Original Article

Effects of Prolonged Oral Intake of Monosodium Glutamate (MSG) on Body Weight and Its Correlation to Stomach Histopathological Changes in Male Rats

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Abstract

Monosodium glutamate (MSG) is a food additive found in many commercial food products. The safe use of MSG has generated much controversy regarding weight gain and health effects. In the present study, the effects of MSG administration for different periods of time on weight gain and on the structure of gastric mucosa in rats were investigated. Thirty-two adult male rats were used and randomly divided into two treated and two control groups (n=8, each). The rats in the treated groups received a daily oral dose of 395 mg/kg bw of MSG for 3 and 6 weeks, respectively, while the control rats received distilled water for similar periods. The body weight and food consumption were measured. At the time of sacrifice, the stomach was dissected and fixed for routine histological procedures. Results revealed a steady increase in body weight and food consumption untill the 4th week in the treated groups. This was followed by a reduction in body weight, although food consumption continued to increase. The gastric mucosa of the rats treated for 3 weeks showed a number of pathological alterations which were more pronounced in the group treated for 6 weeks. These results indicate that prolonged administration of MSG causes an initial increase in weight gain followed by terminal suppression, independent of food consumption. This may be explained by the induced gastric mucosal damage. In conclusion, it appears that prolonged intake of MSG induces gastric damage which, consequently, leads to decreased body weight.

Keywords: additive, consumption, food, gastric, MSG, weight

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Introduction

Monosodium glutamate (MSG), known as AJI-NO-MOTO or white magi, is a salt derivative of the amino acid glutamic acid and is used as a food additive in many commercial food products, *e.g.* meat, fish, cheese and vegetables (IFIC Review of Monosodium glutamate, 2009).

MSG is used to enhance the flavor of many types of food as it stimulates receptors located in the taste buds, providing an expansion of taste (Diniz et al., 2005). MSG is frequently added to processed food, particularly in Asian cuisine, to improve food palatability and acceptance, to increase salivary flow and to reduce oral complaints (Li et al., 2002). Accordingly, the global MSG production and consumption have increased considerably in recent decades (Shi et al., 2010).

Regarding the association of MSG with increasing risk of overweight, information obtained from human or experimental studies in animals is controversial and remains to be verified (Ebert, 2009). Recent studies on healthy Chinese subjects reported that MSG consumption was positively linked to increased risk of overweight (He et al., 2008 and 2011). Additional alarms over MSG have been raised in response to reports of its association with overweight and obesity (Collison et al., 2010). On the contrary, Shi et al. (2010) reported that MSG intake was not associated with a higher prevalence of obesity or with a clinically significant weight gain in Chinese adults. Data from animal studies suggested a possible link between MSG and obesity, where weight gain was found to be significantly greater in MSG-treated animals, and that this might be independent of an increase in appetite (Iwase et al., 2000) or due to an improvement in the palatability of foods by exerting a positive influence on the appetite center (Hermanussen and Tresguerres, 2003; Hermanussen et al., 2006). Other studies demonstrated that MSG did not increase food intake or induce obesity (Boutry et al., 2011), and some even demonstrated that MSG administration to rats was associated with suppression of body weight gain, fat deposition, and plasma leptin levels (Kondoh and Torii, 2008).

In another respect, several studies reported damaging effects of MSG; on small doses of 15 and 30 mg/kg for 10, 20, and 30 days; on the gastric mucosa (Falalieieva et al., 2010), ileum (Eweka and Om'Iniabohs, 2007), and liver (Soliman, 2011). These reports indicate that MSG might have some deleterious effects on the gastrointestinal tract, which in turn might be associated with mal-digestion and mal-absorption of ingested foods.

Since MSG still represents an important ingredient in food preparations, and because of its suspected actions within the gastrointestinal tract, it is necessary to investigate the changes in body weight after oral administration of MSG in correlation with the histological changes of gastric mucosa.

Materials and Methods

Test materials: Monosodium glutamate (MSG) manufactured by SPECTRUM Laboratory Products Inc was purchased from local dealers in Jeddah, Saudi Arabia. It was stored and protected from direct sunlight until time of administration.

Animals: Thirty-two adult male Sprague-Dawley rats (275-300 g in weight) were obtained from, and maintained in the animal house facility of King Fahd Medical Research Center, King Abdulaziz University. The rats were kept in separate metallic cages under standard temperature (25 ± 1°C), humidity (50 ± 5%) and lighting (12h: 12h Light: Dark cycles) conditions. The rats were fed a standard chow diet ad libitum with free access to water for one week before the beginning of the study. This study was approved and registered by the Committee of Animal Investigations, Department of Anatomy, Faculty of Medicine, King Abdulaziz University.

Experimental design: The rats were randomly divided into four groups (n = 8, each), group I rats were given distilled water for three weeks (control for group II animals), group II rats were given MSG for three weeks, group III rats were given distilled water for six weeks (control for group IV animals), and group IV rats were given MSG for six weeks.

In the MSG-treated groups, MSG was administered daily at 10:00 AM through oral gavage at a dose of 395 mg/ kg bw/day (1/40 of rat's oral LD_{50}) (MSDS, 2013). MSG fresh solution was prepared every two days and kept in the fridge until the time of use. In the control groups, distilled water was administered at the same regimen.

Growth and food consumption assessment: Growth assessment was carried out using the following parameters; initial body weight, final body weight, body weight gain and percentage of body weight gain. The body weights of the rats in different study groups were measured individually every three days.

Food consumption assessment was carried out using the following parameters; daily food intake, total food intake, and food conversion ratio (FCR). The amount of daily food intake was calculated as the difference between the weight of food that remained in the food bin (D_a) and the amount placed one day before (D_b). These data were then used to calculate a daily average food intake according to the formula *Average food intake* = $D_b - D_a$.

Food Conversion Ratio (FCR) was determined by the following formula: *FCR = Food intake / Weight gained (Wf – Wo)*, where Wo is the initial body weight and Wf is the final body weight at the end of the study period (Sahzadi et al., 2006; Makni et al., 2008).

Histological study: At the end of the experimental periods (3 & 6 weeks), the animals were anesthetized with ether and sacrificed at 10:00 AM on the assigned day in all groups. The animals were dissected and small pieces from the cardiac portion of the stomach were quickly excised and fixed in 10% formol saline. The specimens were then dehydrated in ascending grades of alcohol, cleared in xylene and embedded in paraffin wax. Serial sections (4-5 µm thick) were

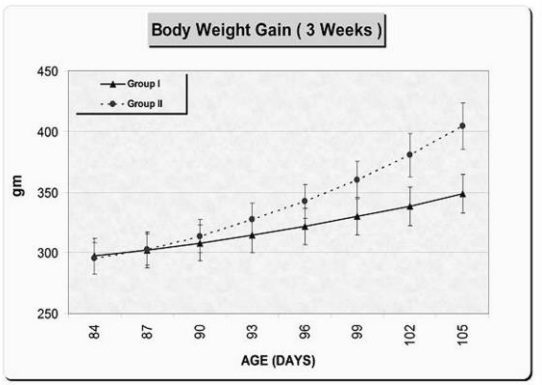

Figure 1 Mean body weight gain of groups I & II. Notice that the rate of weight gain was higher in group II.

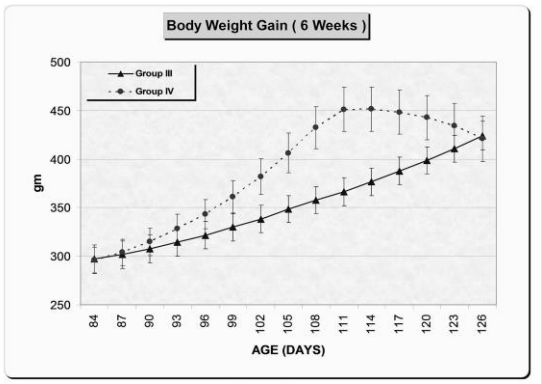

Figure 2 Mean body weight gain of groups III & IV. Notice that the rate of weight gain was higher in group IV until the middle of 4th week, and then followed by a gradual decrease.

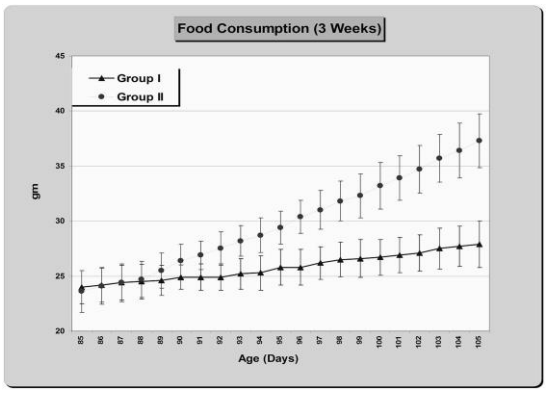

Figure 3 Daily food consumption in groups I & II. Notice that the rate of food consumption was higher in group II.

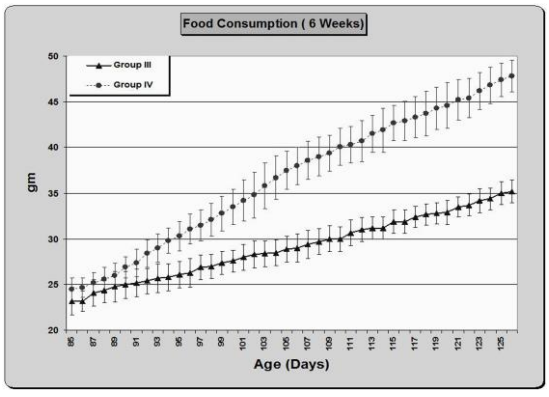

Figure 4 Daily food consumption in groups III & IV. Notice that the rate of food consumption was higher in group I

obtained using a rotatory microtome. The deparaffinized sections were stained with haematoxylin & eosin (H&E) and Masson's trichrome (MTC) stains. Photomicrographs were obtained using a digital research photographic microscope in the Anatomy Department, King Abdulaziz University.

Statistical analysis: Data were represented as means ± SD. The means were compared for statistical significance using Student's *t* test for unpaired data. Differences were considered significant at $p<0.05$. All statistical analyses were performed using SPSS software, version 14.

Results

Growth Assessment

All rats from all groups survived to the end of the study period. The growth assessment for all groups is presented in Table 1, Figure 1 and 2. The initial body weights were approximated among the study groups. The mean of the final body weight in group II was significantly higher than that of group I ($p<0.001$). In group IV, the mean value of the final body weight was not significantly different from that of group III.

A time-dependent steady increase in body weight was observed in groups I, II, and III throughout the study period. The same result was observed in group IV up until the middle of the 4th week (Fig 2), after which a gradual decrease in body weight was observed until the end of the study period. There was a significant increase in the weight gain in group II compared to group I ($p<0.001$) and group IV ($p<0.01$). The percentage of body weight gain in group IV was significantly higher than that in group II ($p< 0.001$). This percentage was significantly higher in group II compared to group I ($p<0.0001$).

Food Consumption

Food consumption increased steadily in all groups. However, it showed a higher rate of increase in groups II& IV (Table 1, Fig 3-4). Daily food intake was significantly higher in group II compared to group I ($p<0.001$). Moreover, this intake was significantly higher in group IV compared to groups III and II ($p<0.001$). Total food intake was significantly higher in group II compared to group I ($p<0.001$). It was also significantly higher in group IV compared to groups III and II ($p<0.0001$). Food Conversion Ratio (FCR) was reduced significantly in group II compared to group I ($p<0.0001$). However, it was significantly higher in group IV compared to groups III ($p<0.001$) and II ($p<0.0001$).

Histological results

Examination of stomach sections from the control rats (groups I&III, Fig 5) showed normal histological features of gastric mucosa which consisted of surface epithelium, gastric glands, lamina propria and muscularis mucosa. The surface epithelium consisted of simple columnar cells with acidophilic cytoplasm and basal oval nuclei. The lamina propria was occupied by simple branched tubular adjacent glands, which were straight and perpendicular to the surface epithelium, and were lined by mucous neck

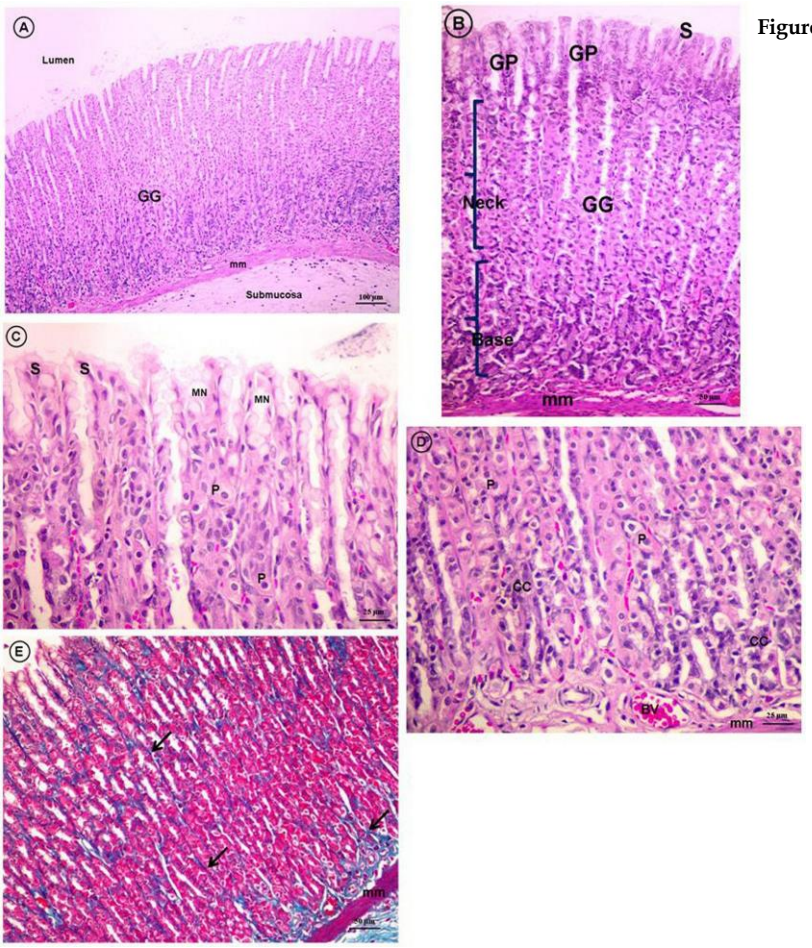

Figure 5 Photomicrographs of stomach from control rats (groups I & III) showing: (A) Mucosa and part of submucosa and muscularis mucosa (mm). The mucosa contains simple branched tubular glands (GG). (H & E, X 100); (B) The full length of gastric glands (GG); neck and base in the mucosa. Notice the superficial epithelial cells (S), gastric pits (GP) and muscularis mucosa (mm). (H & E, X 200); (C) The upper parts of the gastric glands, which were lined by mucous neck cells (MN) with flat basal nuclei and pale cytoplasm, P (parietal cells with central rounded nuclei and acidophilic cytoplasm), and superficial epithelial cells. (H & E, X 400); (D) The lower parts of the gastric glands. Notice the presence of abundant peptic cells (CC) with rounded nuclei and basophilic cytoplasm, parietal cells, BV (blood vessels), and muscularis mucosa (mm). (H & E, X 400); and (E) Presence of little amount of connective tissue (appeared as blue strands) in the lamina propria (LP) and around the basal parts of the glands (↑), and muscularis mucosa (mm). (Masson's trichrome, X 200).

Table 1 Effect of MSG intake on growth and food consumption in male rats (Mean ± SD)

	Group I	Group II	Group III	Group IV
Initial body weight (gm)	297.3 ± 14.8	295.5 ± 12.9	297.0 ± 14.6	296.2 ± 13.3
Final body weight (gm)	348.7 ± 16.1	404.7 ± 19.2[a]	424.2 ± 14.6	420.8 ± 23.3
% of body weight gain	17.21 ± 0.99	36.94 ± 2.05[b]	42.98 ± 4.16	42.01 ± 1.96
Daily food intake (gm)	25.79 ± 0.27	29.81 ± 0.31[a]	29.12 ± 0.37	36.61 ± 0.7[c,e]
Total food intake (gm)	541.6 ± 31.24	626.1 ± 36.7[a]	1223 ± 49.04	1537.7 ± 70.3[c,f]
Food Conversion Ratio (FCR)	10.56 ± 0.66	5.75 ± 0.3[b]	9.67 ± 0.97	12.4 ± 0.89[c,f]

Student t-test:
a- $p<0.001$ compared to group I b- $p<0.0001$ compared to group I c- $p<0.001$ compared to group III
d- $p<0.01$ compared to group IV e- $p<0.001$ compared to group II f- $p<0.0001$ compared to group II

cells, parietal cells and peptic cells. The mucous neck cells were most frequent in the necks of the glands and had flat basal nuclei and pale cytoplasm. The parietal cells appeared large with central pale nuclei and eosinophilic cytoplasm and were numerous at the middle parts of gastric glands. The peptic (chief) cells predominated in the basal parts of gastric glands and appeared as low columnar cells with rounded, condensed, basally located nuclei and strongly basophilic cytoplasm. Sections stained with MTC showed little connective tissue in the thin lamina propria and between the basal parts of the gastric glands.

Examination of stomach sections from the rats in group II (Fig 6) showed various degrees of damage in the gastric mucosa. Some gastric glands appeared irregular with wide lumina. Others were widely separated with the presence of congested blood vessels. The mucous neck cells appeared enlarged and encroached on the lumen. Some of the parietal cells were swollen with pale and granular cytoplasm; while others showed vacuolated cytoplasm with pyknotic nuclei. The peptic cells were markedly condensed and some of their nuclei were flattened and compressed. Sections stained with MTC showed partial increase in the thickness of the connective tissues in the lamina propria around gastric glands compared to the control.

Examination of stomach sections from the rats in group IV (Fig 7) revealed more damage and variable degrees of distortion in the gastric mucosa. Some areas of gastric mucosa showed sloughing or partial loss of surface epithelium. In some other areas the gastric glands showed disorganization in the form of atrophied, distorted and irregular glands with wide lumina. Numerous congested blood vessels of variable size were observed in the mucosa and submucosa. The parietal cells were more accumulated at the bases of the partially- or completely vacuolated cytoplasm and

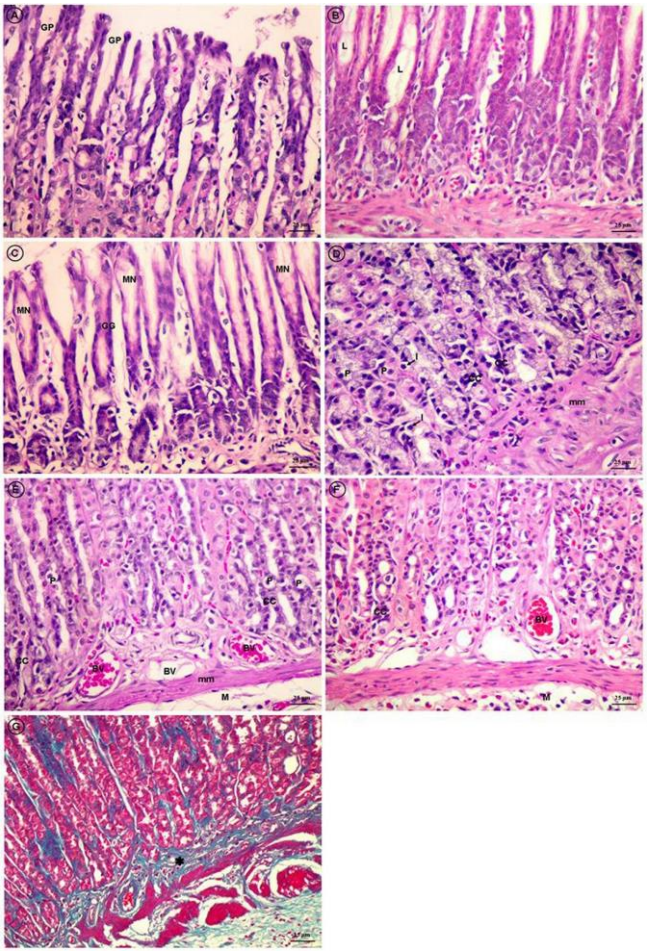

Figure 6 Photomicrographs of stomach from rats in group II showing: (A) Dilated and irregular gastric pits (gp). (H & E, X 400); (B) Irregular gastric glands (GG) with dilated lumina (L). (H & E, X 400); (C) Mucous neck cells (MN) that were enlarged and encroached on the lumen of the gastric glands (GG). (H & E, X 400); (D) Some parietal cells exhibited partial or complete vacuolation of cytoplasm (P), while others exhibited pyknotic nuclei (I). Notice the presence of condensed peptic cells (CC). (H & E, X 400); (E) Parietal cells in the basal glandular part exhibited complete vacuolation of cytoplasm (P). Notice the condensed peptic cells (CC) and congested blood vessels (BV). (H & E, X 400); (F) Peptic cells were markedly condensed and some of their nuclei were flattened and compressed (CC) with the presence of congested blood vessels (BV). (H & E, X 400); and (G) Increase in the amount of the connective tissues in the lamina propria around gastric glands in the form of condensed bluish coloration (✱). (Masson's trichrome, X 400). M= mucosa, mm= muscularis mucosa.

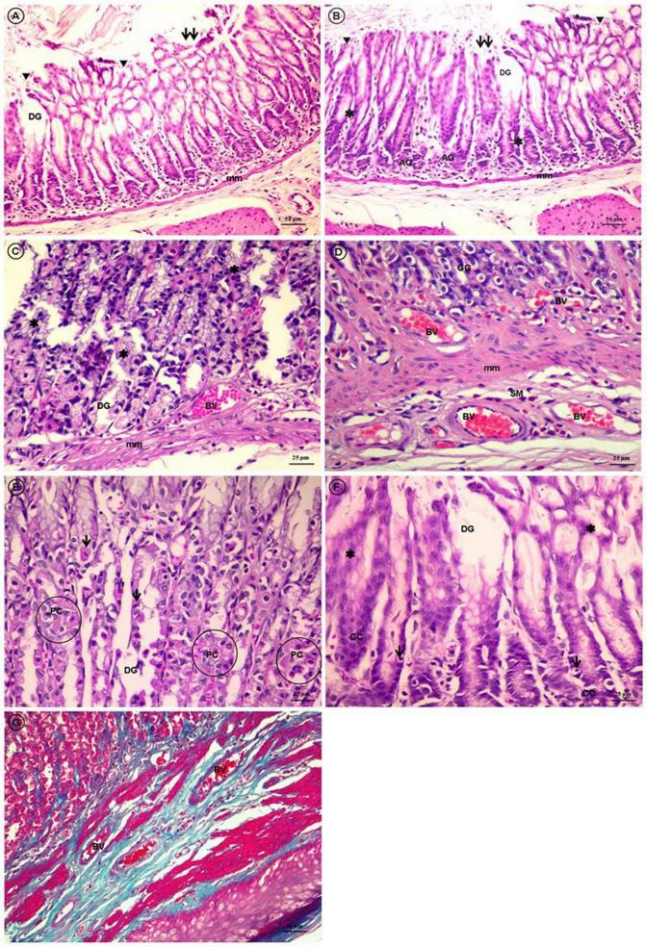

Figure 7 Photomicrographs of stomach from rats in group IV showing: (A) Some sloughed areas of gastric mucosa (⇓⇓) or partial loss of surface epithelium (▼). Notice that some gastric glands appear dilated (DG). (H & E, X 200); (B) Distorted (✱) or atrophied gastric glands (AG) which were separated by irregular spaces and are dilated (DG). (H & E, X 200); (C) Distorted gastric glands (✱), congested and dilated blood vessels in the lamina propria (BV), and dilated glands (DG). (H & E, X 400); (D) Multiple congested and dilated blood vessels (BV) between the lower parts of the gastric glands and in the submucosa (SM), and muscularis mucosa (mm). (H & E, X 400); (E) The parietal cells were more accumulated at the base of the glands (inside the circle), where they coalesced and showed loss of cell boundaries. These cells exhibited either partially or completely vacuolated cytoplasm (PC). Some parietal cells showed pyknotic nuclei (⇓). There are also some dilated glands (DG) (H & E, X 400); (F) Distorted gastric glands (✱). The peptic cells were markedly condensed and some of their nuclei were flattened and compressed (CC). Other cells showed pyknotic nuclei and vacuolated cytoplasm (⇓).There are also dilated glands (DG). (H & E, X 400); and (G) Presence of marked increase in connective tissues in the lamina propria and around the basal parts of the gastric glands. (Masson's trichrome, X 400).

some showed complete vacuolation with small dark pyknotic nuclei. The peptic cells were markedly condensed and some of their nuclei were flattened and compressed. Other cells had small pyknotic nuclei and many vacuoles. Sections stained with MTC showed excessive connective tissue in the thick lamina propria and in between the bases of gastric glands and at the base of the ulcer.

Discussion

In the present study, the effects of short- and long term administration of MSG on body weight and food consumption were assessed in rats. The results show that the intragastric administration of MSG for a short time led to increased weight gain and food consumption, while the prolonged administration of MSG caused an initial increase in weight gain followed by terminal suppression, although food consumption continued to increase. This is supported by the calculation of FCR ratio each group, which exhibited reduction in group II compared to group I and increase in group IV compared in to group III, indicating the continuous increase of food intake in group IV in spite of the decreased body weight. Previous studies on experimental animals suggested a possible link between MSG and obesity, where weight gain was found to be significantly greater in MSG-treated animals compared to controls and that this might be due to an increase in appetite or even with consumption of similar amounts of food (Iwase et al., 2000), and improvement in the palatability of foods by exerting a positive influence on the appetite centre (Hermanussen and Tresguerres, 2003; Hermanussen et al., 2006). On the contrary, other studies reported that long term adminstration of MSG did not increase food intake or induce obesity (Boutry et al., 2011). Furthermore, other studies on rats showed an association of MSG with suppression of body weight gain, fat deposition, and plasma leptin levels which were probably related to the increase in energy expenditure as food intake was not altered by the ingestion of MSG (Kondoh and Torii, 2008; Kondoh et al., 2009).

The mechanisms of action that would allow MSG to promote obesity are not clear. Different studies were carried out in order to understand the relationship between MSG and obesity. These studies reported that chronic MSG intake might intoxicate the arcuate nucleus and disrupt the hypothalamic signaling cascade of leptin action, causing leptin resistance related to overweight/obesity (Hermanussen and Tresguerres, 2003; Hermanussen and Tresguerres, 2005). Morover, the observed weight gain associated with MSG intake might be due to destruction of several brain regions (including the hypothalamus) involved in appetite and energy metabolism (Monno et al., 1995; Kondoh and Torii, 2008; Kondoh et al., 2009).

Regarding the gastric mucosa, the findings of the present study indicate that MSG has deleterious effects on the structure of the stomach, where there were various degrees of damage in the gastric mucosa of the rats treated for 3 weeks that became more pronounced in the group treated for 6 weeks.

The above findings are in agreement with previous studies of both small doses and short periods of exposure to MSG which reported that it led to erosive and ulcerative lesions of the gastric mucosa and an increased secretion of hydrochloric acid (Eweka et al., 2007; Falalieieva et al., 2010), and an increase in the thickness of the gastric mucosa (Numan et al., 2012).

In the present study MSG resulted in marked affection of gastric glands in the form of irregular and distorted glands together with degenerative changes of cells lining the glands. These findings are consistent with clinical findings of many investigators who reported prevalence of gastric symptoms like heart burn, epigastric pain, nausea and vomiting associated with MSG intake (Yang et al., 1997). Furthermore, our findings are in agreement with some researchers (Eweka et al., 2007) who showed evidence of increased degenerative and atrophic changes in the gastric epithelium and glands in rats given MSG.

The reported gastric changes could be explained by the existence of a glutamate-sensing system in the gastrointestinal tract, *e.g.* glutamate receptor subtype 1 is located in the rat gastric mucosa (San Gabriel et al., 2005,2007), as well as vagal gastric afferent fibers, which specifically respond to intra-gastric administration of glutamate (Niijima, 2000; Uneyama et al., 2006). Physiological studies reported that intragastric administration of MSG increased the firing rate of afferent fibers in the gastric branch of the rat vagus nerve resulting in increased acid secretion (Uneyama et al., 2006). In another explanation, it has been established that MSG is a potentiating factor of gastric acid secretion as it enhanced the secretagogue-effect, which acts by releasing histamine, which in turn is required for gastric acid secretion (Prinz et al., 1993).

In conclusion, the results of the present work show that prolonged administration of MSG causes an initial increase in weight gain followed by terminal suppression, despite increased food consumption. This could be explained by the inefficient food digestion and absorption due to gastric mucosal damage induced by MSG administration. It thus should be taken into consideration that long-term intake of MSG may lead to aggravation of gastric damage and decreased body weight.

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บทคัดย่อ

ผลกระทบของการบริโภคโมโนโซเดียม กลูตาเมต (MSG) เป็นระยะเวลานานต่อน้ำหนักตัว และความสัมพันธ์กับการเปลี่ยนแปลงพยาธิสภาพของกระเพาะในหนูแรทเพศผู้

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โมโนโซเดียม กลูตาเมต เป็นสารเติมแต่งอาหารที่พบในผลิตภัณฑ์อาหารเชิงพาณิชย์ ความปลอดภัยในการใช้ MSG ยังมีความขัดแย้งมากเกี่ยวกับน้ำหนักที่เพิ่มขึ้นและ ผลกระทบต่อสุขภาพ การศึกษาครั้งนี้ได้ศึกษาผลกระทบของการบริโภค MSG ในระยะเวลาที่แตกต่างกันต่อการเพิ่มขึ้นของน้ำหนัก และโครงสร้างของเยื่อบุกระเพาะอาหาร (gastric mucosa) ในหนูแรท โดยได้แบ่งหนูแรทเพศผู้จำนวน 32 ตัว เป็นสี่กลุ่มการทดลอง เป็นสองกลุ่มที่ได้รับ MSG และสองกลุ่มควบคุม (n=8) หนูในกลุ่มที่ได้รับ MSG จะได้รับ 395 มิลลิกรัมของ MSG ต่อกิโลกรัมน้ำหนักตัวต่อวัน เป็นเวลา 3 และ 6 สัปดาห์ ในขณะที่หนูในกลุ่มควบคุมได้รับน้ำกลั่นเป็นระยะเวลาที่เท่ากัน ศึกษาน้ำหนักของร่างกาย การบริโภคอาหาร และจุลพยาธิวิทยาของกระเพาะอาหาร พบว่า น้ำหนักของร่างกายและการบริโภค มีการเพิ่มขึ้นอย่างต่อเนื่องจนถึงสัปดาห์ที่ 4 ใน กลุ่มที่ได้รับ MSG ตามด้วยการลดลงของน้ำหนักตัวถึงแม้จะมีการบริโภคอาหาร เพิ่มขึ้น ผลทางจุลพยาธิวิทยาของเยื่อบุกระเพาะอาหารของหนูที่ได้รับ MSG เป็นเวลา 3 สัปดาห์ พบว่ามีการเปลี่ยนแปลงทางพยาธิวิทยาที่เด่นชัดมากกว่าในกลุ่มได้รับ MSG เป็นเวลา 6 สัปดาห์ ผลการศึกษาครั้งนี้พบว่า การให้ MSG เป็นเวลานานทำให้เกิดการเพิ่มขึ้นของน้ำหนักของร่างกายในช่วงแรกตามด้วยการลดลงในตอนท้าย ซึ่งไม่ขึ้นกับปริมาณของการบริโภคอาหาร ซึ่งอาจจะมีความเกี่ยวข้องกับความเสียหายที่เกิดกับเยื่อเมือกในกระเพาะอาหาร ในการทดลองครั้งนี้สรุปได้ว่า การบริโภค MSG เป็นเวลานานก่อให้เกิดความเสียหายในกระเพาะอาหาร ซึ่งส่งผลทำให้เกิดการลดลงของน้ำหนักตัวของหนูแรทเพศผู้ได้

คำสำคัญ: สารเติมแต่งอาหาร การกิน อาหาร กระเพาะ โมโนโซเดียม กลูตาเมต น้ำหนักตัว

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