GRASAS Y ACEITES, 63 (3),
JULIO-SEPTIEMBRE, 303-312, 2012,
ISSN: 0017-3495
DOI: 10.3989/gya.011412

Studies on the hypolipidemic effects of Coconut oil when blended with Tiger nut oil and fed to albino rats

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RESUMEN

Estudios sobre los efectos hipolipidémicos en ratas albinas alimentadas con aceite de coco mezclado con aceite de chufa.

La hiperlipidemia es un factor de riesgo predominante para la aterosclerosis y las enfermedades cardiovasculares asociadas (ECV). Las directrices internacionales emitidas por la Organización Mundial de la Salud recomiendan una reducción de grasas saturadas y colesterol, como medio para prevenir la hipercolesterolemia y las enfermedades cardiovasculares. El principal objetivo de la presente investigación fue evaluar los efectos de una alimentación conteniendo mezclas de aceites, que consiste en aceite de coco (CNO) con diferentes proporciones de aceite de chufa (TNO), sobre los niveles de lípidos en suero en ratas albinas. Se realizó un análisis GLC para determinar la composición de ácidos grasos de los aceites mezclados. Los aceites se obtuvieron mezclando aceite de chufa con aceite de coco en las relaciones:100:0, 70:30, 50:50, 25:75, 10:90 y 0:100 (volumen:volumen). Cincuenta y seis ratas albinas macho se dividieron aleatoriamente en 7 grupos de 8 ratas cada uno, según el tipo de aceite y se alimentaron durante un período de hasta 10 semanas con las mezclas de aceites. Se determinó el colesterol total (T-Ch), colesterol en lipoproteínas de alta densidad (HDL-Ch), colesterol en lipoproteínas de baja densidad (LDL-Ch), triglicéridos (TG) y el índice aterogénico (IA). Los resultados mostraron cambios no significativos en todos los parámetros nutricionales entre el grupo control y las ratas alimentadas con los aceites ensayados. Los resultados también indican que el aceite de coco tiene un 86% de ácidos grasos saturados. TNO por otro lado contiene un 66% de ácido oleico. Por lo tanto, una mezcla de aceite de coco con aceite de chufa reduce la relación de ácidos grasos saturados a insaturados del CNO. Las ratas alimentadas con las mezclas de aceites mostraron niveles significativamente mas bajos de colesterol en suero en comparación con los de CNO. Los niveles de HDL mejoraron ligeramente en las ratas alimentadas con las mezclas de aceites. El colesterol total y colesterol LDL estuvieron controlados cuando las proporciones TNO / CNO variaron entre el 25/75 a 70/30. Esto se reflejó en el índice aterogénico calculado. Cambios similares también se observaron con los niveles de triglicéridos en suero.

PALABRAS CLAVE: Ácidos grasos – Chufa – Coco – Hipercolesterolemia – Lípidos en sangre — Mezclas de aceites – Ratas albinas.

SUMMARY

Studies on the hypolipidemic effects of Coconut oil when blended with Tiger nut oil and fed to albino rats.

Hyperlipidemia is a predominant risk factor for atherosclerosis and associated cardiovascular diseases (CVD). The international guidelines issued by the World Health Organization recommend a reduction in dietary saturated fat and cholesterol intake as a means to prevent hypercholesterolemia and CVD. The main objective of the current investigation was to evaluate the effects of feeding blended oils consisting of coconut oil (CNO) with different proportions of Tiger nut oil (TNO) on serum lipid levels in Albino rats. GLC analysis was performed to illustrate the fatty acid composition of the blended oils. Blended oils were obtained by mixing tiger nut oil with coconut oil at the volume ratios of 100:0, 70:30, 50:50, 25:75, 10:90 and 0:100. Fifty-six male albino rats were randomly divided into 7 groups of 8 rats each according to the oil type. The blended oils were fed to rats for a period of up to 10 weeks. Total cholesterol (T-Ch), high-density lipoprotein cholesterol (HDL-Ch), low-density lipoprotein cholesterol (LDL-Ch), and triglycerides (TG), were determined. The atherogenic Index (AI) was calculated. The results showed that non-significant changes in all nutritional parameters were observed between the control group and the rats fed with the tested oils. The results also indicate that coconut oil had 86% saturated fatty acids. On TNO contains 66% oleic acid. Therefore, blending coconut oil with tiger nut oil can reduce the proportions of saturated to unsaturated fatty acids in CNO. The rats that were fed blended oils showed significantly reduced levels of serum cholesterol as compared to those fed CNO. The HDL levels were marginally enhanced in the rats that were fed blended oils. The total cholesterol and LDL cholesterol levels were controlled when TNO/CNO proportions varied between 25/75 and 70/30. This was reflected in the calculation of the atherogenic index. Similar changes were observed with serum triglyceride levels.

KEY-WORDS: Albino rats – Blood lipid – Coconut – Fatty acids – Hypercholesterolemia – Oil blends – Tiger nut.

1. INTRODUCTION

Hypercholesterolemia and its associated cardiovascular diseases (CVD) represent one of the greatest worldwide economic, social and

medical challenges that we are currently facing (Olshansky *et al.*, 2005). The relationship between plasma lipid and lipoprotein concentrations and the risk of developing cardiovascular disease (CVD) on the basis of dietary fat type is well documented (Krauss *et al.*, 2000). A high plasma concentration of total cholesterol, triacylglycerol, and LDL cholesterol and a low plasma concentration of HDL cholesterol are considered important risk factors for the development of coronary diseases (Kannel *et al.*, 1979), and these plasma indexes or biomarkers must be jointly considered in the assessment of risk for human populations (American Heart Association scientific cholesterol levels, 2004). Dietary fat selection is known to exert a major influence on circulating cholesterol levels; they are raised with the consumption of fats containing saturated fatty acids (SFA), and reduced with fats rich in monounsaturated (MUFA) and polyunsaturated (PUFA) fatty acids (Mensink, and Katan, 1992). Excessive cholesterol builds up in the blood, leading to plaque formation and hardening of the arteries. This condition is known as atherosclerosis, which is the leading cause of heart disease, stroke, and heart attacks (U.S. Food and Drug Administration, 2004).

Coconut oil is an edible oil extracted from the kernel or meat of mature coconuts harvested from the coconut palm (Cocos nucifera). Throughout tropical regions, it has provided the primary source of fat in the diets of millions of people for generations. It has various applications in food, medicine, and industry. Coconut oil is very heat-stable, which makes it suited to methods of cooking at high temperatures such as frying. Because of its stability, it is slow to oxidize and, thus, resistant to rancidity, lasting up to two years due to its high saturated fat content (Fife, 2005). It consists of a mixture of triglycerides containing only short and medium chain saturated fatty acids (92%) and unsaturated fatty acids (8%) (Che Mann and Marina, 2006). Coconut oil raises cholesterol levels when compared with other fats (Keys *et al.*, 1965; Reiser *et al.*, 1985).

Tigernut (Cyperus esculentus L.) is an underutilized crop which belongs to the division-Magnoliophyta, classliliopsida, order – cyperales and Cyperaceae family and was found to be a cosmopolitan perennial crop of the same genus as the papyrus plant. Tiger nut is not really a nut but a small tuber, first discovered some 4000 years ago in ancient Egypt and is cultivated today in China, Spain and West Africa for its small tuberous rhizomes which are eaten raw or roasted, used as hog feed or pressed for its juice to make a beverage. (Belewu and Belewu, 2007). Non-drying oil (usually called chufa) is equally obtained from the rhizome. (Belewu and Belewu, 2007). Tigernuts are valued for their highly nutritious starch content, dietary fiber and carbohydrates (Umeneand Enebeli, 1997) and are rich in sucrose (17.4-20.0%), fat (25.5%), protein (8.0%) (Kordyias, 1990; Temple *et al.*, 1990). Tiger nut is also rich in mineral contents such as sodium, calcium, potassium, magnesium, zinc and traces of copper (Oladele and Aina, 2007). The dietary fiber content of tigernut is effective in the treatment and prevention of diseases such as colon cancer, coronary heart diseases, obesity, diabetes and gastro-intestinal disorders (Anderson *et al.*, 1994). The very high fiber content combined with a delicious taste; make them ideal for healthy eating (Osagie and Eka, 1998). Since the tubers contain 20-36% oil, C. esculentus has been suggested as a potential oil crop for the production of biodiesel. (Zhang *et al.*, 1996).The nut was found to be rich in myristic acid, oleic acid, linoleic acid (Zhang *et al.*, 1996; Eteshola and Oraedu, 1996). Tiger nut oil is a monounsaturated oil similar to olive oil (both contain high levels of oleic acid), therefore, nutritional experiments were performed to evaluate the efficacy of tiger nut (Cyperus esculentus) oil, coconut (Cocos nucifera) oil and binary mixtures of them on the serum lipid profile of Albino rats. Apart from that, the fatty acid compositions of tiger nut (Cyperus esculentus) oil, coconut (Cocos nucifera) oil and binary mixtures of the two were quantified by GLC to indicate their atherogenic effects.

2. MATERIALS AND METHODS

2.1. Materials

Tiger nut tubers (Cyperus esulentus) were obtained from Harraz Spices and Herbs Co. Cairo, Egypt. Coconut oil was obtained from El Hawag Company for extract oils, Badr city, Egypt. Corn oil was obtained from Arma for Food Industries, 10th of Ramadan, Egypt. A diet containing 10% corn oil was used as the control.

All reagents and chemicals used in this work were of analytical grade.

2.2. Methods

Tiger nut oil extraction

Dried Tiger nut tubers (*Cyperus esulentus*) were crushed and pressed using a hydraulics laboratory press model C S/N 37000 – 156 Freds from Carver (WI, USA). Anhydrous sodium sulphate was added to the extracted oil and allowed to stand for 30 min to remove excess residual moisture. The resulting dry oil was centrifuged at 1080 g and filtered through Whitman filter paper No.1 and kept in a brown glass bottle at 4 $\pm$ 0.5 °C.

Preparation of mixtures

Tiger nut oil (TO) was blended with coconut oil (CO) in varying proportions. The following TO: CO (% v/v) blends were prepared; 100:0, 70:30, 50:50, 25:75, 10:90 and 0:100. The oil blends were mixed

with a magnetic stirrer for 20 minutes at 30 °C and stored in brown glass bottles at 4 ± 0.5 °C.

Fatty acid compositions of tiger nut oil, Coconut oil and binary mixtures of them

Capillary gas chromatography (HP 6890) was used for the qualitative and quantitative determinations of the fatty acids of the oil samples and reported in relative area percentages. Fatty acids were transesterified into their corresponding fatty acid methyl esters (FAMEs) by shaking a solution of oil (ca. 0.1 g) in heptane (2 mL) with a solution of methanolic potassium hydroxide (0.2 mL, 2N). The FAMEs were identified using a gas chromatograph equipped with DB-23 capillary column (60 m × 0.32 mm × 0.25 μm film thickness) and a flame ionization detector. The nitrogen flow rate was 3 mL/min; hydrogen and airflow rates were 40 and, 450 $mLmin^{-1}$, respectively. The oven temperature was programmed from 150 °C to 170 °C at a rate of 10 min, then raised to 192 °C at a rate of 5 °C min^{-1} and kept at this temperature for 5 min and then raised again to 220 °C at a rate of 10 °C min^{-1} and kept at this temperature for 3 min. The injector and the detector temperatures were 230 °C and 250 °C, respectively. MEFAs were identified by comparing their retention times with a known fatty acid standard mixture. Peak areas were automatically computed by an integrator.

Nutritional experiment

The animal experiments were performed with the approval of the Ethics Committee for experimental animals of the Food Technology Research Institute; Agricultural Research Center, Giza, Egypt. A total of 56 Albino male rats with an average weight of 90-100 g were raised in the animal house of Food Technology Research Institute; Agricultural Research Center, Giza, Egypt. The rats were housed in temperature-controlled rooms (25 ± 2 °C) with constant humidity (55 ± 5%) and a 12 h/12h light/dark cycle prior to experimental protocols. All animals were allowed to drink water ad libitum. The animals were fed a basal diet for 15 days as an adaptation period. The basal diet was formulated according to (A.O.A.C., 2000) and consisted of casein (15%), corn oil (10%), cellulose (5%), salt mixture (4%), vitamin mixture (1%) and starch (65%). Casein is characterized by its low content of sulphur amino acids; hence L methionine was added to the basal diet at the level of 4.6 g Kg^{-1} diet. The composition of the vitamin and salt mixtures used was similar to that reported by (A.O.A.C., 2000; Reeves *et al.*, 1993) respectively. The rats were randomly divided into 7 groups of 8 rats each according to oil type. The oil samples under study in the nutritional experiments were used instead of the corn oil in the basal diet. During the nutritional experiment, body weight and feed intake were recorded weekly. At the end of the experiment, total food intake, body weight gain and the food efficiency ratio were calculated.

Blood sampling

Blood samples were taken at the start of the experiment and at 2, 4, 6, 8 and 10 weeks from the beginning of the experiment. Blood samples from each rat were obtained from the orbital pleux by means of a capillary tube (1-1.5 m). The blood of the rats from each group were centrifuged at 1100 Xg for 20 min to obtain the sera and kept in a deep freezer (–18 °C) until analysis.

Lipid analysis

Total cholesterol (T-Ch), high-density lipoprotein cholesterol (HDL-Ch), low-density lipoprotein cholesterol (LDL-Ch), and triglycerides (TG), were determined according to Roeschlau *et al.* (1974); Assmann (1979); Levy (1981) and Fossati and Prencipe (1982), respectively. The atherogenic Index (AI) was calculated using the following equation as described by Dobiasova and Frohlich (2001).

$$\text{Atherogenic Index (AI)} = \text{Log (TG/HDL-Ch)}$$

Statistical analysis

Data are expressed as mean ± SD. Data were statistically analyzed in completely randomized design in factorial arrangement according to the procedures outlined by Gómez and Gómez (1984) and the treatment means were compared by least significant differences (L.S.D) and Duncan multiple range using an SPSS program package. Data are presented in text and tables as the means of five determinations.

3. RESULTS AND DISCUSSION

3.1. Fatty acid composition

The results in Table 1 show the fatty acid composition of tiger nut oil, coconut oil and binary mixtures of them. Coconut oil was characterized by the presence of high levels of lower chain fatty acids C8:0, C10:0 and C12:0. The most prominent fatty acids in coconut oil were lauric (42.90%), myristic (20.20%) and palmitic (10.76%) acids, while tiger nut oil has an abundance of oleic (66.22%) acid. Lower, but still considerable, concentrations of C18:2 n-6 (13.10%) were found in the tiger nut oil sample. Coconut oil had the highest saturated fatty acid level (86.28%) and tiger nut oil was very rich in mono-unsaturated fatty acids MUFA (66.22%). Therefore, blending coconut oil with tiger nut oil as a source of MUFA was suggested for improving the nutritional and health properties of coconut oil. The blending of coconut oil with different levels of tiger nut oil caused a

Table 1
Fatty acid compositions of Tiger nut oil (TNO), coconut oil (CNO) and binary mixtures of them

Fatty acid	TNO	CNO	TNO + CNO (v/v)				
			(70/30)	(50/50)	(25/75)	(10/90)	LSD at 0.05
C8:0	ND^{f}	5.12^{a} ± 0.04	1.53^{e} ± 0.03	2.56^{d} ± 0.02	3.84^{c} ± 0.05	4.60^{b} ± 0.02	0.0553
C10:0	ND^{f}	4.30^{a} ± 0.04	1.30^{e} ± 0.04	2.15^{d} ± 0.04	3.22^{c} ± 0.04	3.87^{b} ± 0.04	0.0637
C12:0	ND^{f}	42.9^{a} ± 0.63	12.87^{e} ± 0.29	21.45^{d} ± 0.09	32.17^{c} ± 0.22	38.61^{b} ± 0.28	0.5699
C14:0	ND^{f}	20.20 ± 0.16	6.00 ± 0.08	10.10 ± 0.18	15.16 ± 0.00	18.18 ± 0.23	0.2484
C16:0	15.58^{a} ± 0.11	10.76^{f} ± 0.09	14.10^{b} ± 0.26	13.17^{c} ± 0.05	11.96^{d} ± 0.06	11.23^{e} ± 0.00	0.2225
C16:1	ND	ND	ND	ND	ND	ND	–
C18:0	4.00^{a} ± 0.05	3.00^{f} ± 0.06	3.80^{b} ± 0.04	3.50^{c} ± 0.01	3.25^{d} ± 0.07	3.10^{e} ± 0.02	0.0831
C18:1	66.22^{a} ± 0.82	9.50^{f} ± 0.09	49.20^{b} ± 0.66	37.86^{c} ± 0.72	23.67^{d} ± 0.13	15.17^{e} ± 0.03	0.9399
C18:2	13.10^{a} ± 0.04	4.22^{f} ± 0.01	10.43^{b} ± 0.06	8.66^{c} ± 0.02	6.43^{d} ± 0.01	5.17^{e} ± 0.00	0.0554
C18:3	1.10^{a} ± 0.01	ND	0.77^{b} ± 0.02	0.55^{c} ± 0.01	0.27^{d} ± 0.03	ND	0.0281
C20:0	ND	ND	ND	ND	ND	ND	–
C20:1	ND	ND	ND	ND	ND	ND	–
C22:0	ND	ND	ND	ND	ND	ND	–
C22:1	ND	ND	ND	ND	ND	ND	–
SAFA	19.58^{f} ± 0.16	86.28^{a} ± 1.02	39.60^{e} ± 0.74	52.93^{d} ± 0.39	69.59^{c} ± 0.44	79.59^{b} ± 0.95	1.2286
MUFA	66.22^{a} ± 0.82	9.50^{f} ± 0.09	49.20^{b} ± 0.66	37.86^{c} ± 0.72	23.67^{d} ± 0.13	15.17^{e} ± 0.03	0.9335
PUFA	14.20^{a} ± 0.05	4.22^{f} ± 0.01	11.20^{b} ± 0.08	9.21^{c} ± 0.03	6.70^{d} ± 0.04	5.17^{e} ± 0.00	0.7788

Means within the same row with different letters are significantly different ($P < 0.05$); ± S.D; SAFA, Saturated Fatty Acids; MUFA, Monounsaturated Fatty Acids; PUFA, Polyunsaturated Fatty Acids; ND, refers to not detected fatty acid; TNO, refers to Tiger nut oil; CNO, refers to Coconut oil.

significant ($p \leq 0.05$) decrease in the content of saturated fatty acids consistent with a significant ($p \leq 0.05$) increase in the content of mono-unsaturated fatty acids (MUFA) in the mixtures. Mixing tiger nut oil with coconut oil at levels 90, 75, 50 and 30% resulted in the reduction of the saturated fatty acid contents from 86.28 to 79.59, 69.59, 52.93 and 39.60% respectively. MUFA contents increased from 9.50 to 15.17, 23.6, 37.86 and 49.20% following the above mixing ratios.

3.2. Nutritional parameters

The nutritional parameters of the rats fed with diets containing tiger nut oil, coconut oil and binary mixtures of them are shown in Table 2. The results show that non-significant changes in all nutritional parameters were observed between the control group and the rats that were fed the tested oils.

Total cholesterol levels

Table 3 shows the sera cholesterol rates of rats fed with diets containing tiger nut oil, Coconut oil and binary mixtures of them. As expected, the rats fed the diet containing coconut oil and its mixtures with tiger nut oil at levels 90 and 75% showed significant ($p \leq 0.05$) increases in their total cholesterol levels of 106.9, 82.8 and + 34.4%, respectively at the end of the nutritional experiment (10 weeks). Recent lipid research indicated that not all SFAs have the same impact on serum cholesterol. For instance, lauric acid (C12:0) and myristic acid (C14:0), have a greater total cholesterol raising effect than palmitic acid (C16:0), whereas stearic acid (C18:0) has a neutral effect on the concentration of total serum cholesterol, including no apparent impact on either LDL or HDL. Lauric acid increases total serum cholesterol, although it also decreases the ratio of total cholesterol: HDL because of a preferential increase in HDL cholesterol. (Mensink and Katan, 1992; Kris-Etherton, 1997 and Mensink *et al.*, 2003). The lowest values of cholesterol content were observed in rats fed with diets containing tiger nut oil and its mixture with coconut oil at level of 70%. Diets supplemented with corn oil or olive oil were found to increase bile acid synthesis in rat liver (Botham and Boyd, 1983), and hepatic acyl- CoA-cholesterol acyltransferase activity has been reported to increase in rats fed with n-3 polyunsaturated fat compared with those given a saturated-fat diet (Spector *et al.*, 1980). The data show that there were no significant ($p \geq 0.05$) changes observed in the levels of total cholesterol throughout the experiment for the control rats group and for rats fed with diets containing tiger nut oil and its blend

Table 2
Nutritional parameters of rats fed on diets containing tiger nut oil, coconut oil and binary mixtures of them

Nutritional parameters	Control	TNO	CNO	TNO + CNO (v/v)				LSD at 0.05
				(70/30)	(50/50)	(25/75)	(10/90)	
Initial B.W (g)	$96.4^{a} \pm 5.55$	$94.6^{a} \pm 4.15$	$92.06^{a} \pm 3.03$	$91.22^{a} \pm 2.32$	$89.67^{a} \pm 3.08$	$90.00^{a} \pm 4.76$	$99.78^{a} \pm 2.00$	6.58
Final B.W. (g)	$202.36^{a} \pm 5.45$	$198.71^{a} \pm 2.36$	$196.23^{a} \pm 4.74$	$200.95^{a} \pm 4.64$	$193.04^{a} \pm 3.18$	$190.31^{a} \pm 3.89$	$200.3^{a} \pm 5.96$	7.84
B.W. gain (g)	$107.16^{a} \pm 2.94$	$104.26^{a} \pm 1.63$	$104.16^{a} \pm 1.78$	$106.70^{a} \pm 3.34$	$103.37^{a} \pm 0.57$	$100.31^{a} \pm 0.89$	$106.07^{a} \pm 5.75$	5.12
Total Feed Intake (g)	$997.02^{a} \pm 10.89$	$970.21^{a} \pm 15.49$	958.04 ± 18.95	$964.68^{a} \pm 25.58$	$998.78^{a} \pm 12.50$	$982.33^{a} \pm 10.6$	$987.4^{a} \pm 15.8$	28.81
Feed Efficiency Ratio	0.107^{a}	0.107^{a}	0.108^{a}	0.110^{a}	0.103^{a}	0.102^{a}	0.107^{a}	

Data are expressed as mean ± SD; values followed by the same letter are not significantly different ($p < 0.05$).

Table 3
Serum total cholesterol (mg/dl)) of rats fed on diets containing tiger nut oil, coconut oil and binary mixtures of them

Blood withdrawal Period (week)	Control	Type of oil					
		TNO	CNO	TNO + CNO (v/v)			
				(70/30)	(50/50)	(25/75)	(10/90)
0	$84.52^{fg} \pm 2.21$	$84.55^{fg} \pm 2.03$	$83.60^{fg} \pm 1.63$	$83.10^{fg} \pm 1.09$	$84.20^{fg} \pm 1.12$	$83.00^{fg} \pm 1.54$	$83.31^{fg} \pm 1.58$
2	$84.71^{fg} \pm 2.32$	$84.70^{fg} \pm 1.89$	$86.10^{fg} \pm 1.25$	$83.30^{fg} \pm 1.16$	$84.33^{fg} \pm 1.32$	$84.60^{fg} \pm 1.63$	$85.00^{fg} \pm 2.00$
4	$85.20^{fg} \pm 1.63$	$84.80^{fg} \pm 1.54$	$105.35^{def} \pm 1.58$	$83.35^{fg} \pm 1.26$	$85.65^{fg} \pm 1.24$	$90.00^{efg} \pm 1.52$	$96.02^{efg} \pm 2.64$
6	$84.86^{fg} \pm 1.27$	$85.00^{fg} \pm 1.86$	$140.20^{c} \pm 1.73$	$84.71^{fg} \pm 1.58$	$87.10^{fg} \pm 2.00$	$102.12^{efg} \pm 1.84$	$123.50^{d} \pm 2.04$
8	$84.90^{fg} \pm 1.66$	$85.95^{fg} \pm 2.02$	$165.10^{ab} \pm 2.65$	$86.50^{fg} \pm 1.22$	$92.70^{efg} \pm 1.98$	$105.60^{def} \pm 1.67$	$145.85^{c} \pm 3.32$
10	$84.98^{fg} \pm 2.10$	$85.96^{fg} \pm 2.13$	$171.80^{a} \pm 3.99$	$87.50^{fg} \pm 1.47$	$93.40^{efg} \pm 2.35$	$111.60^{de} \pm 2.01$	$152.30^{bc} \pm 3.22$

LSD at 0.05 = 18.52; data are expressed as mean ± SD; values given represent means of five determinations; values followed by the same letter are not significantly different ($p < 0.05$).

with coconut oil at a 70% ratio. Every one percent of total dietary energy in which oleic acid is substituted for saturated fatty acids, the serum total cholesterol concentration falls by an average of 2.7 mg dL^{-1}. (Keys *et al.*, 1965). Human studies suggest that monounsaturated fats are as effective as polyunsaturated fats in lowering plasma cholesterol concentrations when substituted for saturated fats (Grundy, 1989).

High density-lipoprotein cholesterol (HDL-Ch) content

HDL picks up and transports cholesterol in the blood back to the liver, which leads to its elimination from the body. HDL can help to keep LDL cholesterol from building up in the walls of the arteries (Awan, 1993). The changes in the sera levels of HDL-Ch in rats fed with diets containing tiger nut oil, Coconut oil and binary mixtures of them are shown in Table 4. The results indicate that HDL-Ch levels were significantly ($p < 0.05$) increased in the control and experimental groups at the end of the nutritional experiment. Rats fed diets containing coconut oil and its blend with tiger nut oil at ratio of 90% had a slight but significant ($p < 0.05$) increase in HDL-Ch levels at the end of the nutritional experiment which were approximately 1.11 and 1.15 times as great as their levels at the start of the experiment, respectively. Lauric acid increases total serum cholesterol, although it also decreases the ratio of total cholesterol: HDL because of a preferential increase in HDL-Ch (Mensink and Katan, 1992; Kris-Etherton, 1997 and Mensink *et al.*, 2003). However, rats fed with diets containing tiger nut oil and its mixture at a level of 70% had the highest values of HDL-Ch at the end of the nutritional experiment which were recorded at 52.90 and 49.25 mg dL^{-1}. These values were approximately 1.37 and 1.29 times as great as the values recorded at the start of the experiment. Oleic acid is difficult to oxidize and that is involved in the fluidity of lipoproteins and as a consequence in the generation of HDL (Sola *et al.*, 1990). Reverse cholesterol transport is initiated in extrahepatic tissues by the transfer of unesterified cholesterol from cell membranes to nascent HDL (Fielding, 1984). A portion of this cholesterol is esterified by lecithin:cholesterol acyltransferase (LCAT) tocholesteryl esters, by virtue of their hydrophobicity partition into the core of the HDL particle (Fielding, 1984). HDL cholesteryl esters are returned to the liver through several pathways including: i) Transfer to lower density lipoproteins with subsequent

Table 4
Serum HDL-colesterol (mg/dl) of rats fed on diets containing tiger nut oil, coconut oil and binary mixtures of them

Blood withdrawal Period (week)	Control	Type of oil					
		TNO	CNO	TNO + CNO (v/v)			
				(70/30)	(50/50)	(25/75)	(10/90)
0	$38.45^{jk} \pm 0.19$	$38.40^{jk} \pm 0.28$	$38.00^{jk} \pm 0.17$	$38.00^{jk} \pm 0.26$	$38.60^{jk} \pm 0.09$	$38.75^{hijk} \pm 0.51$	$38.00^{jk} \pm 0.22$
2	$40.50^{fgh} \pm 0.59$	$41.50^{ef} \pm 0.43$	$39.00^{hijk} \pm 0.39$	$40.34^{fgh} \pm 0.28$	$39.00^{hijk} \pm 0.25$	$39.14^{hijk} \pm 0.07$	$39.00^{hijk} \pm 0.48$
4	$45.60^{abcd} \pm 0.57$	$46.00^{abc} \pm 0.38$	$41.50^{efg} \pm 0.26$	$40.86^{fgh} \pm 0.53$	$39.75^{ghi} \pm 0.64$	$41.51^{efg} \pm 0.17$	$41.00^{fgh} \pm 0.19$
6	$47.75^{abcd} \pm 0.43$	$48.35^{abcd} \pm 0.37$	$42.90^{cde} \pm 0.40$	$46.90^{abc} \pm 0.41$	$40.97^{fgh} \pm 0.43$	$41.60^{def} \pm 0.69$	$41.12^{efg} \pm 0.10$
8	$50.80^{abc} \pm 0.34$	$49.72^{abcd} \pm 0.28$	$42.25^{def} \pm 0.19$	$47.00^{abc} \pm 0.60$	$44.32^{bcd} \pm 0.18$	$42.30^{def} \pm 0.11$	$42.00^{def} \pm 0.97$
10	$51.50^{ab} \pm 0.37$	$52.90^{a} \pm 0.16$	$42.50^{def} \pm 0.09$	$49.25^{abc} \pm 0.38$	$46.54^{abc} \pm 0.80$	$43.90^{bcd} \pm 0.83$	$43.90^{bcd} \pm 0.66$

LSD at 0.05 = 6.637; data are expressed as mean ± SD; values given represent means of five determinations; values followed by the same letter are not significantly different ($p < 0.05$).

uptake via the LDL receptor pathway (Tall, 1993), ii) Uptake of the intact HDL particle (Ponsin *et al.*, 1993 and iii) Selective uptake of HDL cholesteryl esters resulting in an HDL particle of reduced size and cholesteryl ester content (Goldberg *et al.*, 1991). The scavenger receptor B1 (SR-BI) plays a major role in selective HDL cholesteryl ester transport (Krieger, 1998). SR-BI was shown to mediate selective cholesteryl ester uptake when transfected into cells (Acton *et al.*, 1996). In mice, overexpressing SR-BI in the liver results in the virtual elimination of HDL cholesteryl ester from plasma (Kozarsky *et al.*, 1997)whereas disrupting the SR-BI gene by targeted mutation leads to a decrease in HDL cholesteryl ester uptake by the liver and an increase in plasma HDL cholesteryl ester concentrations (Varban *et al.*, 1998).

Low density-lipoprotein cholesterol (LDL-Ch) content

Keeping LDL-Ch levels at a healthy level is vital for heart health, because, when LDL cholesterol accumulates in the blood stream plaque formation can occur; this increases the risk for heart attack and stroke (Escott-Stump and Mahan, 2004). Table 5 shows the changes in the levels of LDL-Ch of rats fed diets containing tiger nut oil, coconut oil and binary mixtures of the two. Rats fed a diet containing coconut oil and its mixture with tiger nut oil at a level of 90% had significantly ($p < 0.05$) the highest levels of LDL-Ch at the end of the experiment. The level of LDL-Ch of the rats that were fed diets containing coconut oil was about 3.67 times as great at the end of the nutritional experiment as it was in the beginning of the experiment. It is generally accepted that for every 1% increase in energy from SFA, LDL cholesterol levels reportedly increase by 1.3 to 1.7 mg dL^{-1} (0.034 to 0.044 mmol L^{-1}) (Mensink and Katan, 1992 and Mensink *et al.*, 2003). Studies in animals have shown that saturated fats increase LDL cholesterol by inhibiting LDL receptor activity and enhancing apolipoprotein apo-B-containing lipoprotein production (Grundy, 1989; Dietschy, 1998). On the other hand, the rats that were fed

Table 5
Serum LDL- cholesterol (mg/dl)) of rats fed on diets containing tiger nut oil, coconut oil and binary mixtures of them.

Blood withdrawal Period (week)	Control	Type of oil					
		TNO	CNO	TNO + CNO (v/v)			
				(70/30)	(50/50)	(25/75)	(10/90)
0	$28.00^{rstu} \pm 0.36$	$29.50^{opq} \pm 0.19$	$28.60^{pqrst} \pm 0.13$	$28.30^{qrst} \pm 0.33$	$27.50^{ut} \pm 0.15$	$29.00^{opqrs} \pm 0.44$	$28.50^{pqrst} \pm 0.05$
2	$26.90^{uv} \pm 0.42$	$27.72^{stu} \pm 0.26$	31.00mn ± 0.64	$26.00^{vw} \pm 0.10$	$28.50^{pqrst} \pm 0.27$	$31.30^{m} \pm 0.51$	$29.25^{opqr} \pm 0.23$
4	$25.85^{vw} \pm 0.19$	$25.85^{vw} \pm 0.37$	$49.70^{h} \pm 0.72$	$25.00^{wx} \pm 0.61$	$29.60o^{pq} \pm 0.36$	$35.00^{L} \pm 0.18$	51.11g ± 0.44
6	$25.70^{vw} \pm 0.44$	$24.75^{wx} \pm 0.12$	$75.90^{d} \pm 1.06$	$25.00^{wx} \pm 0.53$	$29.80^{nop} \pm 0.61$	$37.66^{k} \pm 0.34$	63.00f ± 1.13
8	$25.70^{vw} \pm 0.16$	$24.00^{xy} \pm 0.48$	$89.90^{b} \pm 0.24$	$25.10^{wx} \pm 0.56$	$30.00^{no} \pm 0.04$	$43.50^{j} \pm 0.77$	70.60e ± 1.27
10	$24.90^{wx} \pm 0.56$	$23.50^{y} \pm 0.07$	$105.00^{a} \pm 2.41$	$25.00^{wx} \pm 0.37$	$35.00^{L} \pm 0.30$	$46.00^{i} \pm 0.97$	85.90c ± 2.10

LSD at 0.05 = 1.144; data are expressed as mean ± SD; values given represent means of five determinations; values followed by the same letter are not significantly different ($p < 0.05$).

diets containing tiger nut oil and its mixture with coconut oil at a level of 70% had significantly the lowest values of LDL cholesterol and did not show any significant changes from the control group. Mixing coconut oil with different amounts of tiger nut oil caused significant ($p < 0.05$) reductions in the levels of LDL cholesterol. The levels of LDL cholesterol of the rats fed diets containing tiger nut oil at levels 70, 50, 25 and 10% were about 4.2, 3.0, 2.2 and 1.2 times as low as those for rats fed the diets containing coconut oil, respectively.

The replacement of dietary saturated fatty acids (SFAs) with monounsaturated fatty acids (MUFAs) lowers LDL cholesterol, without inducing the hypertriacylglycerolemia sometimes observed when SFAs are replaced by carbohydrates (Mensink, and Katan, 1992; Kris-Etherton *et al.*, 1999). MUFA (predominantly oleic acid, C18:1) was speculated to be either neutral or half as potent as PUFAs in lowering blood cholesterol levels (Yu *et al.*, 1995). In this respect, Kurushimaa *et al.*, (1995) suggested that the suppression of hepatic LDL receptor activity was prevented by either linoleic acid or oleic acid, although linoleic acid was more effective in preventing LDL receptor suppression than oleic acid.

Triglycerides (TG) content

High levels of plasma TG are considered to be an independent risk factor for the development of cardiovascular disease (CVD) (Miller, 2000). The changes in the levels of sera levels of TG in the rats fed diets containing tiger nut oil; coconut oil and binary mixtures of the two are shown in Table 6. The results indicate that rats fed diets containing coconut oil had significantly ($P \leq 0.05$) the highest level of TG at 172.4 mg dL^{-1} at the end of nutritional experiment (10 weeks). On the other hand, the rats fed diets containing tiger nut oil and its mixture with coconut oil at a level of 70% as well as those rats fed the control diet had significantly ($p \geq 0.05$) the lowest values of TG at 82.5, 82.4 and 81.50 mg dL^{-1}, respectively. Mixing coconut oil with various levels of tiger nut oil as a source of MUFA caused significant ($P \leq 0.05$) falls in the levels of TG compared with the group that fed on the diet containing coconut oil. These fatty acids have been shown to maintain a positive lipoprotein phenotype by decreasing plasma TG concentrations, decreasing VLDL synthesis and secretion, decreasing the production of apolipoprotein B100 (apo B100), and increasing the clearance of TG-rich lipoproteins (Kendrick and Higgins, 1999). Elevated levels of triglycerides in the blood stream have been linked to an increased incidence of coronary artery disease. Maintaining a triglyceride level of 150 mg dL^{-1} or less in the bloodstream has been shown to reduce the risk for developing heart disease (Ahmed *et al.,* 2010).

Atherogenic Index (AI)

Atherogenic Index (AI) is an established and efficient indicator of lipid atherogenesis, reflecting the balance of cholesterol transport in and out of the arterial intima (Kannel and Wilson, 1992). Table 7 shows the changes in the values of AI of the rats fed diets containing tiger nut oil, Coconut oil and binary mixtures of them. The results indicate that the rats fed diets containing coconut oil had significantly ($p < 0.05$) the highest value of atherogenic Index (AI) at the end of the experiment at 0.60. A significant increase in the atherogenic index (AI) was observed, primarily due to the decreased serum HDL-Ch levels, suggesting that these rats are possibly exposed to a higher risk of atherosclerosis. The increase of AI values is correlated to the increase in coconut oil levels in the diets. On the contrary, the rats fed the control diet and those that were fed diets containing tiger nut oil at levels 100, 70 and 50% had significantly ($p < 0.05$) the lowest values of Atherogenic Index (AI) at the end of the nutritional experiment which ranged from 0.19 to 0.38. Mixing coconut oil with

Table 6
Serum triglycerides (TG) (mg/dl)) of rats fed on diets containing tiger nut oil, coconut oil and their binary mixtures.

Blood withdrawal Period (week)	Control	Type of oil					
		TNO	CNO	TNO + CNO (v/v)			
				(70/30)	(50/50)	(25/75)	(10/90)
0	$80.23^{qr} \pm 0.36$	$82.00^{pqr} \pm 1.12$	$80.00^{r} \pm 0.38$	$81.30^{qr} \pm 0.77$	$80.25^{qr} \pm 0.46$	$81.00^{qr} \pm 0.97$	$82.10^{pqr} \pm 0.88$
2	$80.55^{qr} \pm 1.02$	$82.10^{pqr} \pm 0.62$	$88.00^{n} \pm 0.53$	$81.35^{qr} \pm 0.63$	$82.12^{pqr} \pm 0.55$	$84.00^{op} \pm 0.82$	$85.30^{o} \pm 1.24$
4	$80.90^{qr} \pm 0.98$	$82.10^{pqr} \pm 0.47$	$118.60^{g} \pm 1.14$	$81.40^{qr} \pm 0.67$	$86.00^{o} \pm 0.86$	$90.66^{m} \pm 0.63$	$106.00^{j} \pm 0.99$
6	$81.00^{qr} \pm 0.47$	$82.00^{pqr} \pm 0.48$	$130.85^{e} \pm 2.07$	$81.50^{qr} \pm 0.95$	$94.88^{L} \pm 1.42$	$96.35^{L} \pm 0.56$	$122.00^{f} \pm 0.47$
8	$81.35^{qr} \pm 0.36$	$82.50^{pq} \pm 0.72$	$160.70^{b} \pm 1.52$	$82.40^{pqr} \pm 1.16$	$101.87^{k} \pm 2.18$	$110.80^{j} \pm 2.08$	$135.80^{d} \pm 0.87$
10	$81.50^{qr} \pm 0.54$	$82.50^{pqr} \pm 0.87$	$172.40^{a} \pm 2.87$	$82.40^{pqr} \pm 1.03$	$113.00^{h} \pm 2.12$	$130.55^{e} \pm 0.17$	$152.90^{c} \pm 2.97$

LSD at 0.05 = 1.955; data are expressed as mean ± SD; values given represent means of five determinations; values followed by the same letter are not significantly different ($p < 0.05$).

Table 7
Atherogenic Index (AI) of rats fed on diets containing tiger nut oil, coconut oil and their binary mixtures

Blood withdrawal Period (week)	Control	Type of oil					
		TNO	CNO	TNO + CNO (v/v)			
				(70/30)	(50/50)	(25/75)	(10/90)
0	0.31	0.32	0.32	0.33	0.31	0.32	0.33
2	0.29	0.29	0.35	0.30	0.32	0.33	0.33
4	0.24	0.25	0.45	0.29	0.33	0.33	0.41
6	0.22	0.22	0.48	0.23	0.36	0.36	0.47
8	0.20	0.21	0.58	0.24	0.36	0.41	0.50
10	0.19	0.19	0.60	0.22	0.38	0.47	0.54

The atherogenic Index (AI) was calculated by using following equation: Atherogenic Index (AI) = Log (TG/HDL-C).

different levels of tiger nut oil caused a significant ($p < 0.05$) reduction in the values of the Atherogenic Index (AI). The increase in the oleic/linoleic acid ratio in LDL has been shown to promote favorable changes in inflammatory markers. Tsimikas *et al.* (1999), observed that an increase in the oleic/linoleic acid ratio in LDL induced less monocyte chemotaxis and adhesion when exposed to oxidative stress.

There are several explanations concerning the mechanisms by which dietary fatty acids affect plasma cholesterol concentrations such as changes in lipoprotein composition (Shore *et al.*, 1981), in LDL production (Turner *et al.*, 1981), and in very low-density cholesterol (VLDL) secretion from the liver and hepatic LDL receptor activity (Hayashi *et al.*, 1993). Tiger nut was reported to have high contents of vitamins E and C (Belewu and Belewu, 2007). Vitamin E (α-tocopherol) inhibited the activation of endothelial cells stimulated by high levels of LDL-cholesterol and pro-inflammatory cytokines. This inhibition is associated with the suppression of chemokines, the expression of cell surface adhesion molecules, and the adhesion of leukocytes to endothelial cells, all of which contribute to the development of lesions in the arterial wall. Moreover, the positive effect of dietary vitamin E on endothelium and vascular functions in animal models of atherosclerosis was demonstrated (Meydani, 2004).

In conclusion, our study shows that coconut oil had the highest saturated fatty acid level at (86.28%); while tiger nut oil has an abundance of oleic (66.22%) acid. There are lower, yet still considerable, concentrations of C18:2 n-6 (13.10%) found in the tiger nut oil sample. Therefore, blending coconut oil with tiger nut oil as a source of MUFA was suggested for improving the nutritional and health properties of coconut oil. Our findings show also that the diets rich in tiger nut oil have more favorable effects on the blood lipid profile and plasma lipoproteins and can be recommended for patients with dyslipidemia diseases.

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Recibido: 18/1/12
Aceptado: 2/4/12