Lipidemic effects of common edible oils and risk of atherosclerosis in diabetic Wistar rats

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Original Article

Abstract

BACKGROUND: Diabetic state potentiates atherosclerosis and the type of edible oil consumed by the individual may affect this further. This study aimed to determine if the common edible oils in Nigeria have any effects on the lipid profiles and arteries of alloxan-induced diabetic male Wistar rats.

METHODS: Thirty male Wistar rats were randomly divided into five groups of normal control, diabetic control, animals on diet enriched with refined, bleached deodorized palm oil (RBD-PO), animals on diet enriched with soya oil, and animals on diet enriched with olive oil. At the end of 8 weeks, the lipid profiles of the animals were determined before sacrificing them. Their aortas were subsequently harvested for histological examination.

RESULTS: The olive oil fed group had the highest level of total cholesterol (TC), non-high-density lipoprotein cholesterol (non-HDL-C), lowest HDL-C, and highest artherogenic index (AI). Diabetic animals fed on RBD-PO had a lower non-HDL-C, higher HDL-C, and lower AI than diabetic animals fed on olive oil or soya oil. However, the diabetic animals fed on RBD-PO had the highest triglyceride level. When the aortas were examined histologically, there were no atherosclerotic lesions in all the control and experimental groups except those fed on 10% soya oil enriched diet that had type II atherosclerotic lesions according to American Heart Association (AHA).

CONCLUSION: The result of our study showed that RBD-PO appears to offer a better lipid profile in the diabetic animals compared with olive oil and soya oil. Soya oil appears to cause the development of atherosclerosis in diabetic state.

Keywords: Diabetes, Wistar Rats, Atherosclerosis, Lipids

Date of submission: 12 July 2016, ***Date of acceptance:*** 12 Oct. 2016

Introduction

Atherosclerosis is the single largest cause of death and disability in the Western world and in the next two decades will be the leading cause of death worldwide.[1] The modifiable risk factors for atherosclerosis include dyslipidemia and diabetes mellitus. However, no risk factor on its own can sufficiently produce an atherosclerotic lesion.[2] The abnormal metabolic state in diabetes includes chronic hyperglycemia, dyslipidemia, and insulin resistance all of which render arteries susceptible to atherosclerosis. This may be worsened by decreased removal of triglycerides (TGs) into fat depots and decreased activity of lipoprotein lipase.[3] Dietary manipulation can exacerbate or ameliorate these processes. Common outbred rat strains such as Sprague–Dawley and Wistar rats have the capacity to develop elevated low-density lipoprotein cholesterol (LDL-C) and atherosclerosis with appropriate dietary manipulations.[4] These animal models are useful in studying the effects of edible oils on the development of atherosclerosis.

The edible oils of interest in our study were palm, soya, and olive oils. Palm oil is the most widely produced edible vegetable oil in the world[5] and its nutritional and health attributes have been well documented.[6] Its components include palm olein a mono-unsaturated fatty acid (MUFA) the liquid fraction and palm stearin a saturated fatty acid, the more solid fraction. Feeding experiments in various healthy animal species and humans have highlighted the beneficial role of fresh palm oil to health. These benefits include reduction in the risk of arterial thrombosis and atherosclerosis, inhibition of cholesterol biosynthesis and platelet aggregation, and

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reduction in blood pressure.[7] Similar benefits have not been well documented in diabetes.

Soya oil contains approximately 60% of polyunsaturated fatty acids (PUFA), 24% MUFA, and 16% of saturated fatty acids.[8] This high level of PUFA dietary intake can improve the blood lipid profile status.[9,10] On the other hand, the composition of olive oil is complex, the major groups of compounds thought to contribute to its observed health benefits include oleic acid, phenolics, and squalene, all of which have been found to inhibit oxidative stress.[11,12]

Most of the studies on lipid profile and atherosclerosis in experimental animals have been conducted using edible oils available in the research location with little data in diabetic states. No work has been done on the effects of the common edible oils available in Nigeria on the lipid profile and the aortic wall of male diabetic Wistar rats. Considering the fact that vascular diseases are the principal cause of death and disability in people with diabetes,[13] a good knowledge of how these common dietary oils affect the lipid profile and the aorta would be of benefit in managing this chronic condition. The aim of this study is to determine if the common edible oils in Nigeria have any effects on the lipid profiles and arteries of alloxan-induced diabetic male Wistar rats.

Materials and Methods

Thirty 10-week-old male Wistar rats were used for this study. The animals were purchased from the common central animal house, University of Ibadan. They were housed in well-ventilated cages and were fed on standard rat pellets obtained from Capsfeed Ltd. They were allowed free access to drinking water. The animals were randomly divided into 5 groups with six rats in each group. Four groups were induced with diabetes using alloxan produced by Kem Light Laboratories, Limited, Mumbai, Maharashtra, India. After a week of acclimatization, animals to be induced were fasted for 48-hours, and weighed. The fasting blood glucose (FBG) of all the animals was taken using venous blood samples obtained from animal tail tips, and blood glucose level was analyzed with a portable glucose analyzer (Accu Chek Glucometer and test trips). This was done to confirm the non-diabetic state of the animals. Following this, a solution of 5% alloxan diluted in distilled water was administered intraperitoneally to each rat at a dose of 120 mg/kg body weight.[14] Two days after the induction, the FBG level of the induced animals was checked, and animals with FBG levels above 200 mg/dl were considered as having severe diabetes[15] and were chosen for the study. Three of the diabetic groups were fed on feeds containing three different common dietary oils at 10% for 8 weeks, based on method of dietary induction of hypercholesterolemia and atherosclerosis in rodent models.[4] The edible oils were obtained from a local market in Ibadan. The remaining two groups which served as the diabetic and normal control, respectively, received normal rat chow diet for 8 weeks.

Animals in Group A (normal control, non-diabetic) were feed on normal basal rat chow diet.

Animals in Group B (diabetic control) were feed on normal basal rat chow diet.

Animals in Group C (diabetic) were feed on 10% refined, bleached deodorized palm oil (RBD-PO) enriched basal rat chow diet.

Animals in Group D (diabetic) were feed on 10% soya oil enriched basal rat chow diet.

Animals in Group E (diabetic) were feed on 10% olive oil enriched basal rat chow diet.

At the end of 8 weeks, 2 ml of blood was collected from the orbital sinus of each animal with capillary tubes into ethylenediaminetetraacetic acid bottles for lipid profile analysis. The atherogenic index (AI) was calculated using the formula:

AI = Non high-density lipoprotein cholesterol (non-HDL-C)/HDL-C

Thereafter, the animals were sacrificed, and their aorta was harvested for histological examination. The tunica intima (TI) and tunica media (TM) thickness were measured using computerized image analyzer (Motic Image Plus, Version 2.0).

The TI thickness was measured from the lumen to the internal elastic lamina while the TM thickness was measured from the internal elastic lamina to the external elastic lamina.

The results were expressed as mean ± standard deviation. The statistical analysis was performed by means of one-way analysis of variance followed by Dunnett's test. $P < 0.050$ was considered as statistically significant. All the data were processed with Graph Pad Prism software, version 5.00.

Results

At the end of the experimental period of 8 weeks, all animals had an increase in body weight and those in the normal control group had the highest percentage weight increase (58.33 ± 37.64, 48%). There was no statistically significant difference in the mean weight gain in all the groups, at P value of 0.0872 ($P > 0.050$). The lipid profiles of the diabetic and control groups are shown in table 1.

Table 1. Lipid profile of experimental and control animals

Group	TC	HDL-C	Non-HDL-C	TG	AI
NC	205.81 ± 96.52	117.16 ± 31.93	88.65 ± 81.25	205.81 ± 96.53	1.06 ± 0.62
DC	220.57 ± 59.06	121.15 ± 28.84	99.42 ± 67.97	226.90 ± 86.01	1.53 ± 0.66
RBD-PO	257.38 ± 57.08	142.07 ± 92.50	115.28 ± 82.99	492.90 ± 214.60*,**	1.426 ± 1.35
Soya oil	217.50 ± 23.50	67.89 ± 65.30*	150.21 ± 46.25	203.40 ± 147.07	17.82 ± 8.45
Olive oil	278.80 ± 58.75*,**	30.69 ± 1.45*,**	248.12 ± 58.83*,**	75.61 ± 27.15	30.07 ± 14.59

* Statistically significant values in comparison with the control group (P < 0.050). ** Statistically significant values in comparison with the diabetic control group (P < 0.050). TC: Total cholesterol; HDL-C: High-density lipoprotein cholesterol; TG: Triglyceride; AI: Atherogenic index; RBD-PO: Refined bleached deodorized palm oil; DC: Diabetic control; NC: Normal control

The total cholesterol (TC) at the end of the experiment was higher in the olive oil group than the RBD-PO and soya oil group. There were no significant differences in the mean plasma TC values of all animal groups at P value of 0.2215 (P > 0.050). The mean HDL-C values of soya and olive oil fed groups (67.89 ± 65.30, 30.69 ± 1.45), respectively, were significantly lower than the mean HDL-C value of the normal control group (117.16 ± 3.93), at P values < 0.050.

The mean HDL-C value of olive oil fed group (30.69 ± 1.45) was also significantly lower than the mean HDL-C value of the diabetic control group (121.15 ± 31.93), at P > 0.050. The mean TG value of RBD-PO fed group (492.9 ± 214.6) was significantly greater than the mean TG value of the normal control group (205.81 ± 96.53) and diabetic control group (226.90 ± 96.53) at P > 0.050. The mean plasma non-HDL-C value of olive oil fed group (248.12 ± 58.83) was significantly greater than the mean plasma non-HDL-C value of the diabetic control group (99.42 ± 67.97) and that of the normal control group (88.65 ± 81.25) at P > 0.050. When the AI was arranged in descending order, diabetic animals fed on 10% olive oil fortified diet had the highest AI value (30.70 ± 14.59), followed by diabetic animals fed on 10% soya oil (17.82 ± 8.45), followed by diabetic animals fed on normal diet (1.53 ± 0.66), followed by diabetic animal fed on RBD-PO (1.43 ± 1.35), and control animals fed on normal diet had the lowest AI value (1.06 ± 0.62). Statistically, the mean AI values of all the groups were significantly different at P value of 0.0001 (P < 0.050).

Table 2 shows the TI/TM ratio of the control and diabetic groups and the ratio of all the groups were not significantly different at P value of 0.1161 (P > 0.050).

Figure 1 shows the histological section of the aorta of the control male diabetic Wistar rat fed on normal chow for 8 weeks and figure 2 is that of male diabetic Wistar rat fed on 10% soya oil enriched diet for 8 weeks.

Table 2. Ratio of the aortic tunica intima (TI) and tunica media (TM)

Animal group	TI/TM ratio	P
Control	0.02883 ± 0.006795	
Diabetic control	0.02333 ± 0.005502	0.114
RBD-PO	0.02038 ± 0.019670	0.286
Soya oil	0.04725 ± 0.023470	0.267
Olive oil	0.02100 ± 0.003606	0.110

TI: Tunica intima; TM: Tunica media; RBD-PO: Refined bleached deodourised palm oil

There were no atherosclerotic lesions in all the control and experimental groups except those fed on 10% soya oil enriched diet that had type II AHA atherosclerotic lesions as shown in figure 2. The aortic endothelium had two to three layers of macrophages without lipid droplets.

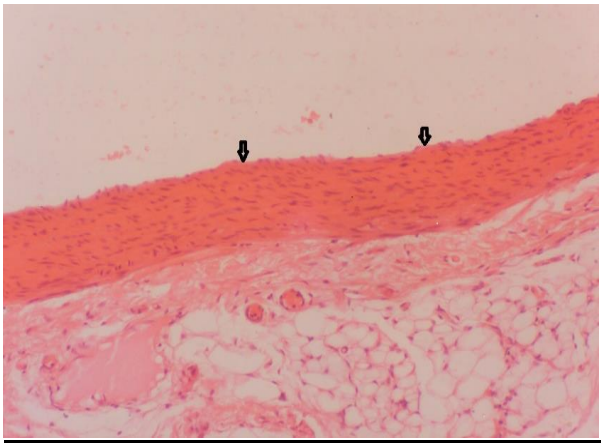

Figure 1. Aortic section of male diabetic Wistar rat fed on normal chow for 8 weeks. The arrowheads point to an intact endothelium without any interruption (stained with hematoxylin and eosin stain)

Discussion

The metabolic disorder of diabetes is characterized by accelerated atherosclerosis with widely distributed vascular lesions.[16] Dyslipidemia in form of hypercholesterolemia, high LDL-C, and low HDL-C in the blood will worsen this atherogenic

process,[17] and dietary manipulation can exacerbate or ameliorate it.

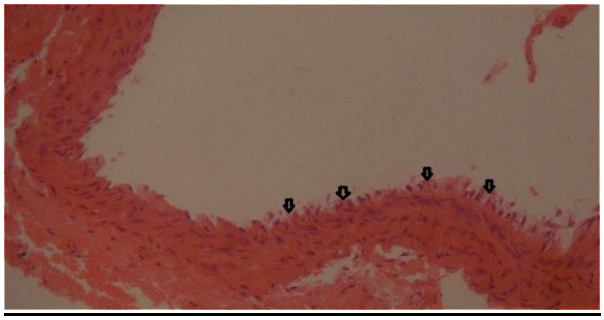

Figure 2. Aortic section of male diabetic Wistar rat fed on 10% soya oil enriched diet for 8 weeks. The arrowheads point to areas of disruption of the endothelium without lipid droplets that represents type II lesions (stained with hematoxylin and eosin stain)

Our study is the first to investigate the effects of edible oils in Nigeria in male Wistar diabetic rats. Diabetic animals fed on olive oil enriched diet had the highest TC, non HDL-C, and AI but the lowest level of TG. These findings from our study suggest that olive oil may be proatherogenic in the diabetic state. This contrasts with an earlier study that reported the antioxidant and hypolipidemic effects of olive oil in normal and male diabetic rats.[18] The non-HDL-C directly reflects the proatherogenic nature of oils. The HDL-C was also significantly low in animals fed on olive oil in our study, and this is the cholesterol that is associated with reduced risk of cardiovascular events when present at appropriately high concentration.[19] Vegetable oils, such as olive and soya oils, are recommended for consumption due to their high content of MUFA and PUFA.[20] A study comparing the effects of sunflower, fish, and virgin olive oils on the progression of experimental atherosclerosis in rabbits found that extra virgin olive oil, and to a lesser extent, fish oil, stops its progression.[21] In another study, the aorta and coronary arteries of albino rats administered olive oil showed less atheromatous lesions compared with animals fed on corn oil[22] or peanut oil.[23] The only explanation that we can give for the proatherogenic state of olive oil in our study is that it is possible that the locally available olive oil may be adulterated, and this is alarming and calls for prompt intervention by the consumer regulatory agency in Nigeria.

In our study, diabetic rats fed on soya oil had high levels of non-HDL-C, and the AI was highest in animals fed on olive oil followed by those fed on soya oil. In addition, only diabetic rats fed on soya oil had atherosclerotic lesions on their aortic endothelium though at an early stage, that is, type II atherosclerosis. Our findings are at variance with a study comparing fresh soya oil with repeatedly heated soya oil which showed that fresh soya oil offered vascular protection in the estrogen-deficient state, as rats had similar features to those of the normal control group, while animals fed on repeatedly heated soya oil had deleterious damage of their endothelium.[24] Another study comparing the effects of soymilk and probiotic soymilk on serum HDL-C and LDL-C in diabetic Wistar rats observed that probiotic soymilk increased HDL-C significantly more than soymilk.[25] The researchers recommended that probiotic soymilk may be considered in managing diabetes complications and atherosclerotic risks. Soya oil is the most widely marketed edible oil in the world, and it contains approximately 60% of PUFA, 24% MUFA, and 16% of saturated fatty acids.[8] This high level of PUFA can improve the blood lipid profile status.[9] In addition, with its high content of tocopherols, soya oil is known to exhibit various antioxidant actions against lipid peroxidation.[26]

In our study, diabetic rats fed on RBD-PO had high levels of HDL-C and low AI. Our study showed that RBD-PO has a better lipid profile and AI than olive oil and soya oil in diabetic rats. This is in line with various studies in animals[26] and humans[27-29] that showed that palm oil has the effect of decreasing TC and "bad" LDL-C and increasing the level of "good" HDL-C. However, in their study, soya oil and peanut oil had no effect on the blood cholesterol. The beneficial roles of fresh palm oil to health include reduction in the risk of arterial thrombosis and atherosclerosis, inhibition of cholesterol biosynthesis and platelet aggregation, and reduction in blood pressure.[30] This may be attributed to the tocopherol and tocotrienols antioxidant property of RBD-PO. A study that examined the effects of palm oil tocotrienol-rich fractions on streptozotocin-induced diabetic rats reported that tocotrienol-rich fraction lowers the blood glucose level and improves dyslipidemia.[31] Another study comparing hamsters fed on RBD-PO with those fed coconut oil found that the former had lower TC and non-HDL-C and higher HDL-C concentrations and accumulated less aortic cholesterol than the later.[32] We observed that the mean TG value of RBD-PO fed group was significantly greater than the mean TG value of the normal control and the diabetic control groups. The reason for the elevated level of TG is not clear but

can be attributed to the high concentration of plasma TG that accompanies uncontrolled diabetic state.

When we studied the effect of oils on the aortic sections, the TI/TM ratios of all the experimental groups were not significantly different from that of the normal control and the diabetic control group. Diabetic animals fed on RBD-PO and olive oil also had an intact endothelial lining, but the aortic endothelial lining of diabetic animal fed on soya oil showed two-three layers of macrophages without lipid droplets, which is the feature of type II atherosclerotic lesion. This may indicate that the antioxidant properties of soya oil were not able to prevent the development of atherosclerotic lesion in diabetic animal. This result is not in agreements with a previous report that soya oil offered vascular protection in the estrogen-deficient state[23] although the animals were not diabetic. Another study showed that Citrus aurantifolia lime peel is more effective than the juice can decelerate the process of atherogenesis in rabbits because it increases plasma antioxidant capacity.[32]

Conclusion

In conclusion, the result of our study showed that RBD-PO offered a better lipid profile in the diabetic animals compared with olive oil and soya oil. This is because the diabetic animals fed on RBD-PO had a lower non-HDL-C, higher HDL-C, and lower AI than diabetic animals fed on olive oil or soya oil. However, animals fed on RBD-PO had the highest TG level that is not significantly different from the diabetic control and normal control groups. We attributed this to the high level of TG that is compatible with diabetic state. The diabetic group fed on olive oil had the highest level of TC, non-HDL-C, and AI. The only explanation we have for this may be that the oil may be adulterated; however, further studies may be needed to confirm or refute this. Type II atherosclerotic lesion was seen only in the soya oil fed diabetic group.

Acknowledgments

None.

Conflict of Interests

Authors have no conflict of interests.

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How to cite this article: Oladapo OO, Ojora KA, Quadri OM, Ajani RS. **Lipidemic effects of common edible oils and risk of atherosclerosis in diabetic Wistar rats.** ARYA Atheroscler 2017; 13(1): 14-9.