

Red Palm Oil (RPO) may Enhance Chronic Inflammation through Disturbance in Lipid Metabolism

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ABSTRACT

Several studies have been published on lipid lowering effect of Red Palm Oil (RPO) with little known about health impact of differential dosage. In this study, we examined lipid profile of hyperlipidemia-induced wistar rats fed with varying quantity of RPO supplementation (15 ml/kg, 20 ml/kg and 25 ml/kg feed). A total of 30 male wistar rats were procured and randomly divided into five groups (A, B, C, D and E) with 6 rats in each group. Group C, D and E received 15 ml/kg, 20 ml/kg and 25 ml/kg of RPO respectively (thoroughly mixed with high fat diet). No force feeding or oral gavage procedures were employed. While rats in group A were fed with standard rat chow, group B animals fed on high fat diet only and neither of the two groups received RPO supplementation. Plasma concentration of Total Cholesterol (TC), Triglycerides (TG), Albumin, Low Density Lipoproteins (LDL-C), High Density Lipoproteins (HDL-C), and Total Proteins (TP) were assessed at the end of the experiment that lasted 4 weeks. In addition to the lipid lowering effect observed in RPO supplementation groups (C, D, E) compared to fatty diet fed only (group B) as widely reported in many studies, both the LDL-C and TG appeared to rise with more RPO supplementation. Findings also revealed the lipid lowering effect more pronounced on triglycerides than the low density lipoproteins. TP in group E was significantly higher compared to group A and B ($P < 0.05$) and RPO supplementation had a tendency to increase plasma TP.

Keywords: Total proteins; Low density lipoproteins; Palm oil

INTRODUCTION

The study of dietary fats and oils consumption has gained recognition during the last twenty years, and still continues to especially with emergence of dramatic expansion in areas of metabolic physiology and nutrition science, primarily to combat risks of excess fat related disorders and attendant dysmetabolism. This is because obesity, insulin resistance, and metabolic syndrome X are all linked to diets with high fat content especially the Low Density Lipoprotein (LDL). Various chemicals that make up lipids exhibit a unique characteristic of being soluble in organic solvents but insoluble in water with fatty acids as building block just as amino acids are to protein [1-3].

Following this, will it be rational to jettison all fat containing diets for fear of potential harmful effects? certainly not.

It turns out that fats are not that really bad as presented in many academic gazettes. The truth is, lack of fats in diets is as dangerous as consuming excess of it. Because of this, they are increasingly recognized for their importance and regulatory roles in numerous physiological functions. Lipids and fatty acids act as components of membranes, energy supply and fuel storage, although certain lipids serve other specific purposes. For instance, some act as cell signaling and very low concentrations could cause disturbance in homeostasis resulting in fatal disease conditions like Schizophrenia, Alzheimer's disease, Cardiovascular diseases, Glomerulonephritis etc [4,5].

One most important type of lipid that is tightly controlled is the cholesterol. Essentially, and except biological derangement subsists in *de novo* synthesis, only exogenous cholesterol has proven terribly challenging for the body regulatory mechanisms and learning to regulate daily intake is now a necessity. A

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number of methods have been identified as interventions, with diet, drugs and exercise of various types and intensities being most favored. However, and for the purpose of this research, we investigated effect of dietary RPO supplementation on lipid profile in hyperlipidemia-induced model of wistar rats.

Excerpts from people's feeding habits reveal attitude as major challenge in related diet controlled experiments in human subjects. Unlike animals, human beings are hard to put and maintain under experimental conditions. Many would not like to forsake preferred diets regardless of disease burden that might ensue. Even when recruited for experiments of this nature, they hardly abide to prescribed nutrition regimen or daily calorie modification. This may be one of the reasons many studies continue to report conflicting findings with so much discrepancy that make it practically impossible to appreciate the true impacts of varieties of diet interventions. To control for this, we resorted to animal study and investigated one of the most widely consumed cooking oil 'red palm oil' having reviewed substantial literatures on its lipid lowering potential [6].

RPO is extracted from a tropical plant *Elaeis guineensis* (commonly known as the oil palm) which produces second largest volume of vegetable oil in the world. Overall biochemical

effects of RPO's most important group constituents which are fatty acids and antioxidants like carotenoids, tocopherol and tocotrienols have been well debated with a view to harness possible therapeutic values in treatment of obesity and hyperlipidemia conditions. Notwithstanding, presence of high percentage of saturated fatty acids (up to 51%) has been cited against its lipid lowering potential and reduction in Cardiovascular Risk Factors (CVR) in obese patients. Hence, we hypothesize that palm oil does not affect lipid profile in diet-induced hyperlipidemia in wistar rat's model [7].

MATERIALS AND METHODS

Thirty male wistar rats were procured from the anatomy department, faculty of basic medical sciences, Ladoko Akintola university of technology, Ogbomoso, Oyo state Nigeria. Rats were weighed and initial weights recorded. After two weeks of acclimatization period, they were further randomly divided and assigned into five groups with six rats in each group. Animals were housed in plastic cages specially designed for this experiment with roof made of net for adequate ventilation. They were daily fed to satisfaction and had free access to water and food throughout four weeks the experiment lasted (Table 1).

Table 1: Rats were weighed and initial weights recorded.

Groups	Treatment (Water ad libitum for all groups)
A	Standard rat chow for 4 weeks (negative control).
B	Fatty diet only for 4 weeks (positive control).
C	Fatty diet+RPO (15 ml/kg feed) for 4 weeks.
D	Fatty diet+RPO (20 ml/kg feed) for 4 weeks.
E	Fatty diet+RPO (25 ml/kg feed) for 4 weeks.

Rats in groups B, C, D and E were fed high fat diet to induce hyperlipidemia while those in group A were fed standard rat chow. In addition, group C, D, and E were fed with fatty diets supplemented with locally refined RPO purchased at Sabo

market, Ogbomoso, Oyo State. The RPO was thoroughly mixed with experimental diets to avoid texture concern. No force feeding or oral gavage [8,9]. Body weight of the animals were recorded on weekly basis until end of the experiment (Table 2).

Table 2: Nutritional content of the high fat experimental diet.

Nutritional constituents	%
Protein	15
Carbohydrate	15
Fat	70
Formula	Unit (gm/kg)
Casein	130
DL-Methionine 3	20

Corn starch	200
Sucrose	90
Vegetable shortening	364
Corn oil	16
Cellulose	150
Vitamin mix	10
Mineral mix	25
Calcium carbonate	5
Total	1000

Blood collection and storage

Blood samples were collected from the heart by terminal bleeding. Venous blood was withdrawn from each rat at the end of the study and collected into sample bottles containing heparin anticoagulant. Samples were centrifuged and analyzed at the Bioethics Laboratory, Institute for Advanced Medical Research and Training (IMRAT), University College Hospital (UCH) Ibadan, Oyo State, Nigeria [10].

Plasma preparation

Blood collected into heparin sample bottles were gently mixed and centrifuged at 4000 g for 10 mins and supernatants collected into plain bottles for analysis.

Estimation of plasma triglycerides

Plasma triglycerides was determined using GPO-PAP colorimetric method according to Jacobs, et al.

Estimation of plasma total cholesterol

Total cholesterol was estimated using the enzymatic endpoint method according to Allain, et al.

Estimation of plasma HDL and LDL

Estimation of plasma HDL and LDL was done using CHOD-PAP method according to Bairaktari, et al. In summary, low density lipoproteins (LDL and VLDL) and chylomicron fraction

were precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium ions. After centrifugation, the cholesterol concentration in the HDL fraction, which remains in the supernatant, was determined using Friedewald formula [11-13].

Estimation of Plasma Total Protein (TP)

Total protein was estimated using Biuret Method (colorimetric end point) according to Lubran.

Estimation of plasma albumin

Plasma albumin was determined by the method of Doumas, et al. Briefly, measurement was based on its quantitative binding to the indicator 3, 3', 5, 5'- tetrabromocresol sulphonephthalein (bromocresol green, BCG). The albumin-BCG-complex absorbs maximally at 578 nm, the absorbance being directly proportional to the concentration of albumin in the sample (Figures 1-8).

RESULTS

Statistical analysis

SPSS package was used for all statistical analyses. Data were expressed as mean \pm Standard Error of Mean (SEM). The statistical analysis was carried out using (ANOVA) and Schiff's post hoc and level of significance was accepted at ($p < 0.05$) (Tables 3-5).

Table 3: Plasma lipid profile and protein level at different doses of RPO supplementation.

Expt. Group	TC (mmol/L)	TP (g/dL)	TG (mmol/L)	Albumin (g/dL)	HDL-C (mmol/dL)	LDL-C (mmol/dL)
A	2.31 \pm 0.10	54.00 \pm 2.59	0.41 \pm 0.16	26.17 \pm 1.82	1.42 \pm 0.07	0.44 \pm 0.06
B	3.14 \pm 0.24*	55.00 \pm 2.42	0.96 \pm 0.18*	27.00 \pm 2.57	1.24 \pm 0.15	1.39 \pm 0.13*

C	2.42 ± 0.16 [#]	53.00 ± 3.31	0.64 ± 0.11	27.00 ± 2.44	1.33 ± 0.16	0.79 ± 0.67 ^{*#}
D	2.59 ± 0.13	65.00 ± 1.87 ^{*#§}	0.69 ± 0.09	30.83 ± 1.49	1.43 ± 0.10	0.83 ± 0.05 ^{*#}
E	2.86 ± 0.18 [*]	65.00 ± 1.77 ^{*#§}	0.73 ± 0.14	32.67 ± 1.43 [*]	1.15 ± 0.21	1.30 ± 0.12 ^{*#§%}

(^{*}) indicates significant difference compared with group A at p < 0.05, ([#]) indicates significant difference compared with group B at p < 0.05, ([§]) indicates significant difference, compared with group C at p<0.05, ([%]) indicates significant difference compared with group C at p<0.05.

All values are expressed as Mean ± Sem (n=6)

Table 4: Weight gain between group A-E showing effect of the high fat diet and RPO supplementation.

Expt group	Initial weight (g)	Final weight (g)	Weight gain (g)
A	47.33 ± 0.62	90.33 ± 1.82	43 ± 1.92
B	47.33 ± 0.62	164.50 ± 6.24	117.17 ± 6.43
C	61.83 ± 0.79	124.83 ± 3.43	63 ± 3.52
D	66.83 ± 0.70	123.83 ± 3.88	57±3.94
E	65.50 ± 1.23	132.67 ± 2.38	67.17 ± 2.68

Table 5: Weight gain comparison between experimental groups.

Expt. group	Weight gain (g)
A	43 ± 1.92
B	117.17 ± 6.43 [*]
C	63 ± 3.52 ^{*#}
D	57±3.94 ^{*#}
E	67.17 ± 2.68 ^{*#}

(^{*}) indicates significant difference compared with group A at p<0.05, ([#]) indicates significant difference compared with group B at p<0.05, ([§]) indicates significant difference compared with group C at p<0.05, ([%]) indicates significant difference compared with group C at p<0.05

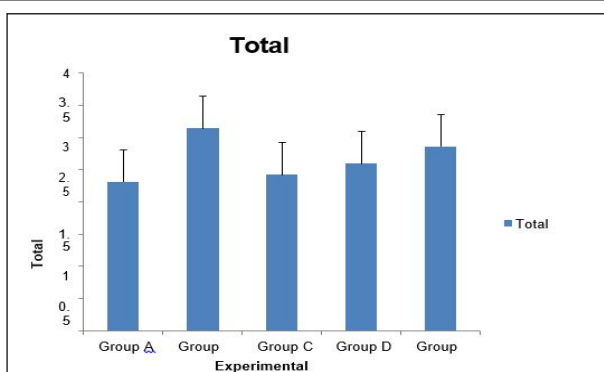


Figure 1: Graph showing comparison of mean plasma TC among experimental groups.

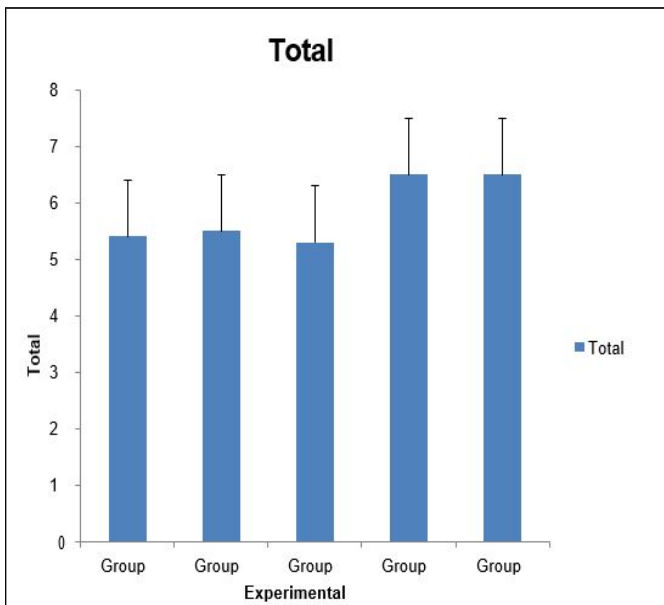


Figure 2: Graph showing comparison of mean plasma TP among experimental groups.

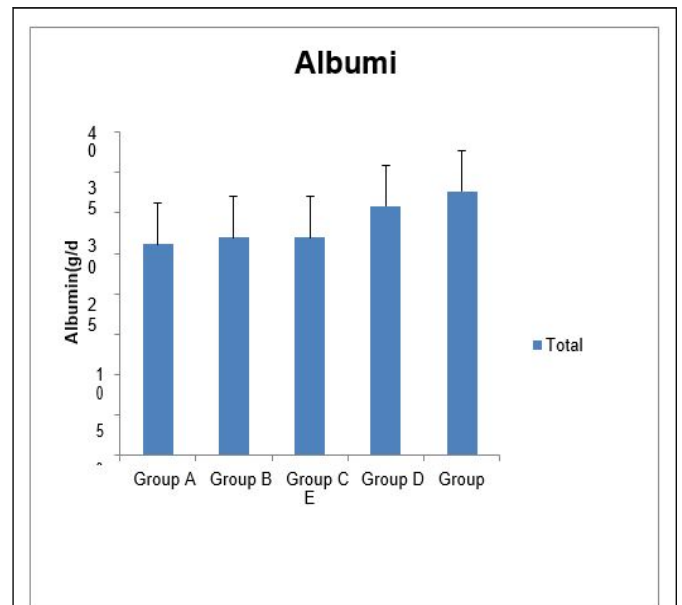


Figure 4: Graph showing comparison of mean plasma albumin among experimental groups.

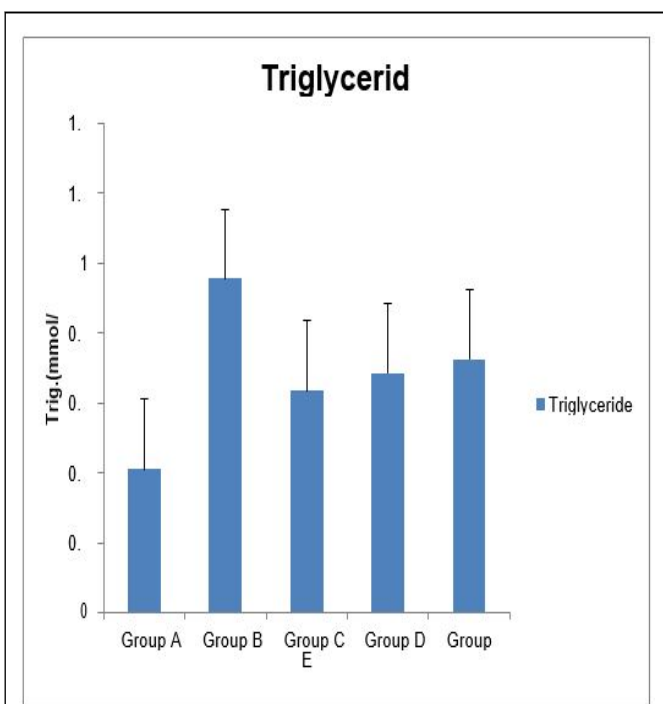


Figure 3: Graph showing comparison of mean plasma TG among experimental groups.

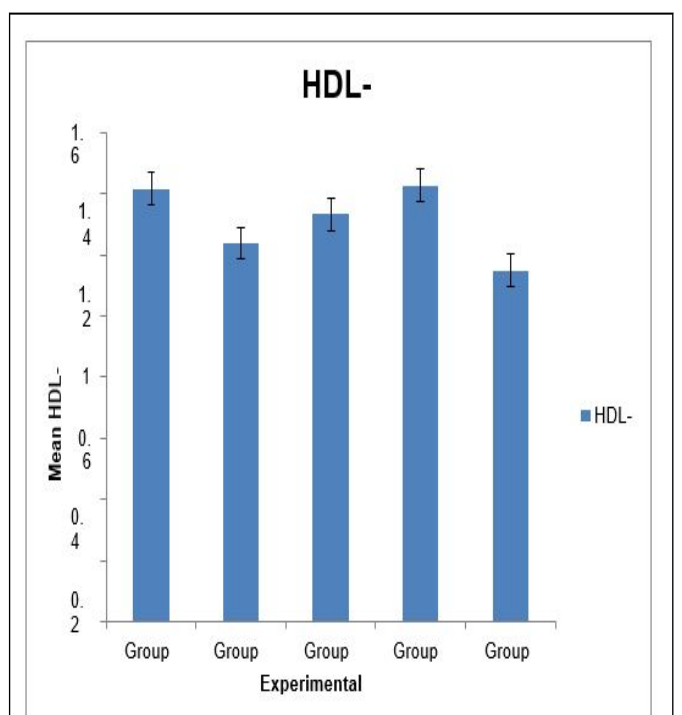


Figure 5: Graph showing comparison of mean plasma HDL-C among experimental groups.

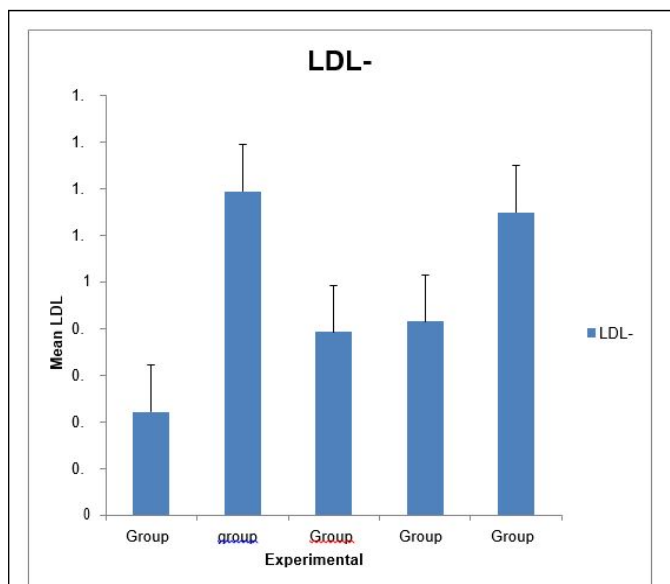


Figure 6: Graph showing comparison of mean plasma LDL-C among experimental groups.

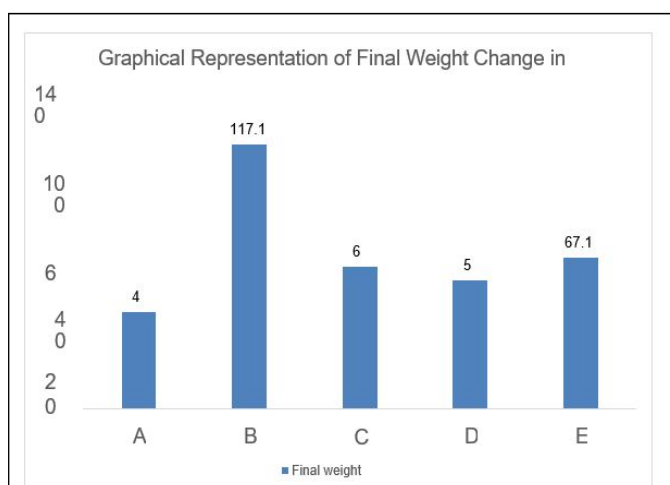


Figure 7: Graph showing comparison of mean weight gain among experimental groups.

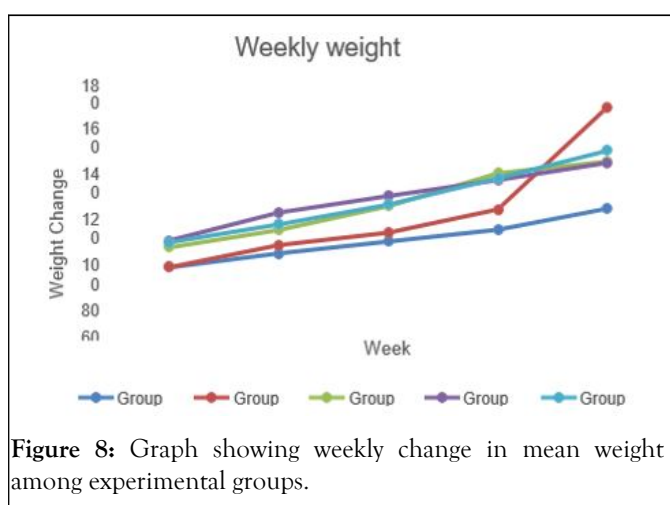


Figure 8: Graph showing weekly change in mean weight among experimental groups.

DISCUSSION

In this study, we found gradual but concomitant increase in plasma TG and LDL-C as RPO supplementation increases. The mechanism by which RPO aids lipid accumulation remains unresolved among authors investigating health impacts. But diets rich in Saturated Fatty Acids (SFA) like RPO are deemed more obesogenic compared to ones with less saturated or Polyunsaturated Fatty Acids (PUFA) and higher serum lipid with RPO was reported by Choi, et al. Although, long-term study shows plasma level remains fairly constant. Significantly higher weight gain observed in fatty diet fed group without RPO fortification compared to groups with RPO supplementation confirms lipid lowering effect of RPO as reported by Oguntibeju, et al. albeit believed to primarily center on prevention of rise in TG and LDL-C level. But tendency of RPO to boost lipid profile at a later time in a dose-dependent manner (as noted in the present study) has received less attention and remains a major public health concern [15,16].

Interestingly, sustained high plasma LDL-C and TG triggers cardiovascular disease risks, having been implicated in a wide range of dysmetabolism as evidenced in several studies. Thus, we are not surprised that plasma albumin follows similar trend as a major transporter for the abundant Free Fatty Acids (FFA). Less soluble in aqueous solution of the body fluid, short to medium chain fatty acids are regularly transported by albumin whose concentration indirectly determines plasma lipid availability.

In normal condition of homeostasis, the highly reactive cholesterol is shielded from longtime interaction with body tissues. Intracellular lipid droplets, *de novo* synthesis, uptake by circulating lipoproteins, storage and extracellular excretion are held under tight control by providing safe passage through esterification. The body employs the esterification process to store and transport cholesterol (both dietary and synthetic) effectively while as well protecting cells against toxicity of excess (otherwise free or unesterified) cholesterol. Although, lipoproteins in this process, are capable of absorbing minute free cholesterol restricted to their outer surface. More cholesterol can, however, be packed inside of them (safe corridor) as inert cholesteryl esters which significantly expands lipoproteins capacity and makes it possible for cholesterol to easily transverse the bloodstream. But this mechanism is most effective for lipid regulation during homeostasis and in conjunction with the HDL-C [17-20].

The HDL-C is functionally antithetical to LDL-C by acting to continuously adjust and restore the critical biological equilibrium making its plasma concentration an important health determining factor. Unfortunately, its restoration effect is often attenuated when overwhelmed by external factors that favor formation of more LDL-C than it is able to offload as apparent in the present study. This further proves RPO may lack the high-end therapeutic values attributed to it in many studies as HDL-C did not improve with further supplementation.

During and prior to metabolic derangement, however, LDL-C (tagged bad cholesterol) is aggressively mobilized to tissues from areas like the digestive system and liver even when they seem warning of it. The Very Low Density Lipoprotein (VLDL) is, in a

similar manner, notorious for enriching tissues with both LDL-C and TG. It primarily effects this by packing TG and Cholesteryl Esters (CEs) together then later shed the former after undergoing steady transformational remodeling. The creeping pile-up in plasma TG and LDL-C observed in this study with RPO increment suggests high plasma VLDL and possible malicious health burden (such as intramuscular lipid infiltration) capable of progressing into full blown age-dependent sarcopenic obesity characterized by impaired muscle power, strength and performance. And by implications, since abundance of and feedback from lipids and metabolites upregulate Pyruvate Dehydrogenase Kinase-4 (PDK₄) and up tissue dependence on them for energy and utilization, elevated plasma lipid has been described as one material precursor for oxidative stress and type 2 diabetes as less glucose are metabolized leading to insulin resistance, cardiovascular disease and arrays of other metabolic dysfunctions that follow the initial subclinical inflammation.

In addition to albumin that helps transport Free Fatty Acids (FFA), observed tendency of TP to rise with dietary RPO signals low-grade but progressive and potentially damaging oxidative stress in the experimental animals, presumptively through the Inflammation Sensitive Proteins (ISP) like fibrinogen, orosomucoid, alpha-1 antitrypsin, heptoglobin, and ceruloplasmin associated with future weight gain. In the same vein, the ISP may be responsible for the rise in cellular oxidative stress and inflammation reported on repeatedly heated RPO. Along with cholesterol, they have also been linked to myocardial infarction, cardiovascular death, and stroke suggesting that chronic inflammation may be associated with rise in plasma lipids.

Overall, we found high quality evidence that RPO may initiate, aid and augment subclinical inflammation, hyperlipidemia, atherosclerosis, cardiovascular disease risks, type 2 diabetes and stroke in wistar rat model and advise that caution be applied in dietary consumption and daily intake.

CONCLUSION

RPO processing takes place in stages which include threshing, (removal of fruit from bunches), sterilization of bunches, digestion of the fruits, pressing (oil extraction), and finally is clarification and drying. Unfortunately, many local RPO processing facilities as common in Nigeria lack financial capacity to acquire technology that can preserve and protect the unsaponified contents (tocopherols, tocotrienols, carotenes, phytosterols etc.) often praised for lipid lowering effect. Hence, the tendency to raise cellular oxidative stress, blood pressure, TC and cause atherosclerosis through excessive heating and overriding effect of the SFA contents. Even though we did not measure plasma antioxidants in this research, we acknowledge its lipid lowering effect and possible agent for differential scientific reportage about lipid lowering potential of RPO.

Furthermore, unprocessed fruit bunches can be kept for days if not weeks in the local processing factories and RPO can be hoarded for months by local marketers to be sold later at expensive price. These factors, among others that we do not

have control over, are capable of compromising RPO quality by increasing the level of free Fatty Acids (FFA) and peroxides through the activities of microbial lipase. But for lack of quality testing and control in Nigeria, no one has kept an eye on FFA content of publicly sold RPO talk more of rancidity status. Although, RPO source employed in this experiment was adjudged to be of highest nutritional quality in the Southwestern Nigeria. We are aware that local processors often change approach based on economic reality especially when no one watches over them. By the way, the wear and tear of the rusty cooking irons used in the heating process coupled with the locally fabricated milling machines can adulterate final products by raising heavy metal contamination known to compromise the body's antioxidant defense system.

For this, results from RPO studies may vary significantly based on many factors including processing culture, region, technological sophistication, and waste disposal strategy. We stress on importance of differential technological advances on RPO quality which authors have ignored. We propose that future research should consider Malaysian RPO as gold standard against locally refined products in other parts of the world. This will not only help unravel the growing inconsistent findings regarding its therapeutic benefits but also signals policy makers on the product quality consumed by the multitudes and thus assist in strengthening public health systems. The RPO adopted in the study of Ayeleso, et al. that reported decreased lipid profile with increased RPO supplementation differs in source and most probably in nutritional composition from the one employed in the present experiment. Generally, we believe the more advanced refining technology is, the better and healthier is the RPO quality.

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