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Biological and Histological Evaluations of Palm Oil and its Fractions

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Abstract: Biological and histopathological effects of palm oil, its fractions (palm olein and palm stearin) and two commercial shortening products (C.Sh.1 and C.Sh.2) were evaluated compared to corn oil. For this purpose, five basal diets were prepared containing the five tested oils at 10%, in addition to the control one and fed to six groups of male albino rats for six weeks. Concerning biological evaluation all the studied dietary oils compared to control (corn oil) caused an increase in blood LDL-c and TC and significantly decreased HDL-c over the feeding period of experimental rat groups, thereby increased the TC/HDL-c and LDL-c/HDL-c ratios. Plasma glutamate oxaloacetate transaminase (GOT) and glutamate pyruvate transaminase (GPT) enzymes in rats were generally increased by the tested oil diets compared to corn oil, except palm olein in the plasma GPT. Meanwhile, blood urea and creatnine levels of all groups through the experimental period didn't have any significant differences compared to control group except rats group fed on palm stearin. The microscopically examination of liver and kidney of rats fed on most tested oils showed histopathological alteration.

Key words: Palm oil · Palm olein · Palm stearin · Biological evaluation · Histological examination

INTRODUCTION

Palm oil is the second largest vegetable oil produced in the world. As it is highly saturated and contains about 50% palmitic acid so, palm oil was discredited like saturated animal fats, such as butter, lard and tallow. The allegation that palm oil raises total serum cholesterol, thereby increasing the risk of coronary heart disease (CHD). Recently, studies in animals and in humans indicate that palm oil is quite different from other hypercholesterolaemic fats such as lard or coconut oil [1]. Therefore, the scientific community needs to conduct controlled studies on the effects of palm oil and its relation to cardiovascular disease (CVD) and maintain a responsible perspective when reporting its findings or making recommendations concerning consumption of this oil. Sundram *et al*. [2] studied the effect of dietary palm oil and its fractions on rat blood lipids using semi synthetic purified diet containing 20% fat for 15 weeks. The dietary fats were corn oil, soybean oil, palm oil, palm olein and palm stearin. No significant difference in the body and organs weights of rats fed on the various diets was evident. Plasma cholesterol levels of rats fed soybean oil were significantly lower than those of rats fed on corn oil, palm oil and palm olein or palm stearin. Grundy [3] showed that, saturated fats raise serum TC and LDL-C, also a raise of serum high density lipoprotein cholesterol (HDL-C) was found. On the other hand medium - chain fatty acids with 8-10 carbon atoms and stearic acid (18:0) don't raise serum TC and considered neutral. The other long-chain fatty acids 12:0, 14:0 and 16:0 have been grouped together as cholesterol raising fatty acids. Keys *et al*. [4] attributed similar cholesterol-raising effects to 12:0, 14:0 and 16:0.

The effects of dietary palm oil, hydrogenated canola oil and soy-bean oil on indexes of cardiovascular risk in free-living healthy subjects were compared by Wahle *et al.* [5]. The three test oils did not differ greatly in their overall effect on plasma risk profiles. Beneficial effects were seen with the palm oil diet, such as significantly reduced plasma triglycerides and a tendency toward increased HDL-C. While, Cater *et al.* [6] found that plasma concentrations of TC and LDL-C are significantly higher after a palm oil-rich diet than those obtained with unsaturated edible oils such as high- oleic acid sunflower

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oil. Others have presented favorable results showing that, concentrations of LDL, HDL and very LDL (VLDL) after palm oil-rich diets are comparable to those obtained after ingesting diets rich in sunflower, peanut, corn, olive and soybean oils in normocholesterolemic and hypercholesterolemic subjects [7, 8] and significantly different from those fed on milk- and butter-rich diets. Still others have shown significant decreases in LDL-C and HDL-C in young subjects with normal body mass indexes (BMIs) [9, 10] even after palm oil-rich diets. When there is a damage of smooth, skeletal or heart musculature, serum level of some enzymes such as lactate dehydrogenase (LDH), alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma glutamyl transpeptidase (γ-GT) are elevated. These diagnostic enzymes are valuable tools could use in early detection of muscle wastage as a result of ischemia, injury or inflammation [11]. On the other hand, Cook *et al.* [12] found that, palmitic acid had no effect on serum lipoprotein profiles in human subjects in the presence of recommended intakes of polyunsaturated fatty acids. Clarke *et al.* [13] showed that, palm oil raised plasma cholesterol only when an excess of dietary cholesterol was presented in the diet. Two meta-analyses examined the effect of palmitic acid of (palm oil) on serum cholesterol. Palmitic acid increased the total: HDL cholesterol ratio more than other saturated fatty acids, including lauric acid and myristic acid, which are abundant in palm kernel oil and coconut oil [14]. This finding indicated that, in terms of blood cholesterol, palm oil was somewhat more harmful than the average U.S dietary fat and much more harmful than such liquid oils as olive, soy and canola. World Health Organization [15] has stated that there is "convincing evidence" that palmitic acid increases the risk of cardiovascular disease. A number of pre-1990 human feeding studies reported that palm oil diets resulted in lower serum cholesterol levels than pre-study values. Indeed, scientists concluded that these studies, although not specifically designed to study palm oil, revealed that, a palm oil diet lowered plasma cholesterol compared with the starting periods during which the subjects were eating their habitual Western diets. These conclusions were questioned because the studies were not designed to measure the effects of palm oil [16].

The consumption of palm oil in Egypt has increased rapidly in recent years, but the information about the relation of palm oil to health is very limited. Few papers show that palm oil maintains the normal growth of rats and causes a significant reduction of serum cholesterol in rabbits compared to lard. The reports on palm oil in human studies are difficult to find, therefore, it is necessary to undertake properly controlled studies on the effects of palm oil on blood lipids and on the risk of CVD. So, this work was carried out to investigate the biological and histobathological effects on rats livers and kidneys of palm oil and its fractions olein and stearin, in addition to two commercial shortenings produced by using palm oil in Egypt.

MATERIALS AND METHODS

Materials

Oils: Refined, bleached and deodorized corn oil, palm oil, palm fractions i.e., palm olien, palm stearin, commercial shortening 1 (C.Sh.1) and commercial shortening 2 (C.Sh.2) without antioxidants were obtained from Savola Company for Edible Oils, 10th of Ramadan City, Sharkia Governate, Egypt.

Animals: The animals used in this study were white weaning male albino rats, weighted between 50-80 grams, were obtained from Animal Experimental House of the Res. Institute of Ophthalmology, Giza, Egypt.

Kits: Total cholesterol, High density lipoprotein cholesterol (HDL-c), Glutamate oxaloacetate transaminase (GOT) and Glutamate pyrovate transaminase (GPT) kits were obtained from Randox Laboratories Ltd., Diamond roal Crumlin, Co. Antrim, United, BT294OY.

Biological Experiment: Thirty sex white male albino rats were housed individually in air cages with screen bottoms. The rats were adapted for seven days. They were fed basal diet. The basal diet was formulated according to AOAC [17], it consisted of 15% protein (casein), 65% starch, 10% corn oil, 5% cellulose, 4% mineral mixture and 1% vitamin mixture. The composition of mineral and vitamin mixtures was according to AOAC [18]. The animals were then divided into six groups, each contained 6 rats. The first group was fed on the basal diet containing 10% corn oil and it considered as control. The other five groups were fed diets each contained 10% of the five tested oils. The formulated diets and water were administrated *ad-libitum* separately to the six groups of rats. Feeding was continued for 42 days, during which each rat was weighted at the beginning of experimental period and after 7 days intervals. Blood samples were taken and centrifuged at 3000 rpm for 15 min. and the obtained serum samples were used for the

biochemical measurements (Total cholesterol, HDL-c, LDL-c and GOT, GPT). At the end of experimental period, the internal organs (heart, liver and kidney) were weighted and corresponded to their body weight.

Methods

Blood Analysis

Determination of Plasma Total Cholesterol (TC): Plasma total cholesterol was determined using enzymatic method according to Richmond [19] and Allain *et al.* [20]. The cholesterol was determined after enzymatic hydrolysis and oxidation. The quinonemine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase. The absorbance of sample (A_{sample}) and standard ($A_{standard}$) were measured against the blank at 500 nm using a Shimadzu UV-120-02 spectrophotometer. Total cholesterol was measured and expressed as mg/dl according to the following equation:

$$\text{Total cholesterol in mg/dl} = \frac{A_{Sample}}{A_{Standard}} \times 200$$

Determination of Plasma HDL-Cholesterol: Kits were used in determination of high density lipoprotein (HDL) cholesterol according to the method of Burstein *et al.* [21] and Lopez-Virella *et al.* [22]. Phosphotungstic acid and magnesium ions selectively precipitating all lipoproteins except the HDL-cholesterol fraction. The cholesterol present in the supernatant was determined by the same method used for total cholesterol as shown previously. HDL-cholesterol was measured and expressed as mg/dl according to the following equation:

$$\text{HDL- cholesterol in mg/dl} = \frac{A_{Sample}}{A_{Standard}} \times 55$$

Determination of Plasma LDL-Cholesterol: Serum LDL-cholesterol was calculated from serum triglyceride, total cholesterol and HDL- cholesterol levels using the Friedewald formula [23] according to the following equation:

LDL- cholesterol = Total cholesterol - HDL-cholesterol - TG/5

Determination of Glutamate Oxaloacetate Transaminase (GOT) and Glutamate Pyruvate Transaminase (GPT) Activities: Serum GOT and GPT also known as asparatate aminotransferase (AST) and alanine aminotransferase (ALT), respectively were determined according to the method described by Reitman and Frankel [24]. The activities of GOT and GPT were calculated and expressed as numbers of international units per liter using standard curve.

Determination of Urea: Urea was determined according to the method of Fawcet and Scott [25]. Urea reacts with water in the presence of urease forming ammonia plus carbon dioxide according to the following equation:

$$\text{Urea} + H_2O \xrightarrow{\text{Urease}} 2\,NH_3 + CO_2$$

The ammonium ions produced were measured by Berthelot reaction. The blue dye formed indophenol produced in Berthelot reaction absorbs the light between 530 and 560 nm proportional to initial urea concentration. Reagents obtained from Biodiagnostic were used to determination of urea. The absorbance of sample (A_{sample}) and standard ($A_{standard}$) was measured against the blank at 550 nm using a Shimadzu UV-120-02 spectrophotometer. Urea was calculated and expressed as mg/dl according to the following equation:

$$\text{Urea in mg/dl} = \frac{A_{Sample}}{A_{Standard}} \times \text{Standard Cone.}$$

Determination of Creatinine: Determination of creatinine was performed using Biodiagnostic Kits according to the method of Schirmeister *et al.* [26]. Creatinine forms a colored complex with picrate in alkaline solution. The amount of the complex formed is directly proportional to the creatinine concentration. The absorbance of sample (A sample) and standard (A standard) was measured against the blank at 520 nm using a Shimadzu UV-120-02 spectrophotometer. Creatinine was calculated in mg/dl according to the following equation:

$$\text{Creatinine in mg/dl} = \frac{A_{Sample}}{A_{Standard}} \times 2$$

Histological Examination: Autopsy samples were taken from the liver and kidney of rats in different groups and fixed in 10% formol saline for twenty four hours. Washing was done in tap water then serial dilutions of alcohol (methyl, ethyl and absolute ethyl) were used for dehydration. Specimens were cleared in xylene embedded in paraffin at 56°C in hot air oven for twenty four hours. Paraffin bees wax tissue blocks were prepared for sectioning at 4 microns thickness by slidge microtome. The obtained tissue sections were collected on glass slides, deparaffinized and stained by hematoxylin and eosin stains [27] for histopathological examinations through the light microscope.

Statistical Analysis: The obtained data were exposed to Statistical Analysis User's Guide by using SAS computer program [28]. Duncan's multiple range test at 5% level of significance was used for comparison between means.

RESULTS AND DISCUSSION

Growth Performance: The results of the mean body weight (BW) and calculated gain in body weight for rats in both the control and experimental groups fed diets containing 10% of each of the tested oils and corn oil as a control, for 6 weeks are presented in Table 1. The data showed a gradual increase in body weight of all the animals with advancing the experimental period. It was cleared that the growth performance of rats was not significantly ($p<0.05$) affected by the dietary oils till five weeks. However, after 6 weeks feeding on diet containing 10% C. Sh.1 lead to significant ($p<0.05$) increase in the final body weight BWG of animals compared to those of rats fed on palm oil and control diets. Animals fed on 10% C. Sh.2 diet, were in the second order followed by rat groups fed on palm olein, palm stearin then palm oil and control rats fed on corn oil diet.

The highest growth performance in rats group fed on C. Sh.1 may be due to the high aroma and tasty of this product, therefore they eat this diet greedily with excessive desire for eating which lead to an alteration of lipid metabolism in the liver and caused its accumulation and more weight gain. The insignificant difference in body weight of rats fed on palm oil, palm olein and palm stearin was also evident by Sundram *et al.* [2]. These results are in agreement with those obtained by Karaji-Bani *et al.* [29].

Internal Organs: The results in Table 2 show the average of internal organs weights and their relative values, expressed on body weight basis of rats fed for six weeks on diets containing 10% of palm oil, palm olein, palm stearin, C. Sh.1and C. Sh.2 compared to those of rats fed on control corn oil diet. Data showed that all rats in the experimental groups fed on any diet had statistically ($p<0.05$) the same mean weights of their livers and kidneys, as well as their relative weights to rats body weights being similar to those of rats group fed on control corn oil diet, except the relative kidney weight of rats fed on palm stearin diet was significantly ($p<0.05$) lesser than that of control rats. However, the mean weights of animal hearts were significantly ($p<0.05$) equal among all rat groups and not statistically differed from that of control rats except hearts weight of animals fed on palm olein and C. Sh.1 showed a significant increases compared to those of rats fed on palm oil diet. While, no significant differences were observed in hearts relative weights among all the tested rat groups and control rats.

Blood Biochemistry: Data presented in Table 3 revealed that plasma TC levels at the beginning of the experiment had an equal values ranging between 79.54 and 79.81 mg/dl. After 3 weeks of feeding, the TC concentration was elevated in the plasma of all the treated groups. At this period, rat groups fed on diets containing palm stearin exhibited significantly ($p<0.05$) the highest serum TC level among all other treated groups and the control one, except rats group fed on palm oil, their serum TC was statistically equal. On the other hand, the plasma TC level of rats fed on palm olein diet showed the lowest increase value being more close to that of control rats. However, rats fed on C.Sh.1 and C.Sh.2 diets showed middling increase in serum TC. All the obtained values after 3 weeks of feeding fall in the normal range levels in rats serum that ranged from 42 to 90 mg/dl [30], except the level of plasma TC of rats fed on palm stearin diet (92.07 mg/dl). After 6 weeks, this level stepping up being 102.39 mg/dl, in addition all the other tested rat groups, their plasma TC levels were also grow being over plus the normal range, except in rats serum fed on palm olein which was in the normal range. The final levels of plasma TC were higher than the start levels descendingly by 23.85, 19.15, 13.73, 12.00 and 9.21% in rats fed on palm stearin, palm oil, C.Sh.1, C.Sh.2 and palm olein diets, respectively compared to 1.74% in control rats group. These increases in plasma cholesterol have been shown to be a major risk factor in the development of atherosclerosis and cardiovascular diseases. It was calculated that a 1% increase in serum TC would increase the risk of death due to CHD by 2% [31]. The great hypercholesterolemic effect of palm stearin was probably due to its higher content of saturated fatty acids which mostly constitute of palmitic acid.

A strong inverse relation between HDL- c and risk of CHD has been advocated. HDL particles controls the lipid metabolism by taking free cholesterol from the peripheral tissue cells, etherifying it and deposit it in the liver for catabolism and from which a portion of cholesterol is put back into circulation and another portion is excreted after conversion to bile acids. This is important to reduce risk for CVD and hypertension. The obtained results illustrated in Table 3 revealed that the levels of plasma HDL-c in animal groups at the beginning were statistically ($p<0.05$) equal being in the range between 41.00 mg/dl and

Table 1: Mean body weight (BW) and body weight gain (BWG) of rats fed for 6 weeks on diets containing 10% of studied oils.

	BW (g) after period in weeks							
Diets containing 10% oils	0	1	2	3	4	5	6	BWG (g)
Corn oil	66.83^{a}	79.50^{a}	86.66^{a}	119.66^{a}	130.83^{a}	138.33^{a}	149.16^{b}	82.33^{c}
Palm oil	69.00^{a}	80.50^{a}	91.16^{a}	132.50^{a}	127.50^{a}	141.16^{a}	152.33^{b}	85.00^{bc}
Palm olien	67.66^{a}	76.16^{a}	84.16^{a}	126.16^{a}	135.83^{a}	146.50^{a}	165.66^{ab}	98.00^{abc}
Palm stearin	67.83^{a}	83.33^{a}	93.33^{a}	129.00^{a}	133.33^{a}	146.33^{a}	164.83^{ab}	95.00^{abc}
C.Sh.1	66.66^{a}	77.50^{a}	87.83^{a}	134.83^{a}	147.50^{a}	158.00^{a}	178.83^{a}	112.16^{a}
C.Sh.2	67.33^{a}	79.33^{a}	95.00^{a}	135.50^{a}	145.00^{a}	154.83^{a}	170.50^{ab}	103.16^{ab}

Means with the same letters in the same column are not significantly different at 5% level

Table 2: Mean internal organs weights (g) and their relative weights (%) of rats fed for 6 weeks on diets containing 10% of studied oils.

		Heart		Kidney		Liver	
Diets containing 10% oils	Final body weight	G	%	g	%	g	%
Corn oil	149.16^{b}	5.83^{a}	3.91^{a}	1.15^{a}	0.76^{a}	0.48^{ab}	0.32^{a}
Palm oil	152.33^{b}	5.58^{a}	3.65^{a}	1.11^{a}	0.72^{ab}	0.41^{b}	0.26^{a}
Palm olien	165.66^{ab}	6.45^{a}	3.90^{a}	1.15^{a}	0.68^{ab}	0.53^{a}	0.32^{a}
Palm stearin	164.83^{ab}	5.86^{a}	3.56^{a}	1.11^{a}	0.67^{b}	0.46^{ab}	0.28^{a}
C. Sh.1	178.83^{a}	6.66^{a}	3.69^{a}	1.31^{a}	0.73^{ab}	0.53^{a}	0.29^{a}
C. Sh.2	170.50^{ab}	6.46^{a}	3.81^{a}	1.26^{a}	0.74^{ab}	0.48^{ab}	0.28^{a}

Means with the same letters in the same column are not significantly different at 5 % level

Table 3: Serum TC (mg/dl), HDL - c (mg/dl) and LDL- c (mg/dl) in rats fed for 6 weeks on diets containing 10% of studied oils

	TC (mg/dl)			HDL-c (mg/dl)			LDL-c (mg/dl)		
	Feeding period (weeks)			Feeding period (weeks)			Feeding period (weeks)		
Diets containing 10% oils	Zero	3	6	Zero	3	6	Zero	3	6
Corn oil	79.71^{a}	80.84^{d}	81.10^{e}	41.25^{a}	40.93^{a}	41.37^{a}	22.18^{a}	23.46^{c}	23.48^{d}
Palm oil	79.69^{a}	88.54^{ab}	98.84^{b}	41.25^{a}	38.55^{a}	35.75^{c}	22.17^{a}	32.77^{ab}	44.93^{a}
Palm olein	79.56^{a}	83.36^{cd}	88.77^{d}	41.50^{a}	39.68^{a}	40.55^{ab}	21.78^{a}	26.93^{bc}	30.94^{c}
Palm stearin	79.54^{a}	92.07^{a}	102.39^{a}	41.00^{a}	35.70^{a}	29.50^{d}	22.24^{a}	38.00^{a}	49.18^{a}
C. Sh.1	79.56^{a}	86.83^{bc}	93.29^{c}	41.25^{a}	38.67^{a}	37.84^{abc}	22.08^{a}	31.20^{ab}	37.11^{b}
C. Sh.2	79.81^{a}	84.98^{bcd}	91.81^{cd}	41.12^{a}	40.10^{a}	36.93^{bc}	22.43^{a}	27.72^{bc}	36.03^{b}

Means with the same letters in the same row are not significantly different at 5 % level.

41.50 mg/dl. It was noticed that, the level of plasma HDL-c of all experimental rats groups except those fed diet contained palm olein showed continuous dropping over the feeding period reaching values ranging between 29.50 and 37.89 after 6 weeks compared to 41.37 mg/dl in control serum rats. In summary, the general picture appear in Table 3 is that all the tested oil diets equally affected plasma HDL-c and that there were an insignificant ($p<0.05$) and slight decreases in serum levels of HDL-c of animals after 3 weeks. In the final of experiment after 6 weeks, the HDL-c levels in serum rats of all the groups significantly ($p<0.05$) decreased compared to that of control rats group except palm olein and C. Sh.1 groups, palm stearin diet caused a considerable and significant ($p<0.05$) drop in HDL-c level in serum rats compared to all the other tested oil diets and the control one.

From the same Table 3 it could be noticed that the calculated plasma LDL-c of rats fed diets contained either 10% of the tested oils or control corn oil diet followed the same trend that did the TC. At the beginning of the experiment, the levels of serum LDL-c were statistically ($p<0.05$) the same. After 3 weeks of feeding, the serum LDL-c concentration raised in all the animals. It could be seen that palm stearin diet frequently raised serum LDL- c, followed by palm oil and C.Sh.1 diets being significantly ($p<0.05$) in the first order. While, C.Sh.2 and palm olein diets were in the second order, they raised serum LDL-c reaching lower levels statistically ($p<0.05$) equal to that in control rats serum. The high level of SFA palmitic in palm stearin and palm oil as well as the commercial shortenings and also the low amount of PUFA linoleic in all tested oil are undoubtedly major factors in

the effectiveness of dietary oils in the more and continuous increasing serum LDL-c after 6 weeks. These increases were by 121.13, 102.66, 68.07, 60.63 and 42.05% for palm stearin, palm oil, C.Sh.1, C.Sh.2 and palm olein, respectively compared to 1.30% only of corn oil. Therefore, these oils had hypercholesterolemic effect and could avoid them in our diets. In this respect, the WHO [15] stated that the palmitic acid consumption contributes to an increased risk of developing of CVD.

Risk Ratios: In the past, the pervious studies revealed that an increase in plasma TC level which is usually due to an increase the level of LDL-c and a decrease in HDL-c have been independently attributed to be associated with increased risk of atheroscelerosis and CHD. However, recent reports indicated that the TC/HDL-c and LDL/HDL ratios are stronger indices of atherogenicity of lipoproteins rather than the lipid profile of the individual lipoprotein fraction [32]. The calculated ratios in Table 4 followed the same trend of those of serum TC and LDL-c during the feeding period of rat groups. At the beginning of the experiment, each of the TC/HDL-c and LDL-c/HDL-c risk ratios was the same for all the animal groups. After 3 weeks, high increases were obtained in both the calculated ratios. With advancing the feeding period, their values were continued in increasing. It was noticed that the highest increase of both TC/HDL-c and LDL-c/HDL-c ratios was in rats group fed on palm stearin diet followed by those fed on palm oil diet then C.Sh.1 and C.Sh.2 diets. While, the risk ratios in rats fed on palm olein were the least values. On the other hand, no change in these ratios was obtained when feeding on corn oil. It was noticed from the calculated ratios that LDL-c/ HDL-c ratio is stronger to differentiate between the potential of oils containing diets in increasing the risk of CVD and atheroscolorsis rather than TC/LDL-c risk ratio.

Liver Enzymes: Aspartate transaminase AST (GOT) and alanine transaminase ALT (GPT) are enzymes catalyze the transfer of an amino group from amino acid to keto acid, one of the important general reactions of protein metabolism; a new amino and keto acids are formed in the process. These two transaminases are of clinical interests that reflect liver function. They occur in most of organs and tissues; the liver is very rich in these two enzymes. Serum transaminases levels in normal subjects are low, but after extensive tissues destruction, particularly in liver, these enzymes are liporated into the serum. Thus, their appearance in serum is a marker to tissue damage. They are found in the serum at very high levels during liver infection with hepatocytes (liver cirrhosis), hemorrhage and inactive hepatitis. From Table 5 the average levels of serum GOT in rats fed on the dietary tested oils at the beginning of the experiment ranged between 33.26 to 33.55 U/L compared to that in serum control rats fed on corn oil (33.48 U/L). It was clearly noticed that feeding on the tested oils containing diets for 3 weeks caused significant ($p<0.05$) increases in levels of the GOT enzyme in plasma of the treated animals reaching an average levels ranging between 42.37and 44.89 U/L compared to 37.40 u/L in rats serum fed control corn oil diet. It could be noticed that palm stearin diet showed the highest serum GOT concentration followed by C.Sh.1 and C.Sh.2 then palm oil diets. While, palm olein diet exhibited significantly ($p<0.05$) the least serum GOT level. After 6 weeks, the level of serum GOT of rat groups fed on the studied oils showed a continuous increase. It was noticed that rats group fed on C.Sh.2 diet take the first order in increasing the level of GOT enzyme followed by palm stearin, C.Sh.1 then palm oil diets, whereas palm olein diet was the least one. These increases were by 52.88, 46.42, 40.13, 35.02 and 23.53% from the baseline levels of the above respective value compared to 12.84% of GOT increase in control serum rats. The levels of serum GPT of rat groups fed on the studied oil (Table 5) showed an increases from a range between 23.15 and 23.28 u/L at the onset of the experiment, reaching statistically ($p<0.05$) an equal values compared to GPT level (24.33 u/L) in serum rats fed on corn oil diet except rat groups fed on palm stearin and C.Sh.2 diets which caused significantly higher levels in serum rats GPT. At the same time, all the tested dietary oils showed an equal effect on raising plasma GPT, except palm stearin diet which significantly ($p<0.05$) exhibited the highest level. Thereafter, feeding on the rested dietary oils for 6 weeks caused continuous increases in levels of GPT enzyme in serum rats. Palm stearin and C.Sh.2 diets were significantly ($p<0.05$) raised the level of plasma GPT in rats. Whereas, C.Sh.1 and palm oil diets showed an ordinary effect. In conclusion, the obtained results showed that there was an adverse effect of all the studied oil diets may account for the high levels of GOT and GPT enzymes monitored in this study, because all the obtained values of either GOT or GPT enzymes over all the feeding periods were elevated in rats plasma indicating disfunction or distraction in liver tissues which caused these enzymes to leak from injured cells.

Table 4: TC/HDL-c and LDL-c/HDL-c Risk ratios in rats fed for 6 weeks on diets containing 10% of studied oils

	Risk ratios in rats fed on diets containing 10% oils											
	Corn		Palm oil		Palm olein		Palm stearin		C. Sh.1		C. Sh.2	
Period (weeks)	A	B	A	B	A	B	A	B	A	B	A	B
0	1.93	0.53	1.93	0.53	1.91	0.52	1.94	0.54	1.92	0.53	1.94	0.54
3	1.97	0.57	2.29	0.85	2.10	0.67	2.57	1.06	2.24	0.80	2.11	0.69
6	1.96	0.56	2.76	1.25	2.18	0.76	3.47	1.66	2.46	0.98	2.48	0.97

A: TC/HDL-c - B: LDL-c/HDL-c oil

Table 5: Serum glutamate-oxaloacetate transaminase (GOT) and glutamate-pyrovate transaminase (GPT) enzymes (u/L) in rats fed for 6 weeks on diets containing 10% of studied oils

	GOT enzyme (u/L)			GPT enzyme (u/L)		
	Feeding period (weeks)			Feeding period (weeks)		
Diets containing 10% oils	Zero	3	6	Zero	3	6
Corn oil	33.48^{a}	37.40^{d}	37.78^{f}	23.28^{a}	24.33^{c}	24.96^{d}
Palm oil	33.44^{a}	42.37^{bc}	45.15^{d}	23.28^{a}	25.18^{bc}	30.51^{c}
Palm olein	33.45^{a}	40.76^{c}	41.32^{e}	23.27^{a}	25.16^{bc}	27.20^{d}
Palm stearin	33.26^{a}	44.89^{a}	48.70^{b}	23.32^{a}	29.65^{a}	34.89^{a}
C. Sh.1	33.47^{a}	43.23^{ab}	46.90^{c}	23.15^{a}	25.54^{bc}	31.49^{bc}
C. Sh.2	33.55^{a}	43.40^{ab}	51.29^{a}	23.15^{a}	26.33^{b}	34.36^{ab}

Means with the same letters in the same row are not significantly different at 5 % level

Table 6: Serum urea and creatinine (mg/dl) of rats fed for 6 weeks on diets containing 10% of studied oils

	Serum urea (mg/dl)			Creatinine (mg/dl)		
	Feeding period (weeks)			Feeding period (weeks)		
Diets containing 10% oils	Zero	3	6	Zero	3	6
Corn oil	40.76^{a}	40.21^{b}	40.91^{c}	0.38^{a}	0.39^{b}	0.39^{b}
Palm oil	40.30^{a}	41.91^{ab}	41.91^{bc}	0.36^{a}	0.40^{b}	0.40^{b}
Palm olein	41.15^{a}	39.88^{b}	41.76^{c}	0.38^{a}	0.39^{b}	0.39^{b}
Palm stearin	41.38^{a}	41.94^{ab}	44.13^{a}	0.39^{a}	0.47^{a}	0.46^{a}
C. Sh.1	41.50^{a}	41.16^{ab}	43.58^{ab}	0.40^{a}	0.41^{b}	0.40^{b}
C. Sh.2	41.53^{a}	43.09^{a}	42.50^{abc}	0.40^{a}	0.43^{b}	0.40^{b}

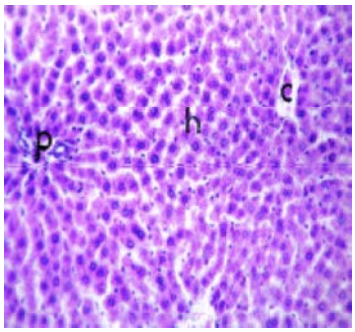
Means with the same letters in the same row are not significantly different at 5% level

Kidney Functions: From the obtained data in Table 6, a slight increases and decreases were observed in serum urea levels in rat groups after 3 weeks. It could be noticed that at the third week, there was no significant ($p>0.05$) differences between all the obtained mean values of urea concentration in the serum of all rat groups except that of rats fed on C.Sh.2 diet which exhibited significantly ($p<0.05$) the highest mean value of serum urea concentration. After 6 weeks the urea levels of rats fed on palm olein, palm stearin and C.Sh.1 diets were rarely stepped up. While feeding on C.Sh.2 caused slightly decrease in urea level from 43.09 mg/dl at the third week to 42.50 mg/dl. On the other hand, the concentrations of urea in rats serum fed on palm oil and the control diet were immutable. It could noticed that all the treated rats groups as well as the control rats had no significant ($p>0.05$) differences in urea levels in their plasma except rat groups fed on palm stearin and C.Sh.1 diet significantly raised urea levels compared to other groups. The same Table 6 show slight increases in serum creatinine levels in all treated rat groups after 3 weeks. It was noticed that palm stearin diet caused a significant ($p<0.05$) increase in serum creatinine level, while, all the other dietary oils and the control showed statistically ($p<0.05$) an equal levels. After 6 weeks the creatinine level in serum rats fed on all the experimental diets fall in the range of the baseline levels indicating no significant ($p>0.05$) effect on creatinine level, one exception was observed when the rats fed on palm stearin diet which caused a significant ($p<0.05$) more raising effect on creatinine.

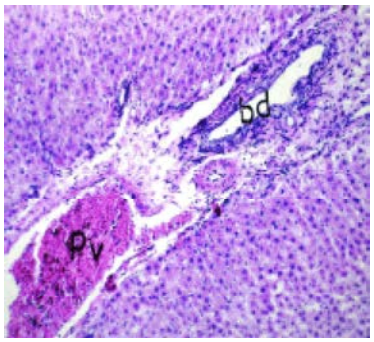
Histopathological Examination: The effect of the tested experimental dietary oils on the histopathology of male albino rats liver and kidney were examined after 6 weeks of feeding and the sections are shown in Fig. 1-12.

Histology of Liver: The section in liver of control rats group fed on corn oil diet for six weeks (Micrograph 1) showed a normal histological structure of the central vein, portal area and the surrounding hepatocytes. However, feeding on palm olein dietary oil caused a dilatation and congestion of the central vein (Micrograph 2), also, a dilatation and congestion of the portal vein with distention bile duct was noticed, while a normal structure of the surrounding hepatocytes were observed in rats liver. Concerning the liver of rats group fed on palm oil, the sections in Micrograph 3 showed a few fatty changes in hepatocytes with inflammatory cells infiltration in between the hepatocytes; in addition of fibrosis with inflammatory cells infiltration in the portal area. As shown in Micrograph 4, the section in liver of rat fed on palm stearin diet showed a more dilatation of the central vein (Fig. 4a) and more fatty change in diffuse manner in most of the hepatocytes with inflammatory cells infiltration in between the hepatocytes. Among the liver of rats fed on C.Sh.1 dietary oil, an inflammatory cells were aggregated in the portal area was happened as shown in Micrograph 5. It was also noticed a fatty change in few hepatocytes with kupffer cells proliferation in between the hepatocytes. However, the sections in lever of rats fed on C.Sh.2 caused fibrosis inflammatory cells and infiltration in the portal area with fatty change in surrounding hepatocytes (Micrograph 6).

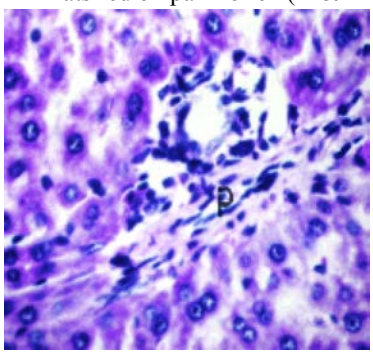
Histology of Kidney: The histopathological study indicated a normal kidney tissues of rat groups fed on control corn oil and palm olien diets as shown in Micrograph 7 and Micrograph 8, respectively, which proved a normal histological structure of the glomerali and tubules. While, feeding the rats on palm oil diet caused a mild cystic dilatation in the tubules in corticomedullary junction (Micrograph 9). Concerning the kidney of rats fed on palm stearin diet, Micrograph 10 showed a proliferation of the endothelial cells lining the glomerular tuft of the glomeruli. However, feeding the rats on C.Sh.1 dietary oil, the kidney sections (Micrograph 11) showed degeneration in the epithelial cells lining the tubules. On the other hand, feeding on C.Sh.2 diet caused a cystic dilatation in the tubules of corticomedullary junction (Micrograph 12).



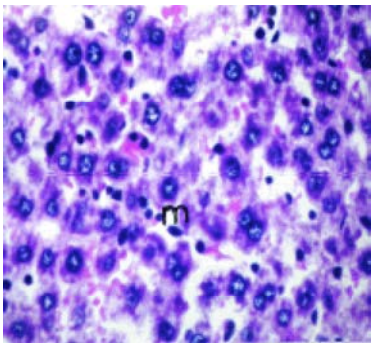
Micrograph (1): Photomicrography section in the Liver of rat fed on corn oil (control sample). (H & E, 40X)



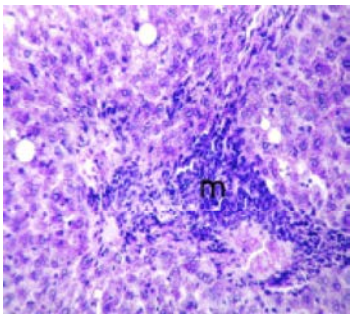
Micrograph (2): Photomicrography section in Liver of rats fed on palm olien (H & E X 40)



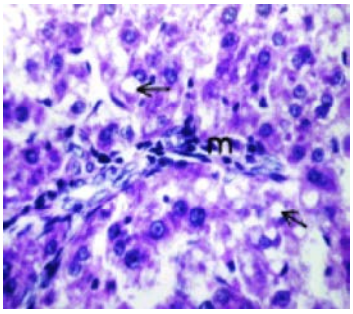
Micrograph (3): Photomicrography section in Liver of rat fed on palm oil (H & E X 160)



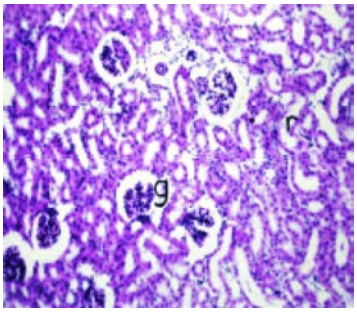
Micrograph (4): Photomicrography section in the Liver of rat fed on palm stearin (H & E X 160)



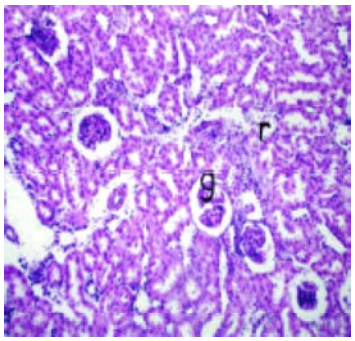
Micrograph (5): Photomicrography section in the Liver of rat fed on Commercial Shortening 1 (H & E X 64)



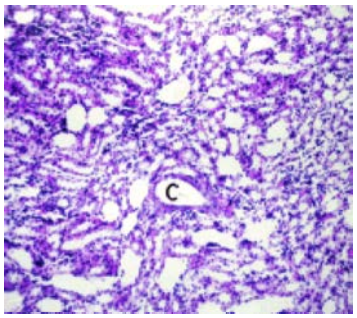
Micrograph (6): Photomicrography section in the Liver of rat fed on Commercial Shortening 2 (H & E X 160)



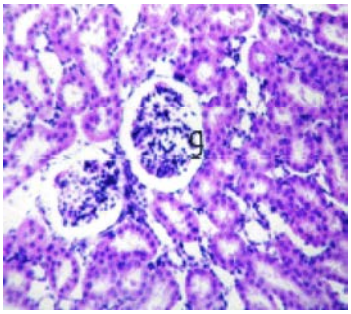
Micrograph (7): Photomicrography section in the Kidney of rat fed on corn oil (control sample). (H & E, 40X)



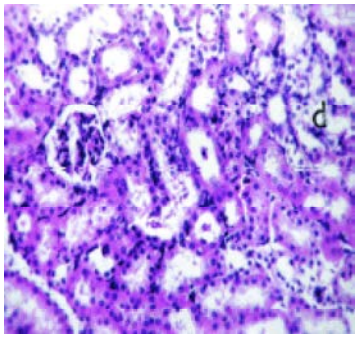
Micrograph (8): Photomicrography section in the Kidney of rats fed on palm olien (H & E X 40)



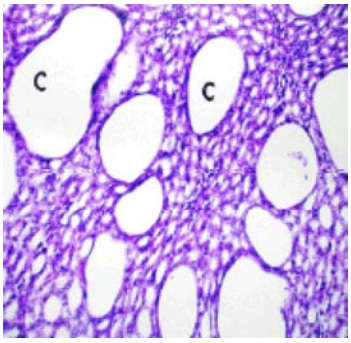
Micrograph (9): Photomicrography section in the Kidney of rat fed on palm oil (H & E X 40)



Micrograph (10): Photomicrography section in the Kidney of rat fed on palm stearin (H & E X 64)



Micrograph (11): Photomicrography section in the Kidney of rat fed on Commercial Shortening 1 (H & E X 64)



Micrograph (12): Photomicrography section in the Kidney of rat fed on Commercial Shortening 2 (H & E X 40)

CONCLUSION

The obtained results conformed that all the studied oils compared to corn oil as control significantly ($p<0.05$) increased LDL-c and TC and decreased HDL-c over the feeding period, thereby increasing the TC/HDL-c and LDL-c/HDL-c risk ratios, where positively associated to the development of CHD. It appeared that palm oil, palm stearin and commercial products could be harmful to health.

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