Heliyon

ELSEVIER

Received:
10 April 2015
Revised:
1 June 2015
Accepted:
30 June 2015

Heliyon (2015) e00010

Comparative alteration in atherogenic indices and hypocholesteremic effect of palm oil and palm oil mill effluent in normal albino rats

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Abstract

The comparative hypocholesteremic effect of feeding palm oil and palm oil mill effluent (POME) was investigated in male albino rats. Diets were prepared and designed to contain 50% of energy as carbohydrate, 35% as fat, and 15% as protein. Groups of six rats were each fed one of these diets, while a group was fed pelletized mouse chow which served as the control. Feeding on palm oil and POME led to a significant increase ($p < 0.05$) in serum total cholesterol, triglyceride, and vLDL. Feeding on POME led to significant increase ($p < 0.05$) in cholesterol, triglyceride and LDL levels in brain tissues. Increased hepatic LDL level was also observed in POME fed rats. Except for hepatic triglyceride and tissues HDL level, a rather reduced level of the studied lipids was observed in the serum and tissues of palm oil fed rats compared to POME. These results indicate the protective potentials of palm oil against cardiovascular disease, as well as hyperlipidemia that characterize obesity and hypertension; as compared to its effluent.

Keywords: Atherogenic indices, Hypocholestermia, Palm oil and POME

http://dx.doi.org/10.1016/j.heliyon.2015.e00010

1. Introduction

Palm oil has been reported to be the most widely produced edible vegetable oil in the world surpassing soybean oil [1]. Its nutritional and health benefits have been well documented. It is very rich in Carotenes which gives it the characteristic red colour. It is also high in tocopherols and tocotrienols, CoQ10, phytosterols, and glycolipids [2]. Tocotrienols and carotenes from palm oil are used in food fortification for specific health use and antiaging cosmetics [3] [4]. It is estimated that for 1 tonne of crude palm oil produced, 5–8 tonnes of water are required, and most of which will end up as palm oil mill effluent (POME) [5]. POME is a colloidal suspension of 95–96% water, 0.6–0.7% oil and 4–5% total solids including 2–4% suspended solids [6].

Elevation of serum total cholesterol, triglycerides and low-density lipoprotein (LDL) cholesterol as well as alteration of other lipid parameters has been implicated as a principal risk factor for cardiovascular ailments [7]. This term commonly described as hyperlipidemia has emerged as an increasingly prevalent risk factor in both adults and children, concomitant with the worldwide epidemic of obesity [8]. Until recently, reduced fat intake was being recommended to decrease its occurrence. However, reports in several studies indicate that the type of fat rather than the total amount of fat in a diet plays a major role in hyperlipidemia [9]. This study aims to investigate the comparative hypocholesteremic effect of feeding palm oil and its effluent on normocholesteremic rats.

2. Results

Fig. 1 depicts serum lipid profile of the experimental groups. Feeding on palm oil and POME led to a significant increase ($p < 0.05$) in total cholesterol,

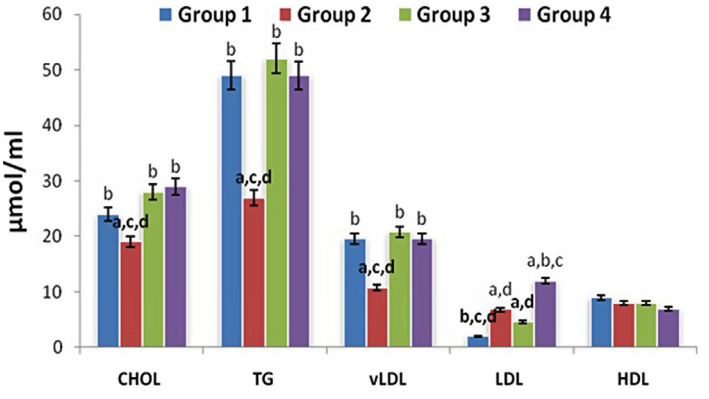

Fig. 1. Serum cholesterol level of experimental groups. Values = mean + SD; n = 6. (a) Statistically significant ($p < 0.05$) as compared with group 1; (b) Statistically significant ($p < 0.05$) as compared with group 2; (c) Statistically significant ($p < 0.05$) as compared with group 3; (d) Statistically significant ($p < 0.05$) as compared with group 4.

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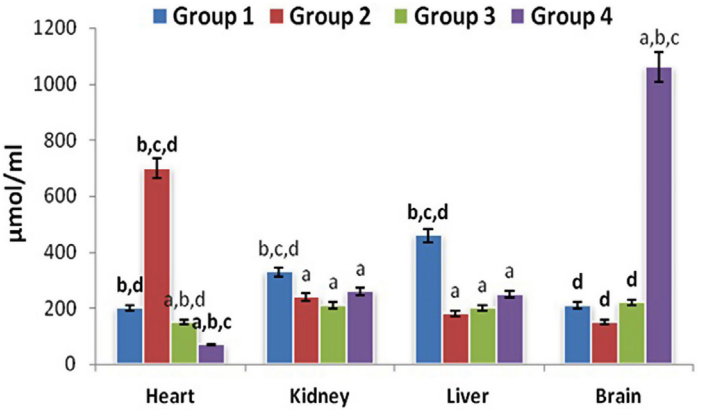

Fig. 2. Tissue cholesterol levels of experimental groups. Values = mean + SD; n = 6.
(a) Statistically significant ($p < 0.05$) as compared with group 1; (b) Statistically significant ($p < 0.05$) as compared with group 2; (c) Statistically significant ($p < 0.05$) as compared with group 3; (d) Statistically significant ($p < 0.05$) as compared with group 4.

triglyceride, and vLDL. No significant difference was observed in the HDL level of the experimental groups.

Feeding on POME led to significant increase ($p < 0.05$) in cholesterol level in brain tissues as shown in Fig. 2. Significant ($p < 0.05$) increased cholesterol level was also observed in the cardiac and hepatic tissues of rats fed on lipid-free diet and palm oil, respectively.

Feeding on palm oil significantly ($p < 0.05$) increased hepatic triglyceride level as depicted in Fig. 3. Palm oil and POME also led to significant increase in brain tissues, respectively. However, a rather reduced level was observed in the cardiac tissues of rats fed on POME.

An increased LDL level was observed in the hepatic and brain tissues of rats fed on POME as shown in Fig. 4. A similar observation was made in the

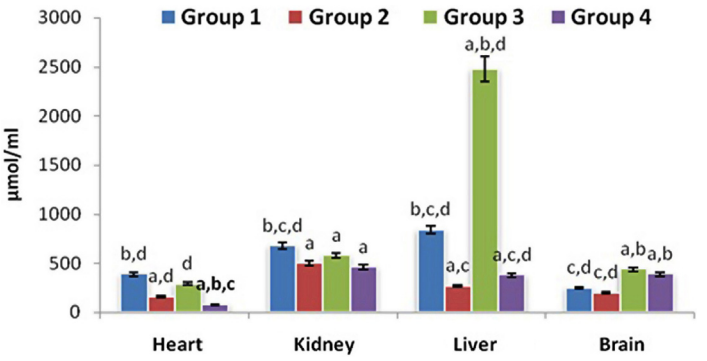

Fig. 3. Tissue triglyceride levels of experimental groups. Values = mean + SD; n = 6.
(a) Statistically significant ($p < 0.05$) as compared with group 1; (b) Statistically significant ($p < 0.05$) as compared with group 2; (c) Statistically significant ($p < 0.05$) as compared with group 3; (d) Statistically significant ($p < 0.05$) as compared with group 4.

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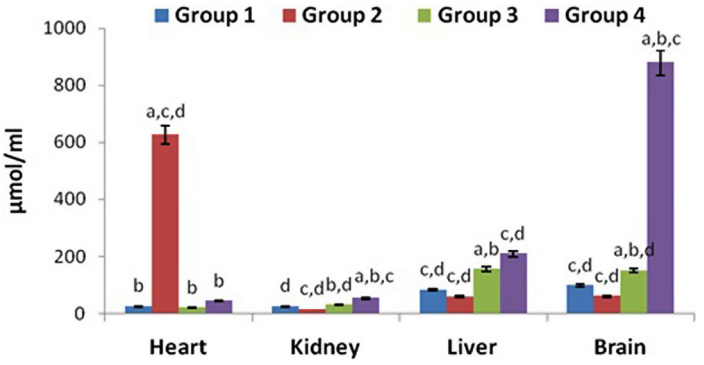

Fig. 4. Tissue LDL levels of experimental groups. Values = mean + SD; n = 6. (a) Statistically significant ($p < 0.05$) as compared with group 1; (b) Statistically significant ($p < 0.05$) as compared with group 2; (c) Statistically significant ($p < 0.05$) as compared with group 3; (d) Statistically significant ($p < 0.05$) as compared with group 4.

cardiac tissues of rats fed on lipid-free diet. There was little or no difference in the renal tissues of the experimental groups.

An increased HDL level was observed in the cardiac, hepatic and brain tissues of rats fed on palm oil as depicted in Fig. 5. Feeding on POME also led to significant ($p < 0.05$) increase in renal HDL level.

Fig. 6 shows the atherogenic indices of the experimental groups. Increased values of the studied indices were observed in POME fed rats. This was followed by the palm oil fed.

3. Discussion

High plasma concentrations of cholesterol, in particular those of low-density lipoprotein (LDL) cholesterol, have been described as one of the principal risk

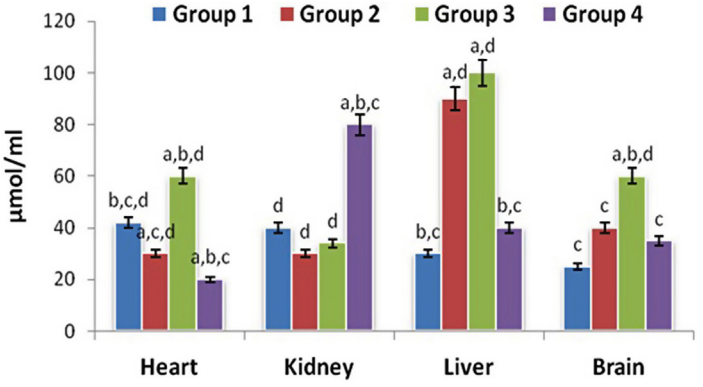

Fig. 5. Tissue HDL levels of experimental groups. Values = mean + SD; n = 6. (a) Statistically significant ($p < 0.05$) as compared with group 1; (b) Statistically significant ($p < 0.05$) as compared with group 2; (c) Statistically significant ($p < 0.05$) as compared with group 3; (d) Statistically significant ($p < 0.05$) as compared with group 4.

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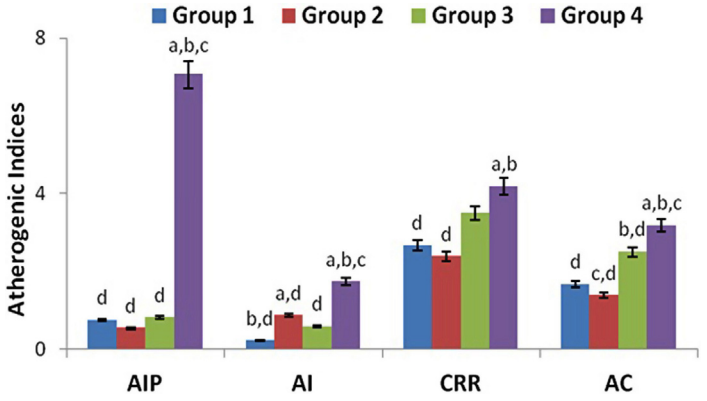

Fig. 6. Serum atherogenic indices of experimental groups. Values = mean + SD; n = 6. (a) Statistically significant ($p < 0.05$) as compared with group 1; (b) Statistically significant ($p < 0.05$) as compared with group 2; (c) Statistically significant ($p < 0.05$) as compared with group 3; (d) Statistically significant ($p < 0.05$) as compared with group 4.

factors for atherosclerotic cardiovascular disease [7] [15]. They can be influenced and modified by the type and amount of dietary fats [16]. Palm oil is the main source of edible fats in most African diets contributing up to 80% of all fat consumed [17]. This study reports the comparative effect of palm oil and POME consumption on serum and cholesterol concentration.

The observed elevated levels of serum cholesterol, triglyceride, and vLDL in the formulated diets indicates their hyperlipidemia potentials. These elevations have been implicated as a primary risk factor for cardiovascular heart disease [14] [18]. The decreased LDL level on consumption of palm oil diet, corresponds to previous reports by Edem [19] that its consumption causes endogenous cholesterol level to drop.

Elevated postprandial cholesterol and triglyceride response to fatty meals have been associated with patients clinically documented atherosclerosis [20]. Recently, cultured neurons have been shown to require glia-derived cholesterol to form numerous and efficient synapses [21]. Cholesterol content of brain tissues has been shown not to be affected by high-fat/cholesterol diet. In contrast, feeding of POME however led to elevated cholesterol in brain tissues. Kurban et al. [22] reported a similar observation on feeding selected vegetable oils to rats. The observed elevated level of TG in hepatic tissues of palm oil fed rats also contradicts previous reports by Ekpo et al. [17] that palm oil do not have adverse effects on tissue lipid profile.

The increased LDL level in brain tissues of rats fed on POME is of major concern. The potential of cells of the blood brain barrier (BBB) to take up LDL through luminal endothelial receptors has been reported [21]. This increased level may be attributed to the high serum LDL level in the POME fed rats.

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HDL has been implicated in the transportation of cholesterol from the peripheral tissues to the liver, thus protecting against atherosclerosis [23]. The increased significant ($p < 0.05$) level observed in the hepatic, brain and particularly the cardiac tissues in the palm oil fed rats portrays a protective effect of palm oil consumption against hyperlipidemia in these tissues.

Atherogenic indices have been described as powerful indicators of the risk of cardiovascular disease; the higher the value, the higher the risk of developing the disease and vice versa [14] [24] [25]. In this study, feeding on palm oil significantly reduced all studied atherogenic indices as compared to POME. Thus, further indicating its cardio-protective potentials.

4. Conclusion

These results are indications of the protective potentials of palm oil against cardiovascular disease, as well as hyperlipidemia that characterize obesity and hypertension; as compared to its effluent. Proper measures should therefore be put in place to avert adulteration of palm oil with POME which could have serious health implications.

5. Materials and methods

5.1. Palm oil and POME

Palm oil and POME were obtained from a palm oil mill industry in Benin City, Nigeria. They were stored in plastic containers until further studies.

5.2. Formulation of diets

Three special diets were prepared using the formula described by Howell et al. [10]. They were designed to contain 50% of energy as carbohydrate, 35% as fat, and 15% as protein, except for lipid-free diet which had distilled water substituted for fat (Table 1). Defatted soybean (15%) was provided for the protein requirement.

Table 1. Composition of diets used.

Ingredients	Mouse Chow	Diet 1	Diet 2	Diet 3
Corn starch	–	50	50	50
Soybean powder (defatted)	–	15	15	15
Palm Oil	–	–	35	–
POME	–	–	–	35
Distilled water	–	35	–	–

Data are present in percentage.

http://dx.doi.org/10.1016/j.heliyon.2015.e00010

5.3. Feeding trials

Twenty four male albino rats, each weighing between 90–120 g were maintained in accordance with and with the approval of the Animal Ethical Committee, Bells University of Technology, Ota, Nigeria. They were acclimatized for one week on pelletized mouse chow (Ladokee® Feeds Nigeria Ltd, Nigeria) with water given ad libitum at room temperature and a 12-h light and dark cycle. They were randomly assigned into groups, each consisting of six animals

GROUP 1 = Pelletized mouse chow

GROUP 2 = Lipid-free diet

GROUP 3 = Palm oil

GROUP 4 = POME

The rats were monitored daily for food and water intake, and body weight. At the end of the sixth week, the rats were fasted overnight and sacrificed by cervical dislocation.

The organs (Liver, Kidneys, Heart and Brain) were removed, rinsed in the ice-cold 1.15% KCl solution to wash off excess blood, blotted dried with filter paper, and weighed. The organs were homogenized in four parts of homogenizing buffer (i.e., 1 g of organ in 4 ml of buffer) and centrifuged at 10,000 rpm for 15 min in an ultracentrifuge at a temperature of $\leq$2 °C to get the mitochondrial fraction. The supernatant (post-mitochondrial fraction) was decanted and stored at $\leq$4 °C for subsequent analysis. Each time the supernatant was outside the freezer, it was kept in ice bags.

5.4. Lipid profile analysis

Serum and tissues total cholesterol, triglyceride and High Density Lipoprotein (HDL) were measured by enzymatic colorimetric method using Randox kits. The concentration of low-density lipoprotein (LDL) cholesterol was calculated by the formula of Friedwald et al. [11].

5.5. Atherogenic indices

Atherogenic Index (AI) and Atherogenic Index of Plasma (AIP) were calculated as described by Takasaki [12] and Onat et al. [13], respectively. Cardiac Risk Ratio (CRR) and Atherogenic Coefficient (AC) were calculated according to the methods described by Ikewuchi and Ikewuchi [14].

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5.6. Statistical analysis

Statistical significance was established using one-way analysis of variance (ANOVA), and data were reported as mean ± standard deviation. Significant difference was established at $p < 0.05$. Statistical analyses were carried out using SPSS for Windows, version 17.0 (SPSS Inc., Chicago, IL).

Declarations

Author contribution statement

Ochuko L. Erukainure: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

John A. Ajiboye, Babatunde A. Lawal: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data.

Viola A. Nwachukwu: Performed the experiments; Contributed reagents, materials, analysis tools or data.

Adesewa O. Tugbobo-Amisu: Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Ebelechukwu N. Okafor: Contributed reagents, materials, analysis tools or data.

Competing interest statement

The authors declare no conflict of interest.

Funding statement

The authors received no funding from an external source.

Additional information

No additional information is available for this paper.

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