EFFECT OF GRANULATED SUGAR AND GARI FEED DIET ON LIPID PROFILE IN ALBINO RATS (*Rattus norvegicus*)

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ABSTRACT

This study was done to determine the effect of sugar and gari on lipid profile of albino rats. Seven groups of five albino rats per group in a treatment were fed normal rat diet mixed with sugar (Granulated) and gari (a dried cassava product) as treatments respectively at concentrations of 10%, 20%, 40%, 60%, 80% and 100% while the last group was fed normal rat diet and distilled water to serve as Control. The Total Cholesterols, triglycerides, HDL cholesterol and LDL cholesterol were monitored in the animals. There were dose dependent variations in the Total Cholesterols, triglycerides, HDL cholesterol and LDL cholesterol concentrations in both sugar and gari treated albino rats compared with their respective Controls. Cholesterol concentrations (Mmol/l) of 1.25 ± 0.22 at 10% increased to 1.44 ± 0.19 at 100%, triglycerides (Mmol/l) of 0.16 ± 0.11 at 10 % increased to 0.57 ± 0.21 at 100% and HDL cholesterol (Mmol/l) at 10% of 0.06 ± 0.00 increased to 0.28 ± 0.13 at 100% while the LDL Cholesterol (Mmol/l) reduced from 1.07 ± 0.25 at 10% to 0.96 ± 0.24 at 100% using sugar diets. Gari diet increased Cholesterol concentrations (Mmol/l) from 1.60 ± 0.20 at 10% to 1.71 ± 0.10 at 100%, while triglycerides concentration(Mmol/l) of 0.16 ± 0.12 at 10% was increased to 0.72 ± 0.23 at 100%.Also HDL Cholesterol concentration(Mmol/l) of 0.06 ± 0.01 at 10% was increased to 0.29 ± 0.10 at 100% by gari diet while the LDL

Cholesterol(Mmol/l) reduced from 1.47 $\pm$0.18 at 10% to 1.09 $\pm$ 0.14 at 100%. The study showed that gari diet caused increase total and HDL cholesterols than sugar. Therefore, feeding sugar and gari can cause synthesis of cholesterols but the increase is more pronounced in gari than sugar.

INTRODUCTION

Nearly all of the energy needed by the human body is obtained from oxidation of carbohydrates and lipids. It was postulated over 40 years ago that dietary composition, particularly carbohydrate content, could affect blood lipid profiles (Ahrens *et al.*, 1960). The finding that high-carbohydrate diet caused an increase in fasting serum triglycerides is not new (Ahrens *et al.*, 1960, Reaven *et al.*, 1965, Nestel *et al.*, 1970, Quarfordt *et al.*, 1970, Nestel 1973, Hayford *et al.*, 1979, Melish *et al.*, 1980).

Sugar cane is one of the most important sucrose sources, containing up to 20% by weight sucrose (Glazer and Nikaido, 1995). Sucrose a form of carbohydrate common in diet is widely consumed by humans (Ahmed *et al.*, 2001) and it consumption has been linked to various disorders such as diabetes (Thomas *et al.*, 1982) metabolic syndrome (Sivabalan and Menon 2008), aging (Lee and Cerami 1992) and Cancers (Dragsted *et al.*, 2002). In spite of the involvement of sucrose

consumption in the aetiology of many diseases of which cardiovascular disease is inclusive, a joint report of WHO/FAO (2003) apparently exonerated sucrose in the aetiology of Cardiovascular disease contrary to investigations by Szanto and Yudkin (1969), Albrink and Ulrich (1986), Johnson *et al.* (2009). WHO/FAO (2003) recommended that sucrose should not supply more than 10% of energy requirements. Sucrose hydrolysis produces a fructose and glucose equimolar mixture named inverted sugar, which has higher edulcorant power. The inverted sugar is incorporated more easily in industrial preparations and has more added value than sucrose (Chou and Jasovsky 1993). Sugar has been reported to reduce haematoxicity caused by petroleum (Braide *et al.*, 2011a) while Granulated sugar was reported to cause changes in renal function based on the concentration of the granulated sugar (Adegoke *et al.*, 2013).

Cassava is a staple food in human diets in over 80 countries (Gomez *et al.*, 1988). Gari a starchy food prepared from cassava (*Manihot utilisima*)tubers is one of the most popular staple foods of the people of the rain forest belt of West Africa and contains mainly starch-20% amylase and 70% amylopectin having lost the soluble carbohydrates (i.e. glucose and sugar) during processing (Ezeji *et al.*, 2009). Gari has been reported to reduce enzymes induction caused by petroleum through the phenomenon of glucose effect (Braide *et al.*, 2011b).

A lipid profile measures total cholesterol, high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, and triglycerides. Lipoprotein disorder is among the most common metabolic disease occurring in human. It may lead to Coronary heart disease (CHD) (Sloop 1999). Excessive levels of blood cholesterol accelerate atherogenesis and lowering high blood cholesterol reduces the incidence of CHD (Grundy 1986). Knowledge about the levels of cholesterol sub fractions is more meaningful than simple plasma cholesterol

purchased from Mile 3 Market, Port Harcourt. Commercially prepared Cholesterol, triglycerides, and HDL

level suggesting that the higher the level of LDL cholesterol, the greater the risk of atherosclerotic heart disease while the higher the level of HDL cholesterol, the lower the risk of coronary heart disease (Truswell,1978). This study was aimed at comparing the effect of feeding various concentrations of sugar and gari on total cholesterol, high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol and triglycerides concentrations in albino rats.

MATERIALS AND METHODS

Test Animals

Seventy Wistar albino rats of 0.195kg average body weight on normal rat diet, were obtained from the Animal House of the Department of Pharmacology and Toxicology, University of Port Harcourt. These rats were fed *adlibitum* with normal rat pellet and water and acclimatized to laboratory conditions for a period of 14 days prior to commencement of study. The granulated sugar (produced by Dangote Sugar Nigeria, PLC) and the 'Gari' used in this study were

Animal Studies

Seventy (70) albino rats averaging 0.195kg in body weight, were divided into fourteen (14) groups of five (5) rats each. The albino

rats were fed with rat diet mixed with sugar and 'gari' at concentrations of 10%, 20%, 40%, 60%, 80% and 100% w/w per group while the last group was fed normal rat diet with distilled water to serve as Control (0.00g/kg) for 3 weeks. Therefore, the animals were sacrificed, blood collected and taken to the Laboratory for analysis.

Biochemical Studies

The cholesterol was determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxides (Allain *et al.*, 1974). Ten microlitre (10) of sample, Control, standard and distilled water was pipette into respective test tubes then 1000 of cholesterol working reagent was added. It was mixed and incubated for 5 minutes at 37^0C. The absorbance of the sample was measured against the reagent blank at 520nm.The concentration of sample was calculated using the absorbance of sample against absorbance of standard multiplied by concentration of standard.

The triglycerides are determined after enzymatic hydrolysis with lipases. The indicator is a quinoneimine formed from hydrogen peroxide, 4-aminophenazone and 4-chlorophenol under the catalytic influence of peroxidase (Buccolo and David, 1973).

Ten microlitre (10) of sample, Control, standard and distilled water was pipetted into respective test tubes then, 1000 of triglyceride reagent was added. It was mixed and incubated for 5 minutes at 37^0C. The absorbance of the sample was measured against the reagent blank at 520nm. The concentration of sample was calculated using the absorbance of sample against absorbance of standard multiplied by concentration of standard.

Low density lipoproteins (LDL and VLDL) and chylomicron fractions were precipitated quantitatively by the addition of phosphotungstic in the presence of Magnesium ions. After centrifugation, the cholesterol concentration in the HDL (high density lipoprotein) fraction, which remains in the supernatant, was determined. Five hundred microlitre (500ul) of sample, Control standard and distilled water was added into respective test tubes, 1000 of precipitant was added into all the tubes. It was mixed and allowed to stand for 10 minutes at room temperature. It was centrifuged for 2 minutes at 12,000 rpm. Then 10 of supernatant from Control, standard and distilled water was added into their respective test tubes and cholesterol concentration of supernatant was determined as shown above by the methods of Allain *et al.* (1974).

LDL-cholesterol was calculated using the formula of Friedwald *et al.* (1972):

LDL-cholesterol(Mmol/L)=Total cholesterol (Mmol/L)-(Mmol/L)+ TG/2.22)(Mmol/L).

Statistical Analysis

Biochemical data values were reported as Mean$\pm$SEM. One way Analysis of variance (ANOVA) was used to test for the individual effect of treatment groups while student's t-test was used to test for significant differences between the diet types, using Statistical Package for Social Sciences (SPSS) version 16.

RESULTS

The cholesterol concentration was reduced when sugar was added to the diet from 10% to 60%. However, at 80% it increases (Table 1). There was no significant difference in the means (P>0.05). Also, the Cholesterol concentrations showed significant dose-dependent increase in gari fed albino rats (P<0.05). The triglycerides concentrations showed dose dependent decrease in 'gari'-fed rats except at 100% which showed increased concentration(P<0.05). The sugar fed albino rats reported variations in triglyceride concentrations with dose dependent increase from 10% to 60% while it reduced at 80% (P<0.05) as shown below in table 1. The HDL cholesterol concentrations showed significant dose-dependent decrease in sugar fed rats which was not significant (P>0.05). Also, gari treated rats showed dose dependent decrease in HDL concentrations with the exception of 80% (P>0.05). There were dose-dependent variations in LDL cholesterol in both sugar and gari fed albino rats as shown in Table 2.

There was significant difference in cholesterol concentration (Mmol/L) of 1.2117$\pm$ 0.11 in sugar fed rats compared with 1.4183 $\pm$0.11 of gari fed rats. Triglyceride (Mmol/L) concentration was 0.4000 $\pm$ 0.07 in sugar treated rats while it was 0.38 $\pm$ 0.08 in gari feeding. The HDL Cholesterol (Mmol/L) concentration of 0.1217 $\pm$ 0.04 in sugar treated rats was not significantly different from 0.17 $\pm$ 0.07in gari treated albino rats. The LDL cholesterol (Mmol/L) was 0.9200 $\pm$ 0.09 in sugar treated rats while it was 1.09 $\pm$ 0.13 in gari treated rats as shown below in table 3.

Table 1: Effect of Sugar and Gari on Cholesterol and Triglycerides concentrations in albino rats

	Cholesterol (Mmol/l)			Triglycerides (Mmol/l)		
Concentration (%)	Sugar	Gari	P value	Sugar	Gari	P value
0.00	1.35 $\pm$ 0. 07	1.35$\pm$0.12	1.000	0.51 $\pm$ 0.19	0.43$\pm$0.09	0.370
10	1.25 $\pm$ 0.22	1.60 $\pm$0.20	0.241	0.16 $\pm$ 0.11	0.16$\pm$0.12	0.703
20	0.78 $\pm$ 0.23	1.00$\pm$0.09	0.337	0.36 $\pm$ 0.10	0.43$\pm$0.10	0.737
40	1.19 $\pm$ 0.13	1.30 $\pm$0.13	0.401	0.37 $\pm$ 0.29	0.36$\pm$0.28	0.980
60	1.06 $\pm$ 0.06	1.35 $\pm$0.12	0.044	0.61 $\pm$ 0.47	0.33$\pm$0.23	0.641
80	1.55 $\pm$ 0.15	1.55 $\pm$0.12	1.000	0.33 $\pm$ 0.20	0.27$\pm$0.21	0.862
100	1.44 $\pm$ 0.19	1.71 $\pm$0.10	0.275	0.57 $\pm$ 0.21	0.72 $\pm$0.23	0.703

Table 2. Effect of Sugar and Gari on HDL and LDL Cholesterol concentrations in albino rats.

	HDL - Cholesterol (Mmol/l)			LDL - Cholesterol (Mmol/l)		
Concentration (%)	Sugar	Gari	P value	Sugar	Gari	P value
0.00	0.07 $\pm$ 0.01	0.31$\pm$ 0.23	0.363	1.14 $\pm$ 0.07	0.84$\pm$ 0.31	0.499
10	0.06 $\pm$0.00	0.06 $\pm$ 0.01	1.000	1.07$\pm$ 0.25	1.47 $\pm$0.18	0.227
20	0.06 $\pm$ 0.01	0.06$\pm$ 0.01	0.391	0.81 $\pm$ 0.04	0.60$\pm$ 0.16	0.802
40	0.07 $\pm$ 0.01	0.063 $\pm$ 0.01	0.911	0.94 $\pm$ 0.14	1.08$\pm$ 0.25	0.735
60	0.06 $\pm$ 0.01	0.46 $\pm$ 0.24	0.182	0.54 $\pm$ 0.22	0.93$\pm$ 0.30	0.499
80	0.20 $\pm$ 0.13	0.065$\pm$ 0.10	0.861	1.20 $\pm$ 0.26	1.36 $\pm$ 0.09	0.663
100	0.28 $\pm$ 0.13	0.29$\pm$ 0.10	0.852	0.96 $\pm$ 0.24	1.09 $\pm$ 0.14	0.674

Table 3: Overall effect of Sugar and Gari on lipid concentrations in albino rats

Parameter (Mmol/L)	Sugar	Gari	T	P value
Cholesterol	1.2117± 0.11	1.4183 ± 0.11	-3.912	0.011
Triglycerides	0.4000 ± 0.07	0.38 ± 0.08	0.363	0.731
HDL Cholesterol	0.1217 ± 0.04	0.17 ± 0.07	-0.600	0.575
LDL Cholesterol	0.9200 ± 0.09	1.09 ± 0.13	-1.850	0.123

DISCUSSION

The results of this study showed that feeding rats with various concentrations of sugar and gari (processed cassava meal) caused dose-dependent increases in triglycerides, HDL cholesterol and cholesterol concentrations. This is similar to results of Salau *et al.* (2012) who reported an increase in lipid indices as sucrose concentrations increases. Association between sucrose intake and cholesterol level has been established (Albrink and Ulrich 1986, Richard *et al.*, 1989). Increase in sucrose consumption follows closely a linear relationship with plasma cholesterol. Excess intake of sugar has been shown to increase energy density by adding empty calories (Kreb-Smith *et al.*, 1997) and this may lead to imbalance diet. Mukherjee *et al.* (2009) also reported increases in lipid profile particularly triglycerides and very low density lipoprotein cholesterol (VLDL-C).

Connor and Connor, (1997) and Katan *et al.*, (1997) reported that lipid profile of humans consuming high carbohydrate low fat (HCLF) diet shows increases in fasting triacylglycerol while HDL-cholesterol, LDL-cholesterol and total cholesterol were reduced. Mclaughlin *et al.* (2000) have also shown that a high carbohydrate diet markedly increase fatty acid synthesis and denovolipogenesis. A high carbohydrate diet will cause fat to be deposited, while slowing down the burning of existing fat. This is linked to a greater potential to store fat (Hudgins *et al.*, 2000). High fructose diets have been reported to elevate serum triglycerides in man (Kaufmann *et al.*, 1966, Nestel *et al.*, 1970) and experimental animals (Laube *et al.*, 1973, Hill, 1970) although some work has been equivocal (Hill, 1970; Abbott *et al.*, 1990). Reiser *et al.*, (1979) found that cholesterol levels increased significantly when sucrose was substituted for starch in the diet suggesting

that the sucrose moiety of the diet is responsible since sucrose is the major contributor to total calories in the high carbohydrate high calorie (HCHC) diet.

The results of this study showed that feeding rats with various concentrations of sugar and gari caused dose-dependent increases in triglycerides. This agrees with studies by Frayn and Kingman (1995) and Salau *et al.* (2012) while Parks and Hellerstein (2000) reported that a diet high in sucrose (>20%) of energy is associated with an elevation of plasma triglycerides concentration. In humans, hypertriglyceridemia can be induced endogenously by a high fat diet (Austin *et al.*, 1998) or it can be carbohydrate induced (Knittle, and Ahrens 1964, Reaven *et al.*, 1965). A high triacylglycerol (TAG) flux rate was observed in subjects fed HCLF diet (Abbott *et al.*, 1990). The TAG increase observed in humans on a HCLF diet may have been from de novo lipogenesis (Parks, and Hellerstein 2000).Most previous studies examining carbohydrate induced hypertriglyceridemia have used a maximum of 10 days feeding protocols (Nestel *et al.*, 1970) or diets with Fresh feed was provided on daily basis while stale simple sugars (Huff and Nestel 1982). This carbohydrate induced elevation of TAG could be explained as a reduced clearance of TAG rich lipoprotein and increased production. There is also a possibility that reduced lipoprotein lipase activity on a low fat diet may limit the conversion of VLDL to LDL.

The study also showed that LDL cholesterol showed dose dependent decrease in both gari and sugar treated rats. This corroborates the fact that LDL cholesterol is influenced by high sucrose diet (Ryu and Cha, 2003). Previous study using gari on LDL are not available. Oboh *et al.* (2007) reported that total serum cholesterol and LDL cholesterol were found to be significantly lowered in the rabbits fed the high-carbohydrate low fat diet (HCLF) while HDL and TAG increased significantly.

The HDL cholesterol in the sugar and gari fed rats in this study showed dose dependent increase in concentration. This is contrary to report of Salau *et al.* (2012) who reported dose dependent decrease HDL in their study which could be due to duration of feeding, as our study was only three weeks feeding compared to twelve weeks feeding. Also Mensink and Katan (1987) and Ullmann *et al.* (1991) have shown similar changes in healthy men and women fed a strictly Controlled diet rich carbohydrate.

CONCLUSION

The study demonstrates the relationship between intake of gari and sugar on lipid

profile. The study showed that sugar and gari (processed cassava meal) which are carbohydrate diet induced increase in cholesterol, triglycerides and HDL cholesterol while reducing the LDL cholesterol. This suggests that feeding on sugar and gari diets (carbohydrate) could increase risk of cardiovascular disorder.

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