SYSTEMIC GLUTAMATE EXCITES TRIGEMINOVASCULAR NEURONS THROUGH ACTIVATION OF PERIPHERAL NMDA RECEPTORS

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INTRODUCTION / AIM

Oral ingestion of 150 mg/kg of monosodium glutamate (MSG) by healthy subjects has been shown to cause headaches. We investigated whether systemic administration of MSG is capable of exciting and/or sensitizing spinal trigeminal subnucleus caudalis (SpVc) neurons with dural receptive fields as part of its mechanism to induce headache.

METHODS

A total of 21 male and 19 female Sprague-Dawley rats were used. Single unit electrophysiological recordings of SpVc neurons that responded to mechanical stimulation of the dura were undertaken. Intravenous MSG (50mg/kg) was administered alone or in combination with the NMDA receptor antagonist 2-amino-5-phosphono-valerate (APV; 5, 50 mg/kg). An electronic von Frey hair was used to assess mechanical threshold (MT).

RESULTS

MSG increased spontaneous action potential discharges. This was attenuated by co-administering 5 mg/kg APV (P>0.05, 2-way ANOVA on RANKS). MSG decreased the MT by 5.7±6.5%. Co-administration of APV with MSG also attenuated this MSG-induced mechanical sensitization in a dose dependent manner (P>0.05, 2-way ANOVA on RANKS). There was no sex-related difference in these effects.

DISCUSSION / CONCLUSIONS

Systemically administered MSG excites and mechanically sensitizes dural afferent fibres through activation of peripheral NMDA receptors. Peripherally restricted NMDA receptor antagonists may be useful in the treatment of headache.

OTHER AUTHORS

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