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Effect Of Dietary Monosodium Glutamate on Some Dipose- derived Hormones in Adult Male Rats

Mohammad R.S.AL-Attabi
Department of Biology- College of Science - Wasit University, Iraq.
Nameer A. Kareem Alzubaidi
Department of Public Health- College of veterinary medicine- University of Basrah, Iraq.
Nasreen H. H. Al-Moussawi
Department of Soil and Water, Marshes researches center , University of Thi-Qar, Iraq
Author for corresponding: E-mail nameer.physiology@gmail.com

ABSTRACT

The current study was conducted to investigate the effect of dietary monosodium glutamate (MSG) on serum adiponectin, leptin, resistin hormone, lipid profile, body weights, food intake, and abdominal fat in adult male rats. Twenty four adult male rats weighing 180 -200 gram and 10 – 12 weeks old were randomly divided in to three equal groups. Group A fed with standard diet and serve as control group. Groups B and C were fed standard rat diet with 2 and 4 g/kg of MSG respectively for thirty days. At the end of experiment the animals were weighted, sacrificed, and blood samples were collected by heart puncture to test lipids profile, adiponectin, leptin, resistin hormones, and relative abdominal fat were calculated. The results showed a significant increase ($p \leq 0.05$) in body weights, relative abdominal fat, serum TC, TAG, LDL-c, and VLDL-c, leptin hormone, and resistin hormone with increasing MSG in the diet, whereas serum HDL-c and adiponectin hormone decreased with increasing MSG in the diet compared with control group.

Key words : Monosodium glutamate; Adipose derived hormone; Male rats;

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1- INTRODUCTION

Monosodium Glutamate (MSG or E 621) is one of the most widely used food additives all over the world which is a part of many commercial foods like bouillon cubes , frozen food , canned food , snack chips, soups , salad dressing as a flavor enhancer . MSG increases the sapidity of food and produces a taste that cannot be provided by other foods. It evokes a flavor described in Japanese as umami, which mean savory [1]. The average intake of MSG in United Kingdom in 1991 was 580 mg/day for general population person , 4.68 gram/day for excessive users [2]. The evaluated average daily MSG intake in industrialized countries is 300–1000 mg / person , it depends on the MSG content in foods and an individual's taste preferences [3]. The LD_{50} in rats and mice is 15 –18 g/kg b. w. [4]. Fat tissues produces several hormones (e.g. leptin, adiponectin, resistin) that involved in regulation of energy balance, metabolism, and neuroendocrine response to changed nutrition [5]. Adiponectin is a protein hormone secreted from adipose tissue , it is exist in human blood in high concentration that accounts for up to 0.01-0.05% of total plasma protein [6,7]. Decreased circulating adiponectin concentration has been associated with several disorders like obesity, dyslipidemia , insulin resistance, type 2 diabetes, essential hypertension , and cardiovascular diseases [8]. Leptin is adipose derived protein hormone control the regulation of appetite and metabolism. Leptin is secreted mostly by white adipose tissue and its concentrations are related to the amount of adipose tissue and affects both hunger and food consumption . Leptin inhibits food intake and stimulates energy expenditure by acting via hypothalamic leptin receptors, and also has peripheral effects, like inhibition of liver and white adipose tissue lipogenesis rate and lipolysis stimulation in adipocytes [9]. The plasma concentration of leptin and its expression in adipocytes are both correlated positively with total adiposity. Adiposity refers to the amount of fat found in adipose tissue . Increased visceral fat has been associated with increase cytokines production and development of insulin resistance [10.11]. Resistin, also is an adipocyte-derived hormone enhance insulin resistance and inflammation , interfere with adipocyte differentiation [12,13]. Resistin originally identified in rats as a protein produced and secreted by adipocytes under the control of different humoral signals, nutrition , nutritional status and metabolism regulate this adipokine. Circulating resistin is increased in obese insulin resistant rodents [14]. whereas resistin is decreased in fasting situations [15] based on the above the aim of the current study to determine the effects of dietary MSG on adipose – derived hormones , lipid profile , body weights and relative abdominal fat in male rats.

2-MATERIALS AND METHODS

2.1.Chemical

Commercial monosodium glutamate Purity 99% (Chinese salt) manufactured by Ajinomoto co.INC. Tokyo, Japan was used in the present study.

2.2. Animals and experimental design

The present study was performed at Department of Biology - College of Science - Wasit University. Twenty four adult male rats weighing about 180–200 grams and 10 – 12 weeks old were used in the current study. Animals were kept under normal temperature (22 - 25 °C) , and controlled lightening . Animals were randomly divided into three equal groups each group consisted of 8 adult male rats as in the following :-

1- Group A : Fed with standard rat diet + 2 g/kg monosodium glutamate for 30 days.

2- Group B : Fed with standard rat diet + 4 g/kg monosodium glutamate for 30 days..

3- Group C : Fed with standard rat diet for 30 days and serve as control group .

All animals were weighted at the beginning of the experiment and at the end of experiment, then animals were sacrificed and blood samples were collected by heart puncture , serum was separated , frozen at – 20 until used .

2.3. Laboratory analysis

Serum Total Cholesterol (TC) , Triglyceride (TAG) , High Density Lipoprotein cholesterol (HDL-c) were estimated by enzymatic colorimetric method using spectrophotometer by use liquicolor kits manufactured by human diagnostic company (Germany) .

The serum Low density Lipoprotein cholesterol (LDL-c), very low density lipoprotein (VLDL-c) were calculated according to (Friedewald *et al.*, 1972)[16].

LDL = Total cholesterol (TC) - HDL - TG/5 (mg/dL).

VLDL-C = TAG / 5 (mg/dL).

Serum Adiponectin ,Leptin , and resistin hormones levels were determined using enzyme linked immunosorbent assay (ELISA) kits manufactured by GenWay BIOTECH INC. (USA).

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2.4. Statistical Analysis

Data were expressed as mean ± SD. The comparisons between groups were performed with analysis of variance (ANOVA) by using computerized SPSS program (Statistical Program for Social Sciences). P<0.05 was considered to be the lest limit of significance.

3- RESULTS

3.1. Effects of dietary MSG on body weight gain

The results in table (1) showed that there were a significant increase ($p \leq 0.05$) in final body weights of animals in groups A and B compared with final weight of animals in group C (control group) , also there were a significant increase ($p \leq 0.05$) in final body weight of group B compared to the final weight of animals in group A.

Food intake increase significantly ($p \leq 0.05$) in male rat fed with monosodium glutamate containing diet (groups A and B) compared with control group (group C) as shown in table (1) ,there were a significant increase in food intake in group B compared with food intake in group A.

Relative abdominal fat increased significantly ($p \leq 0.05$) in group A and B compared with group C , there were a significant increase in relative abdominal fat weight in group B compared with group A

Table (1) effect of dietary monosodium glutamate (2 gram / kg of diet and 4 gram / kg of diet for 30 days) on male rats body weights , food intake and abdominal fat .

Groups / parameters	Group A	Group B	Group C
Initial body weight (gram)	191.12 ± 7.4 a	189.62± 6.3 a	187.12 ± 5.4 a
Final body weight (gram)	218.00 ± 7.7 b	235.75 ± 7.9 a	195.62 ± 6.4 c
Food intake (Rat/day/gram)	19.87 ± 1.8 b	22.37 ± 2.8 a	14.62 ± 2.4 c
Relative abdominal fat (g / 100 g B.W.)	3.35 ± 0.5 b	4.20 ± 0.7 a	1.90 ± 0.4 c

numbers represent the mean ± Standard Deviation .

Different letters indicated significant differences between groups at level p<0.05

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3.2. Effects of dietary MSG on lipids profile

The results presented in table (2) reveals that dietary MSG in both concentrations (2 g/kg of diet in group A and 4 g/kg of diet in group B) caused an increase in serum TC , TAG , LDL-c , and VLDL-c compared with group C , also there were a significant ($p \leq 0.05$) increase in serum TC , TAG , LDL-c , and VLDL-c in group B compared with group A

As shown in table (2) serum HDL-c decreased significantly in group A and B compared with group C , as well as serum HDL-c decreased significantly in group B compared with group A .

Table (2) effect of dietary monosodium glutamate (2 gram / kg of diet and 4 gram / kg of diet for 30 days)on male rats serum lipids profile .

Groups / parameters	Group A	Group B	Group C
TC	145.6 ± 14.5 b	163.8 ± 12.2 a	102.7 ±11.6 c
HDL-c	41.0 ± 3.1 b	31.5 ± 4.4 c	52.5 ±4.2 a
TAG	88.1 ± 3.9 b	99.3 ± 6.1 a	76.9 ± 4.7 c
LDL-c	52.4 ± 3.1 b	58.1 ± 4.2 a	42.0 ± 3.3 c
VLDL-c	22.1 ± 1.4 b	24.6 ± 1.4 a	15.8 ± 2.5 c

numbers represent the mean ± Standard Deviation .

Different letters indicated significant differences between groups at level $p<0.05$

3.3. Effects of dietary MSG on some adipose – derived hormones .

Table (3) shows that serum leptin hormone increased significantly ($p \leq 0.05$) in MSG fed rats (group A and B) compared with group C , as well as serum leptin in group B increased significantly compared with group A .

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Serum adiponectin hormone levels decreased significantly in MSG containing diet to be at the lowest levels in group B and A compared with group C , meanwhile serum adiponectin in group B was decreased significantly compared with adiponectin level in group A .

Resistin hormone serum levels increased significantly in groups A and B compared with group C , there were no significant differences between group A and B when compared with each other .

Table (2) effect of dietary monosodium glutamate (2 gram / kg of diet and 4 gram / kg of diet for 30 days) on some adipose – derived hormones in male rats.

Groups / Hormones	Group A	Group B	Group C
Leptin (ng/ml)	**16.25 ± 2.60 b**	**24.87 ± 3.97 a**	**5.44 ± 1.39 c**
Adiponectin (µg /ml)	**3.65 ± 0.55 b**	**2.50 ± 0.40 c**	**4.77 ± 0.69 a**
Resistin (ng/ml)	**4.17± 0.47 a**	**4.45 ±0.81 a**	**2.46 ± 0.40 b**

numbers represent the mean ± Standard Deviation .

Different letters indicated significant differences between groups at level p<0.05

4- DISCUSSION

The significant increase in body weights in rats fed with both concentrations of MSG (table 1) may be caused by increased food intake as documented in the present study. This increment in food intake may be due to leptin resistance which characterized by hyperleptinaemia [17]. Leptin play a key role in regulation of body weight by increased energy expenditure and reduce food intake [18]. The results of the current study are in agreement with Diniz *et al.* (2005)[19] who reported that dietary MSG cause increase in body weight and food intake . Whereas Kondoh and Torii (2008) [20] suggested that MSG ingestion reduces body weight, plasma leptin levels and body fat mass. The conflicting results may be depend on the duration of experiment or genus of rats . according to Leitner and Bartness (2008)[21] and Collison *et al.* (2010)[22] rats that orally consume MSG exhibit increased adiposity as well as increased leptin resistance .The significant decrease in serum adiponectin shown by the present study could be considered as another reason for the increased body weight , food intake , and abdominal fat based on the known role of adiponectin in increased lipid metabolism , fatty acid oxidation in skeletal muscle [23]. It seem from the results of lipid profile presented in table (2) that serum total cholesterol , bad cholesterol , triglyceride and VLDL-c are elevated in both groups fed with SGM the possible explanations may be due to increased serum leptin which correlated positively with serum cholesterol , LDL-c , triglyceride and consequently VLDL-c whereas it correlated negatively with HDL-c [24]. The decreased serum level of adiponectin in MSG fed rats can also explain the results of lipid profile , according to Izadi *et al.* (2013)[25] who reported that serum adiponectin inversely correlated with VLDL-C and LDL-c and triglycerides and directly correlated with serum HDL-c . The results of the current study regarding to dyslipidaemic effects of MSG are in agreement with Okediran *et al*. (2014)[26]. In contrast Maclewè *et al*. (2013)[27] reported that dietary MSG in rats did not affect the body gain and lipid profile in

experimental groups. With regard to the results related to leptin , adiponectin and resistin hormones that presented in table (3) , the elevated serum leptin hormone represented the best hormone markers for obesity [28] can be attributed to the increased abdominal fat and leptin resistance ,leptin resistance characterized by increase circulated leptin level [29] increase appetite [30](Klok *et al*. , 2007) which documented in the current study. The elevation in resistin hormone can considered as an important sign of obesity and insulin resistance [31]. We can concluded from the present study that dietary MSG can induce obesity and insulin resistance by affecting leptin , adiponectin , and resistin hormone secretion and action .

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