Middle-East Journal of Scientific Research 18 (2): 163-167, 2013
ISSN 1990-9233

DOI: 10.5829/idosi.mejsr.2013.18.2.12430

Evaluation of MSG on Electrolyte Balance and Histology of Gastroesophageal Mucosa

[1]I.G. Ilegbedion, [2]F.M. Onyije and [3]K.A. Digba

[1]Department of Medical Laboratory Science,
[2]Department of Human Anatomy, Faculty of Basic Medical Science, College of Health Sciences,
Niger Delta University, Wilberforce Island, P.M.B. 071 Yenagoa, Bayelsa State, Nigeria
[3]Department of Medical Laboratory Science, Faculty of Health Sciences,
IgbinedionUniversity, Okada, Edo State, Nigeria

Abstract: Commercial MSG is produced by fermentation of starch, sugar, beet, sugarcane or molasses. It is one of the main flavor enhancer used as an ingredient in various food products. Its palatable and favorite flavor is a must in almost all Chinese, South-Asian and Nigerian dishes, where it is known by the names of Ajinomoto and white-Maggi.Twenty (20) female adult wistar rats were used for this experiment. The rats with average weight of 181g were randomly assigned into four groups of five each (Groups A, B, C and O). Group A, B and C served as treatments groups while group O as the control. Each rat in the treatment groups A, B and C received 0.1, 0.15 and 0.20 g kg^{-1} of monosodium glutamate. Results showed that there was elevation of K^+ in groups A and C (6.42 ± 0.40 and 6.07 ± 59) but was not statistically found different from control. While there was a significant elevation of Na^+ in group A (0.1g kg^{-1}), Cl^- in group C (0.20g kg^{-1}), Ca^{2+} in group B, TCa in both groups A and B and pH also increased significantly in group C. In the method used there were no notable distortions of the mucosa architecture of the tissues. Research on MSG should go beyond rudimentary to advance molecular since much have been done on the basic, hoping that the molecular advancement will help to establish clearly what MSG can do to human.

Key words: MSG · Gastroesophageal · Histology · Electrolyte

INTRODUCTION

MSG is a sodium salt of naturally occurring (non-essential) L-form of glutamic acid [1, 2, 3], in pure form it appears as a crystalline powder. L–Glutamic acid is a ubiquitous amino acid present in most foods either in the free form or bound to peptides or proteins [3]. Modern commercial MSG is produced by fermentation of starch, sugar, beet, sugarcane or molasses [4]. It is one of the main flavor enhancer used as an ingredient in various food products. Its palatable and favorite flavor is a must in almost all Chinese, South-Asian [1, 2] and Nigerian dishes, where it is known by the names Ajinomoto and white-Maggi.

It is widely used in restaurants, packaged food industries and household kitchens [4], in Nigeria, most communities and individuals often use MSG as a bleaching agent for the removal of stains from clothes. MSG is sold in most open market stalls and stores in Nigeria as "Ajinomoto" marketed by West African Seasoning Company Limited [5] or "Vedan" marketed by Mac & Mei (Nig) [6].

Since 1960s, Scientist began the analysis of the possible side effects of MSG on humans following its pronounced property of enhancing the taste of the food and massive popularity [7]. Since then research has shown that MSG is dangerous, in high doses can produce neuroendocrine abnormalities and neuronal degeneration [8] and oxidative damage in different organs [9, 10]. Also reported about MSG are retinal degeneration, stroke, epilepsy, brain trauma, neuropathic pain, schizophrenia, anxiety and depression, Parkinson's disease, Alzheimer's disease, Huntington's disease and amyotrophic lateral sclerosis [11]. Others are reduction of the reproductive ability in female and male mice [12] and enhancement hunger [13]. Hence the aim of this research is mainly to evaluate the effect of MSG on the electrolyte balance and mucosa of the stomach.

Corresponding Author: I.G. Ilegbedion, Department of Medical Laboratory Science, Faculty of Basic Medical Science, College of Health Sciences, Niger Delta University, Wilberforce Island, P.M.B. 071 Yenagoa, Bayelsa State, Nigeria.

MATERIALS AND METHODS

Animals: The rats were purchased from Nnsuka, Nigeria. The Wistar rats were kept in a clean plastic cages enclosed with wire gauzes cover according to their groups under standard housing conditions at temperature 25°C-29°C at a 12h light and dark cycle. They were fed with growers marsh produced by grand cereals and oil mills limited, Bukurujos, plateau state, Nigeria, which was obtained from chrys ventures limited, owerri Imo state, Nigeria.

Monosodium Glutamate: The crystal form of monosodium glutamate was bought from Elele Market, Rivers State and grinded to powdered form.

Experimental Design/Administration of Monosodium Glutamate to the Animals: Twenty (20) female adult wistar rats were used for this experiment. The rats with average weight of 181g were randomly assigned into four groups of five each (Groups A, B, C and O). Group A, B and C served as treatments groups while group O as the control. Each rat in the treatment groups A, B and C received 0.1g/kg, 0.15g/kg and 0.20g/kg of monosodium glutamate respectively in 0.5ml of water orally three times daily for two weeks (fourteen days). The rats were sacrificed on the fifteenth day of the experiment.

Histopathological Examination: The rats were sacrificed 24h after the last dose, the ovaries were dissected and fixed in 10% formal saline, dehydrated in ascending grades of alcohol, impregnated and imbedded in paraffin wax. Paraffin sections (5 µm thick) were stained with haematoxylin and eosin (H & E) as a routine stain.

RESULTS

There was elevation of K^+ in groups A and C (6.42±0.40 and 6.07±59) but was not statistically different from control. While there was significant elevationof Na^+ in group A (0.1g/kg), Cl^- in group C (0.20g/kg), iCa in group B, Tca in both groups A and B and pH also increased significantly in group C (Table 1).

DISCUSSION

Serum electrolyte concentrations are among the most commonly used laboratorytests by Medical Laboratory Scientists for assessment of a patient's clinical conditions and disease states. Our result reveals elevation of K^+ in groups A and C (6.42 ± 0.40 and 6.07 ± 59) but was not statistically different from control. While there was significant elevation of Na^+ in group A (0.1g/kg), Cl^- in group C (0.20g/kg), iCa in group B, TCa in both groups A and B and pH also increased significantly in group C. This paper indicates that intake of MSG alters the electrolyte of the body in rats, which could be responsible for stomach cramps as reported by various researchers [14, 15, 16].

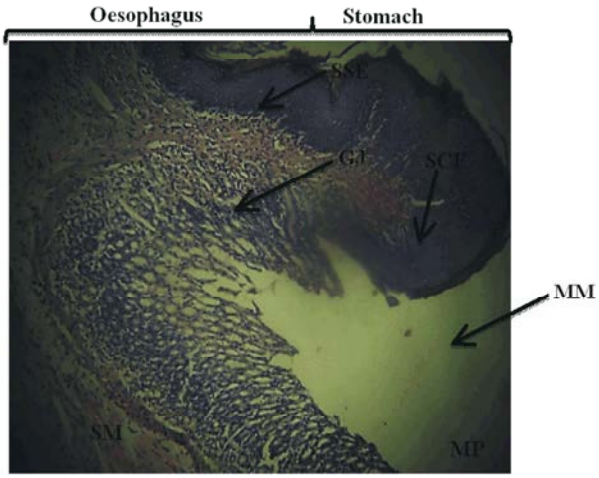
Potassium homeostasis is altered by changes in acid-base balance, insulin, aldosterone and renal function others are gastrointestinal and skin damages [17, 18, 19, 20, 21]. Since renal excretion is the major route of potassium elimination, renal failure is the most common cause of hyperkalemia [21]. On the other hand Hypokalemia occurs when serum potassium concentration less than 3.5 meq/L (<3.5 mmol/L) [19]. Several investigators have reported that hypertensive rats showed a definite increase in the serum Na concentration [22].

Table 1: Electrolyte analysis of rats administered with MSG.

Parameter	Control	A (0.1g/kg)	B (0.15g/kg)	C (0.20g/kg)
K^+(mmol/L)	5.93 ±0.2	6.42 ± 0.40	5.19 ± 0.09*	6.07 ± 59
Na^+(mmol/L)	130.80 ± 0.16	141.50 ± 1.32***	129.54 ± 0.77	139.74 ± 1.60
Cl^-(mmol/L)	99.08 ± 00	103.24 ± 20	94.84 ± 20	107.44 ± 3.7***
iCa(mmol/L)	0.95 ± 0.02	0.91 ± 0.02	0.84 ± 0.007**	0.96 ± 0.04
TCa(mmol/L)	1.84 ± 0.02	1.78 ± 0.05*	1.74 ± 0.01***	1.81 ± 0.04
pH	7.87 ± 0.02	7.86 ± 0.02	7.98 ± 0.11	8.10 ± 0.04**

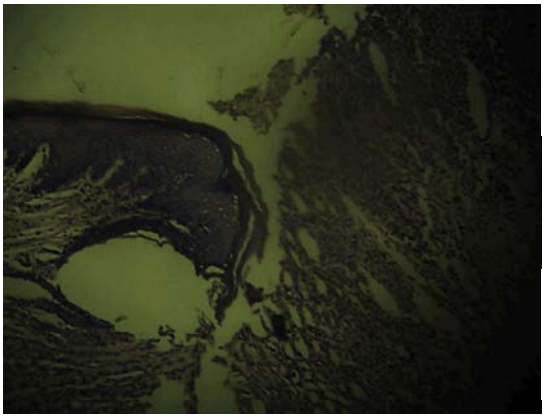
Each value represents the mean ± standard deviation (n = 5), values are statistically different from control at p< 0.05*, 0.01** and 0.001*** one-way analysis of variance (ANOVA) + Tukey –Kramer Multiple Test.

Histopathology

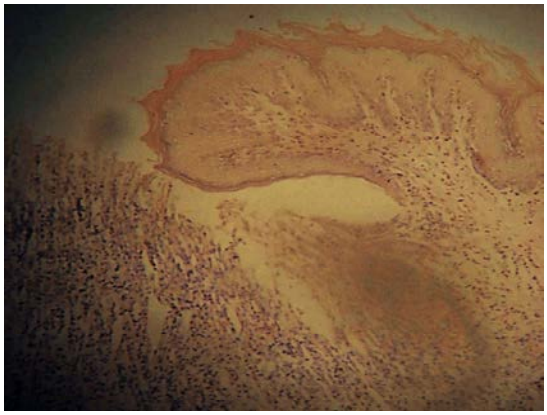


Slide 1: Control, H&E (x10)

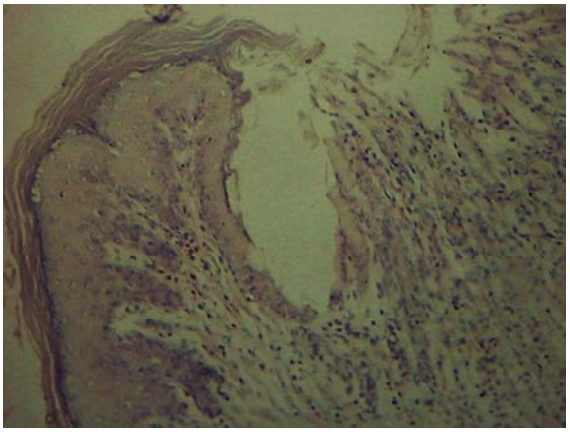
Normal histology of the oesophagus and stomach: SSE= Stratified squamous epithelium, SCE= area of Simple columnar epithelium, GJ= Gastroesophageal junction, MM= Muscularis Musosae, MP= Muscularis Propria, M= Mucosa, SM= Submucosa.



Slide 2: 0.5g/kg, H&E (x10)



Slide 2: 0.15g/kg, H&E (x10)



Slide 2: 0.20g/kg, H&E (x10)

There are no notable distortions of the mucosa architecture of the oesophagus and the stomach when compared with the control using the current methodology.

Campese [23] reported that NaCl-sensitive patients are also more likely than salt-resistant patients to manifest left ventricular hypertrophy, microalbuminuria and metabolic abnormalities that may predispose them to cardiovascular diseases. Although with the current methodology the histology of the stomach did not show evidence of alteration but Eweka [24] reported increased basophilia and cellular hypertrophy of the stomach in animals given 3g of MSG, while degenerative and atrophic changes

CONCLUSIONS

The issue of MSG consumption which has been controversial should not be neglected. Our findings indicate that total elevation of some electrolyte parametersmay not be good for hypertensive patients and others. Therefore, it is recommended that researches on MSG should go beyond rudimentary to advance molecular since much have been done on the basic, hoping that the molecular advancement will help to establish clearly what MSG can do to human.

ACKNOWLEDGMENTS

The authors acknowledge the effort of Chibuike O.O. of Madonna University, Okija, Elele campus, Rivers State Nigeria.

REFERENCES

1. Belluardo, M., G. Mudo and M. Bindoni, 1990. Effect of early Destruction of the mouse arcuate nucleus by MSG on age Dependent natural killer activity: Brain Res., 534: 225-333.
2. Moore, K.L., 2003. Congenital malformations due to environmental; Developing Humans. W.B. Saunders co. Ltd. Philadelphia. 2nd ed. pp: 173-183.
3. Munro, H.N., 1979. Factors in the regulation of glutamate metabolism. Glutamic acid - Advances in Biochemistry, pp: 55-68.
4. Walker, R. and J.R. Lupien, 2000. The safety evaluation of monosodium glutamate. J, Nutr., 130: 1049S-52S.
5. Eweka, A.O., A Eweka and F.A.E. Om'Iniabohs, 2010. Histological studies of the effects of monosodium glutamate of the fallopian tubes of adult female Wistar rats. North Am. J. Med. Sci., 2: 146-149.
6. Eweka, A.O. and J.O. Adjene, 2007. Histological studies of the effects of monosodium Glutamate on the medial geniculate body of adult Wister rat. Electron J. Biomed., 22: 9-13.
7. Garattini, S., 2000. Glutamic acid, twenty years later. J. Nutr., 130: 901S-9S.
8. Moreno, G., M. Perello, R.C. Gaillardand and E. Spine, 2005. Orexina stimulates hypothalamic- pituitv-adrenal (HPA) axis function, but not food intake in the absence of full hypothalamic NPY- ergic activity. Endocrine, 26: 99-106.
9. Farmobi, E.O. and O.O. Onyema, 2006. Monosodium glutamate-induced oxidative damage and genotoxicity in the rat: Modulatory role of vitamin C, vitamin E and quercetin. Hum. Exp. Toxicol., 25: 251-259.
10. Pavlovic, V., D. Pavlovic, D. Kocic, D. Sokolovic and Jevtovic-Stoimenov, 2007. Effect of monosodium glutamate on oxidative stress and apoptosis in rat thymus. J. Mol. CellBiochem., 303: 161-166.
11. Samuels, A., 1999. The Toxicity/Safety of MSG: A study in suppression of information. Acctabil. Resch., 6(4): 259-310.
12. Mozes, S. and Z. Sefcikova, 2000. Obesity and changes of alkaline phosphatase activity the small intestine of 40 and 80-day old rats subjected to early postnatal overfeeding of monosodium glutamate. Physiol Res., 2: 177-86.
13. Micœkowiak, B. and M. Partyka, 1993. Effects of neonatal treatment with MSG (monosodium glutamate) on hypothalamo- pituitary-thyroid axis in adult male rats. Histol Histopathol., 8(4): 731-4.
14. Denice, M., 2011. The Many Faces of Monosodium Glutamate (MSG) Natural Health Technique. www.naturalhealthtechniques.com
15. Baad-Hansen, L., B.E. Cairns, M. Ernberg and P. Svensson, 2010. Effect of systemic monosodium glutamate (MSG) on headache and pericranial muscle sensitivity. Cephalalgia, 30: 68 76.
16. Graham, T.E., V. Sgro, D. Friars and M.J. Gibala, 2000. Glutamate ingestion: the plasma and muscle free amino acid pools of resting humans. Am. J. Physiol. Endocrinol. Metab., 278: E83-E89.
17. Rose, B.D., 2001. Clinical Physiology of Acid-Base and Electrolyte Disorders. 5th ed. New York, NY: McGraw-Hill.
18. Halperin, M.L. and M.B. Goldstein, 1994. Fluid, Electrolyte and Acid-Base Physiology: A Problem-Based Approach. 2nd ed. Philadelphia, PA: W.B. Saunders.

19. Zull, D.N., 1989. Disorders of potassium metabolism. Emerg Med Clin North Am., 7: 771-94.
20. Oh, M.S. and H.J. Carroll, 1994. Electrolyte and acid-base disorders. In: Chernow B, ed. The Pharmacologic Approach to the Critically Ill Patient. 3rd ed. Baltimore, MD: Williams & Wilkins. 957-68.
21. Peterson, L.N. and M. Levi, 2003. Disorders of potassium metabolism. In: Schrier RW, ed. Renal and Electrolyte Disorders. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins. 171-215.
22. Fregly, M.J., F.E. Yates and E.M. Landis, 1955. Proc. Sot. Exp. BioZ. and Med., 90: 695.
23. Campese, V.M., 1994. Salt sensitivity in hypertension. Renal and cardiovascular implications. Hypertension. 23: 531-550 doi: 10.1161/?01.HYP.23.4.531
24. Eweka, A.O., F.A.E. Om'Iniabohs and J.O. Adjene, 2007. Histological Studies of The Effects of Monosodium Glutamate on The Stomach of Adult Wistar Rats Annals of Biomedical Sciences, 6(1): 45-52.