Journal of Medicinal Plants Studies
www.PlantsJournal.com

ISSN (E): 2320-3862
ISSN (P): 2394-0530
https://www.plantsjournal.com
JMPS 2024; 12(1): 214-220

Received: 15-11-2023
Accepted: 23-12-2023

Idongesit Bassey Umoh
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, University of Calabar, Calabar, Cross River State, Nigeria

Imoh Emmanuel Ukoh
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Federal University Otuoke, Otuoke, Bayelsa State, Nigeria

Favour Nyoh Beshel
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, University of Calabar, Calabar, Cross River State, Nigeria

Enene Esu Ukpai
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Cross River State University, Ogoja, Cross River State, Nigeria

Samuel Kelechi Mobisson
Department of Human Physiology, Faculty of Basic Medical Sciences, Madonna University, Elele, Rivers State, Nigeria

Jeremiah Etim Antai
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, University of Calabar, Calabar, Cross River State, Nigeria

Perowei Azosibe
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Federal University Otuoke, Otuoke, Bayelsa State, Nigeria

Azibaobom Karibi
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Federal University Otuoke, Otuoke, Bayelsa State, Nigeria

Ukrakpo Oghenemega Sophia
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Federal University Otuoke, Otuoke, Bayelsa State, Nigeria

Adienbo Emmemodi Nwadighi
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Federal University Otuoke, Otuoke, Bayelsa State, Nigeria

Corresponding Author:
Imoh Emmanuel Ukoh
Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Federal University Otuoke, Otuoke, Bayelsa State, Nigeria

Comparative effects of fresh palm oil and vitamin E on lipid profile in thermo-oxidized palm oil fed albino rat

Idongesit Bassey Umoh, Imoh Emmanuel Ukoh, Favour Nyoh Beshel, Enene Esu Ukpai, Samuel Kelechi Mobisson, Jeremiah Etim Antai, Perowei Azosibe, Azibaobom Karibi, Ukrakpo Oghenemega Sophia and Adienbo Emmemodi Nwadighi

DOI: https://doi.org/10.22271/plants.2024.v12.i1c.1643

Abstract
The human population consuming thermally-oxidized palm oil (TPO) is on the increase and there is no known remedy to it associated diseases. This study evaluates the impacts of fresh palm oil and vitamin E on lipid profiles of rat fed TPO. Sixty albino male rats were grouped into 6 (n=10). Group 2, 3, 4, 5 and 6 were fed TPO, fresh palm oil, vitamin E, TPO + fresh palm oil and TPO + vitamin E respectively. Group 1 acted as the control. 15 g of the oils was added to 85 g rat food to prepare respective diet and vitamin E 200 mg/kg/day orally. After treatment, serum analysis revealed that TPO significantly (p<0.05) alter lipid profiles negatively compared to control. But fresh palm oil and vitamin E significantly (p<0.05) reverses lipid profiles of TPO fed rats. Fresh palm oil and vitamin E may boost lipid indices and lessen the chance of cardiovascular harms.

Keywords: Thermally oxidized palm oil, vitamin E, fresh palm oil, cholesterol, cardiovascular health

Introduction
The most edible vegetable oil in Nigeria is called palm oil, and it comes from the *Elaeis guineensis* fruit. It is eaten raw or after varying degrees of thermal oxidation [1].
When the fresh form is heated to high temperatures and various times, it undergoes thermal oxidation. To make palm oil more palatable, it is typically thermally oxidized [2]. Palm oil in its fresh form contains 50% saturated, 10% polysaturated and 40% unsaturated fatty acids. Triglycerides and small amounts of di- and mono- glycerides forms it major components while phytonutrients and free fatty acids are the minor ingredients [3]. The major phytonutrients include carotenoids, vitamin C and vitamin E (Vit. E). The apparent reddish-black brightness of palm oil is attributed to beta-carotene [4]. These components vitamins act as biological antioxidants network which converts highly reactive radicals to less active species and so help to protect tissues against oxidative damage [3]. Some of the reported benefits of fresh palm oil (FPO) include lowering cholesterol and reducing inflammation while reducing stress [4], inhibition of cholesterol biosynthesis and platelets aggregation, improve immunity, possesses anti-tumorigenic effects, reduction in total cholesterol/HDL levels [4]. FPO was reported to prevents and decreases the risk of an ischemic stroke by reversing the accumulation of plaque in the brain's blood vessels [4]. The antioxidants in FPO have also been demonstrated to lessen peripheral blood flow resistance, dissolve vascular plaques, enhance heart health, and lower the risk of heart attacks and hypertension [4]. Many reports have documented the positive effects of vitamin E under a variety of circumstances. Its antioxidant potential accounts for its effectiveness [5]. According to reports [5, 6], vitamin E stops the synthesis of lipid peroxides, which are hazardous byproducts of numerous metabolic processes in biological membranes.
However, thermal oxidation of palm oil has a deteriorative effect on dietary oils because it changes the physicochemical properties of the oil thereby destroying many of its beneficial components [3], causing the production of harmful and cytotoxic byproducts that harm organs, tissues, and cells [2]. In an earlier study, It was stated that rats fed palm oil diet that has been thermally oxidized (TPO) negatively alters hematological indices [1].

Other studies reported that TPO increases free fatty acid content and density [3], destroys β-carotenes and other phytonutrients and antioxidants in the oil, making it susceptible to peroxidation [7]. It was also reported that TPO deactivate key metabolic enzymes, cause fatty livers and alters liver function and histology [8]. Despite these observations, much of palm oil is still being consumed in the thermo-oxidized form for economic reasons and partly because it is said to improve the taste of food. The human population consuming TPO is on the increase and there is no known remedy to it associated diseases. The function of serum lipids is significant in pathogenesis of many diseases [9]. Among the most common metabolic diseases that affect people is lipoprotein disorder. Understanding the levels of cholesterol sub-fractions is more significant than just knowing the total cholesterol level [10]. Therefore, the current study was started to evaluate the protective impact of FPO and Vit. E against TPO induced changes on lipid profile in albino wistar rat.

Materials and Methods

Experimental animals

Sixty albino males Wistar rats weighing 140-160 grams were used in this study after receiving ethical clearance from the University of Calabar's Faculty of Basic Medical Sciences Ethics Committee. The animals were allowed to acclimate for one week before being randomly split into six groups of ten rats each. As the control group, Group 1 was given regular rat chow and water, while Group 2 received water and fed FPO diet. Group 3 received water and fed TPO diet. Group 4 were fed normal rat chow and water in addition to oral administration of Vit. E. Group 5 were fed TPO diet in addition to FPO diet (TPO + FPO) and Group 6, were fed TPO diet in addition to oral administration of Vit. E (TPO + Vit E). This feeding and treatment lasted for 4 weeks for Group 1-4, while TPO fed rats in Group 5-6 were fed for another 4 weeks with FPO and Vit. E respectively. The Vit. E supplement was administered 200 mg/kg/day by oral gavage.

Preparation of palm oil diets

Twenty liters of FPO that came from *Elaeis guineensis* from the palm tree was bought from Marian market, in Calabar, Cross River State, Nigeria. The palm oil was shared into two black ten liters container to avoid oxidation; one container was FPO, the other container was thermally oxidized to yield TPO. Thermo-oxidation of oil was done following Beshel *et al*., [11] method, which involved heating FPO at 150 °C for 20 minutes at an interval of five and a cooling period of 5 hours prior to commencing the next round of heating in a stainless-steel pot. According to Obembe *et al.*, [12] the FPO and TPO diets were made by combining 15 g of each oil with 85 g of rat feed.

Animal sacrifice and collection of blood

Following an overnight fast, the animals were rendered unconscious at the conclusion of the feeding period using 60 mg kg^{-1} of ketamine-hydrochloride (#50155, Rotex Medica, Trittau, Germany). In order to estimate the various biochemical parameters, blood samples were obtained by cardiac puncture and stored in heparinized screw cap bottles.

Measurement of lipid profiles

Using diagnostic kits from RANDOX Laboratories Ltd. (Crumlin, Co. Antrim, UK), the serum was used for the analyses of lipid profiles, which include total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL-C), low density lipoprotein (LDL-C), and very low-density lipoprotein (VLDL-C).

Statistical Analysis

The data are analyzed using one way analysis of variance (ANOVA) and presented as mean ± SEM. A post hoc multiple comparison test is then performed. Significant values were defined as $p<0.05$. SPSS 16.0 and Microsoft Excel 2010 were used for the statistical analysis.

Results

Total cholesterol concentration comparison among groups

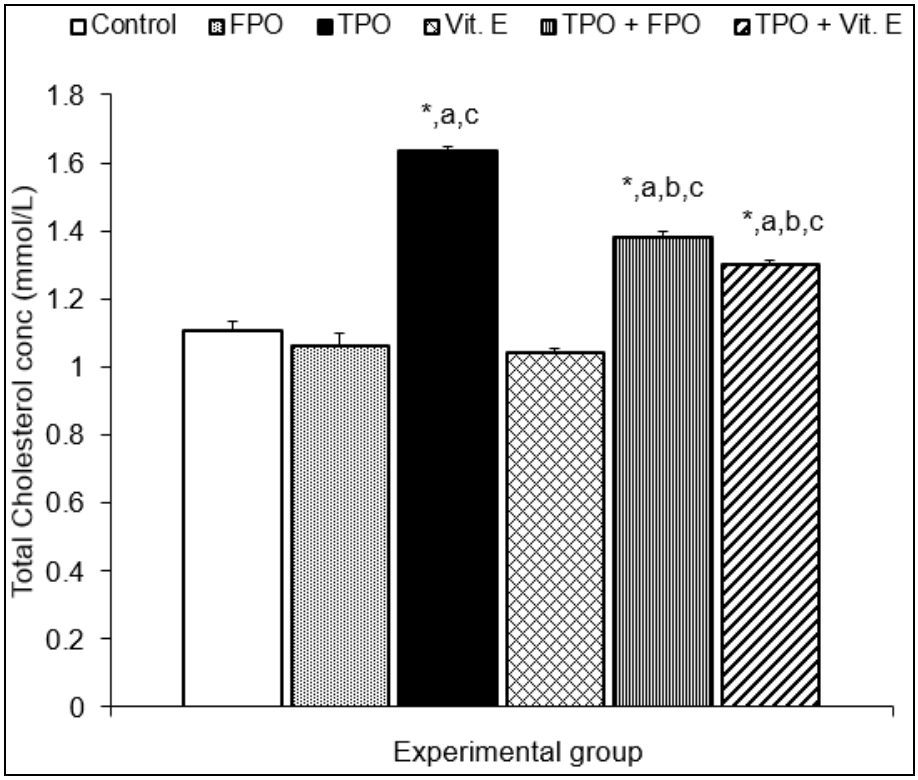
The concentration of total cholesterol (TC) in control, FPO, TPO, Vit. E, TPO + FPO as well as in TPO + Vit. E group was 1.11±0.03, 1.06±0.04, 1.64±0.01, 1.04±0.19, 1.38±0.02 and 1.30±0.01 (mmol/L) respectively. Concentration of TC was increased significantly ($p<0.05$) in TPO, TPO + FPO and TPO + Vit. E groups when compared to the control, but did not differ significantly in FPO and Vit. E groups when compared to the control. TC concentration was increased significantly ($p<0.05$) in TPO, TPO + FPO and TPO + Vit. E groups compared to FPO and Vit. E groups respectively. TC concentration was decreased significantly ($p<0.05$) TPO + FPO and TPO + Vit. E groups when compared to TPO (Fig. 1).

Triglyceride concentration comparison among groups

The concentration of triglyceride (TG) in control, FPO, TPO, Vit. E, TPO + FPO as well as in TPO + Vit. E group was 0.91±0.01, 0.84±0.02, 2.07±0.03, 0.88±0.01, 1.43±0.04 and 1.20±0.01 (mmol/L), respectively. The result showed a significant decreased ($p<0.05$) in FPO group compared to control, but a significant increase ($p<0.05$) in TPO, TPO + Vit. E and TPO + FPO groups compared to control, while Vit. E differs not significantly compared to control. The results for TPO, TPO + FPO and TPO + Vit. E groups showed significant increased ($p<0.05$) when compared to FPO. Vit. E, TPO + FPO and TPO + Vit. E groups showed significant decreased ($p<0.05$) compared to TPO. The TG level was significantly higher ($p<0.05$) in TPO + FPO and TPO + Vit. E groups compared to Vit. E group. The TG concentration was reduced significantly ($p<0.05$) in TPO + Vit. E group in relation to TPO + FPO group (Fig. 2).

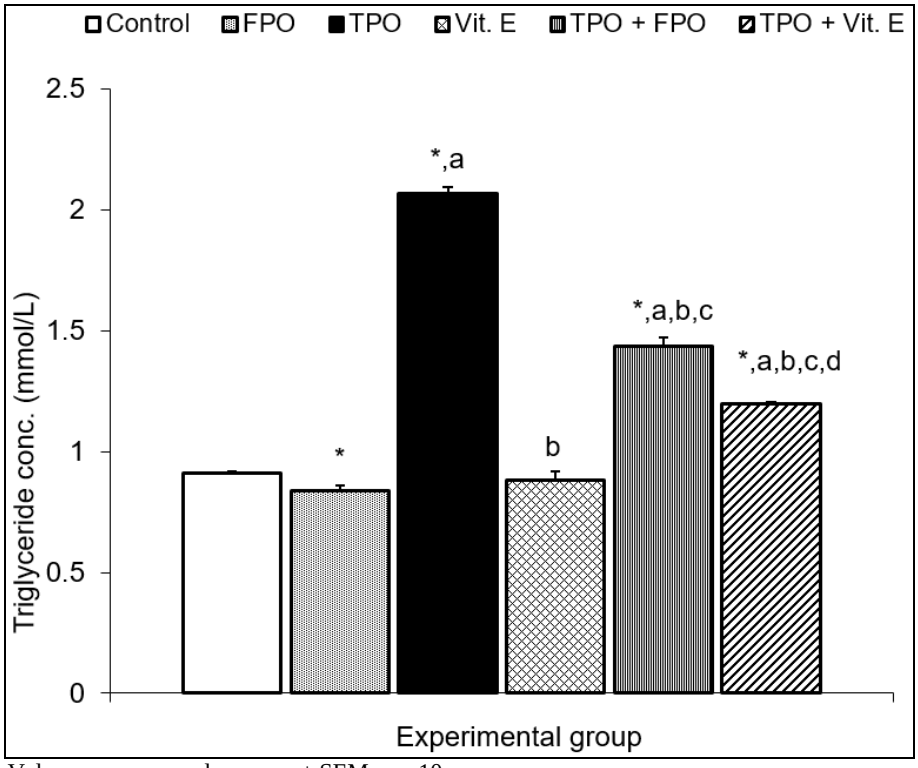
High density lipoprotein cholesterol concentration comparison among groups

The concentration of HDL-c in control, FPO, TPO, Vit. E, TPO + FPO as well as TPO + Vit. E was 0.27±0.01, 0.36±0.01, 0.12±0.01, 0.41±0.00, 0.22±0.00 and 0.25±0.00 (mmol/L), respectively. Concentration of HDL-c was significantly elevated ($p<0.05$) in FPO and Vit. E groups in relation to control, but reduced significantly ($p<0.05$) in TPO, TPO + FPO as well as in TPO + Vit. E groups in relation to control. Concentration of HDL-c was significantly lower ($p<0.05$) in TPO, TPO + FPO and TPO + Vit. E groups in relation to FPO group, but increased significantly ($p<0.05$) in Vit. E group in relation to FPO. HDL-c concentration significantly increased ($p<0.05$) in Vit. E, TPO + Vit. E and TPO + FPO group compared to TPO group. HDL-c concentration was reduced significantly ($p<0.05$) in TPO + Vit. E and TPO + FPO groups in relation to Vit. E group. The concentration of HDL-c significantly increased ($p<0.05$) in TPO + Vit. E group in relation to TPO + FPO group (Fig. 3).



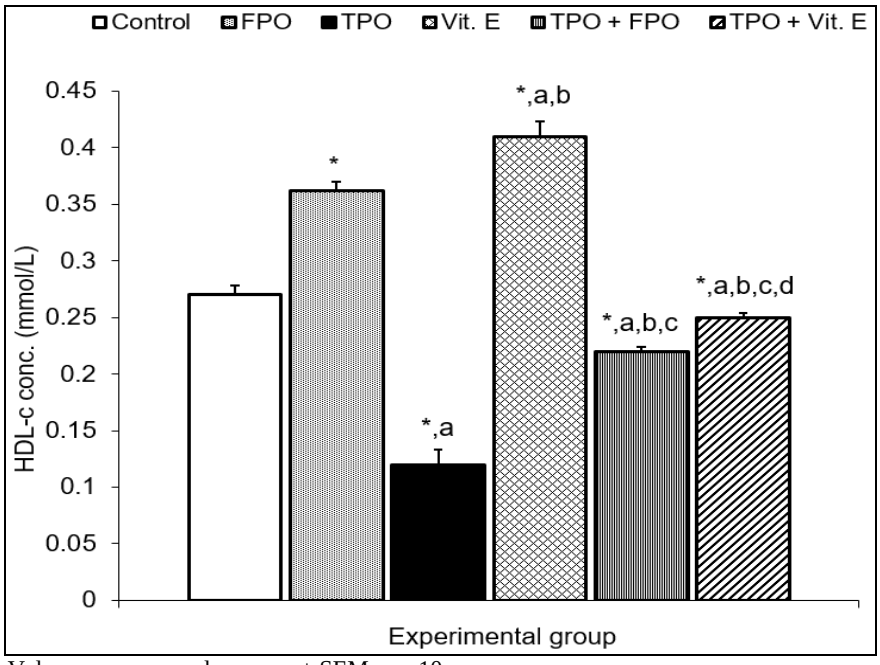
Values are expressed as mean ± SEM, n = 10
* = significantly different from control at $p<0.05$
a = significantly different from FPO at $p<0.05$
b = significantly different from TPO at $p<0.05$
c = significantly different from Vit. E at $p<0.05$

Fig 1: Total cholesterol concentration among groups



Values are expressed as mean ± SEM, n = 10
* = significantly different from control at $p<0.05$
a = significantly different from FPO at $p<0.05$
b = significantly different from TPO at $p<0.05$
c = significantly different from Vit. E at $p<0.05$
d = significantly different from FPO + TPO at $p<0.05$

Fig 2: Triglyceride concentration among groups

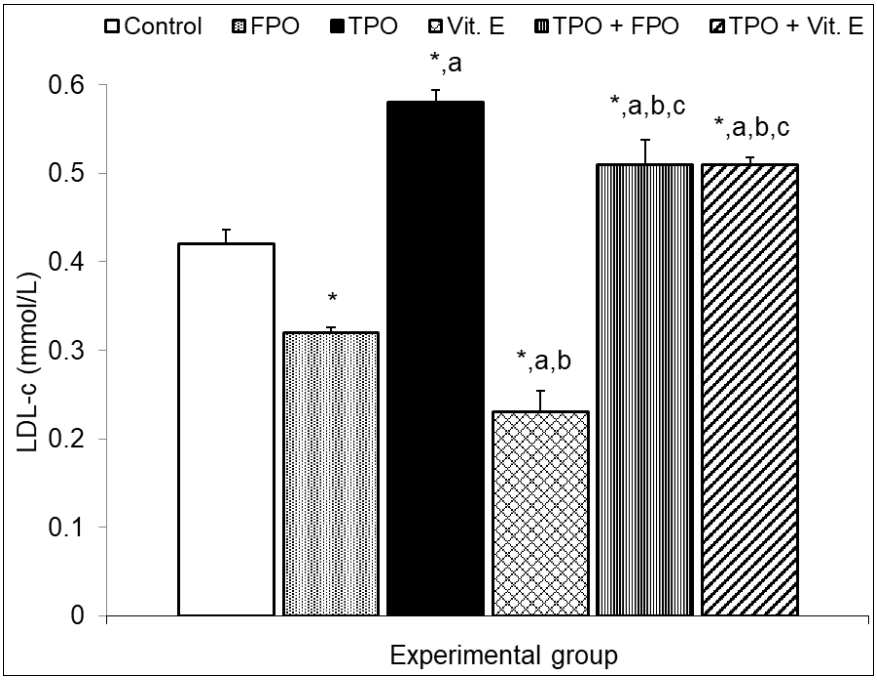


Values are expressed as mean ± SEM, n = 10
* = significantly different from control at $p<0.05$
a = significantly different from FPO at $p<0.05$
b = significantly different from TPO at $p<0.05$
c = significantly different from Vit. E at $p<0.05$
d = significantly different from FPO + TPO at $p<0.05$

Fig 3: High density lipoprotein cholesterol concentration comparison among groups

Low density lipoprotein cholesterol concentration comparison among groups

The LDL-c concentration in control, FPO, TPO, Vit. E, TPO + FPO as well as TPO + Vit. E was 0.42±0.02, 0.32±0.01, 0.58±0.01, 0.23±0.02, 0.51±0.03 and 0.52±0.01 (mmol/L), respectively. The concentration of LDL-c was significantly lower ($p<0.05$) in FPO and Vit. E groups in relation to control, and was elevated significantly ($p<0.05$) in TPO, TPO + FPO and TPO + Vit. E groups compared to control. Concentration of LDL-c was higher significantly ($p<0.05$) in TPO, TPO + FPO and TPO + Vit. E groups compared to FPO group, but significantly reduced ($p<0.05$) in Vit. E group in relation to FPO. LDL-c concentration significantly reduced ($p<0.05$) in Vit. E, TPO + FPO and TPO + Vit. E groups compared TPO group (Fig. 4).

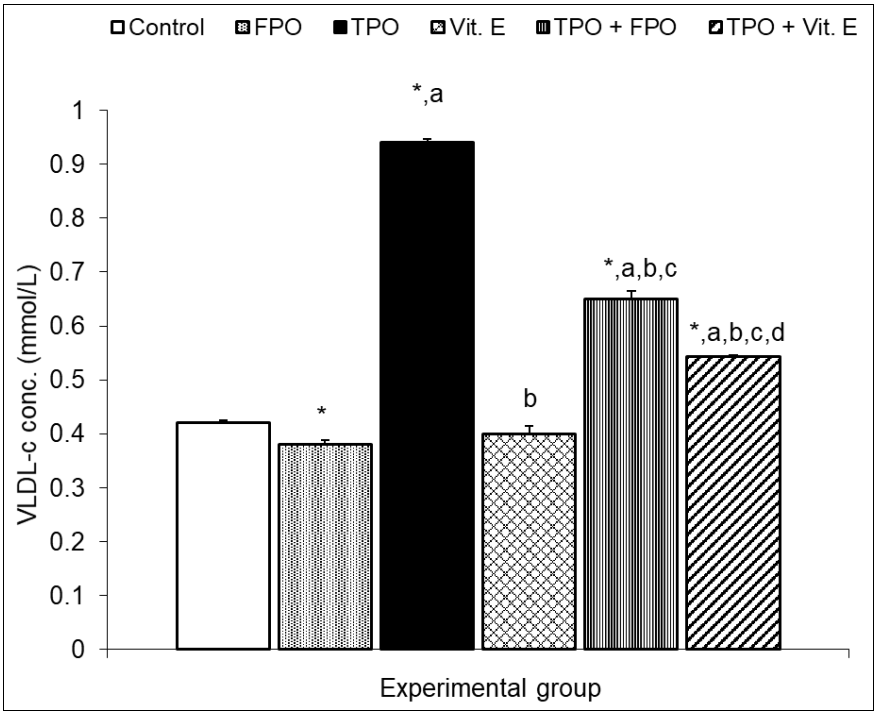


Values are expressed as mean ±SEM, n = 10
* = significantly different from control at $p<0.05$
a = significantly different from FPO at $p<0.05$
b = significantly different from TPO at $p<0.05$
c = significantly different from Vit. E at $p<0.05$

Fig 4: Low density lipoprotein cholesterol concentration comparison among groups

Very low density lipoprotein cholesterol concentration comparison among groups

The concentration of VLDL-C in control, FPO, TPO, Vit. E, TPO + FPO as well as in TPO + Vit. E was 0.42±0.01, 0.38±0.01, 0.94±0.01, 0.40±0.00, 0.65±0.02 and 0.54±0.00 in mmol/L, respectively. The level of VLDL-C significantly decreased (p<0.05) in FPO group compared to control, but significantly increased (p<0.05) in TPO, TPO + FPO and TPO + Vit. E groups in relation to control. Vit. E group did not differ significantly in relation to control. VLDL-C significantly increased (p<0.05) in TPO, TPO + FPO and TPO + Vit. E groups in relation to FPO group. VLDL-C was significantly reduced (p<0.05) in Vit. E, TPO + FPO and TPO + Vit. E groups in relation to TPO group. VLDL-C was significantly elevated (p<0.05) in TPO + FPO and TPO + Vit. E groups in relation to Vit. E. VLDL-C was significantly reduced (p<0.05) in TPO + Vit. E group in relation to TPO + FPO (Fig. 5).



Values are expressed as mean ±SEM, n = 10
* = significantly different from control at p<0.05
a = significantly different from FPO at p<0.05
b = significantly different from TPO at p<0.05
c = significantly different from Vit. E at p<0.05
d = significantly different from FPO + TPO at p<0.05

Fig 5: Very low density lipoprotein cholesterol concentration comparison among groups.

Discussion

This study looked into the protective role of FPO and Vit. E on the lipid profile of albino rats given TPO. It was earlier reported that low TC, TG, LDL-C, VLDL-C, as well as high HDL-C constitute a desirable human serum lipid composition [13]. According to another study, thermally oxidized fats significantly raised the concentrations of TC, TG, and LDL-C in the serum of the experimental animals [14]. The study's findings demonstrated that consuming FPO and Vit. E ameliorated the adverse effect of TPO fed diet in rats lipid profile. Consumption of Vit. E caused a significant improvement in recovery of lipid profile of TPO fed rats compared to FPO.

The increase TC in TPO fed rats in this study is contrary to that of Hur *et al.*, [15], who observed reduced TC level in rabbits of heated corn oil fed diet. Animal type and the oil used might be the causative factors.

Even though cholesterol is essential for the physiological control of membrane fluidity, healthy cell function, it is also a major precursor in the synthesis of bile acids, vitamin D and steroid hormones. Cholesterol has been tagged the "oily killer" [16]. Elevated cholesterol is regarded as a significant risk factor [17]. In our study, level of TC in FPO and Vit. E fed diet did not differ significantly compared to control, but were both significantly reduced compared to TPO fed diet. The decreased TC levels in FPO fed rat in this study when compared to TPO fed rat was in line with the study of Falade *et al.*, [2]. FPO group results in our study shows the protective function of FPO because of the antioxidant vitamin compositions in it.

The decreased TC levels in Vit. E fed diet compared to TPO fed diet is in line with the report of Alam *et al.*, [14], who reported that Vit. E reduces TC level in rabbits and mice. The treatment of TPO fed rat with FPO and Vit. E showed a marked decrease in TC concentration when compared to TPO.

The increase TG levels in TPO fed rats in this study is consistent with that of Rueda-Clausen *et al.*, [16] who observed that eating deep-fried palm oil raised humans serum TG levels. In our study, FPO fed diet decrease TG levels compared to control and TPO fed group. This could be due to the rich antioxidant properties of FPO because Alam *et al.*, [14] research revealed that chicks fed diets with heated vegetable oils had considerably lower serum α-tocopherol levels.

The reduce TG level in FPO in our study is consistent with that of Alam *et al.*, [14] who showed that eating olive oil significantly lowers the amount of TG in rat liver.

The decrease in FPO group compared to TPO group in this study disagrees with the study of Falade *et al.*, [2] which reported an increase in FPO compared to all TPO fed diets at varying time of heating. These contradictory results could be

due to the nature of the FPO and the oil preparation process. In this study, TG level in Vit. E fed group did not differ significantly compared to control but was decreased compared to TPO fed group. The treatment of TPO fed rat with FPO and Vit. E showed a marked decrease in TG concentration when compared to TPO. As observe in this study TPO fed rat treated with Vit. E showed a marked decrease in TG level compared to TPO fed rat treated with FPO. It is expected that FPO which is reportedly rich in Vit. E in addition to its other vital constituents should have been more effective than Vit. E alone in ameliorating the adverse effect of TPO fed diet, but *Ukoh et al.,* [1] reported that most FPO consumed generally must have undergone some level of photo-and-chemical oxidation, a process known to have adverse effect on the oil quality.

However, Alam *et al.,* [14] also noted that tocopherol had a positive impact by bringing serum lipid profile values back to normal when taken either by itself or in conjunction with oxidized olive oils.

The decrease in HDL-C in the TPO fed group in this study suggests that TPO diets reduced HDL-C which is the "good cholesterol". This result is consistent with that of Adam *et al.,* [18], who noted in all heated oil-fed groups a declining trend in serum HDL-C. An essential scavenger of excess cholesterol, HDL-C moves the cholesterol from the cell membrane to the liver, where it is broken down or transformed into bile acids [13]. In our study, FPO fed diet increase HDL-C levels in relation to control and TPO fed group respectively. The increase in FPO is in line with the report of Tan *et al.,* [19]. The increase in FPO group in relation to TPO in this study is in line with the study of Falade *et al.,* [2] and Oyama *et al.,* [4]. Vit. E was observed to increase HDL-C levels compared to control, TPO and FPO fed groups. The treatment of TPO fed rat with FPO and Vit. E showed a noticeable increase in HDL-C levels in relation to TPO. As observe in this study TPO fed rat treated with Vit. E showed a marked increase in HDL-C in relation to TPO fed rat treated with FPO.

The high level of LDL-C in TPO fed group suggests that TPO diets increase LDL-C which is the "bad cholesterol." This result is consistent with that of Jaarin *et al*., [20], who noted that prolonged feeding with heated soya oil and heated once palm oil elevate serum LDL-C. Rueda-Clausen *et al*., [16] reported that exposing oil to deep frying changes its chemical composition by producing other oxidation products and saturating its fatty acids. LDL-C could deliver these oxidized lipids to tissues leading to a deleterious effect. Tan *et al*., [19] reported that palm oil possesses the serum lipid lowering properties on LDL-C. However, palm oil is heat-sensitive, heating it repeatedly weakens its antioxidant qualities.

Ajayi *et al.,* [10] reported that LDL-C increase rate at which TG catabolized by moving fat from the liver into adipose tissue. Since it carries between 60%-70% of TC in the serum, the elevated level of LDL-C in this study implies high circulatory levels of TG and TC which could exacerbate blood lipid related diseases.

In our study, FPO fed diet decrease LDL-C levels in relation to control and TPO fed groups. The decrease in FPO group in relation to TPO group in this study is in line with that of Falade *et al.,* [2]. In our study, Vit. E fed diet decreased LDL-C levels compared to control, FPO and TPO fed groups. Treatment of TPO fed rat with FPO and Vit. E showed a marked decline in LDL-C levels in relation to TPO.

The elevated VLDL-C level in the TPO fed group suggests that VLDL-C could transport these dietary oxidized lipids which are injurious to tissues [15]. In our study, FPO fed diet decrease VLDL-C levels compared to control and TPO fed groups. Vit. E fed diet decreased VLDL-C levels compared to TPO fed group. The treatment of TPO fed rat with FPO and Vit. E showed a marked decrease in VLDL-C level when compared to TPO. As observe in this study TPO fed rat treated with Vit. E showed a marked decrease in VLDL-C level compared to TPO fed rat treated with FPO. LDL-C and VLDL-C synthesis have been linked. It is determined that the decrease in LDL-C synthesis is caused by a decreased VLDL-C synthesis [13].

Conclusion

This study has demonstrated that Vit. E is more potent than FPO in reversing lipid profiles due to consumption of TPO. This may be so because FPO, unless consumed immediately after it is processed is not of the highest quality. Majority of the FPO are subjected to photo-and-chemical oxidation which reduces the potency of the oil.

Reference

1. Ukoh IE, Umoh ID, Ukpai EE, Mobisson SK, Antai JE. Comparative effects of fresh palm oil and vitamin E on some hematological indices in thermo-oxidized palm oil fed rat. World Journal of Pharmaceutical Research. 2023;12(17):15-31.
2. Falade AO, Oboh G, Ademiluyi AO, Odubanjo OV. Consumption of thermally oxidized palm oil diets alters biochemical indices in rats. Beni-Suef University Journal of Basic and Applied Science. 2015;4:150-156.
3. Ekpe OA, Elemi JA, Eme EO, Daniel UO. The effect of long-term consumption of thermo-oxidized palm oil diet on some reproductive parameters in male wistar rats. World Journal of Pharmaceutical Research. 2018;12(7):68-81.
4. Oyama SE, Seriki SA, Mfem CC. Comparative effect of fresh palm oil and thermally oxidized palm oil on cardiovascular risks. Natural Volatiles and Essential Oils. 2021;8(4):9632-9638.
5. Bisong SA, Ukoh IE, Nna VU, Ebong PE. Vitamin E attenuates nicotine- and noise-induced reproductive impairment in male albino Wistar rats. Andrologia; c2018. p. e13050.
6. Kanter M, Aksu B, Akpolat M, Tarladacalisir TY, Aktas C, Uysal H. Vitamin E protects against oxidative damage caused by cadmium in the blood of rats. European Journal of General Medicine. 2009;6(3):154-160.
7. Aribo EO, Nwangwa JN, Udefa AL, Udokang NE. Comparative effects of long-term consumption of thermo- and photo-oxidized palm oil diets on some reproductive parameters in male wistar rats. Saudi Journal of Medicine. 2018;6(3):334-341.
8. Obeten CE, Ani EJ, Ime AU, Kokelu AN, Okon UE. Thermo-oxidized palm oil diet-induced haematological derangements in rats is ameliorated by Aloe vera and Garlic. The Journal of Phytopharmacology. 2018;7(4):353-359.
9. Mawahib BGA, Azhar LJ, Hanaa SK, Mahdi MT. Effect of different types of fat on Lipid metabolism in rats. Indian Journal of Forensic Medicine and Toxicology. 2021;15(3):722-729.
10. Ajayi OB, Ajayi DD. Effect of oil seed diets on plasma lipid profile in albino rats. Pakistan Journal of Nutrition. 2009;8(2):116-119.
11. Beshel FN, Antai AB, Osim EE. Chronic consumption of palm oil diets alters some renal function parameters in

albino rats. Nigerian Journal of Physiological Sciences. 2010;25:200-202.
12. Obembe AO, Ofutet EO, Antai AB, Osim EE. Gastric ulceration: the role of thermoxidised palm oil. Nutrition and Food Science. 2016;46(1):108-119.
13. Hamid NK, Humaira F, Shakir A, Jafar SK. Serum lipid profile and retinol in rats fed micronutrient rich edible vegetable oil blend. Nusantara Bioscience. 2010;2(3):109-115.
14. Alam Z, Ayaz AK. Improvement of serum biochemical parameters and hematological indices through α-tocopherol administration in dietary oxidized olive oil induced toxicity in rats. Frontiers in Nutrition. 2019;5:137.
15. Hur SJ, Du M, Nam K, Williamson M, Ahn DU. Effect of dietary fats on blood cholesterol and lipid and the development of atherosclerosis in rabbits. Nutrition Research. 2005;25(10):925e35.
16. Rueda-Clausen CF, Silva FA, Lindarte MA, Villa-Roel C, Gomez E, Gutierrez R, *et al*. Olive, soybean and palm oils intake have a similar acute detrimental effect over the endothelial function in healthy young subjects. Nutrition, Metabolism and Cardiovascular Diseases. 2007;17(1):50e7.
17. Sun JH, Byungrok M, Ki CN, Eun JL, Dong UA. Effect of dietary cholesterol and cholesterol oxides on blood cholesterol, lipids, and the development of atherosclerosis in rabbits. International Journal of Molecular Sciences. 2013;14:12593-12606.
18. Adam SK, Soelaiman IN, Umar NA, Mokhtar N, Mohamed N, Jaarin K, *et al*. Effects of repeatedly heated palm oil on serum lipid profile, lipid peroxidation and homocysteine levels in a post-menopausal rat model. McGill Journal of Medicine. 2008;11(2):145-151.
19. Tan KX, Noor AO, Low WY, Aniza H, Santhana R, Kamsiah J, *et al*. Reheated Palm Oil Consumption and Risk of Atherosclerosis: Evidence at Ultrastructural Level. Evidence-Based Complementary and Alternative Medicine, 2012, 3(4).
20. Jaarin K, Norhayati M, Norzana G, Nor AU, Ima-Nirwana S. Effects of heated vegetable oils on serum lipids and aorta of ovariectomized rats. Pakistan Journal of Nutrition. 2006;5(1):19e29.