

Palm kernel oil increases the risk of coronary heart disease in rats compared with ghee

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Abstract: The influence of high fat diets (HFD) containing single fat; palm oil (PO), palm kernel oil (PKO), or ghee on feed efficiency, lipid profile, kidney function and histopathological status of liver and heart of rats were studied. Male adult albino rats were fed on basal diet contained 10% corn oil or HFD that contained either of PO, PKO, corn oil or ghee and 1% cholesterol for twelve weeks. Results showed that feeding rats on HFD containing PO or PKO significantly increased serum total cholesterol, triglycerides and VLDL-C levels compared with those of rats that were fed on basal diet or HFD containing corn oil or ghee. The highest LDL-cholesterol level and the lowest total antioxidant capacity level were recorded in rats that were fed on PKO diet. The histopathological examination showed that feeding rats on HFD diet containing ghee for 12 weeks caused minimum damage to liver cells. Feeding rats on PKO diet caused undesirable histological changes in heart cells. These results indicated that PO and PKO caused more harmful effects on rat's blood serum triglycerides and cholesterol concentrations and liver cells than ghee.

Keywords: Ghee, Lipid profile, Palm kernel oil, Palm oil

I. Introduction

PO and PKO, are undoubtedly commercially ideal raw materials for the production of various specialty fats which include cocoa butter substitute, cheese analogue, toffee fats, non-dairy fats and cream filling fats. These fats are used in products such as chocolates/coatings, toffees, coffee whiteners, ice cream, whipped toppings as cream in cake, chocolate bar, cake, candy, jelly, fried snacks such as potato chips, filler creams and non-dairy products [1]. The palm fruit (*Elaeis Guineensis*) is the source of both PO (extracted from palm fruit) and PKO (extracted from the fruit seeds). The PO and PKO are high in saturated fatty acids, about 50% and 80% respectively. The PO contains palmitic acid; while PKO contains mainly lauric acid [2]. Several studies on animals and human indicated that PO could increase both cholesterol levels and the atherogenic risks. Kritchevsky [3] and his colleagues studied the effects of different combinations of corn oil and PO on aspects of lipid metabolism in adult albino rats. They showed that addition of even a small amount of palmitic acid in the presence of cholesterol in diet raised serum cholesterol levels significantly. Feeding rats on diets contained 10% coconut oil or PKO caused a significant increase in serum total cholesterol and LDL-C levels and PKO possessed a greater atherogenic risk than coconut oil [4]. Feeding hamsters on cholesterol-enriched (1g/kg) semipurified diet containing 20% PO increased plasma total cholesterol, non-HDL-Cholesterol and decreased plasma HDL-Cholesterol significantly [5]. Feeding weaning or adult rats on 10% PO, PKO or palm stearin for four or six months significantly increased serum total cholesterol, LDL-C, VLDL-C, TC:HDL and LDL:HDL ratios and caused a significant decrease in HDL-C [6,7]. Fresh PO had no significant effects on blood pressure and cardiac tissue but prolonged consumption of repeatedly heated PO may result in an increase in blood pressure level with necrosis of cardiac tissue of rats [8]. Feeding rats on high PO diet caused hepatic steatosis [9]. Dietary intake of PKO could pose a risk for coronary artery disease on a long term basis [10]. It was suggested that non-extreme short-term changes in PO and dietary cholesterol consumption lead to significant elevations in plasma TC and LDL-C in humans [11]. PO and PKO should be classified as cholesterol-raising fats for humans [12]. In contrast, it was found that feeding rats or hamsters on diets that contained more than 10% PO for 3 weeks or more improved serum and liver lipid profiles and enhanced HDL-C [13-18]. They recommended that it could be useful for preventing cardiovascular disease. Dietary PO, when replacing a major part of the normal fat content in a Dutch diet, may slightly reduce the lipoprotein- and apolipoprotein-associated cardiovascular risk profiles [19]. Consumption of PO as a source of dietary fat did not pose any additional risks for coronary artery disease [2]. There are some controversies about the effect of milk fat on serum lipid and liver status in rats. In this respect, some investigators [20,21] showed a reverse association between milk fat consumption and elevation of serum lipids levels and it also developed fatty changes in liver diseases. However, some studies [22,23] showed the beneficial effects of milk fat consumption on serum lipoprotein cholesterol values and it also reduces the probability of getting cardiovascular disease.

Due to economical reasons, ghee, butter oil and butter are being replaced by PO or PKO either in cooking, bakery products and dairy products analogue. The aim of this study is to explore and evaluate, *in vivo*, the possible harmful effects of these oils on health, mainly cardiovascular diseases, in comparison with the butter oil, i.e. ghee.

II. Materials and Methods

2.1 Oils and fats

Refined bleached deodorized PO and PKO were obtained from Cairo Oil and Soap Company, Egypt. Corn oil (Mazola brand) was obtained from the local market. Freshly prepared Ghee (is a product obtained exclusively from milk, cream or butter by a process that almost completely removes water and nonfat solids; it has a specially developed flavor and physical structure, (codex standards [24])) was purchased from Dairy Department, Faculty of Agriculture, Cairo University, Giza, Egypt. Fatty acids composition of the investigated oils and fats were determined by using gas chromatograph (Thermo Scientific DSQ™ II Series Single Quadrupole GC/MS, USA) equipped with a CDB-23) column (30 m, 0.25 mm i.d, 0.25 um film thickness). Fatty acid methyl esters were analyzed according to A.O.A.C. [25].

2.2 Kits for biochemical analyses

Total cholesterol, HDL-Cholesterol and triglycerides were determined using kits purchased from Analyticon Biotechnologies AG, Germany. Albumin and total protein were determined using kits purchased from EliTech, France. Urea was determined using kit purchased from Diamond, Egypt. Creatinine was determined using kit purchased from Biomed Diagnostics, Egypt. Total antioxidant capacity was determined using kit purchased from Bio-Diagnostic, Egypt. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were determined using kits purchased from Spectrum, Egypt. Alkaline phosphatase (ALP) was determined using kit purchased from Biosystems S.A., Spain.

2.3 Experimental animals

A total of 30 adult male Sprague Dawley albino rats weighing 110±10 grams were obtained from the animal house of National Nutrition Institute, Egypt and housed in individual cages. Water and diet were given *ad-libitum*. The animals were kept under normal laboratory conditions and were fed on a basal diet for 7 days for adaptation. Diets were prepared biweekly and were stored in the deep freezer at -10°C. All animals were humanely treated in accordance with the WHO guidelines for animal care, and the study design was approved by Ethics of animal use in research committee, Cairo University with approval number (Cu F Agry/ S/ FS/ 2013/ 22).

2.4 Experimental design

After the adaptation period, diets and water were introduced to animals for 12 weeks. The rats were divided randomly into the following groups of six rats each: Group 1 (G1): Rats were fed on a standard Basal diet (control). Group 2 (G2): Rats were fed on a HFD containing corn oil. Group 3 (G3): Rats were fed on a HFD containing PO. Group 4 (G4): Rats were fed on a HFD containing PKO. Group 5 (G5): Rats were fed on a HFD containing ghee. The consumed diets and body weight of rats were recorded twice weekly to monitor body weight changes and calculate feed efficiency ratio. Basal diet, mineral and vitamin mixtures were prepared according to A.O.A.C. [26]. The compositions of the diets are shown in Table 1.

2.5 Collection of blood samples and organs

At the end of the experiment, rats were fasted for 12 hours and then they were anesthetized with diethyl ether. Blood samples were collected from the portal hepatic vein and the clear serum was separated and stored in the deep freezer at -10°C for subsequent chemical analyses.

2.6 Preparation of tissue homogenate

Immediately after sacrificing the rats, livers and hearts were plotted free from adhering blood, washed with cold physiological saline (0.9% w/v), dried between filter papers and weighed. Specimens of liver were used for cholesterol determination. Liver tissue was homogenized in 10 ml cold buffer (50 mM potassium phosphate buffer, pH 7.5, 1mM EDTA) per gram tissue, using tissue homogenizer. The liver homogenate was centrifuged at 4000 rpm for 15 min at 4°C. The supernatant was collected and stored at -20°C for analysis.

2.7 Nutritional assessment

Total feed intake and body weight changes were assessed, while feed efficiency ratio was calculated for each group of rats after 12 weeks.

Feed efficiency ratio= weight gain/ Total feed intake.

Relative weight of organs = (Weight of organ / Rat's body weight) X 100

2.8 Biochemical analyses

Determination of total cholesterol, HDL-Cholesterol, triglycerides, total antioxidant capacity, urea, creatinine, albumin, total protein, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP) in the serum of the investigated rats were carried out according to the kits protocols. Total lipids were extracted from liver homogenate according to Bligh and Dyer [27] and then liver cholesterol was measured according to the kit protocol. LDL-Cholesterol and VLDL-Cholesterol concentrations (mg/dl) were calculated according to the following equations of Friedwald et al [28].

LDL-Cholesterol = [Total Cholesterol] - [HDL-Cholesterol] - ([TG]/5)

VLDL-Cholesterol = ([TG]/5)

Globulin concentration was calculated from the following equation

Globulin = total protein – albumin.

2.9 Histopathological examination

Suitable sections of livers and hearts were fixed in 10% formalin and processed for preparation of 5µm-thick paraffin sections. These sections were sequentially stained with hematoxylin and Eosin and examined for any deformation in its normal structure [29].

2.10 Statistical analysis

Results were reported as mean with their standard errors. The data were analyzed by one-way ANOVA; statistically significant effects were further analyzed and group's means were compared using Duncan's multiple range test. Statistical significance was determined at (p< 0.05). Statistical analyses were done with the Costat Statistics software version 6.4 (CoHort software, California, USA).

III. Results

Results in Table 2 show that corn oil contains 86.4% of unsaturated fatty acids. Linoleic acid represents 66.08% of the total unsaturated fatty acids in the corn oil. The same results demonstrate that PKO, PO and ghee contain 78.4%, 49.9% and 64.73% saturated fatty acids, respectively. Lauric acid represents 61.2% of the total saturated fatty acids in the PKO. Palmitic acid represents 88.7% and 50.05% of the total saturated fatty acids in the PO and ghee, respectively.

Results in Table 3 indicate that increasing corn oil level in the diet from 10% to 20% was not accompanied by a significant increase in feed efficiency ratio in rats. Feed efficiency ratio of rats fed on diets containing 20% PO, PKO or ghee for 12 weeks was significantly higher than that of rats fed on basal diet containing 10% corn oil. Increasing the fat level in the diet from 10% to 20% was accompanied by a significant increase in the relative weight of liver of the experimental rats regardless of the fat type used. Meanwhile, relative weight of kidney of rats fed on HFD containing corn oil or PKO was significantly lower than that of rats fed on basal diet. Results in the same Table 3 illustrate that there was no significant difference in the relative weight of kidneys of rats that were fed on HFD regardless of the fat type used. On the other hand, feeding rats on HFD for 12 weeks did not significantly affect the relative weight of the heart, spleen and testis of the experimental rats regardless of the fat source used.

Results in Table 4 show that feeding rats on HFD containing PO or PKO and 1% cholesterol increased significantly serum triglycerides, VLDL-C and total cholesterol levels compared with those of rats that were fed on basal diet or HFD containing corn oil or ghee plus the same level of cholesterol. Results in Table 4 show that increasing the fat level in the diet to 20% was accompanied by a significant decrease in HDL-Cholesterol of the experimental rats regardless of the fat source.

Results indicate that increasing corn oil level in the diet from 10% to 20% was accompanied by a significant increase in LDL-Cholesterol of the experimental rats. LDL-Cholesterol level of rats that were fed on HFD containing ghee was significantly higher than that of rats that were fed on HFD containing corn oil. The highest LDL-Cholesterol level was recorded in rats that were fed on HFD containing PO or PKO. Feeding rats on HFD, in general, increased significantly cholesterol in their livers regardless of the fat type. Results show that increasing corn oil level in the diet from 10% to 20% was not accompanied by a significant increase in the TC/HDL ratio. Replacing corn oil by ghee in HFD did not cause a significant increase in the TC/HDL ratio. Results in the same Table 4 reveal that the highest TC/HDL and LDL/HDL ratios were recorded for the rats that were fed on HFD containing PKO. Results in the same Table 4 indicate that increasing the fat level in the diet from 10% corn oil to 20% (HFD) from corn oil, PO, PKO or ghee did not affect significantly the serum total antioxidant capacity except in the PKO group that had the lowest value.

Results in Table 5 show that increasing the fat level in the diet from 10% to 20% did not affect significantly serum albumin, globulin, total protein, A/G ratio and urea of the experimental rats regardless of the

fat type used. Feeding rats on HFD of corn oil, PKO or ghee caused a significant increase in serum creatinine. Increasing the fat level in the diet from 10% to 20% did not affect significantly serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) of the experimental rats regardless of the fat type used.

In the histopathological examination of rat organs microscopically, normal hepatic parenchyma (Fig.1A) was noticed in the liver specimens of rats from group 1 (fed on basal diet). Meanwhile, cytoplasmic vacuolizations of hepatocytes (Fig.1B) were noticed in liver sections of rats from groups 2 and 5 (fed on HFD containing corn oil and ghee). Fatty degeneration and cytoplasmic vacuolizations of hepatocytes (Figs.1C& 1D) were noticed in the examined sections of rats from groups 3 and 4 (fed on HFD containing PO and PKO).

No histopathological changes were noticed in the heart of rat from groups 1,2, 3 and 5 (fed on basal diet, HFD containing corn oil, PO or ghee) (Fig.2A). Meanwhile, dilatation and congestion of myocardial blood vessels (Fig.2B) were noticed in hearts of rats from group 4 (fed on HFD containing PKO).

IV. Discussion

Present results confirmed previous results by Lai and Ney [30] that feeding rats on HFD containing PO or PKO and 1% cholesterol increased significantly serum triglycerides, VLDL-C and total cholesterol levels compared with rats fed on ghee or corn oil but contradicted with those obtained by Edem [31] who found that the total cholesterol concentration of rats that were fed on 20% corn oil diet or 20% PO diet was similar. LDL-Cholesterol level in rats fed on HFD containing PO or PKO was significantly higher than that of rats fed on HFD containing ghee. The LDL-Cholesterol values reflecting the harmful effect of both PO and PKO. The highest level of hepatic cholesterol was recorded in rats that were fed on HFD containing PKO followed by those fed on PO, corn oil and ghee in a descending order. Present results confirmed previous results by Bolanle and Dais [10] who concluded that dietary intake of PKO could pose a risk for coronary artery disease on a long term basis and also confirmed the Joint FAO/WHO Expert Consultation, [32] which stated that lauric (C12:0), myristic (C14:0) and palmitic acids (C16:0) consumption caused an increase in LDL-Cholesterol whereas stearic (C18:0) had no effect on LDL-Cholesterol and replacing saturated fatty acids (C12:0 – C16:0) with polyunsaturated fatty acids decreased both LDL-Cholesterol concentration and the total cholesterol/HDL-cholesterol ratio. The significantly lower total antioxidant capacity level for rats fed on HFD, compared to rats fed on basal diet, was recorded in rats that were fed on HFD containing PKO which confirmed previous results in this study of the possible health risk of using this oil “PKO” as the major source of fat in the diet. Present results are in accordance with the results obtained by Edem [31] who concluded that feeding rats on 20%PO or corn oil did not affect significantly urea and total protein. Results indicated that feeding rats on HFD from any source increased the alkaline phosphatase significantly in comparison to those fed on basal diet. This increase in alkaline phosphatase reflected the harmful effect of HFD on the liver. However, an increase in alkaline phosphatase when HFD is used for longer periods (more than 12 weeks) could be expected. Present results showed that feeding rats a diet containing 20% ghee caused minimum damage to liver cell compared with feeding rats on diets containing 20% of PO or PKO and the maximum damage in liver cells was noticed in liver cells of rats fed on HFD from PO or PKO. Feeding rats on 20% ghee caused no noticeable changes in heart tissues while dilatation and congestion of myocardial blood vessels were noticed in hearts of rats fed on a diet containing 20% PKO.

V. Conclusion

There are clear biochemical and histopathological evidences that PKO has harmful effects when used as a sole source of fat in a high fat diet.

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Table 1. Composition of basal and high fat diets

Ingredients	Basal diet * g/ 100g diet	High fat diet g/ 100g diet
Casein (85% Protein)	14	14
Corn oil	10	3
Experimental fat (One of the following: Corn oil-PO-PKO-Ghee)	-	17
Salt mixture †	5	5
Vitamin mixture †	1	1
Choline chloride	0.2	0.2
Cholesterol	-	1
Cellulose	2	2
Sucrose	33.4	28.4
Corn starch	33.4	28.4

PO, palm oil; PKO, palm kernel oil.

* Basal diet was prepared according to AOAC (2000) [27].

† Vitamin and Salt mixtures were prepared according to AOAC (2000) [27].

Table 2. Fatty acids composition of the investigated oils and fats

Fatty acids %	Corn oil	Ghee	Palm oil	Palm kernel oil
Capric acid 10:0	----	3.56	---	3.4
Lauric 12:0	----	3.92	---	48.2
Myristic 14:0	----	12.99	1.0	16.2
Palmitic 16:0	11.4	32.4	44.3	8.4
Stearic 18:0	2	11.86	4.6	2.5
Oleic 18:1	28	29	38.7	15.3
Linoleic 18:2	57.1	2.03	10.5	2.3
Linolenic 18:3	1.3	---	----	---
Other/unknown	0.2	4.24	0.9	3.7
Total saturated	13.4	64.73	49.9	78.7
Total unsaturated	86.4	31.03	49.2	17.6

Table 3. Feed efficiency ratio (FER), relative weight (g/100g body wt) of liver, heart, kidney, spleen and testis of rats fed on basal diet or high fat diets for 12 weeks

parameter	n	Basal diet		High fat diet (20 % total fat)							
		Corn oil (10% fat)		Corn oil		Palm oil		Palm kernel oil		Ghee	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
FER	6	0.12 ^c	0.01	0.140 ^{bc}	0.008	0.150 ^{ab}	0.01	0.159 ^{ab}	0.009	0.175 ^a	0.004
Relative weights of organs (g/100g body wt)											
Liver	6	2.37 ^b	0.08	3.37 ^a	0.11	3.19 ^a	0.14	3.18 ^a	0.07	3.37 ^a	0.08
Heart	6	0.269 ^a	0.04	0.239 ^a	0.003	0.239 ^a	0.005	0.252 ^a	0.006	0.284 ^a	0.06
Kidney	6	0.532 ^a	0.01	0.454 ^b	0.03	0.48 ^{ab}	0.02	0.461 ^b	0.007	0.471 ^{ab}	0.01
Spleen	6	0.349 ^a	0.06	0.246 ^a	0.02	0.342 ^a	0.01	0.321 ^a	0.01	0.286 ^a	0.02
Testis	6	0.810 ^a	0.09	0.825 ^a	0.07	0.854 ^a	0.04	0.805 ^a	0.03	0.723 ^a	0.04

*The results are represented as the mean values and their standard errors. Mean values within row with unlike superscript letters were significantly different (P<0.05, one-way ANOVA with Duncan's multiple range test).

Table 4. Serum lipid profile, liver cholesterol and total antioxidant capacity of rats fed on basal diet or HFD for 12 weeks

Parameter	n	Basal diet		High fat diet (20 % total fat)							
		Corn oil (10 % fat)		Corn oil		Palm oil		Palm kernel oil		Ghee	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Triglycerides (mg/dl)	6	62.0 ^b	1.87	61.1 ^b	4.16	128.8 ^a	18.6	119.5 ^a	2.53	75.3 ^b	2.93
Total cholesterol (mg/dl)	6	94.1 ^b	3.22	112.2 ^b	6.08	173 ^a	11.7	177.6 ^a	9.55	117.4 ^b	5.3
HDL-C (mg/dl)	5	59.7 ^a	7.28	46.9 ^b	1.16	42.1 ^b	3.75	33.0 ^b	4.24	34.7 ^b	4.87
LDL-C (mg/dl)	5	15.7 ^d	1.78	42.8 ^c	2.10	105.8 ^a	7.02	104.3 ^a	3.84	58.4 ^b	1.25
VLDL-C (mg/dl)	6	12.5 ^b	0.40	12.2 ^b	0.81	25.7 ^a	3.71	23.9 ^a	0.48	15.0 ^b	0.57
Cholesterol in liver	6	7.39 ^c	0.50	12.68 ^c	0.28	15.19 ^b	0.37	16.52 ^a	0.50	11.29 ^d	0.53
TC/HDL ratio	5	1.678 ^d	0.26	2.058 ^{cd}	0.11	3.008 ^b	0.16	3.942 ^a	0.31	2.58 ^{bc}	0.28
LDL/HDL ratio	5	0.516 ^b	0.19	0.818 ^b	0.09	2.061 ^{ab}	0.44	3.346 ^a	1.23	1.38 ^b	0.30
Total antioxidant capacity (mM/L)	6	2.24 ^a	0.05	1.92 ^{ab}	0.13	1.95 ^{ab}	0.11	1.67 ^b	0.19	1.88 ^{ab}	0.15

*The results are represented as the mean values and their standard errors. Mean values within row with unlike superscript letters were significantly different (p<0.05, one-way ANOVA with Duncan's multiple range test).

Table 5. Serum kidney and liver functions of rats fed on basal diet or HFD for 12 weeks

Parameter	n	Basal diet		High fat diet (20 % total fat)							
		Corn oil (10 % fat)		Corn oil		Palm oil		Palm kernel oil		Ghee	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Albumin (g/dl)	6	3.6 ^a	0.12	3.4 ^a	0.08	3.4 ^a	0.24	3.1 ^a	0.16	3.0 ^a	0.20
Globulin (g/dl)	6	2.8 ^a	0.32	2.0 ^a	0.44	2.4 ^a	0.24	2.6 ^a	0.53	3.3 ^a	0.53
Total protein (g/dl)	6	6.4 ^a	0.44	5.4 ^a	0.40	5.8 ^a	0.16	5.7 ^a	0.40	6.3 ^a	0.48
A/G ratio	6	1.41 ^a	0.15	2.25 ^a	0.30	1.66 ^a	0.27	1.37 ^a	0.44	1.22 ^a	0.18
Creatinine (mg/dl)	6	0.46 ^b	0.02	0.60 ^a	0.04	0.53 ^{ab}	0.01	0.58 ^a	0.02	0.61 ^a	0.036
Urea (mg/dl)	6	31.10 ^a	3.02	29.40 ^a	1.95	29.10 ^a	2.89	31.65 ^a	2.24	32.60 ^a	1.26
AST (U/L)*	5	70.0 ^a	8.94	50.66 ^a	1.86	60.33 ^a	4.05	67.0 ^a	3.13	58.6 ^a	4.49
ALT (U/L)*	5	39.4 ^a	5.18	30.4 ^{ab}	3.38	31.8 ^{ab}	3.91	25.74 ^b	4.06	21.8 ^b	2.07
ALP (U/L)*	6	80.8 ^b	10.81	116.8 ^{ab}	7.87	133.6 ^a	18.20	144.6 ^a	9.43	117.6 ^{ab}	5.55

AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase.

* The results are represented as the means and their standard errors. Mean values within row with unlike superscript letters were significantly different (P=0.05, one-way ANOVA with Duncan's multiple range test).

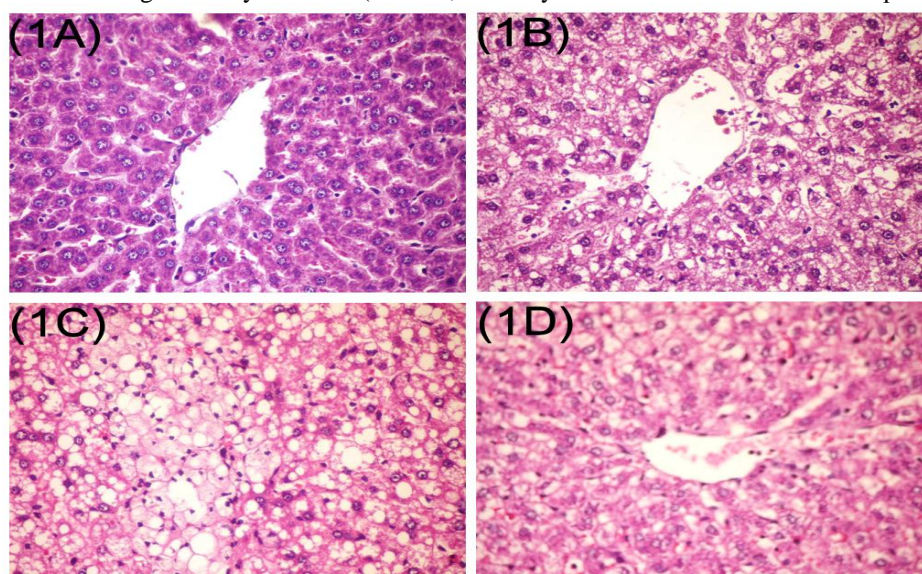


Fig1: photomicrograph of liver cells (hematoxylin and eosin × 400). 1a, liver show normal hepatic parenchyma and no histological changes; 1b, liver show cytoplasmic vacuolization of hepatocytes noticed in rat groups fed on high fat diet containing corn oil or ghee; 1c&1d, liver show cytoplasmic vacuolization and fatty degeneration of hepatocytes noticed in rat groups fed on high fat diet containing palm oil or palm kernel oil.

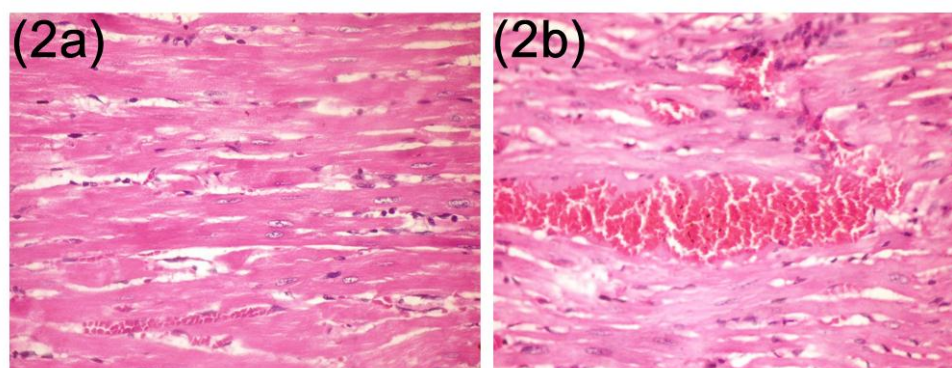


Fig2: photomicrograph of heart cells (hematoxylin and eosin × 400). 2a, heart show no evidence of histopathological changes; 2b, heart show dilatation and congestion of myocardial blood vessels noticed in rat group fed on high fat diet containing palm kernel oil.