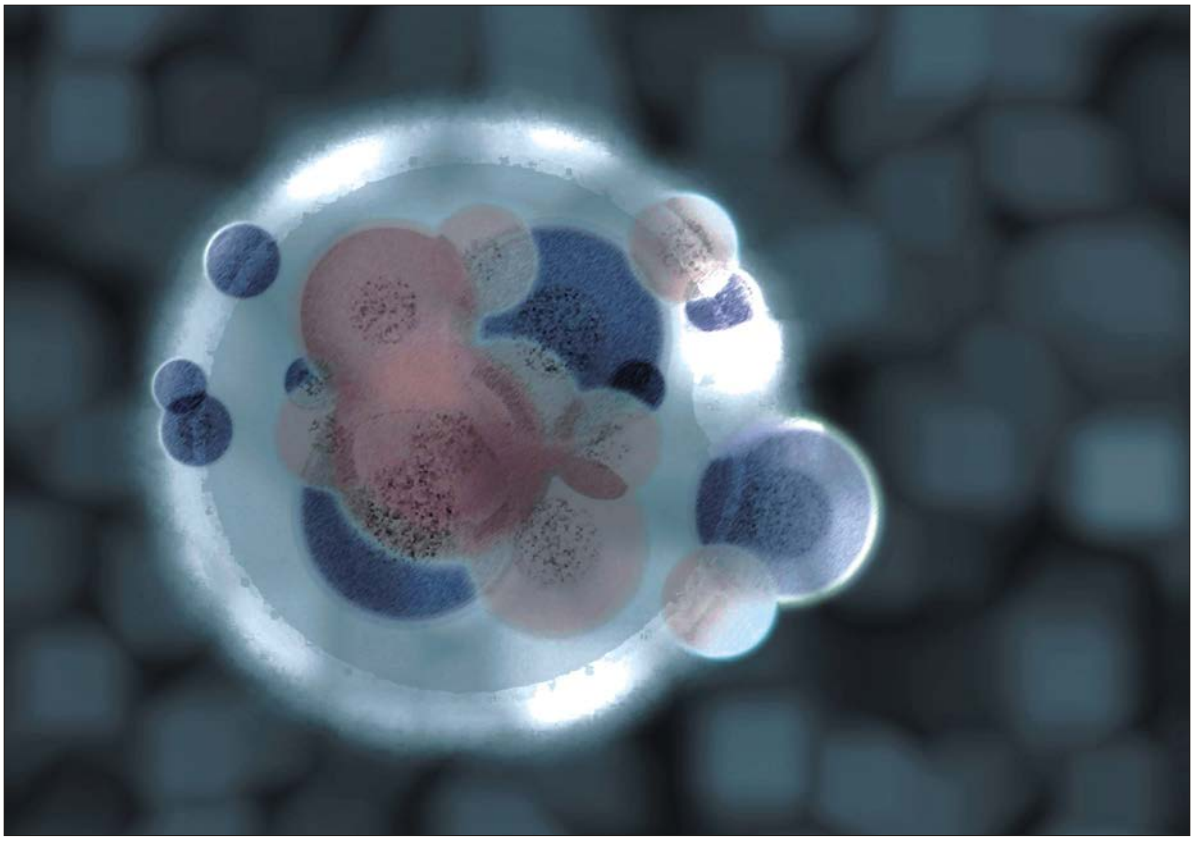

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Monosodium Glutamate, a Possible Threat to Gastric Integrity in Rats

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Abstract: The effects of Monosodium glutamate (MSG) on gastric acid secretion, gastric mucus secretion and gastric ulceration were studied in adult albino rats of Wistar strain. The treated animals were given subcutaneous injection of 0.6 mg g^{-1} MSG for 30 days. Mean basal gastric acid secretion in control versus MSG treated rats were not significantly different; 1.45±0.06 vs 1.40±0.02 ($p>0.05$). However, MSG significantly increased histamine-induced gastric acid secretion with time ($p<0.05$). Mean ulcer scores and gastric mucus secretion were significantly higher in MSG-treated animals than in the control ($p<0.05$). The values are 7.63±1.02/15.75±1.43 and 5.95±0.51/12.75±0.78, respectively. MSG was found to produce nonuniform histopathologic changes in gastric mucosa, which were characterized by edematous lamina propria containing increased numbers of inflammatory cells, vascular congestion and disruption with lifting rupture.

Key words: Monosodium glutamate, gastric acid, gastric mucus, gastric ulceration, gastric pits

INTRODUCTION

Monosodium glutamate (MSG) is a popular condiment in soups, stews, sauces and porridge preparation in several West African countries including Nigeria where the different tribes call it by several names like; kashin zabo, farin maggi and magi funfun.

MSG is used in large quantities as a flavour enhancer throughout the world. The industrial production began in Japan in the early 1900s. In Asian countries, it is used at the table along with salt and pepper. The manufacturer's recommended usage dose of 5 g L^{-1} (30 mM) of hot liquid (Fernstrom, 2000). The majority of the users ignorantly exceed the recommended dose. Reports have it that eating food with MSG could cause a condition known as "Chinese food syndrome" which could mimic a heart attack.

Food additives may have a significant impart on the marketing and acceptability of food but may as well lead to certain side effects. Goldschmiedt *et al.* (1990) reported that addition of 360 mg of MSG to beef consumed in soup was found to have no effect on the meal. However, Monosodium glutamate and settzer, which mongrel dogs do not normally encounter in their diets produced lower gastric acid secretion and pancreatic polypeptide release (Powers *et al.*, 1990).

Since MSG still represents an important ingredient in food preparation across the world, it becomes interesting to examine the effects of MSG on gastric acid secretion, gastric mucus secretion and experimentally induced gastric lesions in rats.

MATERIALS AND METHODS

This study was carried out in the Research Laboratory of the Department of Physiology, University of Ibadan and the Histology section of the Department of Veterinary Anatomy, University of Ibadan, Ibadan, Nigeria between January and September, 2004.

Sixty-four male albino rats of Wistar strain of average weight 200±20 g were used. They were purchased from Preclinical animal house of the College of Medicine, University of Ibadan, Nigeria. They were fed on a commercial rat's diet purchased from Ladokun livestock feed limited, Ibadan, Nigeria.

Experimental design: The animals were divided into four experimental groups of sixteen rats per group. Group I, for gastric acid secretory study; Group II, for experimentally induced gastric ulceration study; Group III, for the study on gastric mucus secretion and Group IV, to assess possible histological changes. In all the groups, eight rats serve as normal control. These were not treated with MSG. The other group also consisted of eight rats were treated daily with subcutaneous 0.6 mg kg^{-1} MSG (Vedan) for 30 days.

Gastric acid secretory study: The animals were starved for 24 h prior to the investigation to allow for clear stomach without debris. The rats were anaesthetized with urethane given intraperitoneally at 0.6 mL/100 g body weight. The stomach was later prepared for perfusion using the continuous stomach perfusion technique described by Ghosh and Schild (1958) as modified by Adeniyi and Oluwole (1990).

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Basal secretion: The stomach was continuously perfused at 1.0±0.1 mL min^{-1} with 0.9% saline warmed to rat body temperature. The effluent was collected in 10 min sample for 1 h and titrate against 0.01 N NaOH.

Histamine-induced secretion: The stimulated secretion was measured after allowing 1hr for the stomach to stabilize post basal collection. Gastric acid secretion was induced with intraperitoneal injection at 1 mg kg^{-1} body weight histamine. This was given via a cannulated femoral vein.

Indomethacin-induced ulceration study: Indomethacin as a model for gastric ulceration has been well documented and was used in this study (Elegbe, 1978; Adeniyi and Oluwole, 1990).

After 24 h fast prior to the experiment, each rat was given 40 mg kg^{-1} body weight indomethacin suspension by oral route using oral dosing needle. The induction of ulcer by this method is rapid requiring a minimum of 4 h. The rats were later killed by a sharp blow to their heads and each stomach was examine macroscopically using a hand lens. The scoring of gastric ulceration was that described by Alphin and Ward (1967).

Gastric mucus secretory study: Following 30 days treatment period, the rats were then fasted overnight and sacrificed. The stomachs were removed and the glandular portion of the stomach was excised, opened along the lesser curvature. The everted stomachs were soaked in 0.1% Alcian blue dissolved in 0.16 M sucrose buffered with 0.05 M sodium acetate for 2 h. The solution was adjusted to pH 5.8 with hydrochloric acid. Uncomplexed dye was removed by two successive washes at 15 and 45 min in 0.25 M sucrose. Dye complexed with mucus was diluted by immersion in 10 mL aliquots of 0.5 M magnesium chloride for 2 h. The resulting blue solutions were shaken briefly with equal volume of diethyl ether and absorbance of aqueous phase was measured at 605 nm with spectrophotometer (Corney, 1974).

The absorbance of each solution was used to calculate the various concentration of dye and the weight of dye (expressed in mg) deduced, using a standard curve. Gastric mucus secretion in mg kg^{-1} was expressed as the weight of the dye against the weight of the stomach.

Histological sections of gastric mucosa: After opening the abdominal cavity of each rat, the esophagus was ligated and the whole stomach was then taken out. An incision was made along the greater curvature to drain contents into a beaker. The stomach was later fixed in 10% formal saline. After complete fixation the blocks were embedded in paraffin and sections cut at 5 μ. Sections were stained with haematoxylin and eosin and mounted in canada balsam. Microscopic examination of the sections was then carried out under a light microscope.

Statistics: The mean and standard deviation for each group were determined. Student's t-test was used to assess significance at $p = 0.05$.

RESULTS

Effects of MSG on gastric acid secretion: The mean basal gastric acid secretion for the control rats was 1.45±0.06 μeq/10 min. This was not different from the value 1.40±0.02 μeq/10 min obtained for MSG treated rats ($p>0.05$). The animals treated with MSG showed significant increase with histamine stimulated acid secretion after 10, 20 and 30 min post-stabilization than those animals not treated ($p<0.05$). The values are 2.80±0.44 versus 1.54±0.12(10), 3.85±0.75 versus 1.98±0.18 (20) and 6.23±0.94 versus 2.75±0.34.

Effect of MSG on indomethacin-induced gastric ulceration: The mean ulcer score of 15.75±1.43 in the MSG treated animals was significantly higher than 7.63±0.02 in the control animals ($p<0.05$).

Effect of MSG on gastric mucus secretion: The mean gastric mucus secretion in control animals was 5.95±0.51 as against 12.76±0.78 in the MSG-treated animals. This observed increase was significant ($p<0.05$).

DISCUSSION

MSG is used worldwide as a flavour enhancer. The average person living in an industrialized country consumes about 0.3 to 1.0 g of MSG per day. MSG is classified by the Food and Drug administration as generally recognized as safe. Indeed, many researchers have questioned the very existence of a true MSG-sensitivity reaction. Most clinical trials, including some double-blind trials, have failed to find any symptoms arising from consumption of MSG, even large amounts, when taken with food (Tung and Tung, 1980; Prawirohardjon *et al.*, 2000). However, clinical trials have found that MSG taken without food may cause symptoms, though rarely the classic symptoms already described (Kenney and Tidball, 1972).

In this study, MSG has been shown to be a potential aggressive factor to the gastric mucosa as it enhances the secretagogue-effect of histamine. It has however

Fig. 1: Micrograph of a normal rat stomach not treated with MSG. The mucosa appears normal with intact layer of surface mucus cells lining the gastric pits. Stained preparation, H and E X10

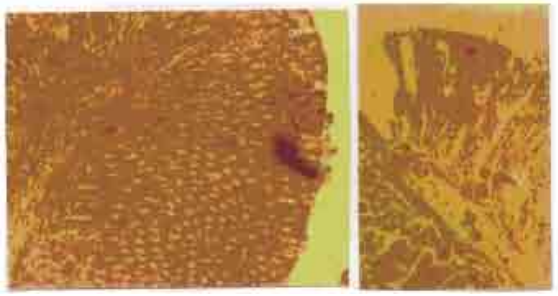

Fig. 2: Micrograph of the stomach of a rat not treated with MSG but gastric ulceration was induced with indomethacin. The gastric pits appear more dilated and a layer of mucus and exfoliated cells are readily apparent covering the mucosa. Stained preparation, H and E; left panel X10, right panel X16

Fig. 3: Micrograph of the stomach of a MSG-treated rat with gastric ulceration induced with indomethacin. The gastric mucosa is showing signs of extreme cellular exfoliation. Most of the surface mucus cells appear detached from the mucosa. Congested blood vessels remain apparent beneath the exfoliated areas. Stained preparation H and E; left panel X10, right panel X16

been established that many secretagogues act by releasing histamine from the ECL cell which in turn is required for gastric acid secretion (Prinz *et al*., 1993). This agrees with the findings that MSG is a potentiating factor of gastric acid secretion in subject of free gastrointestinal disease (Shlygin *et al*., 1991).

Our observations Fig. 2 and 3 in this study lend credence to previous findings of Szabo and Pfeiffer (1984) on the activity of gastric mucosal histamine release; as increasing cell damage, ulceration which are characterized by an edematous lamina propria containing increased numbers of inflammatory cells, vascular congestion, disruption and varying degrees of epithelial lifting and rupture.

The results of the study of MSG and gastric mucus secretion show a significant increase in total gastric mucus secretion with MSG treated animals. This is not surprising since in an attempt to increase the mucosal resistance to acid, there might be an increase in gastric mucus (Adeniyi and Oluwole, 1990). The increase in mucus secretion seen in MSG treated rats in this study was due to exposure of the animals to MSG for 30 days which is a damaging agent to the gastric mucosa and this would result in adaptive cytopotection as earlier speculated by Robert (1979).

MSG might have a causal relationship to the onset of gastric ulcer, this has not been fully validated in human, however, the consumption of large quantities of MSG in diet may not be beneficial to peptic ulcer patients or those prone to have ulcer. This is due to its effects on gastric acidity and gastric mucosal damage.

REFERENCES

Adeniyi, K.O. and F.S. Oluwole, 1990. Influence of thyroid hormones on indomethacin induced gastric ulceration in rats. Nig. J. Physiol. Sci., 6: 192-196.

Alphin, R.S. and J.W. Wards, 1967. Actions of hexapyronium bromide on gastric secretion in dogs and ulceration in rats. Arch. Intl. De. Pharamacodyn. Therap., 168: 82-100.

Corney, 1974. In: Dhuley, J.N. and S.R. Naik, 1998. Protection by rhinax in various models of ulceration in rats. J. Ethnopharmcol., 63: 219-2125.

Elegbe, R.A., 1978. Comparative studies on starvation and indomethacin-induced ulcerations in albino rats. Biochem. Expt. Biol., 2: 159-166.

Fernstrom, J.D., 2000. Pituitary hormone secretion in normal male humans: Acute responses to a large, oral dose of monosodium glutamate. J. Nutr., 130 (4s suppl): 10535-75.

Ghosh, M.N. and H.O. Schild, 1958. Continuous recording of acid gastric secretion in the rat. J. B. Pharmacol, 13: 54-61.

Goldschmiedt, M., J.S. Redferm and M. Feldman, 1990. Food Coloring and Monosdium Glutamate: Effects on the Cephalic Phase of Gastric Acid Secretion and Gastrin Release in Humans. Am. J. Clin. Nutr., 51: 749-757.

Kenney, R.A. and C.S. Tidball, 1972. Human susceptibility to oral monosodium l-gluatamate. Am. J. Clin. Nutr., 25: 140-60.

Powers, M.A., S.S. Schiffman, D.C. Lawson, T.N. Papers and I.L. Taylor, 1990. The effect of taste on gastric and pancreatic responses in dogs. Physiol. Behav., 47: 1295-1297.

Prawirohardjono, W., I. Dwioprahasto and I. Astuti *et al.* 2000. The administration to indonesians of monosodium l-glutamate in indonesian foods: An assessment of averse reactions in randomized double-blind, crossover, placebo-controlled study. J. Nutr., 130 (45 Suppl.): 1068-1074.

Prinz, C., M. Kajimura, D.R. Scott, F. Mercier, H.F. Holander and G. Sachs, 1993. Histamine secretion from rat enterochromaffin-like cells. Gastroenterology, 105: 449-461.

Robert, A., 1979. Cytoprotection by prostaglandin's. Gastroenterology, 761: 767.

Shlygin, GK., L.S. Vasilevskaia, T.I. Loranskaia, A.K. Shakhovskaia and A.M. Kochethov, 1991. Potentiating effect of sodium glutamate on gastric secretion and its possible use as a clinical test. Klm Med. (Mosk). 69: 66-70.

Szabo, S. and C.J. Pfeiffer, 1984. Ulcer disease. Lab. Invest., 51: 121-147.

Tung, T.C. and K.S. Tung, 1980. Serum free amino acid levels after oral glutamate intake in infants and human adults. Nutr. Rep. Intl., 22: 4311-4343.