J. Food and Dairy Sci., Mansoura Univ., Vol. 7 (2): 81 -96, 2016

EFFECT OF DEEP-FAT FRYING ON CANOLA OIL, PALMOLEIN AND SUNFLOWER OIL BLENDS: B) BIOLOGICALAND NUTRITIONAL STUDIES

EL-Reffaei, W.H.M.; A.S. EL-Sebeay; Hanan M.A. EL-Ghandour; Eman M. Ragheb and S.E.A. Badr

Regional Center for Food and Feed (RCFF), Agric. Res. Center, Giza, Egypt

ABSTRACT

Canola oil (CO) is very high in healthy unsaturated fatty acids. It is higher in the omega-3 fatty acid alpha-linolenic acid than any of other oil except flaxseed oil. Because of its light flavor, high smoke point and smooth texture, canola oil is one of the most versatile cooking oils. Oil blending is one way of the most suitable technology to overcome the problem of poor stability of canola oil. It was blend with sunflower oil (SF)and palm olein (PO) to evaluate technological and nutritional qualities. This study was conducted to assess the effects of long-term intake of different fresh edible canola oils, their blends and those frying oils, selected due to their common use in human nutrition. Sixteen groups of (50-70g) albino male rats (n = 5), were fed for 90 days on different fresh and frying oils which drown from different frying cycles of potato chips up to 30th frying. The experiment period was divided into two durations 45- days for each. Blood serum analysis for rat groups were showed; a significant increases in liver, kidney and heart functions consider to 1st experimental duration as compared with the control group which fed on corn oil, While these functions were non-significant difference in the 2nd experimental duration. Serum lipid profile and Atherogenic Index (AI) were recorded a significant different among all groups as compared with the control group (G1) during 1stexperimental duration. However, HDL-C was significant decreased in the 1st duration than that in the 2nd. Otherwise, canola oil in the diets could be increase the HDL level than that in rats group fed on corn oil. A distinct lowering in TG level was recorded among rat groups fed on diets of CO+PO (G12:G16) in the 1st duration. Internal antioxidant of serum coenzyme Q10 was found to decline among later groups fed on frying oils 30th (G6, G11 and G16) ranges < 70mmol/ml.

A blood lymphocyte percentage recorded diminution at 8% among groups which fed on fresh CO + PO (G12) and used frying CO + PO (G13, G14 and G16) oils than other groups as well as the control group G1 through 1st duration. Histological examination for liver, kidney, heart, intestinal and testes tissue of rats were investigated. Most of groups exhibited different histological alteration. these alerts were sever along the later groups fed on 20th and 30th frying cycles oil. Depend on this study, canola oil and palm olein in blend can be used to improve the health status of such fried foods and their oils up to 10th frying cycles.

Keywords: canola oil, frying, sunflower oil, palm olein, histological, lymphocyte, lipid profile, AtherogenicIndex, liver function, coenzyme Q10.

INTRODUCTION

Deep-frying is one of the most popular cooking methods worldwide both for industrial and domestic food preparation procedures (Casal*et al*., 2010). Deep-fried foods, such as potato chips are very popular among Egyptian. In the last 10 years, increase the Junk fried food in Egypt, the percentage of calories ingested from oil and fat has exceeded the value recommended for Egyptian and metabolic syndrome has become more common. Volatile secondary products changes the physicochemical properties and leads to the deterioration of foods quality, though by the occasional production of toxic compounds (Ammawath*et al*., 2006), oxidation reactions (Choe and Min, 2007) and generate toxic compounds, such as polymers and polar compounds. In addition, there is an increase of foaming, viscosity, and density of the oils (Velasco, *et al*., 2004), which affect the sensory and nutritional properties of food as well as their safety for consumption. Resulting in an increase of oxidative products in the frying oil, which may be incorporated into the foods and ingested (Casal*et al*., 2010). Formation of oxidative products depends on the oil type, frying temperature, and number of frying cycles. The oil rich in linoleic acid is more easily polymerized during deep fat frying than the oil rich in oleic acid (Bastida and Sanchez-Muniz 2001).

The drawback of a high content of highly unsaturated fatty acids is the low thermal stability at frying temperatures, which reduces the useful life of the oil for deep-fat frying (Gupta, 2005). Also, weakly oxidized fat and oil at levels of only 100 mequiv/kg of PV are neurotoxic (Gotoh and Wada, 2006).

Considerable efforts in nutrition research and conforming nutritional marketing have firmly established in the minds of health professionals and consumers the knowledge about the nutritional benefits of rapeseed oil.Several researchers have investigated the possibility of using canola oil blended with other vegetable oils (Farhoosh *et al*., 2009).To overcome the problem of poor stability of traditional soybean, sunflower and rapeseed oils, ways of reducing the unstable polyunsaturated fatty acid content were sought (Mariod, *et al*., 2005).

Blend to enhance the stability during the frying process included the use of virgin olive oil or oils with a modified fatty acid composition, such as high oleic and mid-oleic sunflower and high oleic and low-linolenic canola oils (Warner and Gehring, 2009). Despite the potentially harmful effects of deep-fatfrying, few studies have focused on the biological consequences of frying oil consumption. In animal models, it has been shown that consumption of frying oil increases peroxidation in all lipoproteins (Garrido-Polonio*et al*., 2004).

Many different oils can be used for frying, like canola oil, palm oil, soya oil and sunflower oil (Naghshineh*et al*., 2009). One of the most important oils in oil industry is canola oil that after the soybean oil has the highest word production (Przybylski*et al*., 2002). Research indicates that the fatty acid composition of canola oil is especially favorable in terms of health benefits when used as part of a nutritionally balanced

diet. The canola oil has been known as a rich source of oil with a low content of saturated fatty acids (5-7%) and a high content of polyunsaturated fatty acids with about 7-10% α-linolenic and 17-21% linoleic acids. It is the reform considered as very health omega edible oil (Baux, *et al*., 2008)The other nutritionally favorable property of canola oil is a 2:1 ratio of n-6 and n-3 PUFA. Canola oil is also a rich source of antioxidant vitamin E ; it is very effective in reducing the risk of cardiovascular diseases (Ackman 1990). The nutritional value of frying oils is affected by the loss of essential PUFA. This omega-3: omega-6 ratio has become a model for gauging the proper balance of these fats in oils and the diet (Harris, 2006). Diets with greater than a 1:10 ratio of omega-3 to omega-6 are not recommended, whereas a 1:1 ratio is considered perfect. Very unhealthy ratios of 1:25 and 1:50 are common, especially with regular consumption of 'fast-food', high amounts of fried food, and low intake of fresh whole foods.

Wang, *et al*., (2008) suggested that the low-fat diet might play a role in breast cancer prevention. The monounsaturated *Trans* fats may have driven the discrepant associations between types of fat and breast cancer. Recently, much concern has been on the biological effects of frying oxidized lipids, and there is increasing evidence that they may be detrimental to health, especially in connection with the development of atherosclerosis, liver damage, and promotion of intestinal tumors (Dobarganes and Marquez-Ruiz 2003). Oxidative damage to DNA can result from free radical during thermal frying attack after exposure to ionizing radiation or to agents such as H_2O_2 that can produce active oxygen species. Burenjargal and Totani (2009) reported that, commercial deep-fried products are often made with repeatedly used oil with periodically added fresh oil, similar to the present experimental diet, obesity and organ damage may occur in humans. While, the cytotoxic low-molecular-weight compounds bound to gluten from deep-fryingwere ingested in experimental rats, and thus cause organ damage and rapid body weight increase were observed.

Coenzyme Q10 (COQ10) is a vitamin-like nutrient that has a fundamental role in mitochondrial function, especially as it relates to the production of energy (ATP) and as an antioxidant (Hemmi *et al*.,2005). It has attracted attention because it functions as a mitochondrial antioxidant (Kelso *et al*., 2001), decreasing DNA damage and maintaining genome stability (McCarthy *et al*., 2004), scavenges free radicals directly, inhibits biomolecule oxidation, and affects antioxidant defense *in vivo* (Hargreaves 2003). CoQ10 is located in the membranes of cellular organelles such as peroxisomes, lysosomes and, predominantly, the inner mitochondrial membrane, where it is involved in reactions that are necessary to carry out oxidative phosphorylation *via* the electron transport chain (Crane, 2001). Coenzyme Q10, its reduced form (ubiquinol-10) has also been shown serve as a potent antioxidant, protecting phospholipids from peroxidation (Beyer, *et al*., 1987).Meanwhile, Forsmark*et al*., (1995) suggested that, the endogenous content of coenzyme Q10 might protect membrane proteins and DNA against oxidative damage mediated by lipid peroxidation. The antioxidant properties of CoQ10 allow reduction of free radical-induced oxidative damage of the low-density lipoproteins (LDL) and improvement of the endothelial function of the arteries and then a decrease in the susceptibility of cardiovascular disease (Yalcin, *et al*., 2004). It also takes an important role in the prevention and treatment of heart and cardiovascular diseases, brain and neurodegenerative diseases, etc. (Kumar *et al*., 2009). COQ10diet supplementation with the antioxidant cocktail induced a real increase in the plasma levels of coenzyme Q. Under normal conditions plasma coenzyme Q concentrations are not significantly affected by dietary components such as dairy products, eggs, fish and vegetables. The nutritional status participating did not influence the basal levels of antioxidant nutrients in plasma. (Bhagavan and Chopra 2006).

This study was carried out to assess the effects of long-term intake of different edible canola oils and its blends, selected due to their common use in human nutrition. By using canola oil and its blends with most favorable oils (sunflower and palm olein oils) in deep-fat frying process on potato chips, and their effect on biological and nutritional studies on experimental rats.

MATERIALS AND METHODS

Materials:

Fresh canola oil (CO)blend canola +Sunflower oils (CO+SF) and Canola + Palm olein oil (CO+PO) were obtained from Borg El-Arab Company for Oil Extraction, Alexandria, Egypt and Arma Food Industries, 10th of Ramadan, Egypt .These samples were withdrawn after (zero,1,10, 20 and 30 frying cycles for potatoes chips. The volume of oil was not replenished during the frying process for interment 3 days (10 batch every day). The frying oil from each sample was placed in dark glass bottles and promptly stored in the dark at 5 ˚C until experimental using as described before by author Elreffaei *et al*., (2015).

Chemical parameters

Chemical parameters of canola and its used blend oils in frying process such as iodine index , free fatty acid (FFA) peroxide value (PV) ansidine value (An.V), total polar compounds, fatty acid composition totox value and FT-IR measurements were described before (Elreffaei *et al*., 2015). Fatty acid composition of corn oil was analyzed according to AOCS (2013).

Biological experimental animals

Experimental animals:

Eighty male Albino Wister rats, weighing 50-70 g, were purchased from National Research Center "NRC". Rats were kept in ventilated room under controlled laboratory conditions of normal light –dark cycle (12 h light/dark) and room temperature (23 ± 2°C). The rats had free access to a barely and water for one week before starting the experiments as accumulation period according to Regional Center for Food and Feed "RCFF" at Agricultural Research Center "ARC" protocol.

Experimental design:

At same previous conditions ,rats were divided into sixteen groups; five in each, the diets administered into groups *ad-libtium* daily were described in Table A for duration 90 days. This period was divided into two intervals (after 45 days (1st period) and 90 days (2nd period)).

Table (A):Composition of the experimental diets (%)

Diet components	G1	G2	G3	G4	G5	G6	G7	G8	G9	G10	G11	G12	G13	G14	G15	G16
Casein	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20
Sucrose	45	45	45	45	45	45	45	45	45	45	45	45	45	45	45	45
Starch	15	15	15	15	15	15	15	15	15	15	15	15	15	15	15	15
Oil	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10
Cellulose	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
Mineral mix	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5
Vitamin mix	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
DL-Methionine	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Choline citrate	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2

G1: diet containing corn oil (normal control oil).
G2: diet containing canola oil fresh.
G3: diet containing canola 1 st frying oil.
G4: diet containing canola 10th frying oil,
G5: diet containing canola 20th frying oil,
G6: diet containing canola 30 th frying oil,
G7: diet containing canola (CO) + sunflower oil fresh oil (SF).
G8: containing canola (CO) + sunflower oil (SF) 1 st frying oil.
G9: diet containing canola (CO) + sunflower oil (SF) 10th frying oil.
G10: diet containing canola (CO) + sunflower oil (SF) 20th frying oil.
G11: diet containing canola (CO) + sunflower oil (SF) 30 th frying oil.
G12: diet containing canola (CO) + palm olein fresh oil (PO).
G13: diet containing canola (CO) + palm olein oil (PO) 1 st frying oil.
G14: diet containing canola (CO) + palm olein oil (PO) 10th frying oil.
G15: diet containing canola (CO) + palm olein oil (PO) 20th frying oil.
G16: diet containing canola (CO) + palm olein oil (PO) 30 th frying oil.

Blood samples:

The blood samples were collected *via* the retro-orbital plexus (Saka *et al*., 2012). One part of the blood was collected on EDTA tubes for hematological analysis, while another part of blood was submitted to centrifugation at 3500 rpm for 15 min to obtain serum to remove the plasma and Buffy coat (consisting of leukocytes and platelets) as reported by Akhigbe *et al*., (2008).

Biochemical parameters:

Serum analysis

The blood serum samples were used to quantify some biochemical parameters:-

Liver Functions: Glutamic oxaloacetic transaminase (GOT) and Glutamic pyruvic transaminase (GPT)were determined according to Henry (1974) method as liver functions of subjected rat groups for the two interval periods of the experiment.

Table (B): Most of total fatty acids Composition of feeding oil during 1 st and 2 nd period*

treatments	Fatty acids					
	ΣSFA	Σ MUSFA	ΣPUFA	Σ ω-6	Σ ω -3	ω 3/ ω-6
Corn oil	12.3±0.02	27.1±0.08	59.7±0.02	58.9±0.12	0.8±0.03	0.01±0.00
CO	6.4±0.01^{b}	63.9±0.00^{c}	29.1±0.00^{a}	21.1±0.00^{a}	6.2±0.01^{a}	0.29±0.00^{a}
CO F1	6.6±0.01^{ab}	64.6±0.00^{ab}	28.2±0.00 ^{b}	20.8±0.01^{b}	5.9±0.00^{b}	0.28±0.00^{bc}
CO F10	6.6±0.01^{ab}	64.8 ±0.00^{ab}	28.0±0.00^{c}	20.7±0.00 ^{c}	5.8±0.00^{b}	0.28±0.00^{b}
CO F20	6.8± 0.20^{a}	64.2± 0.33^{c}	27.98±0.02^{c}	20.8±0.02^{b}	5.7± 0.02^{c}	0.27±0.00^{d}
CO F30	6.6±0.04^{ab}	65.1±0.13^{a}	27.7±0.08^{d}	20.5±0.00^{d}	5.7±0.05^{c}	0.28±0.00^{c}
CO+SF	8.6±0.10^{a}	47.8±0.02^{d}	43.1±0.04^{a}	39.3±0.03^{a}	3.0±0.05^{c}	0.08±0.00^{c}
CO+SF F1	8.4±0.10 ^{a}	47.9±0.04^{d}	43.2±0.00^{a}	39.4±0.02^{a}	2.97±0.01^{c}	0.07±0.00^{c}
CO+SF F10	8.6±0.00 ^{a}	48.2±0.07^{c}	42.6±0.06^{b}	38.9±0.13^{b}	2.9± 0.03^{d}	0.07±0.00^{c}
CO+SF F20	8.7±0.03 ^{a}	48.8±0.05^{b}	42.2±0.01^{c}	38.3±0.10^{c}	3.2± 0.02^{b}	0.08±0.00^{b}
CO+SF F30	8.7±0.30^{a}	50.5±0.02^{a}	40.4±0.18^{d}	36.2±0.16^{d}	3.5± 0.01^{a}	0.10±0.00^{a}
CO+PO	25.1±0.00^{a}	53.8±0.09^{a}	20.5±0.07^{c}	16.5±0.05^{bc}	3.2±0.02^{a}	0.21±0.00^{b}
CO+PO F1	24.9±0.06^{b}	54.1±0.17^{a}	20.9±0.01^{ab}	16.8±0.00^{b}	3.3±0.01^{a}	0.19 ±0.01^{a}
CO+PO F10	24.5±0.08^{c}	53.8±0.78^{a}	20.8±0.17^{bc}	16.8±0.13^{b}	3.1±0.02^{c}	0.19±0.00^{c}
CO+PO F20	24.4±0.00^{c}	54.2±0.03^{a}	21.3±0.08^{a}	17.1±0.05^{a}	3.3±0.02^{a}	0.19±0.00^{ab}
CO+PO F30	24.9±0.01^{b}	54.1±0.04^{a}	20.9±0.09^{ab}	16.8±0.04^{b}	3.2±0.03^{ab}	0.19±0.00^{ab}

***Reference:Elreffaei, *et al.*, (2015).**

Kidney function: it was identified by creatine analysis in blood serum (Friedman and Young, 1997).

Lipid profile:

Total cholesterol (TC), triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) were evaluated using enzymatic kits (HUMAN kits) according to procedures described by Richmond (1973) and Glick *et al*., (1986). Low-density lipoprotein cholesterol (LDL-C) and VLDL-C were then calculated according to Friedewa l*et al*., (1972) formulas:

VLDL-C = TG/5

LDL-C= TC – (HDL-C + VLDL-C) Calculated Atherogenic lipoproteins according formula described by Ray *et al*.,(2009):

Atherogenic lipoprotein index (AI) = TC/HDL-C ratio

Myocardial infraction: it was analysed by Creatinine kinase (CK) according to the method described by Surtees *et al*., (1979).

Internal antioxidant:

Determination of coenzyme Q10:

The method used was based on that of Grossi*et al*., (1992). Chromatographic analysis was performed in blood serum by using a Shimadzu, Tokyo, Japan, LC 20- equipped with LC-20AD Pump and UV/VIS SHIMADZU spd-m20A operated at 275 nm.

Hematological analysis: Hematological analysis was performed on blood samples using an automated hematology analyzer (model ABC, Vet, user manual RAB 015 A Ind A. Animal blood counter, France). The recorded parameters were hemoglobin, red blood cells (RBC), hematocrit, platelets,white blood cells (WBC), and lymphocytes according toAkhigbe*et al*., (2008).

Histopathological examination

The organs (liver, kidney, heart, intestine and tests) were obtained and placed in well-sealed containers containing 10% formol saline solution for ten hours, at least, then washed in tap water for 12 hours. Serial alcohols(methyl, ethyl and absolute ethyl alcohols) were used for dehydration of the tissue samples. Tissue specimens were cleared in xylene and embedded in paraffin. The paraffin blocks were sectioned at 3 micron thickness by slide microtome .The obtained tissue sections were collected on the glass slides and stained by ematoxylin and eosin stains (Banchroft*et al*., 1996) for histological examination by the light microscope.

Statistical analysis

Data analyses were performed using by of variance (ANOVA) using a Statistical Analyses System SAS version 9.1 (SAS, 2003) under windows. A probability to $P \leq 0.05$ was used to establish the statistical significance. In addition, Duncan post hoc test at 5% probability was used for comparison between mean values.

RESULTS AND DISCUSION

Biochemical parameters:

Data presented in Table 1A As seen in Table 1A and 1B show that rats fed on CO and its blends with SF and PO , used in frying had significantly higher ($P \leq 0.05$) GOT and GPT levels then their fresh counter parts. This was true for both durations of experiments (one and two durations). The GOT of liver in 2nd experimentalperiod (from 60. 7 in group 2 up to 86.67UI/L in group 6) as compared to the group in the 1st experimental period (from 41.50 group 2 up to 54.00UI/L in group 6). Meanwhile, the fed group on used CO+PO had increased in GOT in 1st period of experiment , and extremely reverse hadoccurred in 2nd period in GOT and in GPT along whole experimental period. The GPT was significantly ($P \leq 0.05$) increased among all fed groups on the diet containing unused (fresh) and used (fried) canola oil and its blends as compared to the control group (1) in the2nd experiment period (Table 1A). The GPT concentration among group 12 up to group 16 diet contain unused and used frying CO+PO was significantly lower than other groups, that fed on used canola oil and used CO+SF blend.

Kidney function was determined by creatinine level as illustrated in Table 1A. Creatinine level of the control group was 0.47 and 0.71 mg/dl corresponds to the 1st and 2nd period of studies, respectively. There were no significant changes occurred in the creatinine level among all groups along the first experimental period. Meanwhile, its creatinine level was recorded significantly increase among received groups of fed rats on different used or unused CO and its blends oils, during the 2nd experiment period.

Table (1A): Biochemical blood analysis of rats fed on canola oil and their blends of sunflower or palm olein oils used in deep- fat frying process

Diet treatments groups	Liver function				Kidney function		Myocardial infarction	
	GOT (UI/L)		GPT (UI/L)		Creatinine (mg/dL)		Creatine kinase CK(UI/L)	
	1st period	2nd period	1st period	2nd period	1st period	2nd period	1st period	2nd period
Control G1	40.0±1.4c	17.7±1.5g	15.0±0.0c	14.0 ±1.0 g	0.47±0.10a	0.71±0.02bcd	13.5±2.0bcdef	26.0±2.0jhij
G2	41.5±3.5bc	60.7±1.5de	20.0±1.4abc	20.7 ±0.6ef	0.53±0.01a	0.75±0.05abc	9.5±2.1f	20.0±1.0j
G3	55.0±4.2abc	74.7±0.6b	22.5±4.9abc	26.3±1.5cd	0.51±0.06a	0.58±0.03e	12.5±071cdef	32.0±1.0fgh
G4	55.0±2.8abc	72.0±2.0bc	22.0±4.2abc	26.3±1.2cd	0.49±0.05a	0.73±0.04abcd	17.5±2.1abcd	29.3±2.5fghi
G5	51.0±0.0abc	65.7±3.5cd	20.0±0.0abc	32.3±2.5ab	0.52±0.03a	0.64±0.03cde	18.5±2.1abc	36.3±3.1def
G6	54.0±0.7abc	86.7±2.5a	22.0±1.4abc	33.3 ±2.5a	0.52±0.03a	0.79±0.03ab	20.5±2.1a	40.0±2.0cde
G7	46.5±6.4abc	54.7±2.1e	18.5±2.1ab	16.3±1.5fg	0.49±0.01a	0.72±0.02abcd	11.5±07def	22.7±1.5ij
G8	46.5±0.7abc	62.0±2.0de	20.5±0.7abc	24.0±2.6de	0.52±0.05a	0.81±0.04ab	15.5±07abcdef	35.7±3.1def
G9	50.5±7.8abc	65.7±4.5cd	31.0 ±2.8a	20.67±2.1ef	0.51±0.00a	079±0.04ab	18.0±1.4abc	45.3±2.1bc
G10	52.0±7.1abc	68.7±6.0bcd	20.5±7.8abc	33.7 ±1.2a	0.55±0.13a	0.77±0.03ab	19.0±1.4ab	50.0±2.0b
G11	47.5±07abc	84.7±2.1a	22.0±1.4abc	31.7±2.9abc	0.51±0.00a	0.71±0.02abcd	20.5±2.1a	64.7±4.2a
G12	47.5±0.7abc	19.0±11.0g	27.0±2.8abc	14.5±8.4g	0.41±0.02a	0.63±0.34abcd	11.0±0.0ef	19.5±2.1j
G13	58.0±8.5ab	21.0±1.7g	21.0±0.0ab	14.3 ±1.2g	0.47±0.0.11a	0.62±0.03cde	13.5±2.1bcdef	24.3±4.5hij
G14	50.0±0.0abc	25.0±2.6fg	24.0±1.4abc	19.3±1.2efg	0.43±0.00a	0.77±0.02de	16.0±1.4abcde	29.7±1.5fghi
G15	59.0±2.8a	26.0±3.6fg	24.0±1.4abc	24.3±2.1de	0.60±0.00a	0.83±0.03ab	18.5±0.7abc	33.3±1.5efg
G16	54.0±1.4abc	33.3±3.5f	28.0±0.7ab	27.0±1.0bcd	0.54±0.06a	0.78±0.06a	20.5±0.7a	42.7±3.1bcd
LSD P≤0.05**	SN 6.22	SN 3.46	SN 4.35	SN 2.1	NS 0.09	SN 0.04	SN 4.49	SN 2.99

1st period: First period after 45 day; 2 nd period: Second period at the end of experiment at 90 day. **Significance at P≤ 0.05. Means in a column not showing the same many script are significantly (P≤ 0.05) different.

Tables (1A) illustrate the creatine value CK (myocardial infraction). There was a significant difference in the CK throughout the experiment groups in the all periods of trial. In the 1st period of the experiment period, a significant increase in CK depletion level was observed at the higher ratio among later groups fed on 20th and 30th frying oils used in this study. However, most the groups fed on exhausted oils from 20 th and 30th frying oil from all types of canola oils and its blends with SF and PO revealed higher level of creatine kinase activity than that in groups on different unused oils in the current study after last 90 days of the experiment. On the other hand, a significant increase was observed after 90 days ($P \leq 0.05$) versus unused oils in the same type of frying oil.

The determination of creatine kinase is utilized in the diagnosis and monitoring of myocardial infarction and myopathies. Most of CK ratio was reduced to 23 UI/L) throughout groups fed on fresh CO and its blended oils than the control group. In general, CK was found to decreasein the groups fed on fresh CO, CO+SF and CO+PO by about 13 % than the control group in the first period of feeding trail.The CO containedthe higher ratio of ω-3 fatty acids (6.19%) than corn oil (0.83%) (Table B). Corresponding to feeding the experimental animal in this study on canola oil and its blend, provide animal by higher ratio of omega fatty acid , which improves the membrane phospholipid on myocardial tissues and reduction the CK by about 13% compare to control corn oil group. This also conducted by Hock, *et al.,* (1987) that, the fatty acid composition of myocardial membrane phospholipid is sensitive to the type of fatty acids consumed in the diet. By increase omega -3 fatty acid, the phospholipid of heart was found to change along with reduction of creatine kinase in the feeding rats. These membrane changes may be involved in the observed reduction of ischemic damage in the heart. A similar accepted by Salvatore and McLennan, (2002) the n-3 PUFA reduced oxygen consumption at any given work output and increased postischemic recovery. Thus, direct effects on myocardial function may contribute to the altered cardiovascular disease profile associated with fish consumption. Versa of this result by Sealls, et *al.,* (2008) conducted that, the histological examination of the livers indicated a large accumulation of lipid in animals maintained on the lard and canola oil diets. This corresponded to a 6- to 8-fold higher amount of total triglyceride and 2-fold higher cholesterol levels than measured for mice maintained on the fish/fungal diet.

Table (1B): Biochemical blood analysis of rats fed on canola oil and their blends of sunflower or palm olein oils used in deep- fat frying process

Diet treatments groups	Lipid profile											
	Total cholesterol (TC)		HDL-C		LDL-C		VLDL-C		Triglyceride (TG)		AtherogenicIndex (AI)	
	1st period	2nd period	1st period	2nd period	1st period	2nd period	1st period	2nd period	1st period	2nd period	1st period	2nd period
ContolG1	87.8±1.9^{abc}	99.1±2.6^{a}	52.0±2.8^{a}	45.0±5.3^{b}	20.8±1.7^{abc}	32.5±2.7^{a}	15.0±2.8^{cdef}	21.9±1.4^{de}	99.2±0.3^{de}	59.0±1.9^{i}	21.4^{abc}	34.1^{a}
G2	83.4±1.7^{bcd}	108.7±4.7^{a}	74.0±2.8^{a}	57.0±6.2^{ab}	17.5±3.4^{c}	32.3±3.4^{a}	18.9±1.2^{abcdef}	19.4±0.3^{c}	85.8±1.7^{ef}	79.3±4.7^{h}	17.9^{a}	32.7^{a}
G3	68.8±4.6^{d}	111.3±6.1^{a}	37.5±0.7^{a}	63.7±4.7^{a}	17.6±1.4^{c}	26.2±2.7^{a}	13.8±5.3^{def}	21.5±1.4^{de}	98.6±5.9^{de}	96.5±2.3^{efg}	17.9^{a}	31.6^{a}
G4	83.9±6.6^{bcd}	114.3±10.5^{a}	51.0±7.1^{a}	59.7±5.5^{a}	19.1±1.3^{bc}	30.7±5.3^{a}	13.9±1.8^{cdef}	24.0±0.7^{cde}	117.3±9.7^{cd}	124.9±4.2^{cd}	19.3^{ab}	27.8^{a}
G5	84.5±0.9^{bcd}	115.2±2.9^{a}	47.5±2.1^{a}	54.3±2.1^{ab}	18.6±1.4^{bc}	32.8±3.9^{a}	22.9±1.9^{ab}	28.0±0.9^{abc}	119.6±9.5^{cd}	128.7±2.8^{cd}	19.1^{ab}	26.6^{a}
G6	87.0±1.4^{bcd}	116.1 ± 4.2 ^{a}	49.0± 1.4^{a}	57.7±1.5^{ab}	21.3±1.7^{abc}	26.1±4.8^{a}	16.7±1.7^{bcdef}	32.3 ± 2.6^{ab}	163. 5 ± 6.2^{a}	137.7 ± 3.4^{c}	21.6^{abc}	32.0 ^{a}
G7	85.9±4.0^{bcd}	103.5 ± 5.4 ^{a}	46.0±5.7 ^{a}	55.0 ±5. 6^{ab}	28.6±2.4^{abc}	27.0±6.4 ^{a}	11.0±1.2^{f}	20.2 ± 1.2^{e}	72.5 ± 3.9^{f}	93.6 ± 2.3^{fg}	28.8^{de}	32.1 ^{a}
G8	80.9±5.0^{bcd}	108.8±4. 7 ^{a}	45.5±7.8 ^{a}	55.3±4.2^{ab}	23.7±3.0^{abc}	29.9±6.4 ^{a}	11.8±0.2^{ef}	23.5±0.3^{cde}	101.5±2.9^{df}	107.7±6. 6^{e}	23.9^{abcd}	30.5 ^{a}
G9	76.3±3.2^{cd}	111.6±11.0 ^{a}	33.0±4.2 ^{a}	58.0 ±6.5^{ab}	29.1±5.7^{ab}	26.7±5.0 ^{a}	14.2±1. 8^{cdef}	27.0 ±0.2^{bcd}	126.1±4.3^{bc}	121.2± 2.3^{d}	29.6^{de}	27.4 ^{a}
G10	82.5±5.9^{bcd}	117.5±4.4^{a}	38.5±10.6 ^{a}	52.0±3.6^{ab}	24.3±3.7^{abc}	32.3±5.9 ^{a}	19.7±1.0^{abcd}	33.2±2.2^{a}	146.5±5.6^{ab}	160.7±2.1^{b}	24.9^{bcd}	27.6 ^{a}
G11	88.5±0.3^{abc}	118.4 ±6.12 ^{a}	39.5 ±3.5 ^{a}	58.0±4.6^{ab}	23.5±3.2^{abc}	27.3±3.4 ^{a}	25.5±0.8^{a}	33.2±1.2^{a}	164.6±5.5^{a}	187.0±11.2^{a}	24.1^{abcd}	32.3^{a}
G12	84.2±1.8^{bcd}	100.6±5.8 ^{a}	47.0 ±1.4^{a}	52.0±2.8^{ab}	23.8±1.0^{abc}	27.2±9.1^{a}	13.4±0.1^{def}	19.5±0.2^{e}	72.4±1.9^{f}	102.5±5.19^{el}	24.1^{abcd}	26.7 ^{a}
G13	89.7±1.8^{abc}	97.3±3.2 ^{a}	46.5±4.9 ^{a}	59.0±2.6^{a}	27.1±3.75^{abc}	32.1 ±2.6^{a}	16.2±0.6^{bcdef}	21.7±1.0^{de}	106.3±1.7^{cdf}	65.1±4.8^{i}	27.4^{cde}	25.3 ^{a}
G14	95.6±1.7^{ab}	112.9±4.2 ^{a}	50.5±0.7 ^{a}	59.0±3.0^{a}	24. 7±1.2^{abc}	30.1±2.6^{a}	20.5±1.2^{abcd}	23.8±2.1^{cde}	120.9±4. 7^{cd}	84.0±2.5^{gh}	25.1^{bcd}	35.3 ^{a}
G15	88.3±7.0^{abc}	115.7±6.5 ^{a}	42.0±8.5 ^{a}	54.7±7.6^{ab}	25.0±2.0 ^{a}	29.1±3.7 ^{a}	21.3±0.5^{abc}	25.9±3.5^{cd}	128.0± 0.8^{bc}	92.0±1.3^{fgh}	25.5^{bcde}	29.5 ^{a}
G16	105.8±9.3 ^{a}	115.8±6.8 ^{a}	49.0±7.1 ^{a}	62.0 ±2.0^{a}	31.2±3.1^{abc}	24.9±4.1 ^{a}	25.6±0.9^{a}	28.9±3.9^{abc}	149.3±1.3^{ab}	95.7± 2.2^{efg}	31.7^{e}	27.7^{a}
LSD P≤ 0.05	SN 6.9	NS 173.3	NS 8.0	SN 5.2	SN 4.2	NS 5.4	SN 2.8	SN 2.2	SN 9.1	SN 5.2	SN 5.6	NS 11.4

1st period: First period after 45 day; 2 nd period: Second period at the end of experiment at 90 day. **Significance at P≤ 0.05. Means in a column not showing the same many script are significantly (P≤ 0.05) different.

Changes in serum lipid profile resulted in Table (1B), showed a significantly increased mean cholesterol values among group 12 up to group16 in the 1st experimental period ranged 84.2 -105.8 mg/dL. This result is corresponds to dietary contain a palm olein (PO). Meanwhile, the cholesterol level was

generally either lower amonggroups, which fed on CO and CO + SF used, or unused in frying during first period (1st period).

However, in the second experimental period, there were non-significantly deference between all groups in cholesterol level. The serum cholesterol levels of the control G1 were 87.8 and 99.10 mg/dL corresponds to the 1stand2ndperiod of studies, respectively. The similar trend occur among groups 2, 7, and 12 after 90 days shows a closely cholesterol level for group 1 ranged between 100 up to 108.73 mg/dl.

The serum HDL cholesterol levels of the control group (1) were 52.0 and 45.0 mg/dl corresponds to the 1st and 2nd the experimental period of studies, respectively (Table 1B). Remarkable, a little decrease in mean of HDL-C shown in groups fed on CO+PO either used or unused frying oils at the 1stperiod of the experiment. The other groups showed no significant

changes in serum HDL cholesterol after first period of experiment. However, all groups showed a significant increase in HDL after second period of experiment. By feeding all groups on CO and its blends either SF oils after frying, had a little increased in HDL after the 2nd experiment period when compared to control group (1). In addition, palmolein showed a little increases in HDL cholesterol level than all other treated groups in the 2nd period of the experiment. This result is indicated by Truswell*et al.*, (1992) as reported that,thePO there was a rise of HDL-cholesterol averaging about 10 percent. This was seen and statistically significant in both series of experiments. From, the obvious result indicated that, CO in dietary could be increase the HDL level than corn oil group (1). CO is considered as very health omega edible oil (Baux, *et al.*, 2008) .Canola oil contains a higher ratio of omega-3 fatty acids, which increase good cholesterol such as HDL (Sundram*et al.*, 2003). Therefore, Canola oil contains about 10% α-linolenic acid, an 18-carbon ω-3 fatty acid, and about 20% linoleic acid, an 18-carbon ω-6 fatty acid, whereas corn oil contains about 50% linoleic acid and about 1% ω-3 fatty acids (Johnson *et al.*, 2007). In addition, canola oil contains Lignoceric acid (C24:0), which has negative effects on CVD and brain stroke patients. Also, very long-chain saturates including lignoceric acid (C24:0) have been reported to have a distinctive function such as anti-inflammatory effects (Kihara, 2012).

The LDL values were found to significantly ($P \leq 0.05$) increase among groups fed on CO+SF and CO+PO oils either used or unused in frying process during 1st the experiment period (Table 1B). By feedingthe experimental groups on unused and used frying oil in frying potato corresponds decline in LDL was trace as same period to the control in similar first period of experiment. While, in the second period of experiment, LDL value recorded non- significant changes among all of the groups. However, the mean value of LDL during this second period ranged from 24.9 : 32.8 mg/dl. Our results emphasize that CO and its blends provides an effective means of favorably modulatory lipid profile and decreased LDL value. Despite the canola oil contains higher ratio of monounsaturated,its blend with PO reduced significantly serum LDL more than the control group. This is in agreement with result Most *et al.*, (2005).

The VLDL cholesterol level of the control group was 15.00 and 21.90 mg/dl corresponds to 1st and 2nd of experiment period, respectively (Table 1B). Correspondingly, to the VLDL level, all of groups showed a significant difference ($P \leq 0.05$) during 1 st time and 2nd of the experimental periods as compared to the control group (1). Otherwise, the VLDL of serum cholesterol in groups CO and CO+SF fed on unused and used frying oils; have shown lower values than in unused and used frying CO+PO oils in the first experiment period.

Corresponding triglyceride level in groups (4, 5 and 6) had higher level than the control group in all experiment periods (Table 1B). A noticeable lowering in TG level could be found among groupsfedon CO+PO (G12:G16) in the 1st period of experiment. Meanwhile, TG level recorded a little changes among groups G7:G9 compared to all groups of dietary fed CO+PO. In the 2nd period of experiment after 90 days, there was non-significant decrease in TG among all groups except for G1, G2, G9 and groups from (G13:G16) than occurred in the 1st experimental period.

Table (1B) shows that, the serum Atherogenic Index (AI) in all groups revealed significant ($P \leq 0.05$) difference as compared to the control group (G1), this is accompanied with significant difference between groups lipid profiles during first 45 days of the experiment period. Atherogenic Index was found to decline among groups of unused and used in frying CO (G2, G3, G4, and G5) after administration in the first time of experiment. Therefore, administration CO and its different frying time oils accompanied with decrease LDL cholesterol level compared to the control group. These groups of CO dietary feeding had declined by about 9.60: 16.4 % in the AI when compared to AI ratio of the control group in the first experiment time.

The AI in all groups exhibited no significant difference ($P \geq 0.05$) as compared to the control group in the 2nd period. Most of experimental animals in such groups showed AI ratio ranged from 27.43 up to 35.27 by the end of 2nd period of experiment (Table 1B). There was a relationship between formation and oxidation of LDL and increases of AI. AI indicates the deposition of foam cells or plaque or fatty infiltration or lipids in heart, coronaries, aorta, liver and kidney. Inhibition of LDL oxidation is supposed to be one of the critical steps in retarding the foam cell formation and development of aortic lesion (Chisolm and Steinberg 2000).The AI, relatively increases risk of oxidative damage for the aforementioned organs. These antiatherogenic and cardioprotective effects of canola oil may be due to the presence of polyunsaturated fatty acids.

Considering our pervious study by Elreffaei, *et al.*,(2015), range from 20 -45% total polyunsaturated of the total fatty acids present in the CO and its blends (Table B).Dietary lipids can influence the cardiac function via changes in membrane fatty acid composition, and these changes usually reflect the fatty acid composition of the diet, although important metabolic alterations also occur. Most of these total polyunsaturates contain linoleic acid. Dietary linoleic acid serves as a precursor for biosynthesis of arachidonic acid, the substrate for eicosanoid synthesis through activity of the enzyme cyclo-oxygenase and 5-lipoxygenase. The relationship between major linoleic acid (18:2n6) and it isomers of conjugated linoleic acid (CLA) is considered as an important factor for human health, since 18:2n6 has been associated with decreased atherogenicity in animal models. Therefore, Linoleic acid has attributed as anticarcinogenic properties, as well as anti atherogenic effects, and also known as rumenic acid (Masso-Welch *et al.*, 2004). Attributed to lowering of lipids by CLA in non-hepatic tissues was expected to beneficially affect insulin sensitivity and glucose tolerance (Kennedy *et al.*, 2010). Moreover,Aguila*et al.*, (2005) reported that, the dietary canola oil supplementation has a relative low n-6/n-3

ratio and a high MUFA content. n-3 PUFA provide protection due to its ability to suppress inflammation or coagulation by interfering with the proinflammatory, procoagulation prostanoidsm, thromboxanes, and/or leukotrienes production. Reno protection by n-3 PUFA could be caused by the human being tendency or laboratory rodents ingesting such diets to exhibit lower Blood pressure.

Over all of these results in the current study it can be established that fried canola oil and its blend with SF and PO diet gave a good serum lipid profile and increased the HDL and release LDL ratio, thus helping to prevent intravascular lipid deposition and similar to corn oil in the control group.

Internal antioxidant

Coenzyme Q10 (COQ10)

Serum coenzyme Q10 results are summarized in Table 2. Mean total of coenzyme Q10 was found to decrease among of all groups, which fed on exhaust frying of 30th for all types of oils used in this current study. As expected, coenzyme Q10 was reduced in blood serum after feeding experiment animals on frying oils from F 30th of different frying types of canola oils and its blends by the end of 2nd period of experiment. However, coenzyme Q10 of experimental rats of the control group contained 950 (mmol/ml). This concentration in the control group identify in the healthy group. This is found to with claim that, the coenzyme Q10 is an indigenous cellular antioxidant that may protect bio membranes against some effects of aging, mediated oxidative stress, cancer and immune system abnormal (Hodges *et al.*, 1999). Under normal conditions plasma coenzyme Q10 concentrations are not significantly affected by dietary components such as dairy products, eggs, fish and vegetables. The nutritional status participating did not influence the basal levels of antioxidant nutrients in plasma. (Bhagavan and Chopra 2006).

Some studies suggest that COQ10 may exhibit anti-inflammatory properties, but most of those data have been obtained *in vitro*. This is may contribute a correlation between total coenzyme Q10 and the increase of LDL cholesterol level and ω-3 fatty acid as shown in Tables B and 2B. These decreases in Coenzyme Q10 among G11, G6 and Group 16 were considering to type of fed exhausted oils from F30th. Most of these groups contained less than 70 (mmol/ml) of coenzyme Q10. This reduction in COQ10 among groups of F30 oils have advocated that, a biomarker for increase of oxidative stress throughout groups F 30th fed oils (G6, G11 and G16). Also this was supported by Galinier *et al.*, (2004). Meanwhile, Sohmiya*et al.*, (2004) reported not only a significant reduction in plasma COQ10 in Parkinson Disease patients, but also a significant increase in the percentage that oxidized COQ10comprised of total coenzyme Q10, which was interpreted as evidence of oxidative stress.

Table (2): Coenzyme Q10 (COQ10) in blood serum of feeding groups on F30 of canola oil and its blend oils

Diet treatments groups	G1	G6	G11	G16
Normal range (mmol/ml)	(750-1000)	(750-1000)	(750-1000)	(750-1000)
Coenzyme Q10 (mmol/ml)	950 (mmol/ml)	< 70 (mmol/ml)	< 70 (mmol/ml)	< 70 (mmol/ml)

Therefore, a low ω-6/ ω -3 ratio of foods with antioxidants, micronutrients, minerals, vitamin and coenzyme Q10 may inhibit the generation of superoxide and suppress the pro-inflammatory transcription factors, NF-kB as well as AP-1 and Egr-1, which may inhibit phenotypic expressions. Dietary intakes of wild foods rich in antioxidants and ω-3 fatty acids appear to have important roles in the pathogenesis and prevention of cardiovascular disease (Lai *et al.*, 2006). In accordance,Sohet*et al.*, (2009) concluded that, the COQ10tends to decrease hepatic stress gene expression with obesity in mice in this tissue, independently of any modulation of lipid peroxidation, which was classically considered as its most relevant effect.

Effects on hematological parameters:

As seen in Tables 3 A and 3B , the level of blood hemoglobin (range 13.9-16.6 gm/dl) , RBC (range 7.1-8 10^{12}/L), hematocrit (range 38.6-44.8) , WBC (range 12.9-25.9 10^{9}/L), and lymphocyte (range 53.3- 74.0%) were not significantly($P \geq 0.05$) different among all groups from G1 up to G16in biological assessments at CO and its blends with SF or PO corresponding to different frying cycles during the 1stfeeding period. However, RBC and hematocrit were significantly difference ($P \leq 0.05$) in 2nd experiment among all fed groups than first duration. In previous study by Finlayson, *et al.*, (1999) stated that thermoxidized palm oil causes liver damage which leads to increase in WBC count. While, Elreffaei, et *al.*, (2015) reported frying oil stability among different used CO and its blends with SF and PO oils. This indicates improvement of hematological parameters for such fed groups on different frying oils under this current study. The increase in the hematocrit and RBC contents were due to the feeding of thermally frying oils. However, RBC levels among groups 2 (7.6 10^{12}/L), and 9 (7.4 10^{12}/L) exhibited a slightly difference than other groups fed in the same 2nd period (Table 3A). A remarkable decrease was occurred in blood lymphocyte percentage among groups, which fed on fresh CO + PO (G12) and used frying CO + PO (G13, G14 and G16) oils than other groups in the experiment during 1 st period. This reduction in lymphocyte was about 8% than the control group G1.

Table (3A): Hematological parameters of rats fed on canola oil and their blends of sunflower or palm olein oils used in deep- fat frying process

Diet Treatment Groups***	Hemoglobin gm/dl		RBC 10^{12}/L		Hematocrit %	
	1st period	2nd period	1stt period	2nd period	1st period	2nd period
G1	15.3±1.2^{a}	17.2±0.5^{a}	7.1± 0.1^{a}	8.3±0.3^{abc}	43.9±3.4^{a}	43.7±0.7^{ab}
G2	15.8±0.4^{a}	16.7±0.7^{a}	7.7±0.0^{a}	7.6±0.2^{bc}	44.0±2.1^{a}	73.7±1.8^{ab}
G3	16.0±0.3 ^{a}	16.4±0.9^{a}	7.1±0.4^{a}	8.2±0.3^{abc}	44.5±0.1^{a}	44.3±3.3^{ab}
G4	14.9±0.4^{a}	17.4±0.7^{a}	7.2±0.7^{a}	8.3±0.5^{abc}	44.1±3.3^{a}	43.3±0.2^{ab}
G5	15.3±2.4^{a}	16.6±1.3^{a}	7.1±0.4^{a}	8.4±0.2^{abc}	42.8±4.2^{a}	45.6±3.8 ^{a}
G6	16.1±0.3^{a}	16.7±1.9^{a}	7.2±0.3^{a}	8.2±0.3^{abc}	44.6±0.6^{a}	43.9±1.3^{ab}
G7	15.2±2.3^{a}	17.2±0.6^{a}	7.2±0.4^{a}	8.1±0.1^{abc}	42.7±3.7^{a}	42.0±0.8^{ab}
G8	14.1±0.8^{a}	15.6±2.2^{a}	7.4±0.8^{a}	8.0±0.5^{abc}	38.9±1.3^{a}	42.6±4.1^{ab}
G9	13.9±0.1^{a}	13.7±0.7^{a}	8.0±0.3^{a}	7.4±0.1^{c}	38.6±1.1^{a}	37.8±1.8^{b}
G10	15.1±0.8^{a}	17.3±1.4^{a}	7.3±0.1^{a}	8.2±0.5^{abc}	43.6±0.1^{a}	43.7±1.2^{ab}
G11	16.2±0.3^{a}	18.3±0.9^{a}	7.8±0.3^{a}	8.5±0.4^{abc}	44.8±0.6^{a}	45.8±1.3 ^{a}
G12	15.5±0.0 ^{a}	17.6±1.7^{a}	7.2±0.2^{a}	8.7±0.3^{abc}	43.1±0.5^{a}	43.5±2.1^{ab}
G13	15.1±0.1^{a}	18.0±0.3^{a}	7.7±0.3^{a}	8.2±0.3^{abc}	42.8±0.1^{a}	44.6±1.1^{ab}
G14	15.3 ±0.8^{a}	19.1±0. ^{a}	7.2±0.2 ^{a}	8.8±0.2^{a}	42.7±1.1^{a}	45.8±1.2^{a}
G15	16.6±0.3^{a}	18.4±2.3^{a}	7.7±0.3^{a}	8.5±0.8^{abc}	44.6±0.5^{a}	44.5±4.3^{ab}
G16	15.5±0.6^{a}	17.3±0.6^{a}	7.4±0.0^{a}	8.3±0.2^{abc}	42.1±0.7^{a}	40.9±2.0^{ab}
LSD (P≤ 0.05)	2.08	2.09	0.75	0.63	4.24	3.82
P≤ 0.05	NS (0.48)	NS(0.06)	NS (0.31)	SN (0.015)	NS (0.15)	SN (0.028)

values are expressed as mean ± SD, n = 3 in each group. Means followed by different letters (a–c) in the same column are significantly different (P ≤ 0.05). * G! is the experimental diet group fed on such type of oil**

Table (3B): Hematological parameters of rats fed on canola oil and their blends of sunflower or palm olein oils used in deep- fat frying process

Diet Treatment Groups***	Platelets 10^{9}/L	Platelets 10^{9}/L	WBC 10^{9}/L	WBC 10^{9}/L	Lymphocyte %	Lymphocyte %
	1St period	2ndperiod	1St period	2ndperiod	1St period	2nd period
G1	786±7.1^{abc}	1052.7±109.5^{a}	18.2±2.1^{a}	18.5±6.3^{a}	58.0±12.5^{a}	75.0±4.2^{a}
G2	488±59.4^{abc}	1009.0±248.8^{a}	18.9±0.5^{a}	20.9±0.7^{a}	74.0±3.6^{a}	69.0±12.7^{a}
G3	460±128.7^{bc}	751.3±131.3^{a}	22.9±13.4^{a}	23.7±1.3^{a}	67.3±4.0^{a}	74.0±6.4^{a}
G4	431±16.3^{bc}	826.3±175.3^{a}	23.1±3.3^{a}	22.1±2.1^{a}	68.7±3.1^{a}	74.5±2.1^{a}
G5	660±30.4^{abc}	804.0±150.1^{a}	16.5±2.9^{a}	17.5±2.1^{a}	69.3±7.0^{a}	76.5±4.9^{a}
G6	623±121.6^{abc}	786.3±77.2^{a}	23.1±13.1^{a}	20.0±8.1^{a}	62.7±8.5^{a}	69.5±0.7^{a}
G7	358±412.3 ^{c}	880.3±2.5^{a}	18.0±0.6^{a}	21.6±4.9^{a}	68.3±5.0^{a}	76.5 ±0.7^{a}
G8	779±158.4^{abc}	904.3±169.4^{a}	13.6±0.0^{a}	22.4±3.9 ^{a}	65.7±7.2^{a}	73.5±4.9^{a}
G9	740±108.2^{abc}	1301.7±235.6^{a}	15.8±0.6^{a}	23.0±3.1^{a}	55.0±6.0^{a}	67.5±6.4^{a}
G10	682±43.8^{abc}	947.7±435.8^{a}	15.3±0.3^{a}	21.2±3.5^{a}	69.0±2.6^{a}	76.0±1.4^{a}
G11	805±32.5^{abc}	772.7±74.8^{a}	25.9±2.1^{a}	25.7±8.2^{a}	62.3±2.1^{a}	68.0±8.5^{a}
G12	910±82.0^{abc}	751.0±187.4^{a}	12.9±1.3^{a}	27.1±8.9^{a}	53.3±14.6^{a}	70.5±7.8^{a}
G13	735±82.0^{abc}	852.7±42.2^{a}	24.6±3.1^{a}	22.8±7.9^{a}	53.7±4.7^{a}	78.0±0.0^{a}
G14	1061±122.3^{a}	775.7±204.2^{a}	22.1±2.7^{a}	23.5±2.4^{a}	55.0±16.5^{a}	78.5±4.9^{a}
G15	980±172.5^{ab}	876.7±111.1^{a}	19.1±8.6^{a}	18.6±3.6^{a}	69.3±4.6^{a}	78.0±2.8^{a}
G16	745±136.5^{abc}	836.3±267.7^{a}	22.1±2.3^{a}	15.7±1.4^{a}	55.0±16.5^{a}	71.5±10.6^{a}
LSD (P≤ 0.05)	309.32	317.61	11.59	8.34	14.55	12.93
P≤ 0.05	SN (0.007)	NS (0.116)	NS (0.441)	NS (0.439)	NS(0.086)	NS (0.708)

values are expressed as mean ± SD, n = 3 in each group. Means followed by different letters (a–c) in the same line are significantly different (P ≤ 0.05).* G! is the experimental diet group fed on such type of oil**

Furthermore, there was a significant difference in platelets in all groups in the 1st feeding duration comparing to the control group (G1) even in the different type of fed oils (Table 3B). This shows that supplementation of oxidized CO and its blends produce significant stress on these blood markers. The present results are in agreement with earlier study by Zeb and Ullah (2015) who concluded that, the oxidized ghee produced toxic effects in the liver and hematological parameters.

Igiri *et al*., (1994) stated that intestinal mucosa is severely damaged by thermally oxidized palm oil in rats. This damage may lead to a lower absorption of iron by the intestinal mucosa which results into a decrease bioavailability of iron in the system. However, in the second duration of experiment, there was non-significant difference (P≥ 0.05) among all fed groups for blood platelets concentration. In spite, there was a slight decrease in platelets in groups fed on CO+PO either in fresh or used frying oils during second period of experiment. This behavior of decrease was not

significant among all groups of experiment during this 2nd period.

Histological examination:

The changes in the biochemical of fed rats on different samples of fresh and used in frying canola oil and its blends were further confirmed by histological examination for liver (A) , kidney (B) , heart (C), intestinal (D) and testes (E) tissue from fed rats Figs. 1, 2 and 3. Figure 1 illustrates the liver of histological assessment showed fatty degenerated in hepatocytes with segment ring occurring in rat's tissue among groups 1 and 5, which fed on the control corn oil and COF20, respectively (Fig 1). The hepatic tissue of groups 2, and 3 showed a similar dilation and congested hepato portal blood vessel with hyperplasia in the bile duct. The fat globule and bile duct were in severity formation among hepatic tissue of groups4 and 6, than thoseG1 and G2 (fed on fresh canola oil). For instance, the alterations in the hepatocytes may occurre, in consequence to ingest compounds in frying canola oils. Generally, the accumulation of lipid within the macrophage may be due to an impairment in lipid catabolism (Reasor, 1981) and may occur with G1 and G2 group dietary in their liver.

Also, Sealls, *et al.*, (2008) found that, the lard and canola oil diets resulted in high levels of hepatic triglycerides and cholesterol and elevation of lipogenic gene expression. Hepatic fatty acid analyses indicated that dietary PUFA were efficiently converted to highly unsaturated fatty acids regardless of source. Therefore, differences in hepatic lipid levels and gene expression between dietary groups were due to exogenous fatty acid supplied rather than endogenous pools. These results have important implications for understanding the regulation of hepatic lipogenesis by dietary fatty acids.The worst histological of hepatic tissue had been occurred obviously by fed rats on the higher number of frying cycles COF20 and COF30 as found in G5 and G6. Additionally, the severe fat globule in histological of hepatic group 4, 5 and 6 animals compared with treatment 1, 2, and 3 suggests that the use time of canola frying oil causes further changes and, consequently, worsens the effect in the tissue.

By histological examination of kidney section, the vacuolation glomerular tuft and renal cast formation throughout fed G2 and G3 while the control group has shown a focal area hemorrhage in kidney tissue (Fig 1B). Moderate focal leucocytic cell infiltration showed among G4, which was fed on COF10.

Some vacuolation of renal tubular epithelium showed an increase with associate interstitial blood vessel vasculitis with thick muscular wall was seen among kidneys of G5 and G6, respectively.

Dietary of COF10, COF20 and COF30 caused some intermuscular hemorrhage, focal area of mononuclear cells infiltration and hyalinosis with intermuscular congested blood vessel were seen respectively, with such of those groups in the heart tissue (Fig .1C).

The similar histological investigation was shown among all fed groups on fresh and frying oils up to 30th cycle in their intestine tissues (Fig. 1D). The histology of intestine throughout these G 1, G2, G3, G4, and G5) showed a hyperactivity of the mucous secreting glands.

Teste of dietary group on fresh corn oil G1 (control group) has shown different degenerative of some spermatogonia of the seminiferous tubules (Fig .1E). Furthermore, the G2, G3 and G4 showed a dilation and congested blood vessels and exception for group 4 has an interstitial edema. The gross necrosis of the lining spermatogonia cells of seminiferous and interstitial blood vessel congestion was conducted respectively among group 5 and 6.

From Figure 2 A, shows the liver histology of groups fed on fresh and used blend of CO+SF at different frying cycles up to 30th. In case of fresh fed CO+SF group, the central vein of liver dilated and congested with some normal hepatocyte. While in the (Fig.1 A) liver tissues were fatty and appearance with signet ring among the control normal oil diet (G1). While this previous status of the control group did not appear probably in fed group 10 which was fed on CO+SF F20 (Fig. 2A). Generally , most dilated and congested hepato portal vein shown among groups 8, 9, and 10, which fed on used blends CO+SF in frying at F1, F10, and F30. Interstitial blood vessels, hemorrhage, and vacuolation of glomerular tuft have been shown in kidney tissues among of those groups fed on either fresh or used frying oil CO+SF blend (Fig. 2 B). Heart histological examination showed a muscular hyalinosis with intermuscular congested blood vessel and focal area of leucocytic cell infiltration in the G7 and G8 (Fig. 2C). The later finding was similar to those given in the heart tissue of control group 1. Intestine of the all groups fed on fresh CO+SF and different frying oils cycles CO+SF (G7:G11) showed similar incidence of hyperactivity of the mucous secreting glands (Fig, 2D). The gastric lipase shows a stereo-preference for the hydrolysis of ester bonds, incidence edema and interstitial blood vessel congestion were conducted in all groups teste regarding to feed on fresh CO+SF and different frying oils cycles CO+SF (G7:G11) as illustrated in Fig. 2 E. After theingestion of dietary triglycerides (TGs), the digestion of Medium Chain triglycerides (MCTs) starts in the upper gastrointestinal tract under the action of preduodenal lipase (gastric or lingual depending on thespecie) and is completed in the small intestine by pancreatic lipase. Vanderhoof*et al.*, (1994) concluded that, more unsaturated and short-chain fatty acids are more rapidly adsorbed by the gastrointestinal mucosa. Menhaden oil appears more effective in inducing intestinal adaptation than less highlyunsaturated fats. Consequently, it could be consider digesting omega-3 and PUFA fatty acids of canola oil may stimuli hyperactivity of intestine mucous.

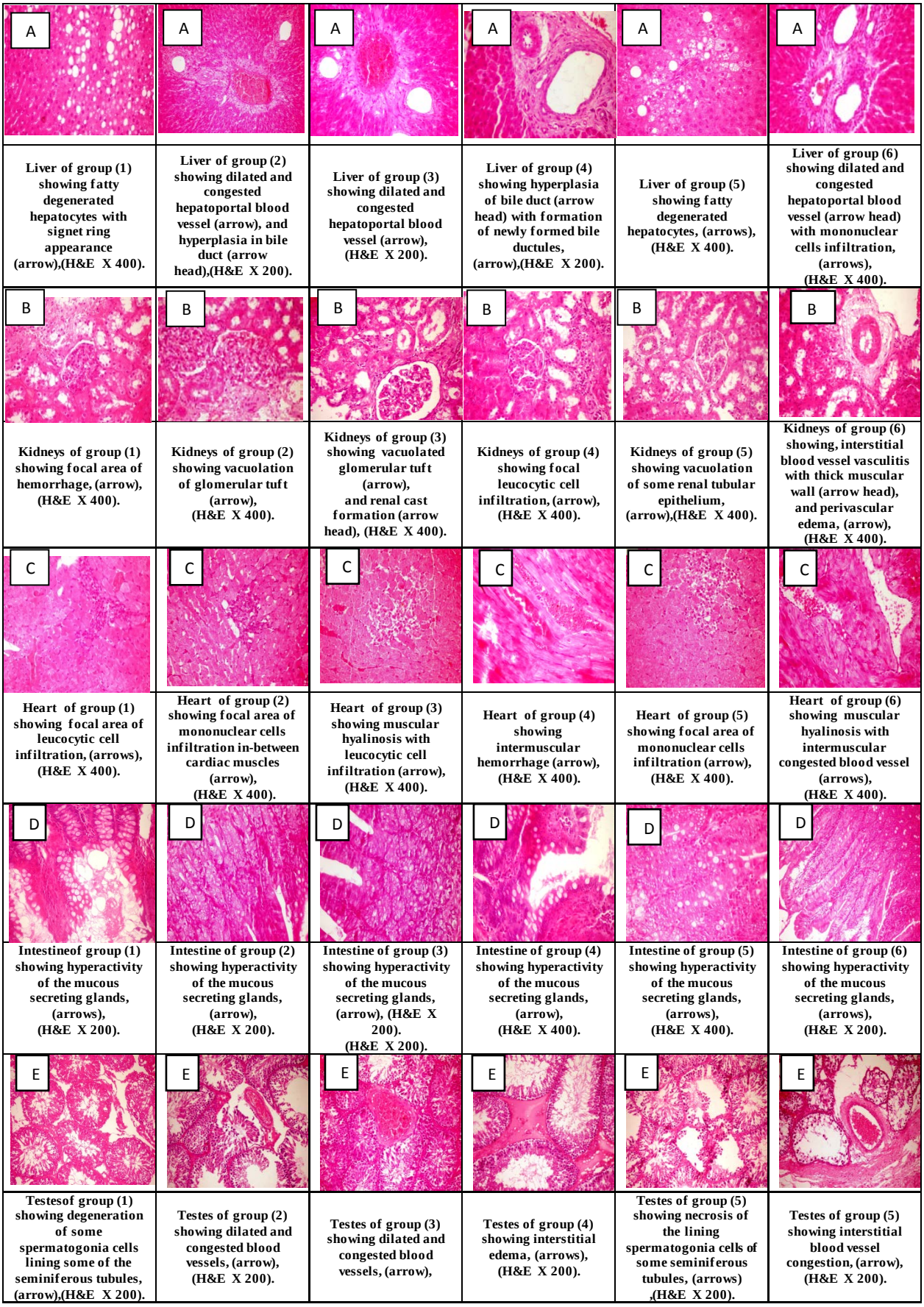


Figure (1): histology organs (liver, kidney, heart, intestine and tests) of rats fed on canola oil and its frying cycles canola oil.

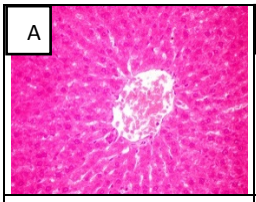
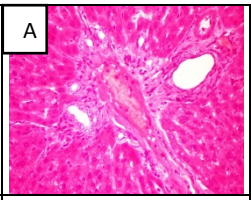
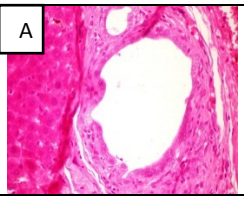
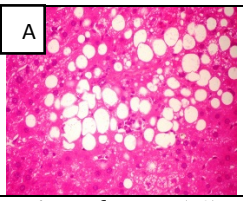
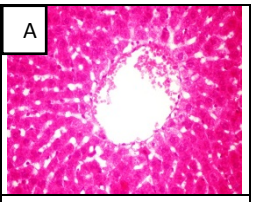
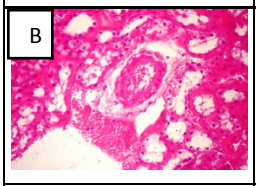
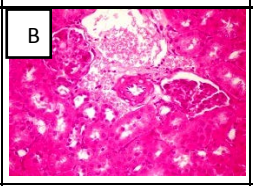
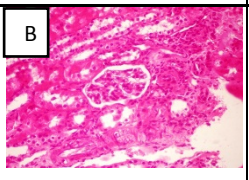
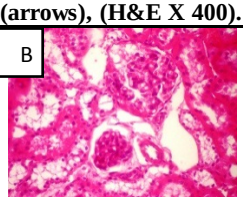
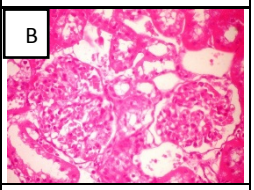
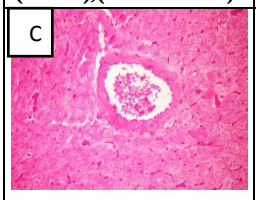
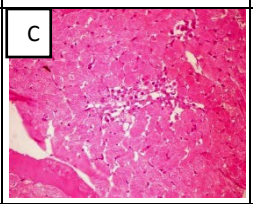
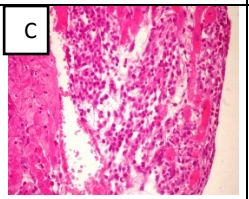
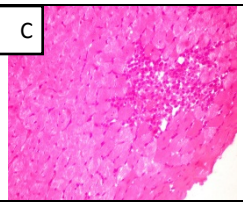
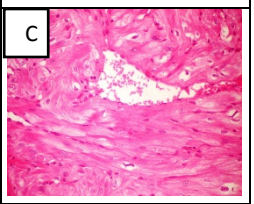
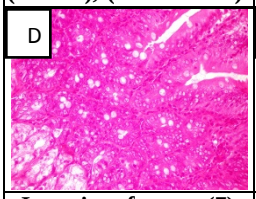
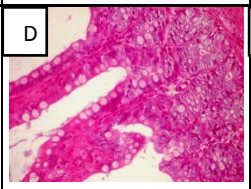
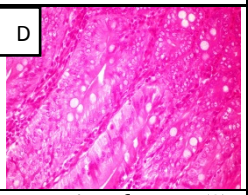
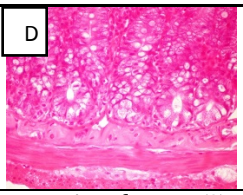
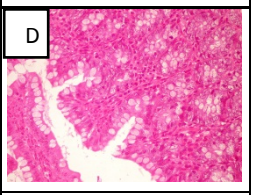
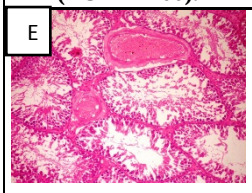
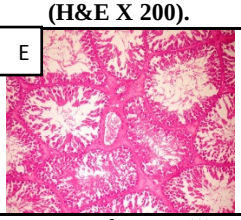
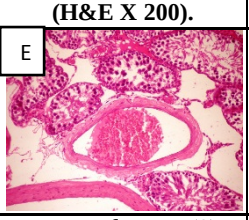
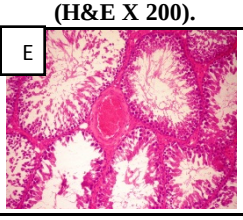
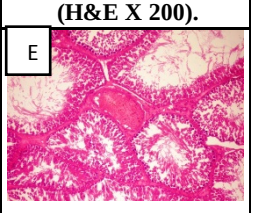
				
Liver of group (7) showing dilated and congested central vein, (arrow), (H&E X 400).	**Liver of group (8) showing dilated and congested hepatoportalvein , (arrow),(H&E X 400).**	**Liver of group (9) showing hyperplasia of the bile duct, (arrow), (H&E X 400).**	**Liver of group (10) showing fatty degenerated hepatocytes with signet ring appearance , (arrows), (H&E X 400).**	**Liver of group (11) showing dilated and congested central vein, (arrows), (H&E X 400).**
				
Kidneys of group (7) showing , interstitial blood vessel vasculitis with thick muscular wall (arrow head), and perivascular edema, (arrow),(H&E X 400).	**Kidneys of group (8) showing , interstitial blood vessel vasculitis together with focal area of hemorrhage, (arrow),(H&E X 400).**	**Kidneys of group (9) showing ,interstitialleucocytic cell infiltration, (arrow),(H&E X 400).**	**Kidneys of group (10) showing,vacuolation of glomerular tuft and tubular epithelium, (arrow),(H&E X 400).**	**Kidneys of group (11) showing vacuolation of glomerular tuft and tubular epithelium, (arrow),(H&E X 400).**
				
Heart of group (7) showing muscular hyalinosis with intermuscular congested blood vessel (arrows), (H&E X 400).	**Heart of group (8) showing focal area of leucocytic cell infiltration (arrows), (H&E X 400).**	**Heart of group (9) showing focal area of leucocytic cell infiltration (arrows), (H&E X 400).**	**Heart of group (10) showing focal area of leucocytic cell infiltration (arrows), (H&E X 400).**	**Heart of group (9) showing dilated and congested blood vessels (arrows), (H&E X 400).**
				
Intestineof group (7) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).	**Intestineof group (8) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).**	**Intestineof group (9) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).**	**Intestineof group (9) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).**	**Intestineof group (11) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).**
				
Testesof group (7) showing interstitial blood vessel congestion, (arrow),(H&E X 200).	**Testesof group (8) showing interstitial edema(arrow head), and congestion (arrow), (H&E X 200).**	**Testesof group (9) showing interstitial edema(arrow head), and congestion (arrow), (H&E X 200).**	**Testesof group (10) showing interstitial congestion (arrow), (H&E X 200).**	**Testesof group (11) showing interstitial congestion (arrow), (H&E X 200).**

Figure (2): histology organs (liver, kidney, heart, intestine and tests) of rats fed on canola oil+ sunflower oil and its frying cycles canola oil + sunflower oil blend.

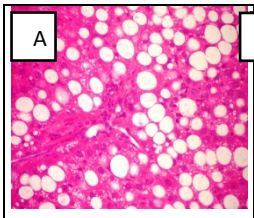
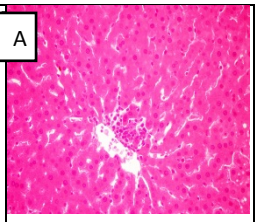
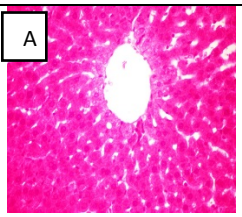
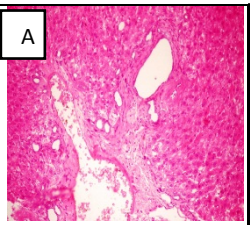
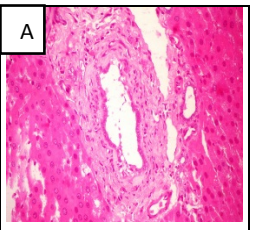
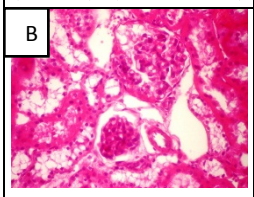
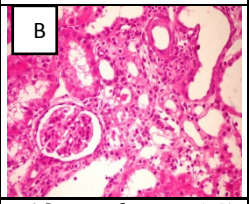
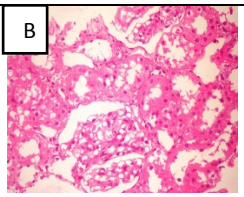
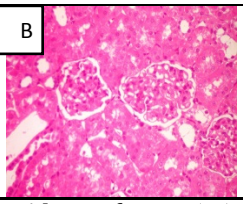
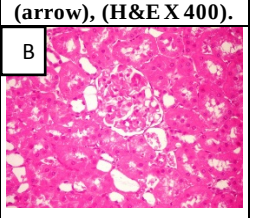
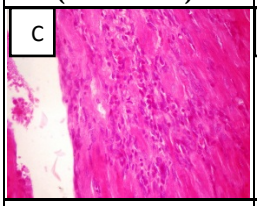
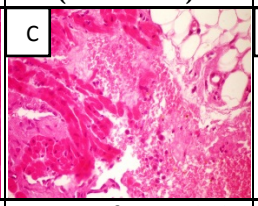
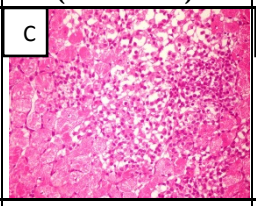
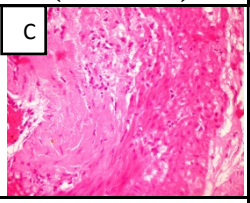
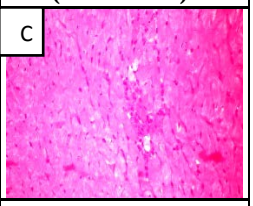
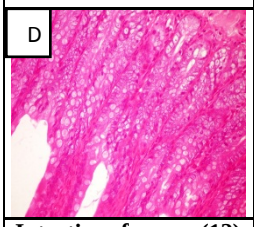
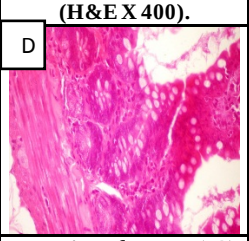
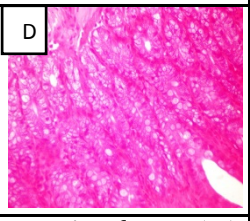
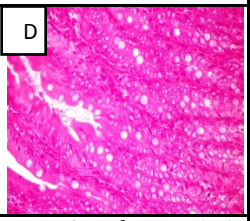
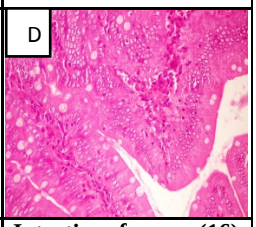
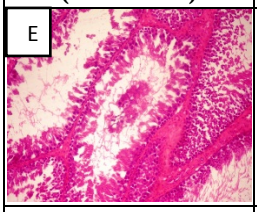
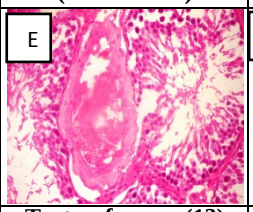
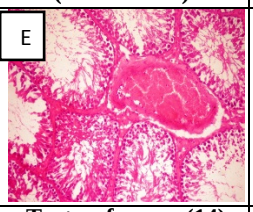
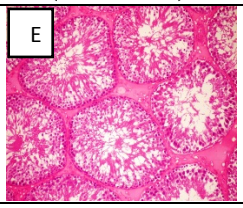
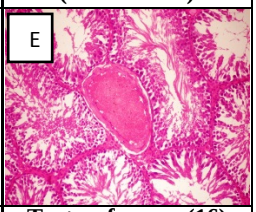
				
Liver of group (12) showing fatty degenerated hepatocytes with signet ring appearance, (arrows), (H&E X 400)	**Liver of group (13) showing focal area of centrolobularleucocytic cell infiltration , (arrows), (H&E X 400).**	**Liver of group (14) showing swelling of hepatocytes leading to disorganized cords, (H&E X 400).**	**Liver of group (15) showing congested hepatoportal blood vessel(arrow), and bile duct hyperplasia(arrow head), (H&E X 400)**	**Liver of group (16) showing cholangitis which characterized by bile duct hyperplasia with thick muscular wall ifiltrated with leucocytic cells (arrow), (H&E X 400).**
				
Kidneys of group (12) showing vacuolation of glomerular tuft and tubular epithelium, (arrow), (H&E X 400).	**Kidneys of group (13) showing degenerated renal tubules with formation of renal cast , (arrow), (H&E X 400).**	**Kidneys of group (14) showing vacuolated glomerular tuft (arrow head) with necrosed renal tubules , (arrow), (H&E X 400).**	**Kidneys of group (15) showing vacuolated glomerular tuft (arrow head) with necrosed renal tubules , (arrow), (H&E X 400).**	**Kidneys of group (16) showing vacuolated glomerular tuft (arrow head) with necrosed renal tubules , (arrow), (H&E X 400).**
				
Heart of group (12) showing focal area of leucocytic cell infiltration , (arrows), (H&E X 400).	**Heart of group (13) showing focal area of hemorrhage (arrow head) with leucocytic cell infiltration ,(arrows), (H&E X 400).**	**Heart of group (14) showing focal area of leucocytic cell infiltration ,(arrows), (H&E X 400).**	**Heart of group (14) showing focal area of necrosis infiltrated with leucocytic cell, (arrows), (H&E X 400).**	**Heart of group (15) showing focal area of leucocytic cell infiltration, (arrows), (H&E X 400).**
				
Intestineof group (12) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).	**Intestineof group (13) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).**	**Intestineof group (14) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).**	**Intestineof group (15) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).**	**Intestineof group (16) showing hyperactivity of the mucous secreting glands, (arrows), (H&E X 200).**
				
Testesof group (12) showing degeneration of the spermatogonia cell of some seminefrous tubules (arrow),(H&E X 200).	**Testesof group (13) showing interstitial congested blood vessel (arrow), (H&E X 400).**	**Testesof group (14) showing interstitial congested blood vessel (arrow), (H&E X 400).**	**Testesof group (15) showing interstitial edema (arrow), (H&E X 200).**	**Testesof group (16) showing interstitial congested blood vessel (arrow), (H&E X 200).**

Figure (3): histology organs (liver,kidney,heart, intestine and tests) of rats fed on canola oil+ palmolein oil and its frying cycles canola oil +palmolein oil blend.

Liver section examination for further confirmed effect of fed CO+PO frying oil among G 12: G16 showed in (Fig. 3A). Fatty degenerated hepatocytes with segment ring appeared obviously in G12 that fed on fresh CO+PO; this case was similar to group 1 of liver control group. In case of CO+POF1frying oil treated group, the section of liver showed focal area of Central tubular leucocytic cell infiltration. The hepatocytes were gross and leading to disorganized cords showed in the liver section of group 14 fed on CO+POF10 (Fig . 3A). Severe congested hepato portal blood vessel and bile duct hyperplasia were shown by investigate of group 15 liver that fed on CO+PO F20. Considerably, the cholangitis of bile duct degenerative and hyperplasia was consider with forming thin wall of muscular and filtrate leucocyticin liver of group 16 as show in fig .3 A. All of these investigate have no evidence of malignancy were seenin (Fig 3A).

Kidney section of group 12 and 13 showed a mark vacuolated of glomerular tuft and developed into formation degenerated renal cast among later group (Fig.3Bc). Severe vacuolated glomerular tuft andnecrotic renal tubules among G 14, G15 and G16 that fed on exhausted frying oils up to 30 frying cycle.

Some infiltration of focal area of leucocytic in heart tissue considers feed on fresh CO+PO. It could developed markedly among later groups of 13, 14, 15 and 16 into focal area of hemorrhage and infiltration of leucocytic cells (Fig .3C).

Similar to previous groups from G1:G11 in this current study are resulting a hyperactivity of the mucous secreting glands in their intestine sections.

Regarding to investigate the teste of groups fed on fresh and frying oils of CO+PO, were similar in formal edema and interstitial congested of blood vessel in G13:G16 (Fig .3D). Except for group 12 show some of degeneration of spermatogonia (Fig .3D). For these previous investigation , similar to study by Farag,*et al*., (2010) using feeding trail of albino rats at blends of sunflower oil and palm oil or canola oil were obtained by mixing the oils at the volume ratios of 60: 40 and 20: 80, (v/v) then heated at 180˚C ± 5˚C for 10 and 20 h in the presence of air and moisture.The nutritional experiments demonstrate that the administration of non-fried sunflower oil alone did not cause any alteration on rat liver, kidney and heart tissues. Whilst, other oils and oil blends both non-fried and fried heated for 10 and 20 h possessed variable detrimental effects on rat organs. In addition, the longer heated oil (20 h) displayed more changes in rat organs than that of heated oil for the shorter period (10 h).

Canola oils are very high in monounsaturated fats and are low risk fats, as shown in animal models and through the finding that the incidence of coronary heart is lower in the Mediterranean region, where such oils are frequently used (Weisburger, 2000). From all of these investigates have no evidence of malignancy were seen among all groups in the current study. The suggestion regard to (Table B), illustrated that, CO and PO contains a higher ratio of monounsaturated fatty acids (MSFA), which shown a higher stability of oils during oxidation and prevent formation a toxic substances in the frying oils. From this later discus, can be used CO and palm olein in blend to improve the health status fromsuch fried foods and their oils. This later result is appreciated by Zeb andUllah (2015) oleic acid is the main fatty acid responsible for stability against the thermal stress. Sea buckthorn seed oil showed lower p-anisidine value compared to oxidized ghee and thus may be helpful in oxidation and more beneficial to health.

CONCLUSION

One of the aim of this study to evaluate the effect of the deteriorative changes on the frying oils. The blending of the canola oil with sunflower SF or palm olein PO in the volume ratio of 50:50 led to a slower degradation to the obtained with PO, based on the chemical parameter. This stability of canola oil was increased to blend with POoil. Canola oil in the blends shown a higher nourished versatile oil, it contain corresponds omega -3 fatty acids. Both canola and palm olein blend improve the nutritional quality of fried oils. Lipid profile of dietary rats on canola oil and palm olein showing improve the nutritional quality. No significant alteration was showed among all groups fed on canola oil and its blends with sunflower and palm olein. Some alteration was occurred throughout received groups on later exhausted frying oil cycles from 20 and 30th. Overall conclusion, it could be claim that, canola oil and palm olein blend can use safe with minimum hazardous of hazardous up to 10th frying cycles.

REFERENCES

Ackman, R. G.(1990).Canola Fatty Acids-an Ideal Mixture for Health, Nutrition and Food Use. In: Canola and Rapeseed (Shahidi, F. "Ed"). Van Nostrand Reinhold, New York, pp: 81–89.

Aguila, M.B. Pinheiro, A.R. Aquino, J.C.F. Gomes, A.P. &Mandarim-de-Lacerda C.A. (2005). Different edible oil beneficial effects (canola oil, fish oil, palm oil, olive oil, and soybean oil) on spontaneously hypertensive rat glomerular enlargement and glomeruli number. Prostaglandins & other Lipid Mediators 76: 74–85

Akhigbe R.E., Azeez O.M., Ige S.F., Oyeyipo I.P., Ajao F.O., &Soladoye A.O.,(2008). "Hemorheological effect of long-term administration of oral contraceptive in rats". International Journal of Pharmacology, 4: 403-406.

Akhigbe, R.E., Azeez, O.M., Ige, S.F., Oyeyipo, I.P., Ajao, F.O., &Soladoye, A.O. (2008). Hematological effect of long-term administration of oral contraceptive in rats. International Journal of Pharmacology 4: 403-406.

Ammawath, W., Chen Man, Y.B., Abdul Rahman, R.B, Baharin, B.S. (2006). A Fourier transforms spectroscopic method for determining butylated hydroxytoluene in palm olein and palm oil. Journal of the American Oil Chemists' Society.83:187–191.

AOCS, (2013). Official Methods and Recommended Practices of the AOCS. Ed. by David Firestone 6th Ed. ISBN978-1893997-74-5. Binder. USA.

Banchroft, J., Stevens, A. & Turner, D. (1996).Theory and Practice of Histological Techniques, fourth ed. Churchill Livingstone, New York, London, San Francisco, Tokyo.

Bastida, S., &Sanchez-Muniz, F.J. (2001). Thermal oxidation of olive oil, sunflower oil and a mix of both oils during forty discontinuous domestic fryings of different foods. Food SciTechnol 7:15–21.

Baux.,A., Hebeisen H.,& Pellet D.(2008).Effects of minimal temperatures on low-linolenic rapeseed oil fatty-acid composition. Europ. J. Agronomy,29 : 102–107.

Beyer, R. E., Nordenbrand, K., Ernster, L. (1987). The function of coenzyme Q in free radical production and as an antioxidant: a review. *ChemicaScripta*. 27:145;

Bhagavan, H.N. & Chopra, R.K. (2006). Coenzyme Q10: absorption, tissue uptake, metabolism and pharmacokinetics. Free Radic Res., 40(5):445-53.

Burenjargal, M. &Totani, N. (2009). Cytotoxic Compounds Generated in Heated Oil and Assimilation of Oil in Wistar Rats. J. Oleo Sci. 58 (1):1-7.

Casal, S., Malheiro, R., Sendas, A., Oliveira, B. P., & Pereira, J. A. (2010). Olive oil stability under deep-frying conditions. Food and Chemical Toxicology, 48(10): 2972–2979.

Chisolm, G. M. & Steinberg, D. (2000). The oxidative modification hypothesis of atherogenesis: an overview. Free Radical and Biological Medicine, 28:1815–1826.

Choe, E. & Min, D. B. (2007). Chemistry of deep-fat frying oils. Journal of Food Science, 72(5): R77–R86

Crane, F.L. (2001).Biochemical functions of coenzyme Q10. J Am College Nutr.20:591–598.

Dobarganes, C. & Marquez – Ruiz, G. (2003). Oxidized fats in foods. CurrOpinClinNutrMetab Care 6:157-163.

Elreffaei, W. H. M., El-Sebeay, A.S., E. M. Ragheb, H. M. Elghandour&Badr, S. E. A. (2015) Effect of Deep-Fat Frying Performance on Canola, Palmolein and Sunflower Oil Blends: A) Chemical Parameters. AlexandriaJournal of Food Scienceand Technology, 12 (2):63-77.

Farag, R. S., Abdel-Latif, M. S., Basuny, A. M. M. &Abd El Hakeem, B. S. (2010). Effect of non-fried and fried oils of varied fatty acid composition on rat organs. Agric. Biol. J. N. Am., 1(4): 501-509.

Farhoosh, R., Kenari, R. E. &Poorazrang, H. (2009). Frying stability of canola oil blended with palm olein, olive, and corn oils. Journal of the American Oil Chemists Society 86:71–76.

Finlayson, N., Hayes P. & Simpson K., (1999). Diseases of the liver and biliary system. Haslett C, Chilvers ER, Hunter JAA, Boon NA, editors. Davidson's Principles and Practice of Medicine, pp: 683-736.

Forsmark, A. P., Dallner, G., &Ernster, L. (1995). Endogenous ubiquinol prevents protein modification accompanying lipid peroxidation in heart submitochondrial particles. *Free Radic. Biol. Med*. 19:749– 756.

Friedewal, W., Levy, R., Fredrickson, D.S., (1972). Estimation of concentration of low density lipoproteins in plasma without use of ultracentrifuge. Clin. Chem. 18:449–502.

Friedman, R.B. & Young D.S. (1997)."Effects of disease on clinical laboratory tests, 3th ed. AACC".

Galinier, A., Carriere, A., Fernandez, Y., Bessac, A.M., Caspar-Bauguil, S., Periquet, B., Comtat, M., Thouvenot, J.P.&Casteilla, L. (2004). Biological validation of coenzyme Q redox state by HPLC-EC measurement: relationship between coenzyme Q redox state and coenzyme Q content in rat tissues. FEBS Lett. 578: 53–57.

Garrido-Polonio, C., Garcia-Linares, M. C., Garcia-Arias, M. T., Lopez-Varela, S., Garcia-Fernandez, M. C. &Terpstra, A. H. (2004). Thermally oxidized sunflower-seed oil increases liver and serum peroxidation and modifies lipoprotein composition in rats. British Journal of Nutrition, 92(2):257–265.

Glick M.R., Ryder K.W., &Jackson SA. (1986). Graphocalcomparisons of interferences in clinical chemistry instrumentation. Clin. Chem. 32:470–474.

Gotoh, N. & Wada, S. (2006). The importance of peroxide value in assessing food quality and food safety. JAOCS, 83: 85.

Grossi, G., Bargossi, A.M., Fiorella, P.L., Piazzi, S., Battino, M. & Bianchi, G.P. (1992). Improved HPLC method for the determination of coenzyme Q. J Chromatogr; 593:217–26.

Gupta, M. K. (2005). Frying oils. In: Bailey's Industrial Oil and Fat Products, Hoboken: John Wiley & Sons 6th edn, Vol. 4 (edited by F. Shahidi). Pp. 1–31.

Hargreaves, I. P. (2003).Ubiquinone: cholesterol's reclusive cousin. Ann ClinBiochem; 40:207–218.

Harris, W.S. (2006). The omega-6/omega-3 ratio and cardiovascular disease risk: uses and abuses. CurrAtherosclerRep . 8(6): 453-459

Hemmi, N., Hagavan, B, & Chopra, R. K.(2005). Potential role of ubiquinone (coenzyme Q10) in pediatric cardiomyopathy. Clinical Nutrition, 24: 331–338.

Henry, R.J. (1974). Clinical Chemistry, Principles and Technics, second ed., Harper and Row, p. 522-525.

Hock, C. E., Holahan, M. A. & Reibel, D. K. (1987). Effect of dietary fish oil on myocardial phospholipids and myocardial ischemic damage American Journal of Physiology - Heart and Circulatory Physiology Published, 252 (3) : H554 - H560.

Hodges, S., Hertz, N., Lockwood, K. & Lister, R. (1999). CoQ10: could it have a role in cancer management? Bio Factors; 9:365–370.

Igiri, A. O., Ibegbu, A. O. &Osim, E. E. (1994). The morphological and histological changes in the small intestines induced by chronic consumption of palm. Journal of Applied Science, 4:144–153.

Johnson, G.H, Keast, D.R. & Kris-Etherton, P.M. (2007). Dietary modeling shows that the substitution of canola oil for fats commonly used in the United States would increase compliance with dietary recommendations for fatty acids. J Am Diet Assoc., 107:1726–1734.

Kelso, G.F., Porteous, C.M., Coulter, C.V., Hughes, G., Porteous, W.K.&Ledgerwood, E.C. (2001). Selective targeting of a redox active ubiquinone to mitochondria within cells: antioxidant and antiapoptotic properties.J Biol Chem,276:4588–96.

Kenneday, A., Martinez, K., Schmidt, S., Mandrup, S., LaPoint, K., & McIntosh, M. (2010) Anti-obesity mechanisms of action of conjugated linoleic acid. Journal of Nutritional Biochemistry, 21:171–179.

Kihara, A. (2012).Very long-chain fatty acids: elongation, physiology and related disorders. J Biochem; 152:387–395.

Kumar, A., Kaur, H., Devi, P.& Mohan, V. (2009). Role of coenzyme Q10 (CoQ10) in cardiac disease, hypertension and Meniere-like syndrome. Pharmacol. Ther., 124:259–268.

Lai, L., Kang J.X., Li, R., Wang, J., Witt, W.T., & Yong, H.Y. (2006). Generation of cloned transgenic pigs rich in n-3 fatty acids. Nat Biotechnol. 4: 433–6.

Mariod, A.A., Matthäus, B., Eichner, K. & Hussein, I.H. (2005) Improving the oxidative stability of sunflower oil by blending with *Sclerocaryabirrea*and*Aspongopusviduatus* oils. Journal of Food Lipids, 12, 150-158.

Masso-Welch, P.A., Zangan, D. Ip, C.., Vaughan, M.M, Shoemaker,S.F., McGee,S.O. &Ip, M.M. (2004). Isomers of conjugated linoleic acid differ in their effects on angiogenesis and survival of mouse mammary adipose vasculature. J. Nutr., 134: 299–307.

McCarthy, S., Somayajulu, M., Sikorska, M., Borowy-Borowski, H., &Pandey, S. (2004). Paraquat induces oxidative stress and neuronal cell death; neuroprotection by water-soluble Coenzyme Q10. ToxicolApplPharmacol, 201:21-31

Most, M. M. ,Tulley, R., Morales S., &Lefevre, M. (2005). Rice bran oil, not fiber, lowers cholesterol in humans. Am J Clin.Nutr., 81:64–8.

Naghshineh, M., Ariffin, A., Ghazali, H., Mirhosseini, H., Kuntom, A.& Mohammad, A.S. (2009) Monitoring the change patterns of physicochemical properties of oil blend as function of storage time. Journal of Food, Agriculture & Environment, 7 (3&4): 1 2 0 - 1 2 5.

Przybylski, R., Mag, T., Eskin, N.A.M. & McDonald, B.E (2002). Canola Oil. University of Manitoba, Winnipeg, Manitoba, Canada.

Ray, K. K., Cannon, C.P., Cairns, R., Morrow, D. A., Ridker, P. M. &Braunwald, E. *(*2009). Prognostic Utility of ApoB/AI, Total Cholesterol/HDL, Non-HDL Cholesterol, or hs-CRP as Predictors of Clinical Risk in Patients Receiving Statin Therapy After Acute Coronary Syndromes Results From PROVE IT–TIMI 22. ArteriosclerThrombVasc Biol., 29: 424-430.

Reasor, M. J. (1981). Drug –induced lipidosis and the alveolar macrophage. Toxicology ,20:1-33

Richmond, W. (1973). Preparation and properties of a cholesterol oxidase from Nocardia sp. Medical analysis. Clin. Chem. 19, 1350 (Ed. AACC).

Saka, W.A., Akhigbe, R.E., Popoola, O.T. &Oyekunle, O.S. (2012). "Changes in serum electrolytes, urea, and creatinine in *Aloe vera*-treated rats". Journal of Pharmacology, 4: 78-81.

Salvatore, P. & McLennan P. L. (2002). Cardiac Membrane Fatty Acid Composition Modulates Myocardial Oxygen Consumption and Postischemic Recovery of Contractile Function. Circulation. 105:2303-2308.

SAS. (2003). Version 9.1, SAS institute INC Cary, NC 27513, USA

Sealls, W., Gonzalez, M., Julia Brosnan, M. P. N.,& Black, C. C. D., (2008) Dietary polyunsaturated fatty acids (C18:2 ω6 and C18:3 ω3) do not suppress hepatic lipogenesis. Biochimica et BiophysicaActa,1781: 406–414

Sohet, F. M., Neyrinck, A.M., Pachikian, B. D., de Backer, F. C., Bindels, L. B., Niklowitz, P., Menke, T., Cani, P. D. &Delzenne, N.M. (2009).Coenzyme Q10 supplementation lowers hepatic oxidative stress and inflammation associated with diet-induced obesity in mice. Biochemical Pharmacology,78 :1391–1400.

Sohmiya, M., Tanaka, M., Tak, N. W., Yanagisawa, M., Tanino, Y.,& Suzuki, Y. (2004). Redox status of plasma coenzyme Q10 indicates elevated systemic oxidative stress in Parkinson's disease. J NeurolSci, 223(2):161–166.

Sundram, K., French, M. A., &Clandinin, M. T. (2003). Exchanging partially hydrogenated fat for palmitic acid in the diet increases LDL-cholesterol and endogenous cholesterol synthesis in normocholoesterolemic women. European Journal of Nutrition, 42:188-194.

Surtees, S. J., Bastable, M. D., Gietzen, T. W., Model, D. G., Carmichael, D. J. S., Bordoli, G., & Kidner, P. H. (1979) . A Biochemical and Clinical Comparison of two Commercially Avail ableCreatine Kinase IsoEne MB Assay Kits Suita ble for use in the RoutineMedical Laboratory.An nals of Clinical Biochemistry, 16(16): 197-204.

Truswell, S. A., Ch B. M. B., Naswrin Choudhury M. B., & Dave C.K. R. (1992).double blind comparison of plasma lipids in healthy subjects eating potato crisps fried in palmolein or canola oil. Nutrition research, 12(1):43-52,

Vanderhoof, J.A., Park, J. H., Herrington, M.K., & Adrian, T.E. (1994).Effects of dietary menhaden oil on mucosal adaptation after small bowel resection in rats. Gastroenterology, 106(1):94-9.

Velasco, J., Marmesat, S., Bordeaux, O., Marquez-Ruiz, G., &Dobarganes, C. (2004). Formation and evolution of monoepoxy fatty acids in thermoxidized olive and sunflower oils and quantitation in used frying oils from restaurants and fried food outlets. Journal of Agriculture and Food Chemistry, 52(14):4438–4443.

Wang, J., Esther, M., John, P. L., Horn-Ross & Ingles.S. A. (2008).Dietary Fat, Cooking Fat, and Breast Cancer Risk in a Multiethnic Population. Nutrition and Cancer, 60(4), 492–504.

Warner, K., &Gehring, M. M. (2009). High-temperature natural antioxidant improves soy oil for frying. Journal of Food Science, 74(6):C500–C505.

Weisburger, J.H., (2000). Eat to live, not live to eat. Nutrition 16:767– 773.

Yalcin, A., Kilinc, E., Sagcan, A.,&Kultursay, H. (2004). Coenzyme Q10 concentrations in coronary artery disease. Clin. Biochem. 37:706–709.

Zeb, A., &Ullah, S. (2015). Sea buckthorn seed oil protects against the oxidative stress produced by thermally oxidized lipids. Food Chemistry, 186:6–12.

تأثيرعمليةالقلي العميق لزيت الكانولا ومخاليطه مع زيت أوليين النخيل و زيت دوار الشمس: ب- دراسات بيولوجية وتغذوية

وائل حلمي موسي الرفاعي ، أشرف صابر السبيعي ، حنان محمد احمد الغندور ، إيمان محمود راغب و شريف السيد علي بدر

مركز البحوث الزراعية - المركز الأقليمي للأغذية والأعلاف - مصر

يعد زيت الكانولا من الزيوتغير مشبعة الصحية لإحتوائه علي نسب أعلي من الأوميجا-٣ وخاصة حمض اللينولينك مقارنة بالزيوت الاخري وهويلي زيت بذور الكتان في ذلكويتميزبخفة الطعم القوام وأرتفاع نقطة التدخين و شيوعأستخدامه في عمليات الطهي. ولكنه يعتبر ذا ثبات حراريمنخفض وقد تؤدي عمليات الخلط بينة وبين زيت دوار الشمس وزيت أوليين النخيل الي زيادة صلاحيتة للقلي وهي من الطرق الشائعة في تغذية الأنسان. تطرقت الدراسة الي تقييم زيت الكانولا مع مخاليطة باردا و بعد قليةلمدد مختلفة حتي ٣٠ قلية معرقائق البطاطس . تم تغذية ١٦ مجموعة من فئران الألبينو وزن ٥٠-٧٠ جم علي تلك الخلطات من الزيوت الناتجة بعد الخلط وبعد القلي لمرات متعددتو لمدة ٩٠ يومامقسمة الي فترتين كل منهما ٤٥ يومامع إجراء التحاليل البيوكميائية والهيماتولوجي والهستولوجي المختلفة. وخلصت نتائج تحاليل الدم البيوكميائية الي حدوث زيادة معنوية خلال المرحلة الاولي من التجربة لكل من وظائف الكبد والكلي والقلب وذلك عند مقارنتها بالمجموعة االضابطة المغذاة علي زيت الذرة (الزيت المعتاد لتغذية الفئران).

أظهرت تلك المجموعات عدم وجود تغير معنوي في المرحلة الثانية من التجربة مقارنة بالمجموعة الضابطةأيضا لنفس الوظائف للكبد والكلي والقلب. كذلك حدث تغير معنوي في المرحلة االأولي من التجربة مابين المجموعات التجريبية و المجموعة الضابطة في كلمن ليبيدات الدم ومؤشر التصلب للشرايين(AtherogenicIndex).

حدث أنخفاض معنوي للدهون عالية الكثافة (الكولسترول الجيد) في بداية المرحلة الأولي عند مقارنتها بالمرحلة الثانية من التجربة وبصفة عامة تعتبر النتائج المتحصل عليها من مجموعات التغذية علي زيت الكانولا أعلي من المجموعات التجريبية الأخري والمجموعة الضابطة . كما تبين حدوثأنخفاض في الجلسريدات الثلاثية مابين مجاميع الفئران المغذاة علي مخلوط الكانولا وزيت النخيل الطازج والمستخدم في عملية القلي مابين المجموعات التجريبية من ١٢ حتي المجموعة ١٦ في المرحلة الأولي من الدراسة .

سجلت النتائج حدوث أنخفاض مابين المجموعات المغذاة علي الزيوت المستهلكه للقلي من القلية ٣٠ في خفض مستوي مضادات الأكسدة الداخلية وهي Coenzyme Q10 وكانت لأقل من ٧٠ ملليمول/ مل للمجموعاتالتجريبية ٦ و ١١ و١٦ .

ومن تحاليل الهيماتولوجي أظهرت النتائج حدوثانخفاض ملحوظ في نسبة خلايا الليمف لاقل من ٨% بين المجموعات المغذاة علي الخليط غير المستخدم للقلي من الكانولا وزيت الأوليين و حتي بين المجموعات المغذاة علي نفس الخليط بعد استخدامه للقلي للمجموعات ١٣ و ١٤ و ١٦ مقارنة بالمجموعة الضابطة وفي المرحلة الاولي من التجربة.

وأظهرت جميع المجموعات تغيرات هستولوجية لمختلف الأعضاء مثل الكبد و الكلي والقلب والأمعاء والخصية وكانت أكثر المجموعات تأثرا هي المجموعات المغذاة علي زيت القلية العشرين والثلاثين .

خلصت هذه الدراسة الي ان خلط زيت الكانولا مع الاوليين يحسن من الحالة الصحية والتغذوية للفئران المغذاة علي تلك الزيوت حتي القلية العاشرة.