wiley and Sons Ltd.pp.191-218.
- Slingerland DW (1955) The influence of various factors on the uptake of iodine by thyroid. J Clin Endocr Metabol 15:131-141.
- Vallee BL, Ulmer DD (1972) biochemical effects of mercury cadmium and lead. Ann Rev Biochem 41:91-128.
- Zel'tser ME (1962) The functional state of thyroid gland in lead poisoning. Tr Inst Kraevoi Pathol Akad Nank. Kaz SSR 10:116-120.

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