ATPase in rat brain under aluminium toxicity

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SUMMARY

The present study deals with evaluation of the impact of aluminium acetate on the ATPase system in rat brain. Male albino rat, *Rattus norvegicus*, weighing 130±2 grams, of 60±2 days age was the experimental animal model and aluminium acetate, the toxicant. Aluminium toxicity (LD50/24h) was evaluated and was found to be 700mg/kg body weight. 1/5th of lethal dose was taken as the 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, the rats were administered with sub-lethal dose of aluminium acetate once in a day for 25 days continuously. The levels of various constituents of the ATPase system *viz.* Total ATPases, Na^+, K^+ - ATPase and Mg^{+2} ATPase were determined in different regions of rat brain such as Cerebral Cortex (CC), Hippocampus (Hc), Hypothalamus (Ht), Cerebellum (Cb) and Ponsmedulla (Pm) at selected time intervals/days under acute and chronic treatment with aluminium. The results revealed that the levels of all ATPases were inhibited to different extent in all the above areas of brain upon aluminium intoxication thus exhibiting region specific sensitivity of rat brain. While under acute treatment, inhibition in ATPase commenced from 3h, reaching maximum at 12h, under chronic exposure, maximum inhibition was observed on 15th day. Further, in acute treatment, the ATPase system never gained normal levels,where as under chronic exposures, a slight recovery was observed in all ATPases from 20th day onwards and by 25th day almost near normalcy was restored.

Key words : Aluminium acetate, Rat brain; ATPase system, Behavioural changes

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Aluminium, the worlds 3rd most common element is widely dispersed in abundance in igneous rocks, shales, clays etc. By virtue of its greatest properties like strength, corrosion resistance, electrical and thermal conductivity, light and heat reflectivity, delibility and formidability, it 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, the increase in aluminium exposure of the general population becomes an increasing concern as evidence by the research reports demonstrating possible association between aluminium exposure and chromosomal aberrations (Roy *et al*., 1991), carcinogenicity (Bhamra and Costa, 1992), hypochromic anaemia (Ward, 1991), impairments in motor function (Strong and Garruto, 1991), bone diseases (Querles, 1991) and so many behavioural impairments (Connor *et al.*, 1988). Further, aluminium is known to cause deficits in immune effectors cell function (Golub *et al.*, 1993), lower grip strength and increased startle response (Oteiza, 1993), post-weaning neurobehavioural changes (Donald *et al*., 1989) and increased mortality (Jensen, 1998) in mice.

Besides, the above effects on various organs and their functions, 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 (Van der Voet *et al.*, 1999); encephalopathy in dialysis patients (Morris, 1989) and implicated in a series of neurological diseases such as amylotropic lateral sclerosis, dementia associated with Parkinson's disease etc. (Altmann *et al.*, 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 ATPase system in the brain of rat subjected to chronic and acute treatment and manifestation of these changes in the behaviour of rat. The reason behind selecting the ATPase system is that the ATPase system plays multiple roles *viz.* as energy transducers (Takao, 1985), as integral membrane proteins facilitating the movement of solutes across the membranes (Boyer, 1976), excitability of neurons (Bonting, 1970), impairing brain energy balance etc. The