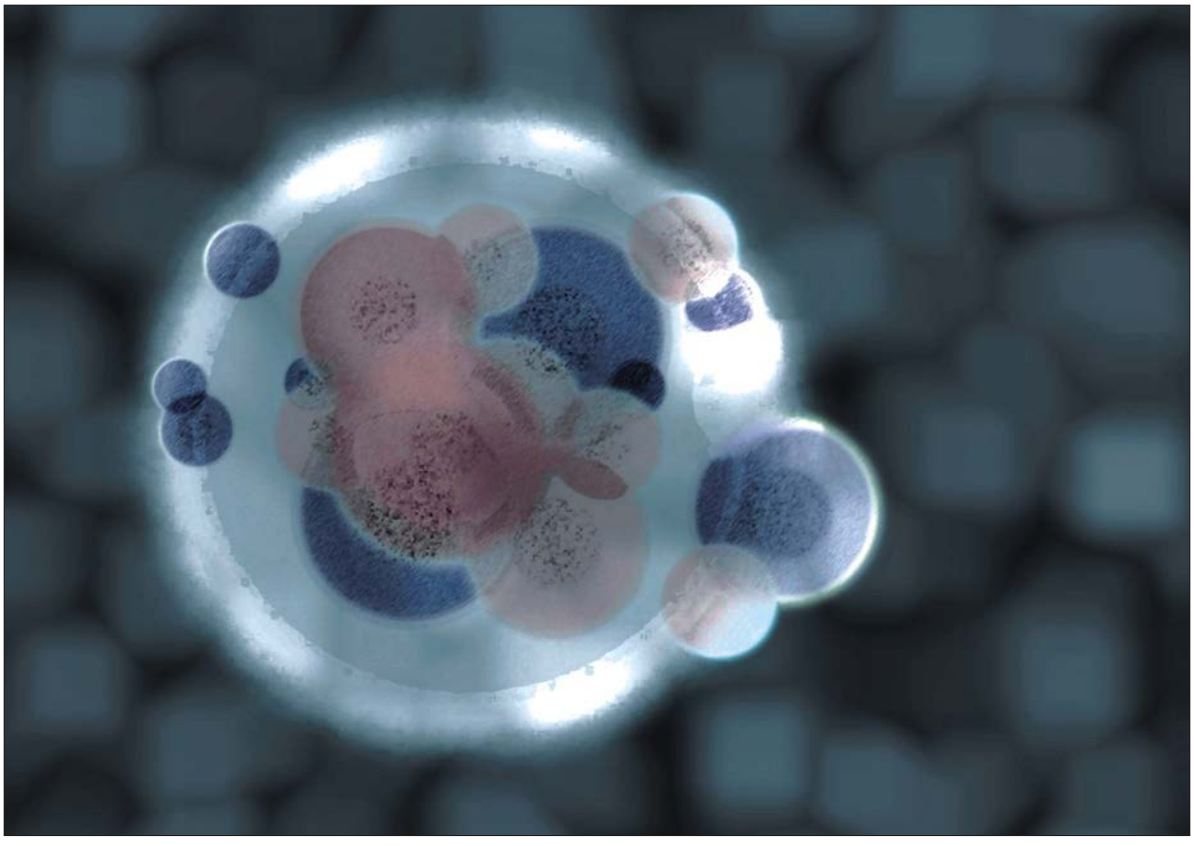

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Research Article
Chronic Sucrose Consumption Adversely Altered Antioxidant Status, Lipid Profile and Peroxidation of Rats Testes

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Abstract

Background and Objective: Sucrose has been implicated in the etiology of many diseases with pronounced effects on various organs, such as liver, kidney and brain to mention but few. However, its effect on reproductive organ appears data-sparse. The study investigated the effect of long term feeding of sucrose diet on the testes of male albino rats. **Materials and Methods:** Twenty four male albino rats grouped into 4 with varying concentrations of sucrose supplying energy at 0% (control), 10% (SUD 10%), 20% (SUD 20%) and 30% (SUD 30%), respectively. Animals were fed for 6 months and the testes were harvested for assessment of lipid profiles and oxidative stress parameters. **Results:** Substantial increase ($p<0.05$) was observed in triglycerides, total, LDL and VLDL cholesterols while decrease ($p<0.05$) was observed in HDL cholesterols as sugar content of diet increases. Whereas significant decrease was observed ($p<0.05$) in glutathione-s-transferase (GST), superoxide dismutase (SOD), catalase (CAT) and reduced glutathione (GSH) levels, substantial increase ($p<0.05$) was observed in malondialdehyde (MDA), an index of lipid peroxidation. **Conclusion:** In conclusion, we opined that the alteration in antioxidant status, lipid peroxidation and lipid profile may adversely affect reproductive functions in male albino rats fed long term sucrose diet.

Key words: Male albino rats, sucrose diet, lipid profile, lipid peroxidation, antioxidant status

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

The involvement of sucrose in the causation of many diseases has always been on a resounding note[1-3]. It is believed that its high consumption mainly when it supplies more than 10% energy content of food[4] may be prejudicial to health and consequently harmful to various organs in the body[5,6]. It has been established that high sucrose consumption affects some organs and tissues adversely by altering its biochemical and physiological integrity. Some of these organs and tissues are heart, liver, brain, blood, bones, testes which high sucrose consumption may alter their biochemical parameters and histochemical architecture[2,5,7-9]. One of the major assaults of high sucrose consumption is the alteration of lipid profile (usually caused by fructose a component of sucrose) by way of dyslipidemia[2]. Some of the lipid components that can be adversely affected by the consumption of high sucrose diet are triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol and VLDL cholesterol[2].

Sucrose diet is also known for the lowering of the antioxidant system and overwhelming the body defence mechanism, consequently leading to oxidative stress[8,10]. Some of the antioxidants that are of veritable importance in the body for counteracting of free radical assaults are catalase, superoxide dismutase, GSH and GST[11]. These 2 pathways of sucrose adversely affecting the body, are closely related in causation and consequence juxtaposed. The oxidative stress and dyslipidemia are therefore, some of the major pathological pathways of sucrose-induced disorders[2,10].

The controversies on sucrose effect on various tissues and organs appeared not yet settled. The optimal level of consumption is still debatable, considering the difference in body response with respect to various tissues and organs. Unfortunately WHO and FAO[4], Food and Nutrition Board[12], American Heart Association Nutrition Committee[13] and Dietary Guidelines for Americans[14] are all discordant on the optimal level of sucrose consumption. In this regard, the need for comprehensive screening of various organs/tissue responses to different levels of sucrose consumption is expedient. Male reproductive organs, especially the testes, could be affected by various factors, including dietary sucrose[6]. However, the amount of sucrose and the magnitude of the effect is still a subject of controversy. In many experiment oral administration of sucrose is mostly through solution which does not depict the way sucrose is being consumed by majority. In fact sucrose is consumed more as in hidden form present in most solid food taken such as bread, candies, pastries etc.

Also many studies is a "one-concentration" approach such as either 30 or 20% sucrose solution which is more of toxicological approach rather than grading the sucrose consumption to mimic the practical situation of different consumption levels in the diet.

Thus this study assessed the different levels of sucrose consumption that would adversely affect the lipid profile, antioxidant status and lipid peroxidation in the testicular tissue.

MATERIALS AND METHODS

Chemicals: All reagents used in this study were of analytical grade, purchased from Sigma-Aldrich Corporation, St. Louis, Missouri, United States and Randox Laboratories, Ireland.

Animals handling: Twenty four weanling male Wistar rats purchased from the animal house, Physiology Department, University of Ibadan, Oyo State, Nigeria were used for this experiment. The whole experiment was carried out in 2018 for a period of 6 months spanning from June to December at Redeemer's University Animal House. Animals weighed between 50-75 g were acclimatized for 2 weeks in individual metal cages where water and rat chow were given *ad libitum*. The care and handling of animals were in accordance with the guidelines established by the National Health and Medical Research Council[15].

Feed preparation: Pellets rat chow, purchased from Ladokun Farms Limited, Ibadan, Oyo State, Nigeria were grounded into powder form and then mixed with granulated sugar at different energy ratios. The mixture was moistened with distilled water and re-pelletized with an industrial pelletizing machine and then dried in an oven at 60°C for 2 days to maintain 5% initial water content of the pellets. The pellets were stored in an airtight container. The sucrose weight to energy equivalent was calculated based on feed label. The gross energy/g of rat chow according to Ladokun feed label was 3.65 kcal and the sucrose energy/g weight is 3.94 kcal g^{-1} (https://en.wikipedia.org/wiki/Sucrose). The resultant weight equivalent to 1 kcal of rat chow and sucrose is 0.27 and 0.24 g, respectively. These factors were multiplied by the percentage rat chow and sucrose required to compound different diets for each group, with respect to their group label. At the end of 2 weeks of acclimatization, the animals were divided into 4 groups of 6 rats per group and fed *ad libtum* with water and their respective feed. Grouping and treatments are as indicated below:

Control : Rats in this group were placed on a commercial diet
SUD 10% : Rats in this group were placed on an experimental diet consisting10% energy supplied from sucrose
SUD 20% : Rats in this group were placed on an experimental diet consisting of 20% energy supply from sucrose
SUD 30% : Rats in this group were placed on an experimental diet consisting of 30% energy supply from sucrose

The rats were maintained on each respective diet *ad libitum* for a period of 6 months. After the 6 months period, the rats were fasted overnight, sacrificed by cervical dislocation. The testes were harvested, kept in sodium phosphate buffer, homogenized using Teflon homogenizer, later centrifuged and the supernatant collected for various analysis.

Biochemical analysis: Plasma cholesterol, HDL cholesterol and plasma triglycerides were determined by enzymatic method using Randox kits. The LDL and VLDL cholesterols were obtained by deduction using the Friedewald[16] equation (VLDL cholesterol was estimated by dividing plasma triglyceride by five). Atherogenic index was calculated using the formula of Abbott *et al.*[17]. The coronary risk index was determined by the method of Alladi and Shanmugasundaram[18]. Catalase was determined by the method of Sinha[19], reduced glutathione by the method of Beutler *et al.*[20], glutathione-s-transferase was determined by the method of Habig *et al.*[21], superoxide dismutase was estimated by the method of Misra and Fridovich[22] while lipid peroxidation was determined by measuring malondialdehyde level according to the method of Varshney and Kale[23].

Statistical analysis: The data were analyzed using one-way analysis of variance (ANOVA). Inter-group comparisons were by Bonferroni *post hoc* test (Graphpad Prism 8). The level of significance was taken to be $p<0.05$ and values were expressed as mean±SEM.

RESULTS AND DISCUSSION

Effect of sucrose diet on triglyceride level in testes of rats: The effect of sucrose diet in the testes is shown in Fig. 1. There was no significant difference between the control and SUD 10% group. However, there was a significant ($p<0.05$) increase in rats fed with SUD 20 and 30%, respectively when compared to the control group. The effect of the SUD was also dose-dependent as the exposure groups were also significantly ($p<0.05$) different from one another. In epidemiological studies, consumption of high sucrose diet has been linked to unfavourable blood lipids[24] that may be scaffold to various disorders such as insulin resistance[25], fatty liver[26], type II diabetes[27,28] and cardiovascular diseases[29]. Triglyceride is one of the lipid parameters that is usually affected by dietary factors. Its high level in the blood which may lead to hypertriglyceridemia and its excess in various tissues have been implicated in the etiology of many diseases[30]. The trend observed in our study is reflective of a previous report by Salau *et al.*[2].

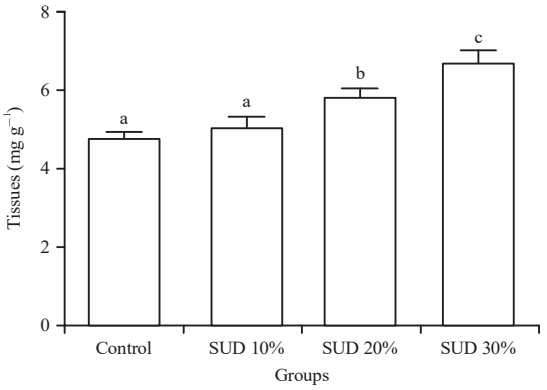

Fig. 1: Triglyceride level in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at $p<0.05$

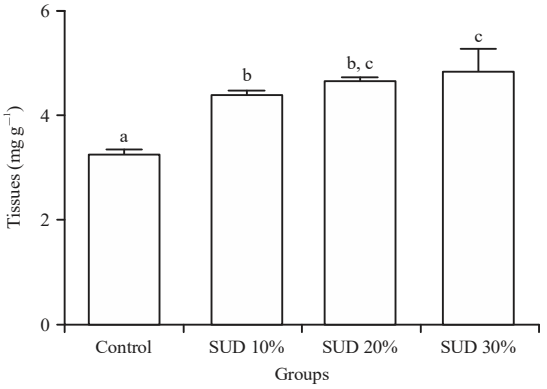

Fig. 2: Total cholesterol level in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at $p<0.05$

Effect of sucrose diet on total cholesterol level in testes of rats: The group of animals fed with SUD 10% significantly ($p<0.05$) increased in total cholesterol levels while the animals fed with SUD 20% and 30% also elicited increase in total cholesterol levels, respectively in comparison to the control group. However, comparisons among the SUD treatment groups did not reveal any significant change (Fig. 2). Relationship between sucrose intake and cholesterol levels has been established by Albrink and Ullrich[31] and

Zhang *et al.*[32]. However, response of various tissues to this lipid differs. For instance, in the previous study, the plasma response followed a close linear relationship to sucrose concentration in more or less a direct form[2] whereas in this study though sucrose consumption increased cholesterol concentration did not follow such linearity as reported earlier by Salau *et al.*[2]. The increase in cholesterol level maybe reasonably linked to the fructose moiety of the sucrose, this has various implications including elevation in oxidative stress[32], male infertility[6,33] and derangement in the tissue chemistry.

Effect of sucrose diet on low-density lipoprotein-cholesterol level in testes of rats: Sub-chronic feeding of rats to SUD 10% did not produce any significant ($p>0.05$) change relative to the control group. However, at the higher doses of SUD 20% and SUD 30%, LDL cholesterol levels were significantly elevated in comparison to control groups (Fig. 3). These treatments were not dose-dependent as there were no significant changes with increase in SUD diet. Elevation in plasma LDL cholesterol usually referred to as bad cholesterol is a good indicator of hypercholesterolemia[34]. It has been shown that high sucrose diet increased LDL cholesterol fractions[35]. In this study there was an increase in LDL cholesterol in thousands of folds down the line of sucrose concentration indicating that a slight shift in concentration of sucrose has a high magnitude effect on LDL cholesterol. This may cause a pronounced adverse effect, as most of this LDL cholesterol may become oxidized thereby leading to increased atherogenicity in tissues[36].

Effect of sucrose diet on very low-density lipoprotein-cholesterol level in testes of rats: The effect of VLDL cholesterol in testes of rats is shown in Fig. 4. The significant difference was observed in SUD 20% and SUD 30% VLDL cholesterol levels, whereas, there was no such significant increment at the SUD 10% diet when compared with control groups. Furthermore, the effect of SUD diet was concentration-dependent with the SUD 10% showing the least effect on VLDL-cholesterol. The VLDL-cholesterol has been implicated in causation of diseases and is known to be influenced by sucrose intake[37,38]. The increased levels of VLDL-cholesterol as energy supply from sucrose increased are indicative of adverse effects and propensity to atherogenicity.

Effect of sucrose diet on high-density lipoprotein-cholesterol level in testes of rats: Figure 5 showed the effect of sucrose on high-density lipoprotein cholesterol. Increasing concentrations of SUD elicited reduction of HDL cholesterol. The HDL cholesterol, otherwise known as "good cholesterol" is beneficial to the body. The HDL-cholesterol's ability to mop up cholesterol from tissues for breakdown in the liver and excretion through bile duct to

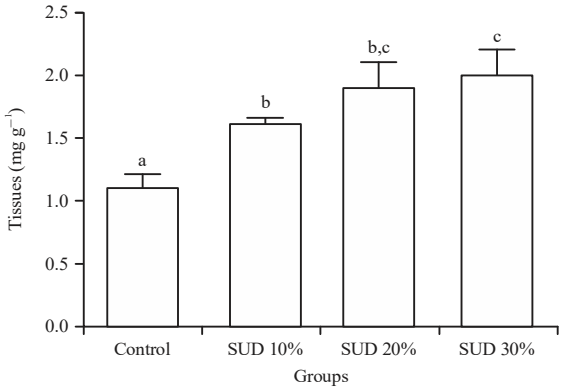

Fig. 3: Low-density lipoprotein-cholesterol level in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at $p<0.05$

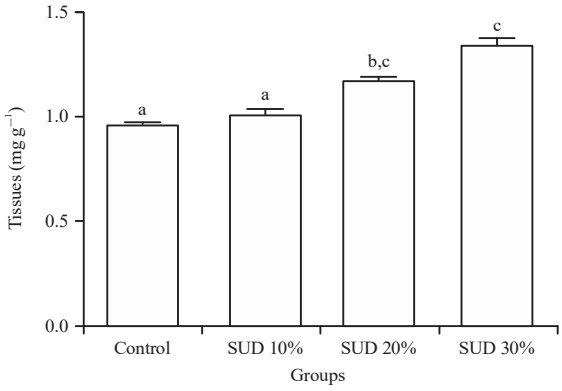

Fig. 4: Very low-density lipoprotein-cholesterol level in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at $p<0.05$

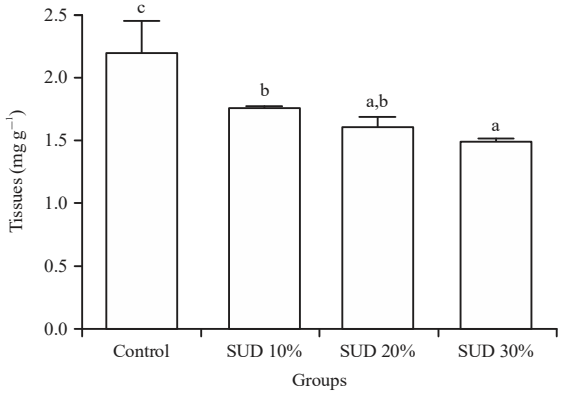

Fig. 5: High-density lipoprotein-cholesterol level in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at $p<0.05$

the intestine for egestion contributes to its benefits. In this study, the concentration-based depletion of HDL-cholesterol has been touted as a causal effect of plaque formation and increased risk of atherogenic incidence[2,39,40].

Effect of sucrose diet on atherogenic and coronary risk indices in testes of rats: The atherogenic index (AI) and the coronary risk index (CRI) in rats treated with various concentrations of SUD are shown in Fig. 6. The AI and CRI correlated with the lipid profiles observed in this study. The atherogenic susceptibility increased with increasing concentration of sucrose diet. Similarly, migration from SUD 10 and 20% gave a percentage coronary risk of 1.4, while a dietary transition from SUD 10 and 30% produced a percentage coronary risk of 2.2.

Effect of sucrose diet on superoxide dismutase activity in testes of rats: Figure 7 showed the SOD activity in the testes of rat fed sucrose diet for 6 months. No significant difference was observed between control and SUD 10%. However, there was a significant reduction in SOD activity in the testes of rats exposed to SUD 20 and 30%, respectively. Equally, there was a significant inter-group difference between the groups fed SUD 10 and 30%. Superoxide dismutase, a metallic antioxidant enzyme plays an active role during the appearance of stress as a result of free radical generation. It is considered as the first line of defence against deleterious effects of oxyradicals in the cell by catalyzing the dismutation of superoxide radicals to hydrogen peroxide and molecular oxygen[41]. Studies have shown an inverse relationship between sucrose consumption and SOD level[5,42]. Testes, in addition to the conventional cytosolic (Cu/Zn) and mitochondrial (Fe/Mn-SOD), also features extracellular SOD which is produced by sertoli and germ cells[43] as part of its defensive armoury against free radicals. In this study, reduction was observed as sucrose consumption increased. One of the major culprits of sucrose diet was believed to be fructose moiety of the sucrose molecule which is in equal ratio to glucose and majorly responsible for oxidative stress.

Effect of sucrose diet on catalase activity in testes of rats: Figure 8 showed the catalase activity in the testes of rat fed sucrose diet for 6 months. No significant difference was observed between control and other groups except the SUD 30%. A decrease in catalase activity has been associated with oxidative stress[44]. Its alteration in testes could be as a result of various factors such as exercise, cold, toxins, xenobiotic, dietary factors which sucrose is inclusive[45]. One of the major components of sucrose believed to be responsible for the oxidative stress in sucrose diet is the fructose moiety. In this study, catalase activity was found to be significantly reduced at 30% energy supply from sucrose which was about 30% activity reduction when compared with the control.

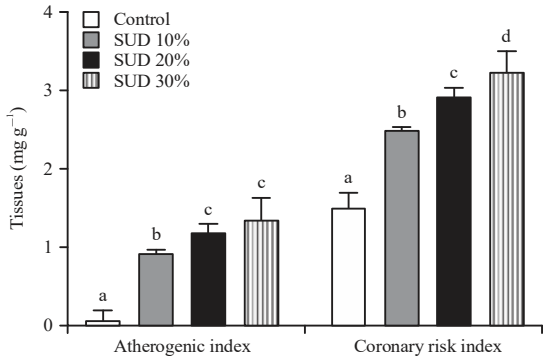

Fig. 6: Atherogenic index and coronary risk index in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at p<0.05

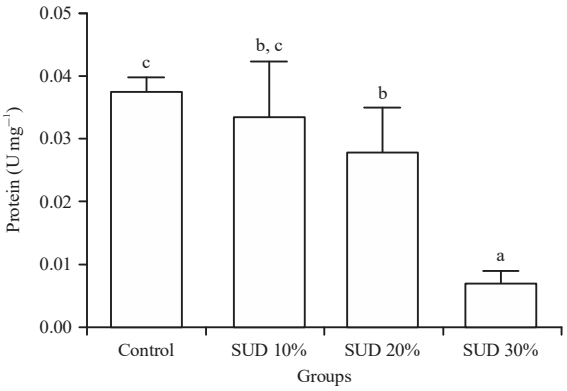

Fig. 7: SOD activity in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at p<0.05

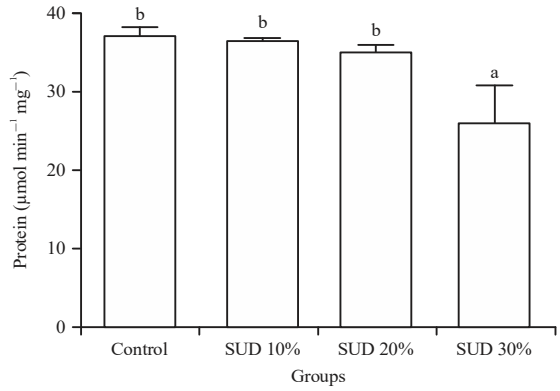

Fig. 8: Catalase activity in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at p<0.05

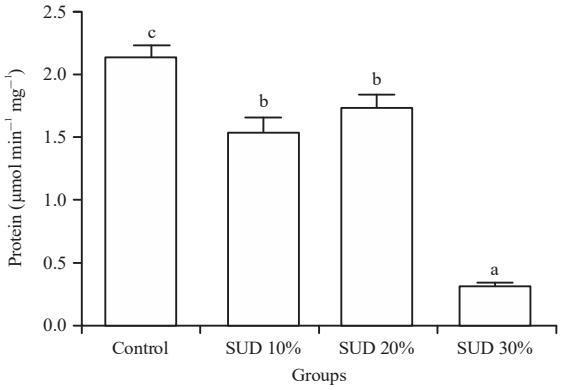

Fig. 9: Glutathione-s-transferase activity in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at p<0.05

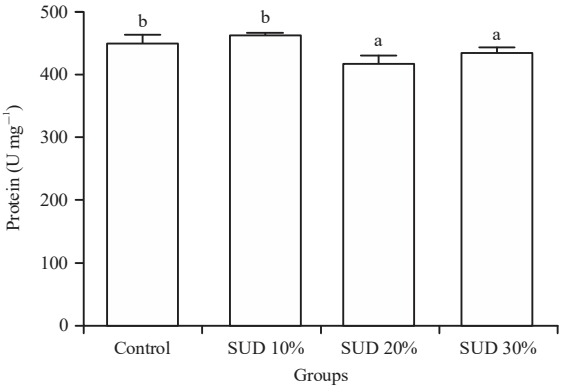

Fig. 10: Reduced glutathione level in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at p<0.05

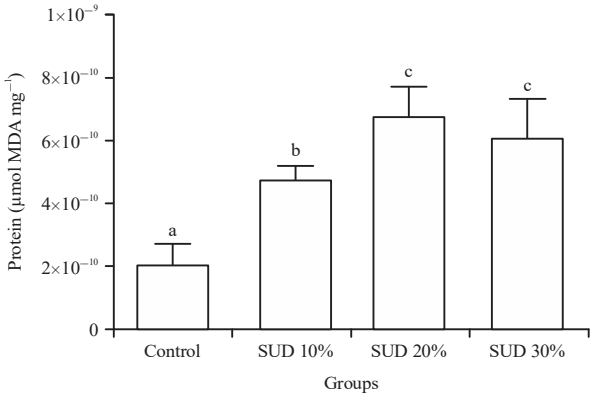

Fig. 11: Lipid peroxidation level in the testes of rats sub-chronically fed sucrose diet
Different letter on bars indicate significant difference at p<0.05

Effect of sucrose diet on glutathione-s-transferase (GST) activity in testes of rats: There was no observable difference in SUD 10 and 20%. However, a significant decrease was observed when the SUD 10, 20 and 30% were compared with the control group (Fig. 9). The GSTs are a group of antioxidant enzyme family, which renders epoxide, hydroperoxides and unsaturated aldehydes harmless. In humans, low GST activity is associated with diseases incidence, such as colon cancer[45]. The GST may be active in the detoxification of xenobiotics in human testes and also active in the epididymis of rat testes[46]. Reduction in GST as the sucrose concentration increases may portend an adverse effect on the testicular functions and spermatogenesis[47]. The trend is almost similar to that of GSH which could be as a result of the relationship between GST and GSH in detoxification process[48].

Effect of sucrose diet on reduced glutathione (GSH) level in testes of rats: Introduction of sucrose at 10, 20 and 30% energy supplies did not affect GSH level (Fig 10). The GSH is a major cellular antioxidant required for several numbers of cellular functions and a potent free radical scavenger[49,50]. Adebayo *et al.*[51] reported that decreased GSH/GSSG ratio is an index of redox imbalance. Glutathione deficiency contributes to oxidative stress and, therefore, may play a key role in ageing and the pathogenesis of many diseases. It protects plasma membrane from lipid peroxidation, scavenges superoxide and prevents singlet oxygen formation[51]. It is postulated that ROS has a greater affinity for the thiol groups of bio-molecules, thus depleting intracellular thiols inducing reduced GSH content in testicular tissue[52]. The result is suggesting that the effect of sucrose consumption on GSH might not be well pronounced as the other antioxidants.

Effect of sucrose diet on malondialdehyde (MDA) level in testes of rats: There were significant changes in malondialdehyde levels at all SUD concentrations when compared with control. In addition, only the SUD 10% versus SUD 20% showed significant inter-group change (Fig. 11). Malondialdehyde, a major breakdown product of lipid peroxidase is an index of the extent of oxidative damage to cellular structures[53]. Thus, oxidative stress and damage that free radicals inflict on cells might be quantitatively determined using MDA as a biomarker of lipid peroxidation.

Mammalian cells contain antioxidants such as glutathione peroxidase and catalase which can detoxify free radicals by converting them to more stable molecules within the cell[54]. Overwhelming antioxidant enzymes with free radicals results in the reduction of the antioxidant's defense and induction of lipid peroxidation evident in elevation of MDA levels[55]. It is reported that elevated production of ROS in the testes has the propensity to cause significant alteration in tissue physiology or induce oxidative damage to DNA, which is a potential risk to male reproduction.

CONCLUSION

This study revealed that an increase in sucrose consumption especially at 20 and 30% energy supply adversely altered lipid profiles, reduced the antioxidant level and consequently increased oxidative stress as assessed by lipid peroxidation. Alterations of these parameters may have a dire consequence on male reproductive organs as changes in these biochemical parameters might also affect the physiology, morphology and consequent functions of this tissue among which male infertility maybe of high probability. Thus, it is suggested that sucrose intake should not be more than 10% of energy supply as recommended by WHO/FAO report of 2003.

SIGNIFICANCE STATEMENT

This study highlighted the adverse effects of long term feeding of sucrose diet (concentrations of 10, 20 and 30%) in the testes of male rats. This study also provided baseline data that will help other researchers explore other molecular mechanisms of adverse outcomes of sub-chronic diets. Possibly, a novel hypothesis involving impaired spermatogenesis and endocrine imbalance after long term sucrose intake could be postulated.

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