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# BEHAVIOURAL EFFECTS OF MONO SODIUM GLUTAMATE ON ALBINO RATS

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### **ABSTRACT**

Monosodium Glutamate (MSG) and it is sold in most open markets and stores as Ajinomoto. MSG improves the palatability of meals and thus influences the appetite centre positively with it resultant increase in body weight.MSG is toxic to human and experimental animals. It has been reported that MSG has neurotoxic effects resulting in some behavioral effects like locomotor activity, rearing activity, learning behavior, drinking and feeding habit. The aim of the present study is to investigate the role of MSG on behavior of rats. Rats were assigned to four groups. One control group and three treated groups. All the treated groups were supplemented with 75gm/kgb.w. (Low dose), 100gm/kgb.w. (Intermediate dose) and 125gm/kgb.w. (High dose) of MSG for 28 days. Behavioural impacts were examined by looking at changes in activity patterns. Lacrimation was found to be decreased in the high dose group, urination was found to be normal in low dose and intermediate dose treated animals but it was slightly decreased in high dose treated animals, decreased feeding and increased drinking was found with increasing dose of MSG.

Key words: Mono sodium glutamate, Neurotoxic, Behavior.

#### INTRODUCTION

Monosodium glutamate (MSG) is one of the commonest food additives in the developed world and it is a commonly used flavour enhancer. MSG can be found in various concentrations in numerous food products (Walker and Lupien, 2000). MSG is a sodium salt of glutamic acid, a naturally occurring non-essential amino acid with trade names such as Ajinomoto. It is now made mostly from bacterial fermentation (Leung *et al.*, 2003). In its pure form, it appears as a white crystalline powder that, as a salt, dissociates into sodium cations and glutamate anions while dissolving (glutamate is the anionic form of glutamic acid) (Sano, 2009).

Glutamate is a naturally occurring amino acid

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that is one of the most abundant amino acids in the central nervous system (CNS). Glutamate is the primary excitatory amino acid neurotransmitter in the human brain. It is important in synaptic plasticity, learning, and development. Over the last four decades, a direct correlation between the neuroexcitatory and neurotoxic properties of glutamate has been linked to activation of excitatory amino acid receptors. This stimulation leads to an enzymatic cascade of events ultimately resulting in cell death (Maragakis and Rothstein, 2001).

The neurotoxic properties of glutamate were first demonstrated by Lucas and Newhouse (1957) who showed that systemic administration of glutamate to infant mice caused retinal degeneration, following this discovery, a series of studies available now have demonstrated serious neurotoxicologic effects of MSG on animals at significantly high doses. Park *et al.*, (2000) found that single intraperitoneal injection of MSG caused significant damage to hypothalamic neurons in the

arcuate nucleus and impaired memory retention in adult mice. Gonzalez-Burgos *et al.*, (2001) also found that subcutaneous administration of MSG to male neonate rats induced excitotoxicity, leading to cell death in prefrontal cerebral cortex. MSG treatment has been known to cause hormonal alterations, which in turn affect the physical state and behavior, especially in neonates who have an immature blood-brain barrier (Larsen *et al.*, 1994).

#### MATERIAL AND METHOD

### **Experimental animals**

White Albino Rats of both sexes weighing between 200-250gms were purchased for the experiment and kept in standard hygienic conditions. They were maintained in cages and fed with pellet diet and water *ad libitum*. rats were quarantified for 10 days and it was confirmed that they were free of pathogen and any other disease.

Rats were equally divided into four groups. The animals of control group were administrated with 0.9% NaCl daily. Low dose group rats were treated orally with the dose of monosodium glutamate (75gm/kg body weight). Intermediate dose group rats received orally with the dose of monosodium glutamate (100gm/kg body weight). High dose group rats were administrated orally with the dose of monosodium glutamate (125gm/kg body weight) for 28 days.

## **Behavioural studies**

The animals were observed in their cages for behavioral symptoms daily for the entire experimental period between 8.00A.M. to 8.00P.M.

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# RESULT AND DISCUSSION

Lacrimation was found to be decreased in the high dose treated group when compared to control. As far as urination is concerned when compared to control it was found to be normal in low dose and intermediate dose treated animals but it was slightly decreased in the animals which were being treated with high dose of MSG. No prominent change was found in the body activity of the treated animals when compared to control. Feeding of the animals decreased continuously with the increasing dose of MSG where as drinking was increased when compared to control animals. It was found to be maximum in the animals which were being treated with the high dose of MSG. Contrary to our results Carter and Levesque (1979) studying the effects of MSG on neurobehaviour in rats described a significant decrease in open-field activity MSG treated group.

Dubovicky et al., (1999) investigated the effect of MSG administration in the neonatal period on habituation of exploratory behavior related to gender differences, rats of both sexes received intraperitoneal injection of MSG to test their habituation with respect to exploratory behaviour, compared to intact controls, there were no significant differences found in interrupted habituation, neither in males nor in females. Kiss et al., (2007) studied the changes in open field activity and novelty-seeking behavior in periadolescent neonatally treated with monosodium glutamate; newborn rats were treated with MSG subcutaneously and found that MSG administration led to only temporary increases in locomotor behavior. In addition to regulating eating behavior, a number of CNS neuropeptides regulate the neuroendocrine pathways (Rindi et al., 2004; Ronaii et al., 2004; Annane, 2003; Puskas et al., 2006).

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