



Effects of food flavour enhancer (Monosodium Glutamate and Maggi Poulet) supplementation on glucose tolerance in *Sprague Dawley* rat

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ABSTRACT

This study was designed to evaluate the effects of purified monosodium glutamate and 'Maggi Poulet', on body weight gain, lipid profile, hepatic lipid peroxidation and glucose tolerance in rats. Thirty five young male *Sprague Dawley* rats were divided into five groups and fed by oral route as follow: group I (distilled water), group II (monosodium glutamate solution), group III ('Maggi Poulet' solution), group IV (monosodium glutamate solution. and high fat solution) and group V ('Maggi Poulet' solution and high fat solution). During the experimental period, fasting glycemia was taken and an oral glucose tolerance test has been performed at the end. Blood samples were then collected in all groups and serum cholesterol and triglyceride were assayed. Animals were killed after and abdominal adipose tissue, liver and heart were excised and weighed. Liver samples were also used to estimate hepatic malondialdehyde level in rats. The results proved that the dietary feeding did not affect the body gain and lipid profile in experimental groups. The hepatic lipid peroxidation has also increased in all experimental groups and at the same time, rats in group II, group IV and group V present a two-hour plasma glucose level significantly higher. However, purified monosodium glutamate and 'Maggi Poulet' at the dose of 1500 mg/kg. b. wt. have not impaired fasting glycemia in *Sprague Dawley* rat. All changes observed in the glycemia of rats in experimental groups do not allow to qualify them to be glucose intolerant, nevertheless monosodium glutamate consumption in association or not with high fat is hepatotoxic and may contribute to the emergence of prediabetes in human being.

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INTRODUCTION

Impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) are intermediate states of abnormal glucose regulation that exist between normal glucose homeostasis and diabetes (David et al., 2007). These two features of glucose intolerance differ in their site of insulin resistance. IFG is

associated with hepatic insulin resistance whereas IGT manifest itself by muscle insulin resistance (Brohall et al., 2006). According to the recommendations of the American Diabetes Association; IFG is defined for fasting plasma glucose level in the range of 100 to 125 mg/dl and a two-hour plasma glucose level less than 140 mg/dl and IGT is

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characterised by a fasting plasma glucose less 100 mg/dl, a two-hour plasma glucose level going from 140 to 199 mg /dl and the glycated haemoglobin A_{1c} rate included between 5.7% to 6.4% (Buyschaert and Bergam, 2011).

It is generally agreed that genetic background, dietary habits and physical inactivity may contribute to the pathogenesis of these disorders. In order to improve the understanding of the mechanisms involved in the onset of insulin resistance and obesity; various rodent models have been developed. Based upon genetic susceptibility, there are close to 50 different types of genetic models of obesity in rodents among which, obese mice (ob/ob, db/db) and Zucker rats (fa/fa) are used as sample animals of diabetes in biological and medical studies (Von Diemen et al., 2006). There are also diabetic rodent models obtained through chemical induction using gold-thioglucose, alloxan or streptozocin (Huang et al., 2004) and experimental ablation of the pancreas or the arcuate nucleus (Von Diemen et al., 2006).

However, those previous models do not sufficiently manifest prediabetic state when we need to extrapolate results from rodent to human being. Dietary rodent models are therefore proposed and several substances such as high fat, trans-fat, sucrose, fructose and recently monosodium glutamate (MSG) have been proposed (Diniz et al., 2004; Collison et al., 2010, Singh et al., 2011). In most cases, MSG subcutaneous injections or large oral dose of MSG dietary supplementation in rodent have shown an impairment of glucose homeostasis, a dyslipidemia and weight gain (Hermanussen et al., 2006).

In contrast to previous studies, Boutry et al. (2011) have reported that 2% MSG diet chronic supplementation had no effect on food intake, body weight, and adipose tissue mass in *Wistar* rat. Furthermore, Kondoh and Torii (2008) reported that 1% MSG dissolved in the drinking water was able to reduce weight gain, body fat mass, and plasma leptin

concentrations in *Sprague Dawley* rats. The mechanism underlying MSG-induced obesity and insulin resistance in non physiological situation remains unclear and controversial (Boutry et al., 2011).

The use of bouillon cube containing MSG in spite of purified MSG in household preparation of food is very popular in Sub-Saharan African countries (Sop et al., 2008; Akpamu et al., 2011). 'Maggi Poulet' (MP) is one of the most commonly cube-type seasoning added to soups in Lomé (Togo). Sop et al. (2008) have estimated the average free MSG content of MP to 13.5% (wt./wt.). The aim of this study is to investigate the effect of dietary MSG and MP with or without high fat supplementation on weight gain, lipid profile, hepatic lipid peroxidation and glucose tolerance in *Sprague Dawley* rat.

MATERIALS AND METHODS

Chemicals

Lard, MSG (West African Seasoning Company Limited, Lagos Nigeria) and MP (Nestlé Company, Dakar Senegal) were purchased from 'Gbonsimé' market, Lomé (Togo). Other chemicals such as thiobarbituric acid, phosphoric acid and cholesterol were from Sigma-Aldrich, France. Elitech/Biolabo kits, France, were used for estimation of total cholesterol, HDL cholesterol and triglycerides.

Animals

Male *Sprague Dawley* (n=35) strain weighing about 160 g were used for the experiments. The animals were provided by Nogoshi Medical Institute (University of Legon, Ghana). They were kept on a 12:12-h light-darkness cycle at the Animal House of the University of Lomé (Togo). They had free access to the standard diet and water *ad libitum*. All animals were acclimated for 2 weeks before starting dietary feeding.

Experimental protocol

The rats were divided into five groups of seven animals each and treated as follow:

-Group I (control): the rats received distilled water (5 ml/kg b. wt.) and thirty minutes after another dose of 10 ml/ kg b. wt of the same distilled water

-Group II (MSG): the rats received monosodium glutamate aqueous solution (1500 mg/kg b. wt.) and thirty minutes after another dose of 10 ml/kg b. wt of the distilled water

-Group III (MP): the rats received 'Maggi Poulet' aqueous solution (1500 mg/kg b. wt.) and thirty minutes after another dose of 10 ml/kg b. wt. of the distilled water

-Group IV (MSG-HF): the rats received monosodium glutamate aqueous solution (1500 mg/kg b. wt.) and thirty minutes after another dose of 10 ml/kg b. wt. of high fat solution

-Group V (MP-HF): the rats received 'Maggi Poulet' aqueous solution (1500 mg/kg b. wt.) and thirty minutes after another dose of 10 ml/kg b. wt. of high fat solution.

High fat solution was prepared by dissolving 400 mg of cholesterol in 10 ml of melted lard. The total treatment period was sixty days and substance administration daily lasted one hour (9:00 a. m-10:00 a. m).

During the experimentation animals' body weight were measured daily while fasting glycemia was taken the first day and two weeks later using One Touch Ultra 2 glucometer. At the end of the treatment (60 days), all the rats were fasted overnight (12-14 h) for the oral glucose test tolerance (OGTT). Eighty per cent glucose aqueous solution was orally administered by gastric tube at the dose of 4 g/kg b. wt per rat. Blood glucose rates were measured at tail vein before glucose administration and at 30, 60, 120 min after the glucose administration. After the OGTT, blood samples were collected with a capillary tube from retro-orbital plexus of rat under ether anaesthesia. The collected blood was incubated at 37 °C until clotted and then, it

was centrifuged to separate the serum. The serum samples were frozen to be used for measurement of total cholesterol (TC), HDL cholesterol (HDL-cholesterol) and triglyceride (TG). Animals were killed by cervical decapitation and the abdominal cavity was opened. Liver, heart and intra-abdominal adipose tissue were dissected and weighed. Liver samples were frozen during 48 hours and hepatic lipid peroxidation was estimated by quantification of malondialdehyde thiobarbituric acid adduct (MDA-TBA). Briefly, liver samples (150 mg) were homogenised with cold 1.5% KCl in order to make 10% homogenate. Three millimetres of phosphoric acid and 1 ml of 0.6% thiobarbituric acid aqueous solution were added to 0.5 ml of the homogenate. The mixture was heated for 45 min. After cooling, 4 ml of n-butanol was added to the homogenate and mixed. Absorbance of butanol phase was measured at 535 and 520 nm. The difference of the two measurements was used as MDA concentration expressed in nmol/g tissue (Sepici-Dincel et al., 2007; Bakoma et al., 2011)

Data analysis

All results were analysed using Graph Pad Prism 4.5 and expressed as mean \pm S.E.M. The significance of the difference between each group was determined by one way analysis of variance followed by Turkey post-hoc test ($p < 0.05$).

RESULTS

As shown in Table 1, the final body weight, the body weight gain and the relative heart weight did not differ into the groups. Relative intra-abdominal fat deposition (mesenteric, retroperitoneal and epididymal fat) and relative liver weight were markedly higher in MSG-HF and MP-HF treated groups compared to the control group.

Overnight fasting glucose was not influenced neither by controlled diet nor MSG and MP ingestion. However, mean fasting glucose in MSG-HF and MP-HF groups had

significantly increased from 15th day onward till the end of the experimental period (Table 2). The OGTT showed that there were no differences in glucose rate after loading in controlled and MP groups. Otherwise, the glucose level after loading in MSG-HF group peaked and was the highest among all groups during the test (Table 3). At 120 min post-glucose challenge, glucose concentrations were significantly higher in MSG, MSG-HF and MP-HF rats groups compared to the control ($p < 0.05$). All rats in experimental groups tended to have elevated glucose area

under curve (AUC) during the OGTT compared to the control; nevertheless only MSG-HF rats presented a glucose AUC significantly higher (+23% versus control group).

In addition, there were no differences in total cholesterol, HDL cholesterol, LDL cholesterol, triglyceride into the five groups after dietary feeding (Table 4). Liver tissue MDA levels in contrast were significantly increased in all experimental groups compared to the control group ($p < 0.05$).

Table 1: Effect of diets on body weight, and relative tissue weight after 60 days supplementation.

Parameters	Groups				
	Control	MSG	MP	MSG-HF	MP-HF
Initial body weight (g)	171.8±3.16	169.71±2.86	169.57±4.79	170.66±3.6	169±3.17
Body weight gain (g)	102.4±5.65	128.71±7.66	101.57±2.82	105.50±3.36	102±7.67
Final body weight (g)	274.2±8.83	289.42±10.54	271.14±7.62	275.16±6.96	271±5.83
Relative liver weight (g)	2.42±0.08	2.53±0.08	2.54±0.07	2.85±0.04 ^a	2.7±0.06 ^{a,c}
Relative heart weight (g)	0.33±0.01	0.35±0.02	0.40±0.01	0.36±0.01	0.40±0.01
Relative intra-abdominal adipose tissue weight (g)	1.54±0.09	1.83±0.17	1.50±0.14	2.04±0.06 ^{a,b}	2.58±0.17 ^{a,c}

Each value is expressed as mean ± standard error mean for seven rats per dietary group. The data were evaluated by ANOVA followed by Turkey post-hoc test at 5%. Relative liver weight was expressed as g liver x 100/ g body weight. Relative intra-abdominal adipose tissue weight was expressed as g intra-abdominal adipose tissue weight x100/ g body weight. (^a Significantly different from controlled group, ^b significantly different from MSG group, ^c significantly different from MP group).

Table 2: Effect of diets on fasting glycemia during experimentation.

Groups	Blood glucose levels (mg/dl)				
	1 st day	15 th day	30 th day	45 th day	60 th day
Control	67.40±0.97	60.40±1.24	65.20±2.97	63.00±1.00	64.20±2.20
MSG	71.14±1.76	67.1±2.00	68.00±2.32	66.57±2.18	67.00±1.5
MP	69.28±1.68	64.85±1.53	63.42±2.33	64.42±2.42	65.00±1.48
MSG-HF	69.00±1.75	79.40±0.60 ^{a,b}	76.40±1.80 ^{a,b}	75.66±2.87 ^{a,b}	80.16±2.8 ^{a,b}
MP-HF	72.14±1.98	72.85±2.26 ^{a,c}	76.40±1.88 ^{a,c}	78.00±2.6 ^{a,c}	78.28±1.4 ^{a,c}

Blood glucose concentration was taken from conscious fasting rat at tail vein. Each value is expressed as mean ± standard error mean for seven rats per dietary group. The data were evaluated by ANOVA followed by Turkey post-hoc test at 5%. ^a Significantly different from control group, ^b significantly different from MSG group, ^c significantly different from MP group.

Table 3: Blood glucose rates after 60 days treatment on fasting conditions and 30, 60, 120 min after oral glucose intake.

Parameters	Groups				
	Control	MSG	MP	MSG-HF	MP-HF
Fasting glycemia (mg/dl)	66.20±1.93	67.28±1.52	65.00±1.48	80.14±2.43 ^{a, b}	79.00±1.21 ^{a, c}
Glycemia 30 min after glucose intake (mg/dl)	113.00±6.39	117.42±3.81	107.57±2.14	135.85±7.97 ^a	120.85±1.54
Glycemia 60 min after glucose intake (mg/dl)	111.80±6.32	112.57±5.91	120.57±4.70	134.16±4.49 ^{a, b}	107.00±1.55
Glycemia 120 min after glucose intake (mg/dl)	70.00±3.54	93.71±3.86 ^a	83.85±4.61	99.85±3.35 ^a	94.28±5.87 ^a
Area under curve (mg x min/ dl)	11514±370.6	12409.2±418.1	12143.5±334.8	14185.7±488.6 ^a	12454.2±228.7

Eighty per cent glucose solution was directly introduced by gavage into the stomach of fasted rat at a dose of 4g/ kg b. wt. Blood glucose concentration was taken on conscious rat at tail vein using One Touch Ultra Two Glucometer before, 30, 60, 120 min after glucose load. Each value is expressed as mean ± standard error mean for seven rats per dietary group. The data were evaluated by ANOVA followed by Turkey post-hoc test at 5%. ^a Significantly different from controlled group, ^b significantly different from MSG group, ^c significantly different from MP group.

Table 4: Effect of diets on lipid profile and hepatic oxidative stress after 60 days supplementation.

Parameters	Groups				
	Control	MSG	MP	MSG-HF	MP-HF
Total cholesterol (g/l)	0.67±0.06	0.68±0.06	0.67±0.02	0.63±0.03	0.75±0.05
Triglycerides (g/l)	0.44±0.02	0.45±0.04	0.42±0.02	0.43±0.02	0.52±0.02
HDL cholesterol (g/l)	0.46±0.02	0.41±0.05	0.51±0.03	0.47±0.03	0.52±0.02
LDL cholesterol (g/l)	0.11±0.05	0.18±0.06	0.19±0.13	0.02±0.02	0.17±0.03
Malondialdehyde (nmol/mg tissue)	1.40±0.27	2.98±0.52 ^a	2.62±0.16 ^a	2.97±0.08 ^a	2.88±0.12 ^a

Total cholesterol, triglycerides and HDL cholesterol were assayed by enzymatic colorimetric method using Elitech/Biolabo kits. LDL cholesterol was estimated by Friedewal et al. (1972) empirical equation. Malondialdehyde concentration was estimated by thiobarbituric acid method (Bakoma et al., 2011). Each value is expressed as mean ± standard error mean for seven rats per dietary group. The data were evaluated by ANOVA followed by Turkey post-hoc test at 5%. ^a Significantly different from controlled group.

DISCUSSION

Monosodium glutamate is a worldwide used flavour enhancer with a level of consumption that has increased during the last two decades (Insawang et al., 2012). It can be used as purified glutamate salt or as a component of a mixture of amino acids and small peptides commonly called bouillon cube. This study was performed to analyse the effects of oral dose of purified MSG and MP (a bouillon cube containing 13% of MSG) supplementation in combination or not with high fat on weight gain, body fat, lipid profile, glycemia and hepatic lipid peroxidation in *Sprague Dawley* rat. From our viewpoint, this is the first study that analyses systemic and morphological effects of MP compared to purified MSG ingestion in rat. We tested in sub-chronic conditions (60 days) a supra-nutritional dose of 1500 mg of MSG or MP per kilogramme body weight in growing *Sprague Dawley* rat.

As expected, MSG or MP sub-chronic supplementation had no effect on body weight, gain, liver weight, heart weight and abdominal fat deposition. Our results are in accordance with those of Tordoff et al. (2012) who reported that 3% MSG in diet or in drinking water did not influence body weight and body fat store in three different species of rodents (*Sprague Dawley* rat, Obesity-prone *Sprague Dawley* derivated rat and C57BL/6J mouse) These findings are also in line with previous data from Boutry et al. (2011) who used a similar dose of 1295 mg MSG/ kg b. wt in diet or drinking water of *Wistar* rat. However, it has been suggested in several animal studies that MSG is able to induce overweight or obesity. In these MSG-induced obesity rat models; MSG is injected subcutaneously to newborn rats or administered by oral route at higher doses (5000 mg MSG/day) to young rats (Hermanussen et al., 2006). The induction of obesity in animals treated in this manner required that plasma GLU concentrations be elevated to very high values, sufficient enough

to penetrate into the brain and exert deleterious effects on the arcuate nucleus neurons. Plasma hyperglutamataemia thereby, can disrupt the hypothalamic signaling cascade of leptin action and cause hyperphagia and obesity (Diniz et al., 2005; Kondoh and Torii 2008; Boutry et al., 2011). In our conditions, since rats did not manifest deleterious effects of overweight and obesity; it seems therefore that GLU derived from MP or MSG ingested is almost entirely (up to 95%) catabolised by the enterocyte of intestinal mucosa (Beyreuther et al., 2007).

The combination of MSG and high fat or MP and high fat feeding resulted in an increase of relative liver weight and abdominal fat deposition. This result is not unexpected as far as lard-based diets induced hepatic steatosis and are recommended as one of the standard fats to be used for the generation of a valid rat model for the metabolic changes associated with obesity (Buettner et al., 2006)

The present work demonstrates that both MSG and MP sub-chronic ingestion in *Sprague Dawley* rat did not increase fasting glycemia during the supplementation. However, the oral glucose challenge generated a normal blood glucose response in MP rats whereas a hyperglycemic response was observed in MSG rats. In this way, MP fed rats' plasma glucose concentration declines to near baseline values at 120 min after load while it remained significantly higher in MSG fed *Sprague Dawley* rat. These results highlight the reduction of glucose utilisation by peripheral tissues in postprandial conditions in MSG fed rats. Elevated plasma glutamate is well known to enhance insulin secretion which can lead to hyperinsulinemia and insulin resistance in sub-chronic conditions (Chevassus et al., 2002). The low amount of MSG contained in the MP diet may explain the relative normal glucose homeostasis observed in this group. In MSG-HF and MP-HF diet groups; lard has exacerbated glucose intolerance by increasing

fasting glucose levels and glucose AUC during the OGTT. Huang et al. (2004) pointed out in their report that prolonged high fat diet supplementation can cause abnormality of the pancreas function and impaired insulin secretion. However, the alteration of glucose homeostasis in all experimental rats is not elevated to such point to be considered as prediabetic animals while basing exclusively on glucose criteria (Buyschaert and Bergam, 2011).

Serum lipids were all within the normal range and did not differ into the five groups whereas hepatic lipid peroxidation (MDA) had increased in all experimental groups. Such findings indicate that MSG and MP oral ingestion do not cause dyslipidemia but induce oxidative stress in *Sprague Dawley* rat. Kondoh and Torii (2008) also observed that the voluntary ingestion of 1% MSG solution had no effect on lipid profile in *Sprague Dawley* rat. However, lipid profile in MSG fed rats is very different from data obtained by Singh et al. (2011) who reported that the oral ingestion of MSG at dose level of 4 and 8 mg/b. wt. significantly increased the level of serum lipid by 19.93% and 29.37% in male *Wistar* rat during 7 days supplementation. They suggest that glutamate favours lipogenesis by converting to glutamine. This hypothesis is confirmed by Boutry et al. (2011), who demonstrated that after 15 days of 2% MSG dietary adaptation in *Wistar* rat, the circulating glutamine concentration has increased. We suggest therefore that *Wistar* rat could be more sensitive to the effects of MSG than *Sprague Dawley* rat. On the other hand, Egbuonu et al. (2009) found that oral intake of low dose of MSG (5 mg/b. wt.) by gastric tube in *Wistar* rat may be destructive to the liver. The mechanism involved in the production of hepatic MDA may be the generation of reactive oxygen species by MSG which is also an endogenous excitotoxin (Ortiz et al., 2006; Eweka and Om'Iniaobohs 2011).

Conclusion

To sum up, this study shows that sub-chronic MP or MSG administration (60 days) at a dose of 1500 mg/b.wt. does not impair glucose tolerance but it promotes hepatic lipid peroxidation in male *Sprague Dawley* rat. A combination of MSG and high fat or MP and high fat supplementation in *Sprague Dawley* rat during growth may predispose to a glucose homeostasis impairment. The findings of this work are supplemental data confirming earlier reports on the hepatotoxicity and the potential role of MSG in the increasing prevalence of prediabetes and metabolic syndrome in human being. Further investigations using long term supplementation of MSG at various doses in rats and humans are needed to raise the ambiguity.

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