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RESEARCH PAPER

THE EFFECT OF MONOSODIUM GLUTAMATE (MSG) ON BLOOD GLUCOSE IN ADULT RABBITS AS MODELS

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ABSTRACT

This study investigates the effect of monosodium glutamate on fasting blood glucose. 18 adult rabbits (1.6 ± 0.20 Kg), procured from the animal house at the College of Medicine, Ambrose Alli University, Ekpoma, and transferred to the Physiology Laboratory of the same institution were used for this study. The animals were grouped into two: groups A (control) and B (Test B1 and Test B2). Group A received water and feed (grower's mash) with grass supplementation. Group B (Test B_1 and B2) received 3.33mg/ml and 6.66mg/ml of MSG respectively at libitum. At the end of each week, fasting blood glucose levels were determined using glucose test strips by Betacheck, Australia, according to manufacturer's instructions. Blood samples were also collected via venipuncture. Statistical analysis showed that there was no significant difference in blood glucose levels during acclimatization. However, B1 and B2 presented significant weekly increases in blood glucose levels especially amongst group B2 rats. This study suggests therefore, that MSG has the potential to induce elevations in blood glucose level and consequently, diabetes mellitus in animals.

Key words: ***Monosodium glutamate, Ajino-moto, Blood glucose level, Diabetes mellitus, Obesity.***

INTRODUCTION

Diabetes mellitus (DM) is recognized as the world most common endocrine disorder (WHO, 1999; 2003; Ingrid and Mathias, 2006) and a major degenerative multi-factorial disorder (Barham and Trinder, 1972; Ogbonnia *et al*., 2008). It is characterized by hyperglycemia-the primary clinical manifestation (Nodestgarrd et al., 1998), and raised metabolic rate (Owu *et al*., 2006). Associated factors include imbalance and/or abnormalities in carbohydrate, fat and lipoprotein metabolism (Ugochukwu et al., 2003; Scoppola *et al.*, 2001), reactive oxygen species and oxidative stress (Kesavulu *et al.*, 2002; Nayeemunnisa, 2009).

Epidemiological data reveals that about 173 million people in the world are suffering from DM and there are postulations that this will be doubled by the year 2030 (WHO, 1999; 2003). Specifically, over 18.5 million people in Europe, 15.1 million in North America, 12.6 million in Latin America, 6.6 million in USSR and 5.3 million in Africa suffer from DM (Barnett and O'Gara, 2003; Greenfield and Chisholm, 2004). Abnormalities in insulin secretion and/or cells/tissue response to insulin are reported as the major etiology (WHO, 1999; 2003). Oxidative stress has also been reported to play a role in the progression of the disease (Giugliono *et al*., 1996).

The fact that food additives is a major constituent of Nigerian diets (Akpamu et al., 2011b) remains a source of concern considering the controversies about their risks and benefits (Moore; 2003; Gaby; 2005; McCann et al.,

2007). Of particular interest is the use of monosodium glutamate (MSG), which, according to Eweka and Om'Niabohs, (2011) is popularly known in Nigeria as *Ajino-moto* or *White maggi*. It is the sodium salt of glutamic acid (Pavlovic and Sarac, 2010; Egbuonu *et al*. 2010) reported to enhance flavour (IFTEPFSN, 1989; Filers and Stergink, 1991; Yamaguchi and Ninomiya, 2000), through stimulation of the orosensory receptors (Fuke and Shimizu, 1993). There are assertions too, that MSG enhances appetite and palatability of meals (Yamaguchi, 1987; Rogers and Blundell, 1990).

On the other hand, some studies have shown that MSG is toxic to humans and experimental animals (Ariano *et al*., 2005; Belluardo *et al*; 1990; Egbuonu et al; 2009a,b, 2010a, b,c,d). It induces seizures, liver damage (Gonzalez-Burgos *et al*; 2004; Egbuonu et al; 2009a, b; 2010a, c,d), brain damage (Nwaopara *et al*., 2009; 2010a, b; 2011), obesity (Rogers and Blundell, 1990; Mozes *et al*; 2004, Egbuonu *et al*; 2009b, 2010b, c, d; Akpamu *et al*; 2011a) and anemia (Ashaolu et al., 2011; Akpamu *et al*; 2011b). This study therefore, determines the effect of chronic ingestion of MSG on blood glucose level using rabbits as models.

MATERIALS AND METHODS

Substance of Study: MSG (white maggi or Ajinomoto) was purchased in sachets of 3g each from an open market in Ekpoma, Edo State Nigeria. Grower's mash used for this experiment was purchased from Bendel Feeds and Flour Mills, Ewu, Edo State.

Experimental animals: Adult rabbits (n=18) of comparable weight (1.60±0.20 kg) were procured from the animal house of the College of Medicine, Ambrose Alli University, Ekpoma, and transferred to the Physiology Laboratory of same institution. The animals were separated into three groups (A, B1 and B2) and allowed to acclimatize for two weeks. Group A (n=6), served as the control while group B (B1 and B2; n=6 respectively) served as the experimental group. The study was conducted from July to November, 2007. However, the actual animal experiment lasted for a period of 10 weeks.

Substance Preparation and Administration: During the acclimatization period, animals were fed ad libitum with grower's mash, grass supplements and water. After acclimatization, group A (control) continued with the acclimatization diet, while 3.33mg/ml and 6.66mg/ml of MSG were added and mixed with the water of group B1 and B2 respectively.

To obtain 3.33mg/ml and 6.66mg/ml of MSG, 30g (10 sachets) and 60g (20 sachets) of MSG was dissolved in 1000ml of water respectively. Administration of the MSG solution was ad libitum on daily basis for eight weeks.

Sample Collection and Data Analysis: With a sterile lancet, blood samples were obtained by venipuncture at the animal's ear. Fasting blood glucose was determined using glucose test strips (National Diagnostic Products, Sydney, Australia and marketed by Betacheck; Ref: AS BAM EVCL6) according to manufacturer's instruction. The process was repeated 3 times for all the animals on weekly basis and the value recorded.

Statistical Analysis: Data obtained were subjected to statistical analysis using SPSS software. The ANOVA test ($P \leq 0.005$) was used to test the level of significance.

RESULTS

Table 1 shows the time representation of FBG level of rabbits fed MSG. The weekly FBG for the control varies between 128.00±4.47mg/dl (in the 5th week) and 123.20±3.03mg/dl (in the 10th week) MSG). In group B1 which received 3.33g/L of MSG, there was a sharp increase in FBG level after acclimatization, and this increase was sustained in the acute state but became irregular in the chronic state in a duration dependent manner. Similarly, the observed increase in FBG levels in group B2 (6.66g/L of MSG) was duration dependent until the 8th week, but in the 9th week (3rd week of chronic period) a slight drop in FBG level was observed but rose again in the 10th week.

Comparatively, there was no difference in FBG levels in the control group (group A) except for that observed in the 5th week, which was significantly different from those of the 1st and 10th week. However, as time progressed, FBG levels in group B1 became significantly different. On the contrary, group B2 presented FBG levels that were significantly-different between the periods particularly during the chronic period.

Table 2 shows the dosage dependent variations in FBG levels of rabbits fed MSG. During acclimatization when the animals were not fed MSG, FBG levels were similar as no significant changes were observed. However, during the acute treatment of 0mg/ml (group A), 3.33mg/ml (group B1) and 6.66mg/ml (group B2) of MSG, there was a dose dependent increase in the levels of FBG. Similar dose dependent changes in FBG levels were observed in the chronic treatment periods. In the acute and chronic treatment periods, the observed increases in FBG levels were significantly different ($p>0.05$) from those obtained for the corresponding control (group A). Also, the FBG levels for the 6.66mg/ml treated group were significantly different from those of 3.33mg/ml treated group.

Table 1: Time course activities of varying doses of MSG on fasting blood glucose of rabbits

Period	Week	Fasting Blood Glucose (FBG) Levels (mg/dl)		
		Group A; control (0mg/ml MSG)	Group B1 (3.33mg/ml MSG)	Group B2 (6.66mg/ml MSG)
Acclimatization (mg/dl)	1	123.60±26.1^{a}	125.20±1.10^{a}	125.60±2.19^{a}
	2	126.80±1.79^{ab}	126.80±2.28^{a}	125.60±1.67^{a}
Acute (mg/dl)	3	126.40±0.89^{ab}	156.00±8.49^{b}	168.80±6.69^{b}
	4	127.20±1.79^{ab}	160.40±4.16^{bc}	175.80±7.89^{bc}
	5	128.00±4.47^{b}	160.00±9.70^{bc}	178.60±5.18^{c}
	6	125.60±0.89^{ab}	163.20±2.68^{c}	179.80±7.43^{c}
Chronic (mg/dl)	7	126.00±0.00^{b}	164.40±2.61^{c}	183.40±10.48^{cd}
	8	125.60±0.89^{ab}	160.80±2.28^{bc}	188.80±5.22^{de}
	9	126.00±0.00^{ab}	161.00±4.24^{bc}	185.60±4.34^{de}
	10	123.20±3.03^{a}	162.40±2.19^{c}	192.20±2.49^{de}

Values are mean ±Std. Deviation. Mean in a column having different superscripts indicate significantly different ($P\leq 0.05$).

Table 2: Dose dependent activities of varying concentration of MSG on fasting blood glucose of rabbits

Period	Week	Fasting Blood Glucose (FBG) Levels (mg/dl)		
		Group A; control (0mg/ml MSG)	Group B1 (3.33mg/ml MSG)	Group B2 (6.66mg/ml MSG)
Acclimatization (mg/dl)	1	123.60±2.61^{a}	125.20±1.10^{a}	125.60±2.19^{a}
	2	126.80±1.79^{a}	126.80±2.28^{a}	125.60±1.67^{a}
Acute (mg/dl)	3	126.40±0.89^{a}	156.00±8.49^{b}	168.80±6.69^{c}
	4	127.20±1.79^{a}	160.40±4.16^{b}	175.80±7.89^{c}
	5	128.00±4.47^{a}	160.00±9.70^{b}	178.60±5.18^{c}
	6	125.60±0.89^{a}	163.20±2.68^{b}	179.80±7.43^{c}
Chronic (mg/dl)	7	129.20±8.32^{a}	164.40±2.61^{b}	183.40±10.48^{c}
	8	125.60±0.89^{a}	160.80±2.28^{b}	188.80±5.22^{c}
	9	126.00±0.00^{a}	161.00±4.24^{b}	185.60±4.34^{c}
	10	123.20±3.03^{a}	162.40±2.19^{b}	192.20±2.49^{c}

Values are mean ±Std. Deviation. Mean in a row having different superscripts indicate significantly different ($P\leq 0.05$).

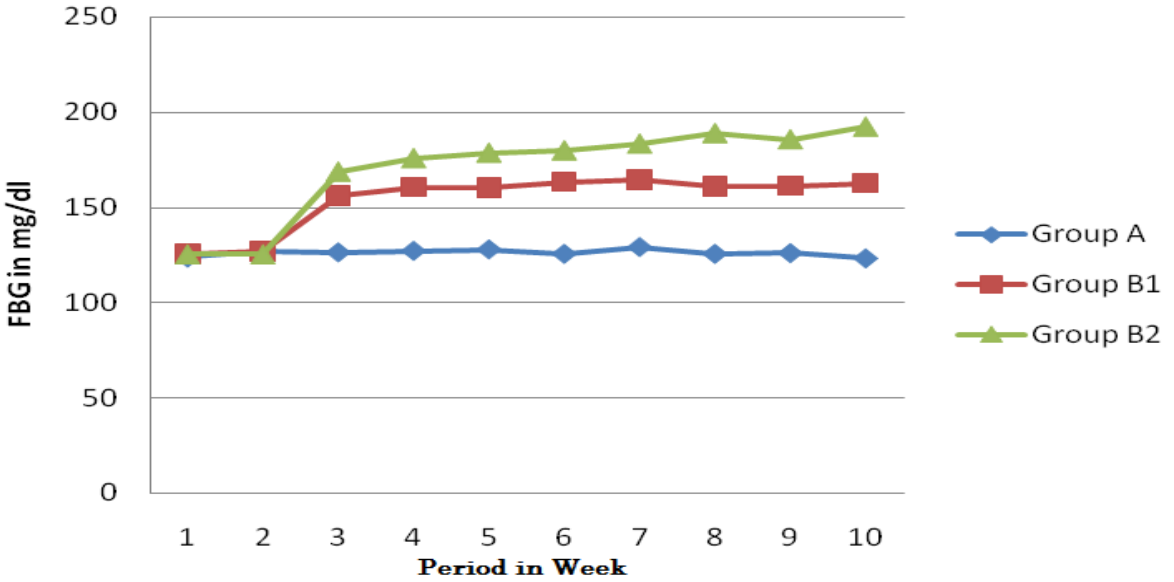

Fig 1: Line graph of the pattern of change in FBG with ingestion of varying doses of MSG

Key: Group A = control, 0mg/ml MSG; B1= 3.33mg/ml of MSG; B2= 6.66mg/ml of MSG; FBG= fasting blood glucose.

DISCUSSION

The present study on the effect of MSG treatment on FBG, demonstrated that duration and dosage have significant effects on glycemic index. Specifically, the greatest impact on FBG was presented in group B2 at the 10th week (192.20±2.49mg/dl) while group B1 was at the 7th week (164.40±2.61mg/dl) as against that of the control in the 5th week (128.00±4.47mg/dl) which was far lower than those of B1 and B2. Although an earlier study by Go et al. (1973), reported that the use of MSG has no effect on blood glucose, cholesterol level and weight. It is interesting however, to note that more recent studies have proven its deleterious effect. In fact, there is overwhelming evidence on MSG toxicity (Samuels, 1999).

As reported in several literatures, MSG is toxic to humans and experimental animals (Ariano *et al*., 2005; Belluardo *et al*; 1990; Egbuonu et al; 2009a,b; 2010a,b,c,d). It induces seizures, liver damage (Arauz-Contreras and Feria-Velasco, 1984; Beas-Zarate et al., 1989; Gonzalez-Burgos *et al*; 2004; Egbuonu et al; 2009b; 2010), neurotoxic and brain damage (Robinzon et al., 1975; Rascher and Mestres, 1980; Hu et al., 1998; Gill et al., 2000; Nwaopara *et al*., 2009; 2010a,b; 2011), obesity and hyperinsulinaemia (Bunyan et al., 1976; Iwase et al., 1998; Rogers and Blundell, 1990; Nakagawa et al., 2000; Iwase et al., 2000; Racek et al., 2001; Dolnikoff et al., 2001; Mozes *et al*; 2004, Akpamu *et al*; 2011a) and anemia (Ashaolu et al., 2011; Akpamu *et al*; 2011b).
Taking a cue from the obesity-inducing potentials of MSG (Rogers and Blundell, 1990; Mozes *et al*; 2004, Akpamu *et al*; 2011a) following its appetite and palatability enhancing potentials (Kanarek et al., 1979; Rogers and Blundell, 1990; Bellisle et al., 1991; Colucci and Grovum, 1993; Hermanussen et al., 2006; He *et al*., 2008; Tsang, 2008; Bellisle, 2008; Carter *et al*., 2011) and by implication, increased energy intake, may explain the observed increases in FBG.

On the other hand, MSG has been reported to increase the secretion of insulin (Niijima et al., 1990; Macho et al., 2000; Mourtzakis and Graham, 2002; Chevassus et al., 2002), which in our thinking should counter the increased blood glucose level reported in the present study. On the contrary, certain possibilities may have accounted for this observed outcome. These include: 1) the fact that MSG alters the regulatory mechanisms that affects fat metabolism (Tsang, 2008); 2) MSG inhibits ketone secretion, resulting in an obese rat with a propensity for creating adipose tissue (fat) (Nakai et al., 1986; Vice et al., 2005); 3) The weakening potential of fats on insulin action (Guyton and Hall, 2006) and the attendant increase in FBG as shown by the results of this study. Moreover, obesity is a major factor for a number of co-morbidities such as noninsulin-dependent diabetes mellitus (Haslam and James, 2005).

Another line of thought is deducible from the neurotoxic and brain damaging effects of MSG (Robinzon et al., 1975; Rascher and Mestres, 1980; Hu et al., 1998; Gill et al., 2000; Nwaopara *et al*., 2009; 2010a, b; 2011). Recall the involvement of the hypothalamus in the regulation and action of insulin (Guyton and Hall, 2006); a hormone that metabolizes glucose. Since the brain cells are damaged by MSG, they may be dormant to blood glucose level and or the feedback regulatory mechanism may not function to regulate insulin secretion. Backing up these assumptions is the report of Ashaolu et al., (2011).

Furthermore, our findings are in agreement with those reported by Cameron et al., (1976), Komeda et al. (1980) and Nagata et al. (2006) that MSG has the potentials to trigger diabetes mellitus. Recall once more, that diabetes mellitus is a multi-factorial degenerative disorder (Barham and Trinder, 1972; Ogbonnia *et al*., 2008). In the same vein, MSG induces a number of organ-damage (Moses and Sefcikova, 2004; Farombi, 2006; Eweka, 2007; Vinodini *et al.*, 2010) and has immune compromising potentials (Ashaolu et al., (2011). Thus, indiscriminate MSG ingestion and the incidence of diabetes mellitus are two inseparable phenomena. It is our opinion therefore, that the there is an urgent need to inform populations about the health dangers associated with indiscriminate consumption of MSG.

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AUTHORS' CONTRIBUTIONS

Oriaghan EA, is the head and coordinator of the experiment and provided the first draft of this manuscript. Inegbenebor U, supervised the study and provided necessary technical assistance. Oriaghan EA, Shelu OJ., Obhimon O, Idonor EO. and Ekhoye I, were involved in the day to day activities throughout the experiment.