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Sucrose Diet Elevates Cardiovascular Risk Factors in Male Albino Rats

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ABSTRACT

Effects of various concentrations of sucrose diet were assessed on thirty weaning male albino rats divided randomly into five equal groups as follows: G1 (baseline group); G2 (control group given rat chow only); G3, G4 and G5 (groups with energy supply from sucrose at 10, 20 and 30%, respectively). The four groups were fed for twelve weeks and then fasted overnight. They were then anaesthetized with diethyl ether and venous blood was collected using cardio puncture method. Plasma was collected by centrifugation and total plasma cholesterol, HDL cholesterol and serum Triglycerides were assayed using Randox enzymatic kit while VLDL cholesterol, LDL cholesterol, Atherogenic index and coronary risk indices were calculated. Sucrose diet increased energy density. It also increased significantly ($p<0.05$). Plasma total cholesterol, LDL cholesterol, VLDL cholesterol, Triglycerides, Atherogenic and Coronary risk indices while it decreased HDL cholesterol. Present results indicated that sucrose diet at present level of consumption (about 25% energy supply) elevated cardiovascular risk factors in male albino rats and may predispose one to cardiovascular diseases.

Key words: Sucrose diet, lipid profile, cardiovascular disease, rats

INTRODUCTION

Cardiovascular disease remains one of the leading causes of morbidity, mortality and major public health problem in the developed and developing countries (Ignarro *et al*., 2007; Burta *et al*., 2008; Badaruddoza *et al*., 2011; Noroozi *et al*., 2011).

An expanding body of evidence indicates that certain dietary patterns can influence the aetiology, progression and treatment of cardiovascular disease (Osadolor *et al*., 2005; Amadou *et al*., 2009). This may occur by modifying risk factors such as obesity, dyslipidaemia, as well as factors involved in systemic inflammation, oxidative stress and thrombosis (Parikh *et al*., 2005; Napoli *et al*., 2006, 2007; Laleye *et al*., 2007).

Sucrose, a form of carbohydrate common in diet is widely consumed by humans (Ahmed *et al.*, 2001) and its 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, the last joint report of WHO/FAO (WHO/FAO, 2003) apparently exonerated sucrose in the aetiology of cardiovascular disease. This however is contrary to indictment by some other investigators (Szanto and Yudkin, 1969; Albrink and Ullrich, 1986; Johnson *et al.*, 2009). The WHO/FAO (2003) recommended that sucrose should not supply more than ten 10% of energy requirement. The clause which sugar company rejected.

Death from cardiovascular disease may be sudden and sometime without warning; however, there are some risk factors such as plasma total cholesterol (Richard *et al.*, 1989; Mazier-Patrioia and Jones-Peter, 1991), plasma triglycerides (Austin *et al.*, 2000; Van Lennep *et al.*, 2002), HDL cholesterol (Asia Pacific Cohort Studies Collaboration, 2004; Barter, 2005), LDL Cholesterol (Staprans *et al.*, 1996), VLDL cholesterol (Rosenfeld *et al.*, 1987), Atherogenic index (Abbott *et al.*, 1988) and coronary risk index (Alladi and Khada, 1989) which may serve as warning signals. Thus, the assessment of these biochemical parameters may go a long way in predicting cardiovascular diseases, thereby giving the opportunity to abate or control the disease at earlier stage. Also, it may help to establish cause-effect relationship with respect to a particular dietary factor such as sucrose. In view of the above, we set to investigate the implications of excess sucrose consumption on these cardiovascular risk factors.

MATERIALS AND METHODS

Experimental animals: Thirty weanling male Wistar rats purchased from the animal house, Physiology Department, Olabisi Onabanjo University, Ago-Iwoye were used for this experiment. The whole experiment was carried out in 2010 for a period of 15 weeks. The rats' weight ranged between 57 to 63 g. The rats were acclimatized for 2 weeks in individual metal cages where water and rat chow were given *ad libitum*.

Feed preparation: The pellets (rat chow) obtained from Ladokun feed Ibadan, Nigeria were grounded with pestle and mortar and the powdery form was mixed with granulated sugar at different ratios. The combined mixture was moistened with distilled water and then palletized with the palletizing machine. It was then oven dried at 60°C for 2 days to maintain 5% initial water content of the pellets. These pellets were then kept in an air tight polythene bags and given to the animal according to their grouping.

Grouping and management of experimental animals: The experimental animals were randomly divided into 5 groups, labeled and managed as indicated below:

- **Group 1 (G1, baseline):** Rats in this group were sacrificed at onset of investigation to serve as the baseline data
- **Group 2 (G2, normal control):** Rats in this group were placed on commercial diet (rat chow)
- **Group 3 (G3, test group 1):** Rats in this group were placed on experimental diet consisting 10% energy supplied from sucrose

- **Group 4 (G4, test group 2):** Rats in this group were placed on experimental diet consisting of 20% energy supply from sucrose
- **Group 5 (G5, test group 3):** Rats in this group were placed on experimental diet consisting of 30% energy supply from sucrose

The rats were maintained on each respective diet *ad libitum* for a period of twelve weeks. After the 12 week period, the rats were fasted overnight, anaesthetized with diethyl ether and sacrificed. Blood was then withdrawn from the rats by cardiac puncture into EDTA bottle, properly mixed and processed for further analysis. The blood was centrifuged at 3,000 rpm for twenty minutes and the plasma was collected.

Biochemical analysis: Plasma cholesterol, HDL cholesterol and plasma triglycerides were determined by enzymatic method using Randox kits. LDL cholesterol was obtained by deduction using Friedewald equation (Friedewald *et al.*, 1972). Atherogenic index was calculated using the formula of Abbott *et al.* (1988). Coronary risk index was determined by the method of Alladi and Khada (1989). VLDL cholesterol was estimated by dividing plasma triglyceride by 5.

Statistical analysis: All data were analyzed using one-way ANOVA, Level of significance was assessed using Duncan Multiple Range Test at $p<0.05$. SPSS 14.0 software was used for data analyses.

RESULTS

Table 1 showed the inclusion of sucrose in the diet of the rats which linearly increased the energy contents of the feed, thereby increasing the energy density. Table 2 is the lipid profile of albino rats placed on diets supplying various percentage of energy from sucrose. Significant differences ($p<0.05$) were observed in total plasma cholesterol. An increasing trend in plasma total cholesterol from (G1 to G5) with increase in percentage energy from sucrose was observed. A

Table 1: Energy content and energy density of the diet

Rat groups	Percentage energy from sucrose (%)	Total feed energy in 100 g (kcal)	Energy density (kcal/100 g diet)
G1	0	364.00	3.64
G2	0	364.00	3.64
G3	10	369.00	3.69
G4	20	373.94	3.74
G5	30	379.16	3.79

Sucrose 1 g supplies 3.98 kcal

Table 2: Plasma cholesterol profile of rats fed with different concentrations of sucrose diet

	Plasma T-C	Plasma LDL-C	Plasma VLDL-C	Plasma HDL-C	Plasma TG
Rat groups	---------- (mg dL^{-1}) ----------				
G1	78.60±0.70^{a}	21.51±1.27^{b}	26.98±0.09^{a}	63.35±1.22^{e}	134.25±0.57^{a}
G2	91.09±3.30^{b}	15.27±1.53^{a}	26.46±0.11^{a}	60.56±1.27^{d}	132.36±0.57^{a}
G3	104.92±1.23^{c}	30.58±1.61^{c}	28.17±0.17^{b}	49.55±0.76^{c}	145.87±5.13^{b}
G4	114.43±1.07^{d}	41.96±1.46^{d}	30.19±0.19^{c}	39.53±0.55^{b}	149.31±1.45^{b,c}
G5	122.55±1.68^{e}	63.78±1.72^{e}	31.17±0.11^{d}	26.25±0.55^{a}	155.07±0.68^{c}

Values are expressed as Mean±SEM for 6 rats per group, Mean values were compared using one way-ANOVA, Level of significance 0 was assessed using Duncan's multiple range test (DMRT) of $p<0.05$, Values with different superscript (in the same row) are significantly different

reduction was observed in the LDL cholesterol concentration of normal control group (G2) when compared with the baseline value group (G1). When the plasma LDL concentration of the normal control rat was compared with those of the test groups, significant increases ($p<0.05$) were observed in LDL cholesterol with increasing percentage energy source from sucrose. Results of the VLDL cholesterol indicated that there was no significant difference ($p>0.05$) in VLDL cholesterol between the baseline and the normal control group. The VLDL cholesterol however continued to increase with increase in sucrose diet.

Though the HDL cholesterol concentration did not vary between the baseline and the control group, increases in sucrose diet significantly decreased the plasma HDL concentration. This decrease was also observed to be concentration dependent. Triglyceride concentration was also observed to increase with increase in percentage energy source from sucrose, however, no significant difference ($p>0.05$) was observed in the triglyceride concentration of the rats placed on diet that supplied 10% energy source from sucrose and that which supplied 20% energy from sucrose. In addition, no significant difference ($p>0.05$) was observed in the triglyceride concentration of the rats placed on diet that supplied 20% energy from sucrose and that which supplied 30% energy from sucrose but a significant increases ($p<0.05$) was observed between rats placed on diet that supplied 10% energy from sucrose and that of 30% energy from sucrose. Table 3 showed average percentage risk. The table indicated that average percentage risk increases with increase in percentage energy source from sucrose. Results of atherogenic and coronary risk indices of albino rats consuming different concentration of sucrose in the diet is shown in Fig. 1. A significant increase ($p<0.05$) in atherogenic index was observed when G2 was compared with the test groups G3 to G5. A similar trend was observed in coronary risk index.

Table 3: Plasma lipid profile percentage risk of rats fed with different concentrations of sucrose diet

	Increment (%)				
Rat groups	T-C risk	LDL-C	VLDL-C	HDL-C	TG
G2	0	0	0	0	0
G3	15.18	100.26	6.46	18.18	10.21
G4	25.62	174.79	14.10	34.73	12.81
G5	34.54	317.68	17.80	56.65	17.16

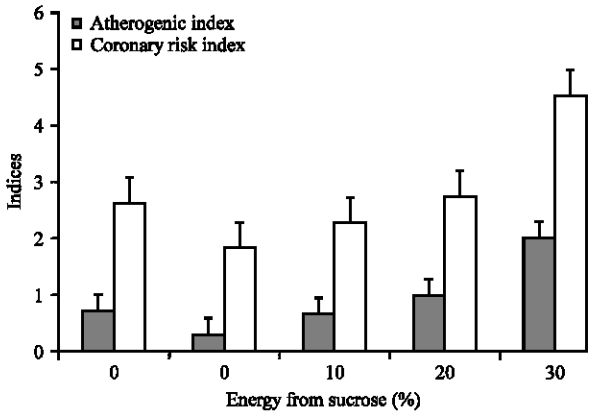

Fig. 1: Atherogenic and coronary risk indices of rats with fed different concentrations of sucrose diet

DISCUSSION

In previous reports, contradictory claims abound as to what level of sucrose consumption is optimal for good health. While WHO/FAO recommend intake of sucrose not more than ten percent of energy supply (WHO/FAO, 2003). The institute of medicine of Food and Nutrition Board reported 25% in energy intake from sucrose may not cause any adverse effect (Food and Nutrition Board, 2005). However, the present study showed the adverse effect of high sucrose diet on lipid profiles that are associated with cardiovascular disease.

Excess intake of sugar has been shown to increase energy density by adding empty calories (Krebs-Smith *et al*., 1997) and this may lead to imbalance diet. The prepared diet mimics sucrose diet consumed by many individuals, thus such diet increases energy density of the food as the sucrose concentration increases and decrease in other nutrient density (Salau *et al*., 2011) which may lead to energy saving pathway such as lipogenesis or sparing effect on other energy yielding nutrients such as protein and lipids and may consequently increase body fat.

Association between sucrose intake and cholesterol level has been established (Albrink and Ullrich, 1986; Richard *et al*., 1989). Increase in sucrose consumption follows closely a linear relationship with plasma cholesterol. Ten percent energy supply from sucrose led to 15.18 percentage risk in plasma cholesterol when compared with the control, while 20 and 30% energy supply from sucrose led to 25.62 and 34.54% plasma cholesterol risk, respectively. One of the possible mechanisms could be as a result of increase in fructose consumption (a component of sucrose) which increases oxidative stress (Faure *et al*., 1977). This in effect may depress the antioxidant status and it is in this process that other molecules such as cholesterol picked up the oxidant, thereby becoming oxidized (which are naturally not recognized by normal cell receptors molecules for cholesterol but picked up by macrophages). In effect, there is dire need for cholesterol in the cell leading to consequent increase in its production. The other probable mechanism is that cholesterol production is a way of disposing excess energy by the individual who consumes more than required energy.

LDL cholesterol was observed to increase as sucrose consumption increases in a more profound manner. Intake at 10, 20, 30% energy supply from sucrose shows increase in plasma LDL at about 100, 174 and 317%, respectively. This corroborates the fact that plasma LDL cholesterol is influenced by high sucrose diet (Ryu and Cha, 2003) and also clarifies that the fraction of cholesterol that increased significantly is LDL cholesterol.

A similar trend was observed in plasma VLDL cholesterol. Increase VLDL cholesterol has been implicated in cardiovascular risk (Rosenfeld *et al*., 1987) and has also been reported to be influenced by high sucrose diet (Frayn and Kingman, 1995).

On the other hand, a striking inverse relationship was observed in sucrose in-take and HDL cholesterol. As sucrose supplies energy at 10, 20, 30%, HDL cholesterol was observed to reduce by 18, 34 and 56%, respectively. This agrees with previous studies of various investigators (US. USSR Steering Committee, 1984; Archer *et al*., 1998; Mensink *et al*., 2003; Appel *et al*., 2005).

The result also indicates that as the concentration of sucrose increases, plasma triglyceride also increases. This agrees with previous study (Frayn and Kingman, 1995) and the report of Parks and Hellerstein (2000) that a diet high in sucrose (>20%) of energy is associated with an elevation of plasma triglycerides concentration. This could be as a result of increase hepatic secretion of triglyceride and decreased clearance of plasma triglyceride (Xve *et al*., 2001). It is important to point out that it appears that increase in sucrose intake does not increase triglycerides concentration proportionately. Ten percent of energy supply from sucrose increases triglyceride by 10.21% while 20% sucrose intakes leads to 12.81% increase and 30% sucrose increased triglycerides by 17.16%.

Analysis shows that when sucrose supply 10% of energy, lipid profile risk was at 30.06% while at 20%, lipid profile risk rose to 52.41%. At 30% of energy supply from sucrose, lipid profile risk increased to 88.77%.

Atherogenic and coronary risk indices are ratios that can be used as quick assessment of cardiovascular disease and have been shown to be influenced by diet (Alam *et al*., 2011; Ibegbulem and Chikezie, 2012). Increases in these indices are noticed as sucrose consumption increased. Though, Ryu and Cha (2003) reported no significance difference in the atherogenic index of sucrose diet and normal diet the difference observed could be due to duration of feeding which was four weeks compared with twelve weeks in our study.

CONCLUSION

The study demonstrated the relationship between intake of sucrose and cardiovascular risk factors (lipid profiles). Increase in sucrose consumption increased cardiovascular risk factors upsetting the balance between energy intakes and other nutrient. Consequently, it increases plasma triglycerides, total cholesterol, LDL cholesterol VLDL cholesterol, atherogenic and coronary risk indices, while decreasing HDL cholesterol. Our result corroborates the fact that energy supply from sucrose should not be more than 10% as stated by WHO/FAO at least for optimal cardiovascular health.

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