

PARASYMPATHOMIMETIC EFFECTS OF MONO SODIUM GLUTAMATE*

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Summary: The effects of Mono sodium glutamate on smooth muscles were studied using the guinea-pig isolated ileum. Mono sodium glutamate produced spasmogenic effect. Atropine blocked the contractile response elicited by Mono sodium glutamate whereas mepyramine and hexamethonium failed to do so. These findings suggest a cholinergic involvement at post ganglionic site of action.

Key Words : Ajino Moto or Mono Sodium Glutamate

Sin. Cib. Syn. or Chinese Restaurant Syndrome

C₆ = Hexamethonium

A.Ch. = Acetyl Choline

Nic. = Nicotine

INTRODUCTION

The side effects of glutamic and aspartic acids were reported as early as in the mid-forties by Smyth *et al.* (9), Levey *et al.* (3) and in 1968 Ho Man Kwok (2) and Schaumberg *et al.* (7) established the syndrome as Chinese Restaurant syndrome. The name derived from the fact that a reaction occurred in susceptible subjects after ingesting Mono Sodium Glutamate (MSG), the usual seasoner and flavouring agent commonly used in many non-vegetarian preparations of Chinese restaurants. The evanescent symptom complex reported were nausea, vomiting, headache, sweating, weakness in the facial and temporal region over upper back, neck and arms, thirst, flushing of the face, formication and abdominal pain (post sin-cibal syndrome). The toxic effects of MSG on the inner layers of retina were studied by Lucas and Newhouse in 1957 (4) in albino mice and found to be more extensive in young animals sparing the (rods) visual receptor. MSG fed exhibited hyper irritability without any change in the levels of glutamate in brain and with a decrease in GABA levels which may be related to this effect (6).

Although a limited study has been done on the Chinese Restaurant Syndrome, no conclusions have been drawn as to whether there is any cumulative or long term ill-effects by regular ingestion of these foodstuffs. In 1965, Le Sch and Nyhan (5) recognized a disease as x-linked hyperuricemia with mental retardation and Ghadimi *et al.* (1) showed the significance of MSG when added to food (10 gm/day) in the treatment of the deficiency state in these cases with the resultant increased appetite and the personality change.

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The present study has indicated that MSG acts on post ganglionic cells as shown by spasmogenic effect on guineapig isolated ileum which is abolished by atropine.

MATERIALS AND METHODS

Mono sodium glutamate as available in the market is "AJINO MOTO" and it is a crystalline powder. It was dissolved in distilled water and used in all the experiments.

Guinea pigs and rats were killed by a blow on the head and bled. The ileum was removed, washed free of food residue and placed in Tyrode solution. 6 cm from the ileocecal junction segments of about 3 cm long were cut and suspended in 25 ml organ bath at 37°C and aerated with a mixture of 95% O₂ and 5% CO₂. Contractions were recorded on the smoked kymograph paper by using isotonic lever.

Although characteristic response could be observed at doses lower than 10 µg/ml, due to the relatively short lasting effect, we preferred to employ a higher concentration to obtain more prolonged response. The contact time for Mono sodium glutamate was 2 minutes in the experiments after which Mono sodium glutamate was removed by washing it out several times.

Drugs used :

Acetylcholine HCl; Nicotine sulphate; Histamine HCl, Atropine sulphate, Hexamethonium bromide and Mepyramine.

RESULTS

Mono sodium glutamate produced tonic contractions immediately in 80% of the preparations while in the remaining the spasmogenic response was delayed for a 2-5 minutes.

Antagonistic actions :

Atropine: The concentration of atropine necessary to suppress the response of acetylcholine equally abolished completely the spasmogenic action of Mono sodium glutamate. (Fig. 1)

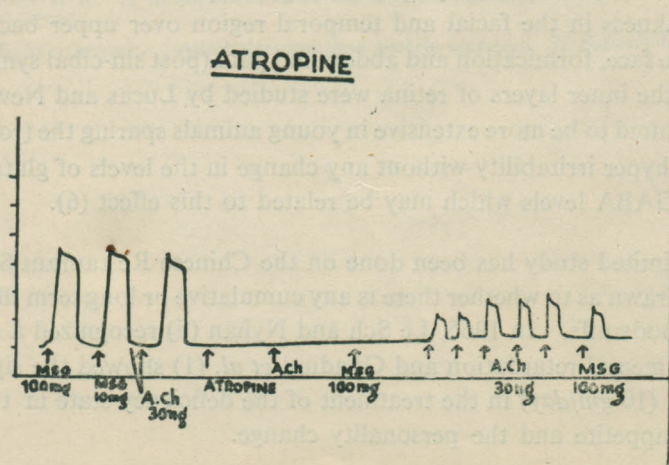


Fig. 1 : Shows Atropine antagonising the effect of A.Ch. and MSG equally.

This antagonistic effect was reversed after washing the tissue containing atropine.

M. pyramine: Produced no inhibitory effect on the action of Mono sodium glutamate on ileum. On addition of atropine at the height of contractions of Mono sodium glutamate, there was an immediate relaxation. (Fig. 2).

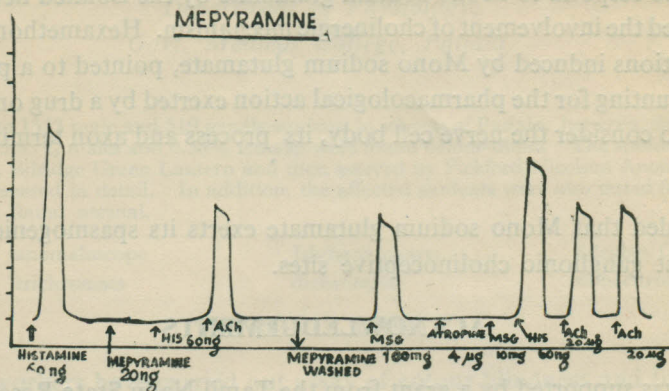


Fig. 2 : Shows the lack of inhibiting response on MSG by Mepyramine.

Hexamethonium : 2 μg of nicotine produced contractions of smooth muscle and this action was blocked by hexamethonium 50 μg dose. Subsequent addition of 100 μg of Mono sodium glutamate recorded the usual spasmogenic effect, which was not blocked by hexamethonium (Fig. 3).

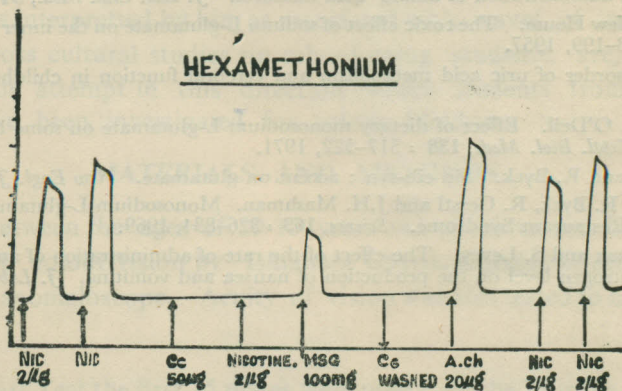


Fig. 3 : Shows Hexamethonium blocking Nicotine response but not spasmogenic action of MSG.

DISCUSSION

It has been reported that 5 g of Mono sodium glutamate can increase the appetite in addition to its use as a seasoner and flavouring agent. Above results have indicated that Mono sodium glutamate acts on the post ganglionic fibres of para sympathetic and not on ganglion cells

as shown by the spasmogenic effect on guinea pig isolated ileum. Lucas and Newhouse (4) have showed extensive destruction of the ganglion cells and inner nuclear layers of retina in suckling mice but spared the visual receptors.

The failure to respond to Mono sodium glutamate by the isolated ileum, in the presence of atropine suggested the involvement of cholinergic mechanism. Hexamethonium failed to block the typical contractions induced by Mono sodium glutamate, pointed to a post ganglionic site of action. In accounting for the pharmacological action exerted by a drug on the post ganglionic neurone, one has to consider the nerve cell body, its process and axon terminals and anticholinesterase activity.

It is concluded that Mono sodium glutamate exerts its spasmogenic effect through the stimulation of post ganglionic cholinceptive sites.

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