

RESEARCH PAPER:

Aluminium toxicity to catecholamines in rat brain

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Asian Journal of Environmental Science (December, 2009 to May, 2010) Vol. 4 No. 2 : 223-230

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SUMMARY

The present study demonstrates toxic effects of aluminium on catecholamines of albino rat brain. LD50/24h for aluminium as per probit method was 700mg/kg body weight. 1/5th of lethal dose was taken as sub-lethal dose. For acute dose studies, rats were given a single lethal dose of aluminium acetate orally for one day only and for chronic dose studies, rats were administered with sub-lethal doses once in a day for 25 days continuously. Various constituents of catecholamines were determined in selected regions of rat brain at selected time intervals and days. The results revealed that the levels of all catecholamines were inhibited differentially in different areas of brain showing region specific response of brain to both modes of exposures to aluminium. However, all these constituents exhibited recovery trend which more pronounced under chronic exposure when compared to acute exposure. Further, these changes in catecholamines were finally manifested in behaviour of rat.

Key words :

Aluminium acetate, Rat brain, Catecholamines, Behavioural changes

Aluminium, the world's 3rd most common element, dispersed in abundance in igneous rocks, shales, clays etc. by virtue of its greatest properties like strength, electrical and thermal conductivity, light and heat reflectivity, delibility and formidability, has an ever increasing number of applications ranging from structural materials to thin packaging foils and electrical transmission appliances. Though dietary aluminium is ubiquitous, in small quantities (30-50mg per day-National Library of Medicine, 2000). It is not a significant source of concern in persons with normal elimination capacity. However, there is the prolonged exposure and increased mortality (Jensen *et al.*, 1998) in mice.

Further, aluminium is also known to exert its toxic effects on the nervous system as well such as degeneration of astrocytes (Suarez-Fernandez, 1999), interfering with the metabolism of the neuronal cytoskeleton encephalopathy in dialysis patients (Morris, 1989) and implicated in a series of neurological diseases such as amyotrophic lateral sclerosis, dementia associated with Parkinson's disease etc. (Altmann, 1999).

In view of the above observations, in the present analysis an attempt has been made to evaluate the toxic effects of aluminium on the catecholamines in the brain of rat subjected to chronic and acute treatment and manifestation of these changes in the behaviour of rat.

MATERIALS AND METHODS

Male albino rats, *Rattus norvegicus*, weighing 130±2 g., 60±2 days age obtained from Sri Venkateswara Enterprises, Bangalore were selected as experimental animals and aluminium acetate as the toxicant. The rats were fed with food pellets (Sri Venkateswara Enterprises, Bangalore) and drinking water *ad libitum*. The animals were housed in polypropylene cages under hygienic conditions with photoperiod of 12 hours light and 12 hours dark.

Parameters studied:

– Toxicity evaluation: Probit method of Finney (1964).

– Aminergic system:

Dopamine, Norepinephrine and Epinephrine (Kari *et al.*, 1978).

All the above biochemical estimations were done under both acute and chronic exposures. For acute exposures, the animals were sacrificed at 1h, 3h, 6h, 12h and 24h intervals after oral administration of a single lethal dose of aluminium acetate and for chronic exposures, the animals were treated with sub-lethal doses of aluminium acetate every day up to 25th day and sacrificed on 5th day, 10th day, 15th day, 20th day and 25th day. After cervical dislocation, the brain was isolated quickly and placed in ice. Different areas of the brain (Fig.1) such as Cerebral

Accepted :
November, 2009