

Original Research Article

Palm oil improves lipid metabolism and cardiovascular health of Wistar rats

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Abstract

Purpose: To investigate the effect of fresh and stored palm oil of varied free fatty acids (FFA) levels on lipid metabolism and cardiovascular health in Wistar rats.

Methods: Thirty-six (36) rats, randomly divided into six groups of six rats each, were used in this study. Group I comprised rats fed normal diet (control group) while Group II served as control and was given fresh palm oil containing 0.4 % FFA. Groups III – VI were administered palm oil (480 mg/kg body weight) of varied FFA levels (4.8, 8.4, 21.9, and 42.7 %), respectively. Body weight, lipid profile, cardiovascular disease risk indices, expression of peroxisome proliferator activator receptor gamma (PPAR- γ) and LDL-receptor genes as well as heart histology of the rats were determined.

Results: There was a significant increase in body weight of normal and study groups, with the fresh palm oil group gaining more weight ($p < 0.05$). However, groups exposed to moderate FFA levels (4.8 and 8.4 %) exhibited reduced weight changes. Administration of palm oil led to a non-significant reduction in serum total cholesterol concentration ($p > 0.05$). Also, a significant reduction in the triglyceride concentrations of study groups was observed compared to normal control rats ($p < 0.05$). Furthermore, palm oil improved the lipid profile by reducing serum concentrations of LDL-cholesterol and increasing the synthesis of HDL-cholesterol of the animals. In addition, cardiovascular disease risk factors decreased significantly in study groups compared to normal rats while an upregulation of PPAR- γ and LDL-receptor genes was observed in the palm oil groups ($p < 0.05$). Furthermore, normal heart histology was observed in all the animals.

Conclusion: This study suggests that fresh and stored palm oil improves lipid profile and cardiovascular disease risk factors and is therefore beneficial for cardiovascular health.

Keywords: Palm oil, Lipid metabolism, Cardiovascular diseases, Wistar rats

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INTRODUCTION

Palm oil, extracted from the ripened fruit of *Elaeis guineensis*, is one of the most widely consumed edible oils globally. It contains a good mix of saturated fatty acids, primarily palmitic acid, alongside monounsaturated and polyunsaturated

fatty acids, making it a key dietary lipid source [1]. The effects of palm oil consumption on health, particularly its influence on lipid metabolism and cardiovascular disease risk have been widely debated. While fresh palm oil is known to possess antioxidant properties due to its rich content of tocotrienols and carotenoids,

stored or repeatedly heated palm oil undergoes oxidative degradation, potentially forming harmful lipid peroxidation products [2]. Fresh palm oil has also been reported to have cardioprotective properties, due to its anti-oxidative stress and anti-inflammatory ability [3].

Cardiovascular diseases (CVDs) are the leading cause of death globally, accounting for nearly 17.9 million deaths annually [4]. These conditions, including coronary artery disease, stroke and heart failure, are strongly linked to dietary fat intake. Excessive consumption of saturated fats raises low-density lipoprotein (LDL) cholesterol, commonly known as "bad cholesterol," which contributes to atherosclerosis, a condition where fatty plaques accumulate in arteries, thereby restricting blood flow and increasing the risk of heart attacks and strokes. Conversely, high-density lipoprotein (HDL) cholesterol, or "good cholesterol," plays a protective role by removing LDL cholesterol from the bloodstream. Maintaining a healthy balance between LDL and HDL is essential for reducing cardiovascular diseases.

Some studies have shown that dietary intake of fresh palm oil has hypolipidemic effect, reducing LDL cholesterol (LDL-C) and increasing HDL cholesterol (HDL-C), thereby contributing to better lipid homeostasis [5]. However, when palm oil is stored for prolonged periods or exposed to repeated heating, it undergoes thermo-oxidative degradation, resulting in lipid peroxidation which may elevate LDL-C and promote cardiovascular diseases [3,6]. Overall, monitoring lipid indices and maintaining a healthy balance of cholesterol through diet and lifestyle choices are crucial for preventing cardiovascular diseases. This study investigated the effects of fresh and stored palm oil on the lipid profile and cardiovascular disease risk factors in Wistar rats.

EXPERIMENTAL

Preparation and collection of palm oil samples

Freshly-milled palm oil sample (Sample A) and palm oil samples stored at room temperature for sixteen months (Samples B – E) were obtained from the Nigerian Institute for Oil Palm Research (NIFOR), Benin City, Edo State, Nigeria.

Determination of FFA levels

The FFA composition of the palm oil was determined according to the American Oil Chemists' Society's (AOCS) official method Ca

5-40 [7]. Results are expressed as percentage of palmitic acid.

Animal grouping and administration of palm oil samples

Female adult albino Wistar rats, weighing between 130 – 150 g, were selected for this study. The animals were obtained from the Animal House of the Department of Biochemistry, Faculty of Life Sciences, University of Benin, Benin City, Edo State, Nigeria. They were kept in clean cages in a 12-hour light/dark cycle room with daily litter change. The animals were acclimatized for two weeks before the experiment commenced and were fed with Grower's mash and water *ad libitum*. Thirty-six (36) Wistar rats were divided into six groups of six rats per group and treated as follows: Group I (Normal Control) was given feed and water only, while Group II (Control) was fed freshly-milled crude palm oil containing 0.4 % FFA. Groups III – VI were given palm oil stored for sixteen months and containing varied FFA levels, viz, 4.8, 8.4, 21.9 and 42.7 %, respectively. Palm oil was administered at a dosage of 480 mg/kg body weight to the animals orally for four weeks. Rat weights were monitored throughout the duration of the experiment.

Ethical approval

Ethical approval for the use of laboratory animals was granted by the Ethical Committee of the Faculty of Life Sciences, University of Benin (approval no. FLS/REC/2025/001).

Animals were euthanized under mild halothane anaesthesia at the end of treatment period after an overnight fast and blood was collected by cardiac puncture into plain sample bottles for biochemical analyses, while a portion of heart was excised and used for histopathological examination.

Estimation of total cholesterol

Total cholesterol was determined using the method of Trinder [8]. One millilitre of cholesterol reagent (containing 50 mmol/L PIPES buffer, 100 U/L cholesterol oxidase, 150 U/L cholesterol esterase, 0.3 mmol/L 4-aminoantipyrine, 800 U/L peroxidase and 6.0 mmol/L phenol) was dispensed into three test tubes labelled blank, standard and sample. Then, 10 µL each of distilled water, standard cholesterol solution and serum were added into tubes labelled blank, standard and sample, respectively. The contents of each tube were mixed and incubated for 10 minutes at 25 °C and absorbances of standard

(A₀) and sample (A_s) were read at 500 nm against the blank within 1 h. Concentration of cholesterol in the serum (CCS) was calculated using Eq 1 and expressed in milligrams per deciliter of sample.

$$CCS = (A_s/A_0)C_0 \dots\dots\dots (1)$$

Where C₀ is the concentration of standard

Assessment of serum triacylglycerol

Triacylglycerol concentration in serum was assayed according to the procedure of Buccolo and David [9]. Briefly, 1 mL of triglyceride reagent was added to two test tubes containing 10 µL of standard triglyceride solution and serum, respectively. A reagent blank composed of 1 mL of triglyceride reagent was also prepared. The tubes were incubated for 10 min at 25 °C and absorbances of standard and sample (A₀ and A_s, respectively) were read at 500 nm against reagent blank within 60 min. The concentration of triacylglycerol in sample (CTS) was calculated using Eq 2 and expressed in mg/dL.

$$CTS = (A_s/A_0)C_0 \dots\dots\dots (2)$$

Estimation of HDL-cholesterol

Concentration of high-density lipoprotein-cholesterol in serum (HDL) was determined using cholesterol CHOD-PAP assay as described by Lopes-Virellas [10]. An aliquot (0.1 mL) of each of distilled water, standard supernatant and sample supernatant was added to test tubes labelled blank, standard and sample, respectively. Thereafter, HDL-cholesterol reagent (containing 0.55 mmol/L phosphotungstic acid and 25 mmol/L magnesium chloride) was further added to all tubes. Each tube was mixed and incubated for 10 min at 25 °C after which the absorbance of standard and sample was read at 500 nm against the blank within 60 minutes. Concentration of HDL was calculated using Eq 3.

$$HDL = (A_s/A_0)C_0 \dots\dots\dots (3)$$

Determination of LDL-cholesterol

The concentration of LDL-cholesterol was calculated using a modification of Friedewald's formula represented in equation 4 [11].

$$LDL-C = TC - HDL-C - (TG/5) \dots\dots\dots (4)$$

Where TC = Total cholesterol, TG = triglycerides

Estimation of atherogenic and coronary risk indices

Atherogenic index (AI) was calculated as the ratio of LDL-cholesterol and HDL-cholesterol (Eq 5), while Coronary Risk Index (CRI) was calculated as the ratio of total cholesterol (TC) and HDL-cholesterol (Eq 6) [12]. Castelli's Risk Index III (CRI-III), atherogenic coefficient (AC) and atherogenic index of serum (AIS) were calculated using Eqs 7 – 9 [13,14].

$$AI = LDL-C/HDL-C \dots\dots\dots (5)$$

$$CRI = TC/HDL-C \dots\dots\dots (6)$$

$$CRI-III = TG/HDL-C \dots\dots\dots (7)$$

$$AC = TC - HDL-C/HDL-C \dots\dots\dots (8)$$

$$AIS = \log (TG/HDL-C) \dots\dots\dots (9)$$

Gene expression studies

The mRNA expression levels of PPAR-γ and LDL-receptor genes were determined using RT-qPCR as described by Elekofehinti and co-workers [15]. One microgram of DNA-free RNA was converted to cDNA by reverse transcriptase reaction with the aid of cDNA synthesis kit based on ProtoScript II first-strand technology (New England BioLabs) in a condition of 3-step reaction: 65 °C for 5 min, 42 °C for 1 h, and 80 °C for 5 min. Polymerase chain reaction (PCR) for the amplification of PPAR-γ and LDL-receptor genes was carried out with OneTaqR2X Master Mix (NEB) using the primers in Table 1 (Inqaba Biotec, Hatfield, South Africa). Amplification was performed in a 25 µL reaction mixture containing cDNA, forward and reverse primers and Ready Mix Taq PCR master mix under the following conditions: Initial denaturation at 95 °C for 5 min, followed by 30 cycles of amplification (denaturation at 95 °C for 30 s, annealing at 55 °C for 30 s and extension at 72 °C for 60 s) and ending with final extension at 72 °C for 10 min. The amplicons were resolved on 1.0 % agarose gel. The GAPDH gene was used to normalize the relative level of expression of each gene, and quantification of band intensity was done using ImageJ software.

Histological assessment of the heart

Histological examination of the heart tissue was carried out according to the method of Drury *et al* [16]. Portions of the heart were fixed in 10 % neutral buffered formalin for histology.

Table 1: Primer sequences used for the amplification of PPAR- γ and LDL-receptor genes

Gene	Forward primer sequence (5'- 3')	Reverse primer sequence (5'- 3')
PPAR- γ	CGAGCTGGGAGTAGCCTGA	GATCACCAGCAGAGGTCCAG
LDL-receptor	CATCTTCCTCCCCATTGCA	CCTCAGCCGCCAGTTCT
GAPDH	CCTCTATGCCAACACAGTGC	CATCGTACTCCTGCTTGCTG

Thin sections of the heart were dissected and processed using Leica TP2010 automatic tissue processor for 18 hours. The processor passed the tissues through fixation, dehydration, de-alcoholization, and paraffinization. Ultra-thin sections (5 μ m) were sliced from the paraffinized sections using a Thermo Scientific semi-automated rotary microtome. The tissues were then subjected to Hematoxylin and Eosin (H&E) staining and viewed under a microscope using x400 magnification.

Statistical analysis

Statistical analysis was carried out using Statistical Package for Social Sciences (SPSS) version 21.0 for Windows. Results are expressed as mean $\pm$ standard error of the mean (SEM) of six replicates. The levels of homogeneity amongst groups were tested using one-way analysis of variance (ANOVA), with $p < 0.05$ considered significant. Duncan's multiple range test was used to separate homogenous groups.

RESULTS

Free fatty acid (FFA) levels of palm oil samples

The result of FFA levels of the palm oil samples is presented in Figure 1. A significant difference ($p < 0.05$) in the FFA levels of the samples was observed, with the increase in FFA directly proportional to the duration of storage. The FFA concentrations for samples A – E were recorded as 0.4, 4.8, 8.4, 21.9 and 42.7 %, respectively.

Effect of palm oil on body weight and heart weight of Wistar rats

Table I shows the mean body and heart weights of rats. There was a significant increase in the body weight of the animals. The rats fed with fresh palm oil sample (group II) gained more weight compared to normal control rats and the rats fed with stored palm oil samples (groups III – VI). On the other hand, there was no significant difference ($p > 0.05$) in the weight of hearts of normal control rats and those of rats fed fresh and stored palm oil samples. The same trend was observed in the organ-body weight ratios of rats.

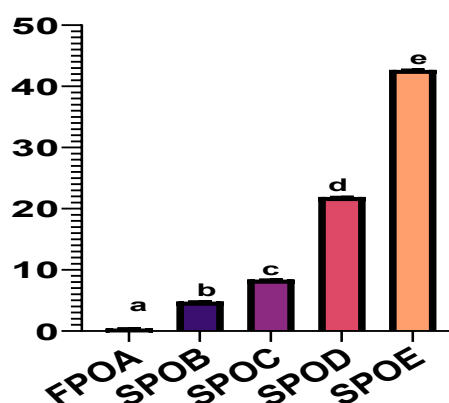


Figure 1: Percentage free fatty acid levels of palm oil samples A – E. Values with different superscripts are significantly different ($p < 0.05$). FPOA = Fresh palm oil sample A; SPOB = Stored palm oil sample B; SPOC = Stored palm oil sample C; SPOD = Stored palm oil sample D; SPOE = Stored palm oil sample E

Table 1: Mean values of the body weight and heart weight of rats

Group (% FFA)	Body weight before treatment (g)	Body weight after treatment (g)	Difference in weight (g)	Weight of heart (g)	Heart-to-body weight ratio (g)
I	136.50 $\pm$ 0.11 ^a	148.06 $\pm$ 0.14 ^b	8.54 $\pm$ 0.05 ^e	0.5200 $\pm$ 0.00 ^c	0.0035 $\pm$ 0.00 ^b
II (0.4% FFA)	141.30 $\pm$ 0.11 ^c	157.00 $\pm$ 0.11 ^e	11.10 $\pm$ 0.08 ^f	0.5400 $\pm$ 0.00 ^c	0.0034 $\pm$ 0.00 ^b
III (4.8% FFA)	149.50 $\pm$ 0.11 ^f	161.60 $\pm$ 0.11 ^f	8.06 $\pm$ 0.06 ^d	0.5333 $\pm$ 0.00 ^c	0.0032 $\pm$ 0.00 ^a
IV (8.4% FFA)	140.30 $\pm$ 0.11 ^b	144.70 $\pm$ 0.11 ^a	3.13 $\pm$ 0.08 ^a	0.4800 $\pm$ 0.00 ^a	0.0034 $\pm$ 0.00 ^b
V (21.9% FFA)	142.30 $\pm$ 0.11 ^d	150.70 $\pm$ 0.11 ^c	5.90 $\pm$ 0.08 ^b	0.4800 $\pm$ 0.00 ^a	0.0031 $\pm$ 0.00 ^a
VI (42.7% FFA)	143.00 $\pm$ 0.11 ^e	154.00 $\pm$ 0.11 ^d	7.69 $\pm$ 0.08 ^c	0.5067 $\pm$ 0.00 ^b	0.0032 $\pm$ 0.00 ^a

Values with different superscripts within a column indicate significant differences ($p < 0.05$) between groups

Effect of palm oil of varied FFA levels on lipid profile

The effect of palm oil on the lipid profile of rats is presented in Table 2. There was a non-significant reduction in the serum total cholesterol concentration of rats fed fresh and stored palm oil (Groups II – VI) compared to the normal control (Group 1; $p > 0.05$). In addition, serum triglyceride concentration of rats fed with fresh palm oil samples was significantly reduced ($p < 0.05$) compared to that of normal rats, but was not significantly different from the stored palm oil group ($p > 0.05$). Furthermore, there was a significant increase in HDL-cholesterol levels of rats fed with palm oil compared to normal control rats ($p < 0.05$). On the other hand, LDL-cholesterol levels were significantly reduced in rats fed with stored palm oil samples compared to control group and fresh palm oil group ($p < 0.05$).

Effect of palm oil on cardiovascular disease risk indices of rats

There was a non-significant decrease in the atherogenic and coronary risk indices of rats fed with fresh and stored palm oil (Groups II – IV) compared with control group, as presented in Table 3. However, the decrease was very significant in rats fed with palm oil of higher FFA concentration (Groups V and VI) compared to control group. Similarly, there was decrease in the CRI, Castelli index III, atherogenic coefficient and atherogenic index of serum of rats fed with palm oil compared to control group, with the decrease being very significant in rats that received palm oil samples of higher FFA levels (Groups V and VI).

Effect of palm oil on peroxisome proliferator activator receptor gamma (PPAR- γ) and LDL-receptor genes

There was a significant increase ($p < 0.05$) in the expression levels of PPAR- γ and LDL-receptor genes of rats that received fresh and stored palm oil (Groups II – VI) compared to control group (Figure 2).

Effect of palm oil on the histopathology of heart

Assessment of the histopathology of the hearts of normal rats and those fed palm oil of varied FFA levels (0.4 – 42.7 %) for a period of four (4) weeks revealed no observable changes in the heart myocytes. The features are indicative of normal myocytes (Figure 3 A – F).

DISCUSSION

This study provides crucial insights into how palm oil, particularly of different FFA levels, influences lipid metabolism and cardiovascular disease risk indices in Wistar rats. There were significant differences in the FFA levels of the palm oil samples used in this study. The increase in the FFA levels of the samples was due to the storage duration of fresh fruit bunches (FFB) prior to processing. The FFA levels of the freshly processed palm oil obtained from freshly harvested FFB was 0.4 %, while those of stored palm oil samples ranged from 4.8 – 42.7 % and corresponded with the storage time of the fresh fruit bunches before processing. These results show that post-harvest storage time of FFB directly affects the FFA levels of palm oil and corroborates earlier reports [17].

Table 2: Lipid profile of rats administered palm oil of varied concentrations of FFA

Group (% FFA)	Total Cholesterol (mg/dL)	Triglycerides (mg/dL)	HDL-cholesterol (mg/dL)	LDL-cholesterol (mg/dL)
I	180.16±17.04 ^a	181.16±15.37 ^b	25.36±1.81 ^a	118.56±12.16 ^b
II (0.4% FFA)	170.12±2.24 ^a	155.84±0.75 ^a	26.50±0.82 ^a	112.45±2.92 ^b
III (4.8% FFA)	178.78±2.80 ^a	178.56±4.12 ^a	32.20±3.08 ^b	111.83±0.97 ^b
IV (8.4% FFA)	137.00±25.86 ^a	168.18±1.12 ^a	22.51±0.16 ^a	80.84±25.80 ^a
V (21.9% FFA)	173.05±2.06 ^a	173.18±4.75 ^a	38.19±2.30 ^c	100.22±5.3 ^a
VI (42.7% FFA)	163.31±4.3 ^a	181.49±4.30 ^b	57.25±0.84 ^d	69.75±2.60 ^a

Values with different superscripts are significantly different ($p < 0.05$)

Table 3: Mean values of cardiovascular disease risk indices of Wistar rats

Group	Atherogenic index	Coronary risk index	Castelli risk index	Atherogenic coefficient	Atherogenic Index of serum
I (Normal)	4.65±0.16 ^b	7.08±0.17 ^c	7.13±0.10 ^d	6.07±0.16 ^c	0.85±0.01 ^d
II (0.4% FFA)	4.25±0.24 ^b	6.43±0.29 ^c	5.89±0.2 ^c	5.43±0.29 ^c	0.76±0.01 ^c
III (4.8% FFA)	3.55±0.40 ^b	5.64±0.50 ^c	5.63±0.46 ^c	4.65±0.50 ^c	0.75±0.04 ^c
IV (8.4% FFA)	3.61±1.17 ^b	6.10±1.19 ^c	7.47±0.10 ^d	5.10±1.19 ^c	0.86±0.01 ^e
V (21.9% FFA)	2.86±0.17 ^a	4.57±0.33 ^b	4.55±0.15 ^b	3.57±0.33 ^b	0.66±0.0 ^b
VI (42.7% FFA)	1.17±0.03 ^a	2.85±0.34 ^a	3.17±0.03 ^a	1.85±0.03 ^a	0.50±0.0 ^a

Values with different superscripts are significantly different ($p < 0.05$)

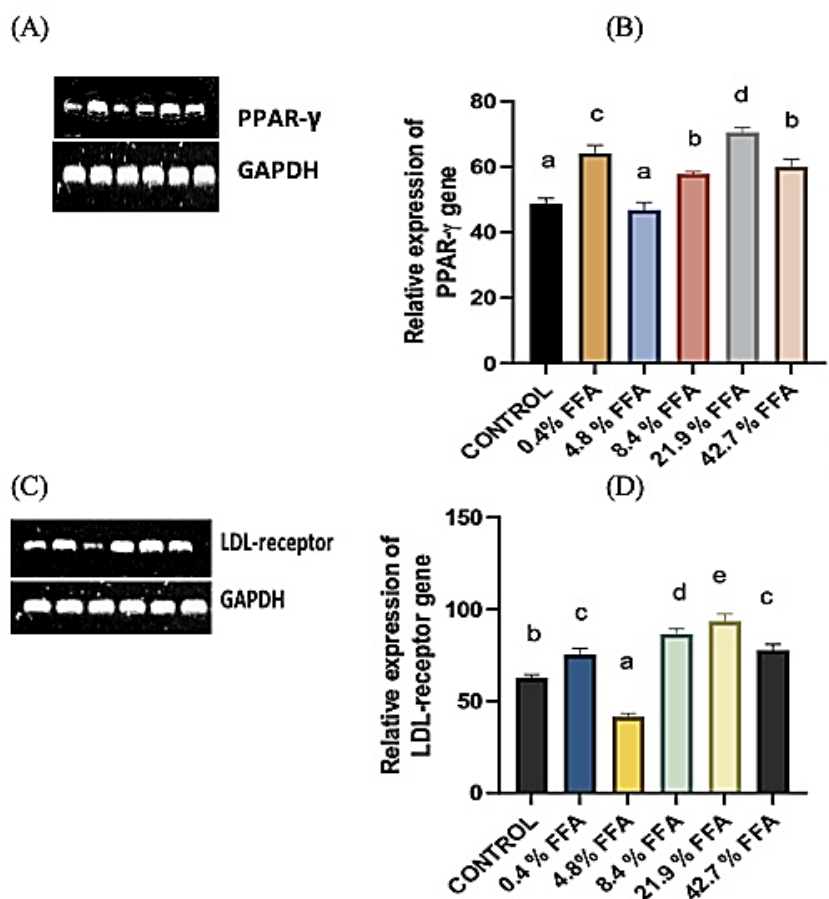


Figure 2: Expression levels of (A) PPAR-γ and (C) LDL-receptor genes. The quantification of each band on the gel is represented as bar charts in (B) and (D), respectively. Data with different superscripts are significantly different ($p < 0.05$)

The results of body and organ weights determination show a clear relationship between the FFA levels in palm oil and the body/organ weight changes in Wistar rats. While control group exhibited an 8.54 % increase in weight, rats fed with fresh palm oil (0.4 % FFA) had the most significant weight gain (11.10 %). However,

Groups III and IV, exposed to moderate FFA levels (4.8 % and 8.4 %, respectively), exhibited reduced weight changes, with the 8.4 % FFA group showing the least weight gain (3.13 %). Interestingly, the highest FFA group (42.7 %) showed a moderate weight gain (7.69 %).

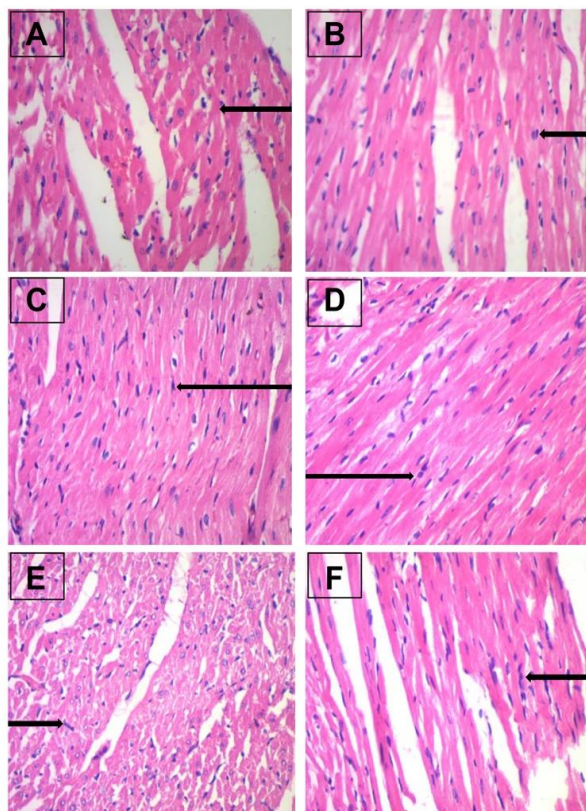


Figure 3: Sections of cardiac muscle showing myocytes (arrow) with peripherally placed nuclei surrounded by eosinophilic cytoplasm. Features are in keeping with normal myocytes (H&E; $\times 400$)

These outcomes suggest a potential threshold for FFA toxicity that hinders normal metabolic processes. On the other hand, results of heart weight determination revealed that heart weight was highest in the 0.4 % FFA group and lowest in the 8.4 % FFA and 21.9 % FFA groups. Heart-to-body weight ratio showed a different trend, with normal control group showing the highest ratios. Significant differences in the heart-to-body weight ratio were observed between groups ($p < 0.05$), indicating that FFA levels might have positive impact on heart size.

Lipid profile indices such as total cholesterol, LDL-cholesterol, HDL-cholesterol and triglycerides serve as critical markers for assessing cardiovascular disease risk. The results of lipid profile and cardiovascular disease risk indices from this study align with a previous study demonstrating that fresh palm oil (0.4 % FFA) has hypolipidemic properties, leading to a decrease in the pro-atherogenic lipoprotein (LDL-cholesterol) and an increase in the anti-atherogenic lipoprotein (HDL cholesterol), which are key indicators of improved lipid homeostasis [18]. Interestingly, results of this study revealed that stored palm oil samples had greater effect in further improving the lipid profiles of the animals. In the highest oxidation groups (21.9 % and 42.7

% FFA), a significant decrease in total cholesterol and LDL-cholesterol was observed, coupled with significant increases in HDL-cholesterol, indicating lower atherogenic and cardiovascular disease risks.

Several lipid-based indices help evaluate cardiovascular disease risk more precisely. The atherogenic index (LDL/HDL ratio) is a widely used marker for assessing the likelihood of atherosclerosis and related conditions, with higher values indicating greater risk, while CRI identifies individuals at risk of coronary artery disease (CAD). Elevated CRI values suggest an imbalance between protective and harmful cholesterol levels, increasing the likelihood of arterial plaque build-up and cardiovascular events. Also, the Castelli index, atherogenic coefficient, and atherogenic index of serum (AIS) are other lipid-based indices; higher values indicate increased susceptibility to atherosclerosis and CAD, making them valuable tools in preventive cardiology [12-14]. In this study, cardiovascular diseases risk indices, such as atherogenic index, coronary risk index, Castelli indices, atherogenic coefficient and atherogenic index of serum (AIS), showed significant decreases in rats fed fresh and stored palm oil samples compared to normal control

group, with highly oxidized palm oils (21.9 and 42.7 % FFA) having highest effects, which suggest a reduced cardiovascular disease risk. However, similar findings in earlier studies suggest that these effects could be a result of metabolic stress responses rather than true cardioprotective effects [19].

The PPAR- γ is a transcription factor belonging to the superfamily of nuclear receptors and its natural activating ligands are fatty acids and lipid-derived substrates whose expression induces differentiation of pre-adipocytes to adipocytes and stimulates triglyceride storage [20]. The LDL receptor (LDLR) facilitates LDL-cholesterol clearance from circulation, and mutations in the LDLR gene lead to familial hypercholesterolemia, a condition characterized by elevated LDL-cholesterol levels and increased risk of atherosclerosis [21]. The gene expression result showed a significant increase in the levels of PPAR- γ and LDL-receptor in rats that received fresh and stored palm oil of varied FFA levels compared to the normal control group. The upregulation of the proteins in the palm oil group further confirms the positive effects of palm oil in improving the lipid profile and cardiovascular disease risk factors of the animals.

Finally, histopathological examination of the heart revealed no observable cardiac tissue damage across all groups, indicating that within the four-week study period, palm oil consumption, regardless of FFA levels, did not induce any structural damage to the heart. This is consistent with the findings of Boon *et al*, who showed that fresh and moderately oxidized palm oils did not cause significant cardiac tissue alterations in hypertensive rats [20]. Overall, this study reinforces the understanding that fresh and stored palm oil have beneficial lipid-modulating effects.

Limitations of this study

While no immediate histopathological damage to the heart was observed in the time frame used in this study, the long-term impact of oxidized palm oil on cardiovascular health warrants further studies. Given the widespread use of palm oil globally, public health policies should focus on promoting proper processing and storage of the product.

CONCLUSION

This study demonstrates that palm oil consumption has a significant positive effect on lipid metabolism and cardiovascular health. Palm oil consumption exhibits favourable lipid profile

and cardiovascular disease risk indices, supporting its potential cardioprotective properties. Additionally, upregulation of PPAR- γ and LDL-receptor genes in this study further confirms the positive effects of palm oil on lipid metabolism.

DECLARATIONS

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Conflict of interest

No conflict of interest is associated with this work.

Data availability

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be obtained from the corresponding author upon request.

Contribution of authors

We declare that this work was done by the authors named in this article and all liabilities pertaining to claims relating to the content of this article will be borne by the authors. All authors have read and approved the manuscript.

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