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Effects of fibre-enriched diets on tissue lipid profiles of MSG obese rats

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ABSTRACT

In order to investigate the influence of some fibre-enriched diets on tissue lipids in an animal model of obesity induced by the administration of monosodium glutamate (MSG), obese rats were fed diets containing 30% of *Acha*, Cassava, Maize and Plantain for five weeks and weight gain, feed intake and lee index were recorded. The lipid profiles of plasma, erythrocytes, kidney, heart and liver as well as hepatic 3-hydroxyl-3-methylglutaryl-CoA (HMG-CoA) reductase activity were measured. The diets significantly ($p < 0.05$) reduced weight gain and lee index in the obese rats. Obesity-induced increase in plasma and erythrocytes lipid levels was significantly ($p < 0.05$) reduced by these diets. MSG-induced obesity also resulted in a significant increase ($p < 0.05$) in hepatic cholesterol level which was reduced by the diets. MSG-obesity was characterised by a significant ($p < 0.05$) increase in cholesterol, triacylglycerol and phospholipids in kidney and this was reversed by the diets except Maize which did not reverse the increased cholesterol level. Only *Acha* reversed the obesity-induced increase in heart cholesterol and phospholipids. The increased activity of hepatic HMG-CoA reductase associated with obesity was also significantly ($p < 0.05$) reduced by the diets. In conclusion, dyslipidemia associated with MSG-induced obesity could be attenuated by consumption of fibre-enriched diets.

1. Introduction

Increased food consumption in excess of energy requirements, coupled with excessive body fat present a growing human health problem (Mozes et al., 2004). Obesity, which is defined as body fat excess, usually develops slowly as a result of long-term alterations in energy balance. In general, when food intake exceeds energy expenditure, the retained energy is deposited as fat (Dolnikoff et al., 2001). Obesity is a highly prevalent disorder associated with decreased life expectancy and increased morbidity because of its combination with a variety of other disorders including hyperglycemia, hyperlipidemia, hypertension and consequently cardiovascular diseases carrying significant economic cost (Guven et al., 1999). These disorders are often associated with both qualitative and quantitative changes in lipid composition of several tissues in the body.

Dietary habits are considered one of the factors contributing to obesity; hence, a scrupulous dietary intervention is indicated in its management (De Filippo et al., 2010). This dietary intervention should include diets that regulate food intake and energy balance. African diets are known to be high-fibre and low-fat (De Filippo et al., 2010; Thian et al., 2006). While the rural African population live agrarian life style, urban population is becoming westernized (De Filippo et al., 2010). In Nigeria, for example, the rural populations consume more of carbohydrate and fibre-rich food like Cassava, *Acha*, Plantain and Maize more than western–type processed food and beverages (Olumakaiye et al., 2010). Thian et al. (2006) reported that in sub-saharan Africa, obesity is becoming a problem with 10–30% of men and 15–45% of women in West Africa being either overweight or obese.

In general, it is believed that humans are more suited to resist famine than overabundance of food (called the "thrift gene hypothesis") and; hence, it has been argued that the easy and related inexpensive availability of energy-dense food is responsible for the current obesity epidemic (Das, 2010). The energy balance is very tightly controlled by hypothalamic factors. Hence, the gut-brain axis and the cross-talk between gut hormones and hypothalamic factors are important in the regulation of food intake, energy balance, and development of obesity (Das, 2010). This knowledge has been under utilised in developing experimental murine model of obesity by intraperitoneal administration of neonatal rats with monosodium glutamate (MSG). This study was therefore aimed at studying the effects of some fibre-enriched diets on MSG – obesity – induced perturbations in lipid metabolism.

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2. Materials and methods

2.1. Animals and diets

Male neonatal albino rats were used for the experiment and obesity was induced as described by Nakagawa et al. (2000) with slight modification. Briefly, the neonates were treated with MSG (4 mg/g body weight) intraperitoneally five times (2,4,6,8 and 10 days after birth). Physiological saline was administered in a similar fashion to the control animals. The rats were weaned on the 21st day and raised normally thereafter, and studied at the age of 10 weeks. At the tenth week, Lee indices of the animals were calculated as $\frac{\sqrt[3]{\text{body weight}(g)}}{\text{nasal}-\text{anal length}(cm)}$. 25 rats with Lee index of 0.3 or more were considered obese. A total of 30 rats (25 obese surviving rats, 5 normal rats (not treated with MSG)) were used and they were divided into 6 groups of 5 rats each and randomly assigned to the experimental diets (Table 1). The 6 groups of rats were designated as follows:

Group 1. Normal-C: Rats fed normal diet.
Group 2. MSG-C: Obese rats fed normal diet.
Group 3. MSG-*Acha*: Obese rats fed *Acha*.
Group 4. MSG-Cassava: Obese rats fed Cassava flakes.
Group 5. MSG-Maize: Obese rats fed Maize.
Group 6. MSG-Plantain: Obese rats fed unripe Plantain flour.

The rats were fed for five weeks on the experimental diets. The composition of the experimental diet (Table 1) was based on the AIN-93 semisynthetic diet and were prepared to contain 30% of the fibre enriched diets. The animals were given food and distilled water ad libitum during the experimental period. Food consumption was measured daily while weight gain was measured weekly. The Ethical Committee for Conduction of Animal Studies at the Department of Biochemistry, University of Agriculture, Abeokuta, Ogun State approved the experimental protocol and all animals were cared for in accordance with the principles and guidelines of the committee.

2.2. Tissue collection

At the end of the experimental period, blood was collected from the animals into heparinised tubes by cardiac puncture under light ether anesthesia after an overnight fast. The plasma was seperated from the erythrocytes by centrifuging the whole blood at 5000 rpm for 10 min. Erythrocytes were washed three times with normal saline. The organs were excised, rinsed with normal saline, blotted dry and weighed immediately. The erythrocytes, plasma and organs were stored at −20 °C until analyzed.

2.3. Biochemical analyses

2.3.1. Plasma lipid profiles

Plasma concentrations of total cholesterol and triglycerides were determined with commercial kits (Cromatest linear chemicals, Montgat Spain). HDL cholesterol and triglycerides were determined in plasma with same commercial kits for total cholesterol and triglycerides after very low density lipoproteins (VLDL) and LDL were precipitated with heparin–$MnCl_2$ solution as described by Gidez et al. (1982). Total phospholipids in plasma and HDL were extracted with chloroform–methanol mixture (2:1, v/v) as described by Folch et al. (1957). Phospholipid content was then determined according to the method of Stewart (1980), which is based on the formation of a complex between phospholipids and ammonium ferrothiocyanate.

2.3.2. Erythrocyte lipid profile

Lipids were extracted from the erythrocytes as described by Rose and Oklander (1965) using chloroform–isopropanol (7:11, v/v). Aliquots of the chloroform–isopropanol extract were then used for the determination of cholesterol and triglyceride. Determination of total phospholipids in the chloroform–isopropanol extract of the erythrocyte followed the same procedure as described for plasma (Stewart, 1980).

2.3.3. Organ lipid profiles

Organ lipids were extracted according to the method of Folch et al. (1957). After washing with 0.05 M KCl solution, aliquots of the chloroform–methanol extract were then used for the determination of cholesterol, triglycerides and phospholipids concentrations. Cholesterol determination followed the same procedure as described for erythrocytes while determination of phospholipids followed the same procedure as described for plasma. Triacylglycerol concentrations in aliquots of the chloroform–methanol extracts of each organ were determined following the procedure described by Kriketos et al. (2003).

2.3.4. 3-Hydroxy-3-methylglutaryl (HMG) coenzyme A (CoA) reductase activity

The activity of HMG-CoA reductase was determined using an indirect method described by Rao and Ramakrishnan (1975). In this method, the concentrations of HMG-CoA and mevalonate are determined in the liver homogenate and the ratio of HMG-CoA/mevalonate is taken as an index of the activity of HMG-CoA reductase. An increase in the ratio indicates decrease whereas a decrease in the ratio indicates increased HMG-CoA reductase activity.

2.3.5. Estimation of VLDL-cholesterol and LDL-cholesterol

The concentrations of very low density lipoproteins (VLDL) Cholesterol and Low Density (LDL) Cholesterol were calculated by a modification of the Friedewald formular (Sandkamp et al., 1990).

VLDL–Cholesterol was calculated as triacylglycerols/5 and LDL-cholesterol was calculated by the equation: LDL-cholesterol = Total plasma cholesterol-(HDL + VLDL).

2.3.6. Estimation of atherogenic and coronary risk indexes

Atherogenic Index (AI) was calculated as the ratio of LDL-Cholesterol:HDL-Cholesterol while Coronary Risk Index (CRI) was estimated as the ratio of plasma total Cholesterol:HDL-Cholesterol as described by Ademuyiwa et al. (2008).

2.4. Statistical analysis

Calculations were made using the software "SPSS 13.0 for Windows" and data were expressed as Mean ± SEM of five readings. Analysis of Variance (ANOVA) was carried out to test for the level of homogeneity at $p < 0.05$ among the groups using Duncan's Multiple Range Test (DMRT) to separate the heterogeneous groups.

Table 1
Composition of diet in g/100 g.

Composition	Control	Acha	Cassava	Maize	Plantain
Fish Meal	20	20	20	20	20
Groundnut oil	5	5	5	5	5
Mineral mix*	3.5	3.5	3.5	3.5	3.5
Vitamin mix*	1	1	1	1	1
Cellulose	5	5	5	5	5
Maize starch	50	25	25	25	25
Sucrose	15	10	10	10	10
Choline bitartate	0.2	0.2	0.2	0.2	0.2
DL-Methionine	0.3	0.3	0.3	0.3	0.3
Acha	–	30	–	–	–
Cassava	–	–	30	–	–
Maize	–	–	–	30	–
Unripe Plantain flour	–	–	–	–	30

* Mineral mix and vitamin mix contains the following in g/100 g:
Calcium phosphate 49.50, sodium powder 11.80, potassium sulphate 5.20, sodium chloride 7.40, magnesium oxide 2.40, potassium citrate 22.40, ferric citrate 0.60, manganous carbonate 0.35, cupric carbonate 0.03, zinc carbonate 0.16, chromium potassium sulfate 0.055, potassium iodate 0.001, sodium selenate 0.001, choline chloride 0.50, thiamine HCl 0.06, riboflavin 0.06, niacine 0.30, calcium pantothenate 0.16, biotin 0.01, vit D3 0.025, vit B12 0.10, vit E acetate 1.00, pyridoxine 0.07, folic acid 0.02, vit A acetate 0.08.

3. Results

The MSG treated rats gained more weight and consumed less feed than the control rats. There was no significant ($p < 0.05$) difference in the Lee indices among all the MSG treated groups before treatment with the diets. However, the diets brought about a significant ($p < 0.05$) decrease in the Lee indices of the animals. *Acha* and unripe Plantain flour containing diets brought the lee index to levels similar that of the normal rats (Table 2). There was no significant ($p < 0.05$) difference in heart weight among all the groups. Kidney weight of the MSG-treated rats and that on Cassava containing diet were also significantly ($p < 0.05$) lower than the normal rats. Liver weight of the MSG treated rats were significantly ($p < 0.05$) lower than that of the normal rats and the other groups (Table 2).

MSG intake induced dyslipidemia in both plasma and lipoproteins and this was characterised by increased cholesterol, triacylglycerol and phospholipids. Intake of the diets attenuated the MSG-induced dyslipidemia (excluding total and HDL-phospholipids where *Acha* further significantly ($p < 0.05$) increased their concentrations). Unripe Plantain flour containing diet had the most