

## Does Monosodium Glutamate Induce Obesity in Female Mice?

Tabassum Zafar<sup>1\*</sup>, Vinoy K Shrivastava<sup>2</sup>

1. PhD Scholar, Masters of Philosophy in Biochemistry, Masters in Biochemistry, Laboratory of Endocrinology, Department of Biosciences, Barkatullah University, Bhopal (M.P), India.

2. Prof. & Head, PhD in Zoology, Masters in Zoology, Laboratory of Endocrinology, Department of Biosciences, Barkatullah University, Bhopal (M.P), India.

### \*Correspondence:

Tabassum Zafar, Laboratory of Endocrinology, Department of Biosciences, Barkatullah University, Bhopal (M.P), India-462026

Tel: (91) 755 266 5071

Email: tztabassumzafar@gmail.com

Received: 07 October 2017

Accepted: 20 November 2017

Published in December 2017

### Abstract

**Objective:** Female metabolism is highly responding towards diet associated changes. Monosodium glutamate is a popular flavor enhancer, which is widely used to develop umami taste. Young female generation nowadays deals with more issues related to metabolism and fertility. Present study is an effort to monitor the effect of monosodium glutamate oral consumption on female mice health and obesity.

**Materials and Methods:** Adult female Swiss albino mice *Mus musculus* were divided into two groups named control and treatment. Treated group received 4 gm/kg body weight/day dose of monosodium glutamate dissolved in double distilled water by oral gavage. Control group received only double distilled water. After the completion of experiment, lee index was calculated to determine the induced level of obesity.

**Results:** Present study states that lee index of monosodium glutamate treated mice were significantly higher than control mice. This increment of lee index indicates that monosodium glutamate is contributing factor for induction of obesity in female mice.

**Conclusion:** Obesity is the main cause of metabolic syndrome, which comes with many associated feminine health issues. Our findings strongly discourage prolonged consumption of high doses of monosodium glutamate to avoid obese young female population.

**Keywords:** Obesity, Lee index, Monosodium Glutamate

## Introduction

The adverse effects of obesity on female health are documented in literature since past many decades (1). Overweight is a primary alarming symptom that indicates nutritional imbalance, which converts easily into obesity. Central abdominal obesity is not just a disorder but also a contributing factor for progression of metabolic syndrome (2). Metabolic syndrome is a group of symptoms, which commonly include abdominal obesity, dyslipidemia, glucose intolerance, and hypertension along with many other clinical symptoms (3).

Because of several circumstances specific to women like pregnancy, polycystic ovarian syndrome, oral contraceptive therapy use, and menopause females are more prone to metabolic syndrome progression. Due to specific circumstantial change in hormones levels, mood swings females have more tendencies to satisfy their taste buds with variety of foods incorporated with food additives, food colours and other processing materials (4). Busy working schedule of independent women followed by sedentary life style also facilitate the consumption of pre

packed, commercially available processed foods. Monosodium glutamate (MSG) is an iniquitous flavor enhances incorporated in various Chinese, Japanese, South-Asian, ready to eat foods, noodles, soups, sauces, chips, packed, processed and branded foods. Monosodium glutamate is a sodium salt of naturally occurring (non-essential) L-form of glutamic acid. It is commercially available under many brand names like Ajinomoto, Sasa, Vetsin, Miwon and Weichaun (5).

MSG is responsible to generate a specific kind of umami taste which seems protein rich and provides great satisfaction to consumer through improving taste stimulation and enhances appetite (6). Apart from its extensive use, status of MSG is still controversial. MSG has evident connections with many clinical disorders like Chinese restaurant syndrome, neurodegeneration, endocrine disorders, stress and behavioral disturbances. Many researchers studied the effect of MSG on different health issues via injecting MSG directly into the blood (7-10). Initially MSG was supposed to be more harmful to males. No clear evidence has been accounted to report the effect of high oral MSG treatment on lee index of female rodents. As mice are widely accepted research models in biomedical sciences, present study aimed to determine the effects of oral consumption of MSG on female obesity and weight pattern in female mice *Mus musculus*.

## Materials and Methods

### Animal care and handling

Adult female albino mice were procured from Jawaharlal Nehru Cancer Hospital and Research Center, Bhopal, India and maintained at the animal house of Department of Biosciences, Barkatullah University, Bhopal, India. Mice were maintained in polypropylene cages on paddy husk bedding under controlled conditions of temperature and light along with standard mice feed and water. Present study is a part of the research plan approved by institutional ethical committee with reference number 1885/GO/S/16/CPCSEA/IAEC/BU/05.

Animal care and handlings were performed according to guidelines issued by CPCSEA, (Committee for the purpose of control and supervision of experiments on animals) New Delhi, India.

### Experimental design

Young female Swiss albino mice were divided into two groups named control and MSG treated. Mice belongs to treatment group were given oral dose of MSG 4 gm/kg/bw dissolved in double distilled water to minimize interference of any other external factor for thirty and sixty days, while control groups received double distilled water from the similar route. At different predetermined durations (day one, day thirty, day sixty) body weight, naso-anal lengths were measured and Lee index was calculated for mice belongs to both group. Lee index is a formula calculated index, used to identify the obesity in rodents. Lee index could be used as the scale of wellbeing similarly as body mass index (BMI) is used in human beings to identify the health status and obesity.

After thirty and sixty days of oral MSG gavage, mice were subjected to calculation of lee index. Naso-anal length (mm) and weight (grams) of each mouse was measured using appropriate tool. Lee index were calculated by the below mentioned formula using previous reference method given by Bernardis and Petterson in 1968 (11,12).

Lee Index= cube root of body weight in gram divided by Naso-anal length in millimeter, multiplied by 10

### Statistical analysis

The collected data were subjected to statistical analysis for monitoring difference between control group and treated group. Means  $\pm$  standard deviation (SD) and standard error of mean (SEM) were calculated using Excel-mac operating system software. Student's t-test was used for statistical comparison between the groups and significance level was determined for different parameters using Excel-mac operating system software. *P*-value less than

or equal to 0.05 was considered as significant.

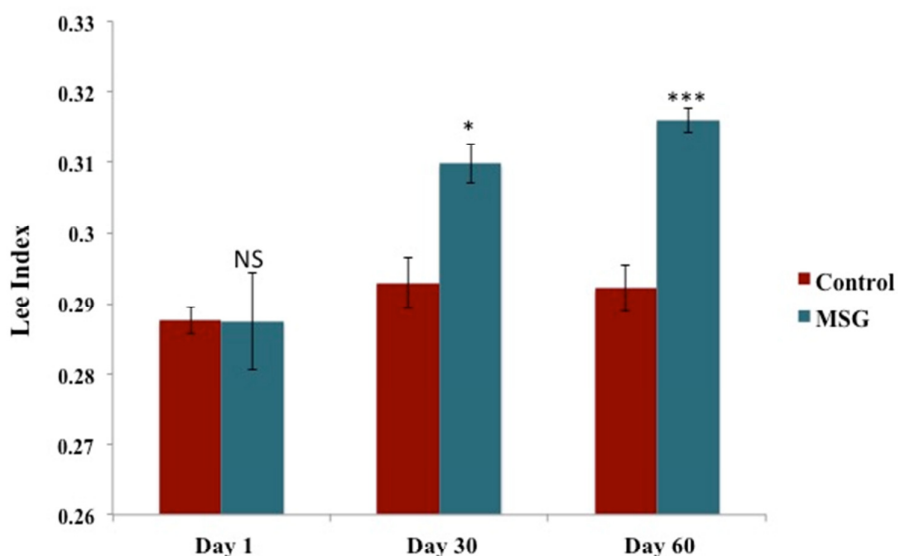
## Results

When value of lee index is below 0.29 it determines the nutritive status of rodent while lee index more that 0.3 is considered as a marker of obesity. It was observed that female mice belongs to group one, which were given double distal water without any treatment have shown normal level of activity with moderate increment in body weight while female mice belongs to group two, which were given MSG orally showed signs and symptoms of stress along with significant increase in body weight. As the duration of MSG oral consumption reaches to thirty day the body weights of treated animals were increased very significantly in compare to control group ( $P$ -value=0.007). When control and treated groups have been compared at the completion of sixty day MSG treatment the difference between body weights become more significant ( $P$ -value=0.009) with a highly significant increment in lee index ( $P$ -

value=0.0001). Lee index of MSG treated group was found to increase significantly in compare to control mice (Table 1) (Figure -1).

## Discussion

Obesity is an emerging health issue for females across the globe (13). Present findings are alarming about use of MSG. The Joint FAO/WHO Expert Committee on Food Additives (JECFA) in 1988 had validated the use of glutamic acid and MSG without any specified daily intake. During the subsequent review of the safety evaluation of MSG by the Federation of American Societies for Experimental Biology (FASEB) and the Federal Drug Administration (FDA) the addition of MSG was restricted to 10 gm/ kg food. 3 grams MSG is allowed to add in 1 kg potato chips. 3–6 grams MSG is allowed to add in each kg of meat products (14,15). Even though there are available guidelines, still population consume unrestricted and unidentified amount of MSG from various sources nowadays.



**Figure 1** Significant increment in lee index after monosodium glutamate treatment at different intervals. NS represents non significant increment between control and treated group. Single star (\*) represents significant increment in compare to control group. Triple star (\*\*\*) represent highly significant increment in compare to control groups.

**Table 1. Comparison of body weight between control and monosodium glutamate treated groups at different durations**

Groups	Body weight (Mean±SD)	P-value
Control initial day pre treatment	23.42±1.227	Non-significant
MSG initial day pre treatment	23.42±0.978	
Control 30 day post treatment	25.39±1.559	0.0073
MSG 30 day post treatment	30.386±2.166	
Control 60 day post treatment	28.22±2.312	0.009
MSG 60 day post treatment	32.098±1.251	

Higher lee index of MSG treated mice indicates that oral MSG treatment could either leads to polyphagia or it slows down the metabolic rate, which will further contribute in progression of obesity. Eating behavior is an integral part of nutritional research. MSG consumption is known to enhance the desire to eat more (16). Coordination in energy intake and energy expenditure is evolved in the regulation of body weight and prevention of obesity. Increment in lee index after oral dosage of MSG indicates that intake–expenditure adjustments were delayed or altered after oral consumption of MSG (17).

Unnecessary increment in weight is usually not taken seriously in count but it could be an alarm about many other oncoming disorders. Overweight and obesity actually have a direct correlation with a variety of disorders like hypertension, diabetes, endocrine disturbance, low fertility rate and sometime increased mortality rate. Obesity is a prominent risk factor for the evolution of dyslipidemia and oxidative stress (18,19). Increased caloric intake and obesity is responsible for decreased mitochondrial membrane fluidity and increasing the generation of reactive oxygen species (20). Raised concentrations of reactive

oxygen species alter the homeostatic balance within the body and disrupt many physiological processes (21). However many obese individuals are multiparous but still obesity is a leading cause of female infertility and related health issues (22).

## Conclusions

In light of present findings it is concluded that oral consumption of high dose of MSG is responsible for increment of weight, which further contribute in progression of obesity. Authors strongly recommend the strict monitoring of quantity of MSG consumed per day from all sources. Further studies are advisable to investigate the biological pathway related to MSG induced obesity.

## Acknowledgements

Authors are thankful to Department of Biotechnology, Ministry of Science and Technology, New Delhi, India for providing fellowship to Ms. Tabassum Zafar under DBT Builder program. Authors appreciate the facilitation received from Mr. Ab Qayoom Naik during experiment.

## References

- Hu FB. Overweight and obesity in women: health risks and consequences. *Journal of Women's Health* 2003;12(2):163-72.
- Bentley-Lewis R, Koruda K, Seely EW. The metabolic syndrome in women. *Nature Clinical Practice. Endocrinology & Metabolism* 2007;3(10):696-704.
- Ford ES, Giles WH, Mokdad AH. Increasing prevalence of the metabolic syndrome among US Adults. *Diabetes Care* 2004;27(10):2444-9.
- Moore KL. Congenital malformations due to environmental; developing humans. W.B. Saunders Co. Ltd. Philadelphia. Second edition; 2003;173-83.
- Geha RS, Beiser A. Review of alleged reaction to MSG and outcome of a multicentre double blind placebo- controlled study. *Journal of Nutrition* 2000;130(4):1058S-62S.
- Biodun D, Biodun A. A spice or poison? Is monosodium glutamate safe for human consumption? *National Concord*; 1993;5.

7. Fox GJ, Barthold WS, Davisson TM, Newcomer EC, Quimby F, Smith LA. The Mouse in biomedical research: normative biology, husbandry, and models. Second edition volume 3, 2007;109-10.
8. Hirata AE, Andrade IS, Vaskevicius P, Dolnikoff MS. Monosodium glutamate (MSG)-obese rats develop glucose intolerance and insulin resistance to peripheral glucose uptake. *Brazilian Journal of Medical and Biological Research* 1997;30(5):671-74.
9. Daabees TT, Finkelstein MW, Stegink LD, Applebaum AE. Correlation of glutamate plus aspartate dose, plasma amino acid concentration and neuronal necrosis in infant mice. *Food and Chemical Toxicology* 1985;23(10):887-93.
10. Macho L, Fickova M, Zorad, SJ. Late effects of post-natal administration of monosodium glutamate on insulin action in adult rats. *Physiological Research* 2000;49:79-85.
11. Bakke JL, Lawrence N, Bennett J, Robinson S, Bowers CY. Late endocrine effects of administering monosodium glutamate to neonatal rats. *Neuroendocrinology* 1978;26:220-8.
12. Bernardis LL, Petterson BD. Correlation between lee index and carcass fat content in weanling and adult female rats with hypothalamic lesions. *Journal of Endocrinology* 1968;40(4):527-8.
13. Bunyan J, Murrel EA, Shah PP. The induction of obesity in rodents by means of monosodium glutamate. *British Journal of Nutrition* 1976;35(1):25-39.
14. Walker R, Lupien JR. The safety evaluation of monosodium glutamate. *Journal of Nutrition* 2000;130(4):1049-52.
15. Hermanussen M, Garcia AP, Sunder M, Voigt M, Salazar V, Tresguerres JAF. Obesity, voracity, and short stature: the impact of glutamate on the regulation of appetite. *European Journal of Clinical Nutrition* 2006;60(1):25-31.
16. Diniz YS, Cicogna A, Padovani C, Santana L, Faine L, Novelli ELB. Diets rich in saturated and polyunsaturated fatty acids: metabolic shifting and cardiac health. *Nutrition* 2004;20(2):230-4.
17. Commerford SR, Pagliassotti MJ, Melby CL, Wei Y, Gayles EC, Hill JO. Fat oxidation, lipolysis and free fatty acid cycling in obesity-prone and obesity-resistant rats. *American Journal of Physiology Endocrinology and Metabolism* 2000;279(4):875-85.
18. Acheson KJ. Carbohydrate and weight control: where do we stand? *Current Opinion in Clinical Nutrition and Metabolic Care* 2004;7(4):485-92.
19. Rogers P, Webb GP. Estimation of body fat in normal and obese mice. *British Journal of Nutrition* 1980;43(1):83-6.
20. Feuers RJ. The effects of dietary restriction on mitochondrial dysfunction in aging. *Annals of New York Academy of Science* 1998;20(854):192-201.
21. Nishiyama Y, Ikeda H, Haramaki N, Yoshida N, Imaizumi T. Oxidative stress is related to exercise intolerance in patients with heart failure. *American Heart Journal* 1998;135(1):115-20.
22. Brewer CJ, Balen AH. The adverse effects of obesity on conception and implantation. *Reproduction* 2010;140(3):347-64.