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Hypolipemic effect of Kuub (*Gundelia tournefotii* A.) oil and clofibrate on lipid profile of atherosclerotic rats

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ABSTRACT

Two major groups of albino adult male rats were used as a biological model for this study. The first group contained five healthy rats (healthy control group), and the second group twenty-five atherosclerotic rats induced by hydrogen peroxide and cholesterol, which were divided equally into five subgroups. The first subgroup was orally treated with sunflower oil (atherosclerotic rats control group). The second subgroup was treated with 300 mg clofibrate drug/kg rat body mass (hypolipidemic reference group). The third, forth and the fifth subgroups were orally treated with 60, 90 and 120 mg Kuub (Kanger) oil/kg body mass, respectively. Blood samples were collected from retro-ocular eye vein using a heparinized pack cell volume (PCV) tube. In plasma, total lipid (TL), total cholesterol (TC), high density lipoprotein-cholesterol (HDL-C) and liver total cholesterol (TC) were determined. Nutritional status of the rats was also estimated. Results showed that the Kuub oil caused an improvement in the nutritional status of the rats, with hypolipidemic effect in plasma TL, TC, very low density lipoprotein-cholesterol and low density lipoprotein-cholesterol (VLDL-C + LDL-C), and decreased atherogenic indices, On the other hand, there was an increase in plasma HDL-C value and a decrease in liver TC level of atherosclerotic rats. Oil extract of Kuub seeds with a concentration of 90 mg/kg body mass is considered to give high efficacy concentration than the other tested ones. However, its hypolipidemic effect is lower than the clofibrate.

Key words: rat, atherosclerosis, hypolipidemia, lipid profile, cholesterol, clofibrate, hydrogen peroxide, Kuub

Introduction

Atherosclerosis can be influenced by dietary cholesterol. Excessive ingestion of fats caused the initial deposition of cholesterol and led to initial lesion of atherosclerosis. Numerous epidemiological and laboratory studies have reaffirmed

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the positive correlation between elevated serum cholesterol levels and the risk of coronary heart disease (CHD). Several clinical studies have shown that modification of serum cholesterol level by diet or drugs can reduce that risk. Patients with high–risk of low density lipoproteins (LDL) serum levels, and those with borderline high–risk levels, benefit from modifying serum cholesterol level (WOLF, 2000).

The TC/HDL (total cholesterol/high density lipoprotein) ratio is an important factor in assessing the risk of CHD. In order to ascertain patient's risk of CHD, it is more beneficial to direct efforts toward, and improving, patients' cholesterol ratios (TC/HDL and LDL/HDL) rather than the straight LDL level alone. The impact on changes in risk were quite substantial from raising HDL through life style modifications as from lowering LDL. Therefore, the best method of determining the risk of CHD is to assess changes in initial LDL and HDL (KINOSIAN, 1996).

Preventing and treating CHD effected by reducing plasma LDL concentration and by cutting off the source of atherosclerosis plaque, lowering LDL with a statin drug in high risk patients reduced the number of cardiac events by about 30%. The presence of high levels of HDL and its major protein apolipo and apo A-1, may inhibit LDL oxidation. In addition, HDL may be involved in promoting "reverse cholesterol transport", which is the migration of cholesterol from the peripheral cells back to the liver for disposal (LENFANT et al., 1997). KWITEROVICH (1997) concluded that for each 1% increase in HDL-C, there is a 2-3% decrease in CHD, and in patients who have isolated low HDL level, niacin has been shown to increase the level by 27%. Other measures to raise HDL include stopping smoking and increasing aerobic exercise.

The odds ratio for a coronary event was 1.15 for a 10% increase LDL and 0.84 for a 10% increase in HDL. However, the odds ratio rose to 1.17 with a 10% increase in the LDL/HDL ratio and to 1.21 with a 10 % increase in the TC/HDL ratio (KINOSIAN, 1996).

A powerful class of cholesterol lowering drugs has been introduced in the past, especially statins (decadehydroxymethyl glutaryl coenzyme A reductase inhibitors). Several large clinical trials have shown that these drugs reduce major cardiovascular events by 20-30% (ROSS et al., 1999). These trials also indicated that treatment with statin for 5-6 years does not affect mortality from cancer, but it is important to establish that long term (>5 years) cholesterol reduction might increase cancer mortality and deaths (MULDOON et al., 2001).

Although there is no conclusive evidence for the beneficial effect of correction of hyperlipo-proteinemia, several drugs have been introduced for lowering serum lipid concentration.

Clofibrate may inhibit both the release of lipoproteins from the liver and cholesterol biosynthesis. Some anabolic steroids are used in some patients investigationally for atherosclerosis therapy. Also, sitosterols compounds isolated from plants can be used as absorbable competitive with cholesterol (GOTH, 1984).

Kuub *Gundelia tournefotii* A. (kuub: Arabic or Kanger: Kurdish names) is an introduced weed in South Africa and countries of the Middle East. Kuub seeds yield oil regarded as an edible oil and have been found to contain tyramine (a cholestyramine drug which is considered as hypocholesterolemia). The physiologically active principles of Kuub seeds are alkaloids and saponin. The green plant of Kuub is regarded a dangerous to livestock, especially cattle and sheep, and has caused many death in Australia due to hydrocyanic acids (WATT and BREYER-BRANDWIJK, 1962).

The aim of this study was to establish the effect of Kuub-seed oil on the blood total lipid and lipoproteins of atherosclerotic rats.

Materials and methods

Experimental laboratory animals. Five albino adult male (normal) rats were used as a healthy control group, and twenty–five adult male atherosclerosis rats induced by 0.5% hydrogen peroxide and 1% cholesterol (SHARAF and ALI, 2003) all rats were aged 3-4 months and with 165-255g body mass. Treated rats were divided equally into five groups: the first group was orally treated with sunflower oil alone and considered as atherosclerotic rats group; the second group was orally treated with clofibrate in a concentration of 300 mg/kg body mass (MOHAMMAD et al., 2001) and considered to be an atherosclerotic rats reference group. The third, fourth and the fifth groups were orally treated with 60, 90 and 120 mg Kuub-seed oil/kg body mass, respectively. All rat groups were housed at room temperature (25 °C). Dark/light cycle was 12/12 hrs for the 15-day experimental period. The healthy rats group received an ordinary diet and fresh water, while the atherosclerotic rat groups received 1% cholesterol in diet and 0.5% hydrogen peroxide in drinking water received *ad libitum*.

Diet preparation. Broiler diet was introduced from the Iraqi IBA Agricultural Research Centre in order to meet the nutritional and physiological requirements of rats. Suitable quantities of vitamins and minerals were added according to the suggestions of American National Academy of Sciences/Nutritional Research Council (NAS/NRC), (1978) to meet the animals' physiological and nutritional requirements. In addition, the diet was modified by adding wheat flour containing cholesterol to obtain a 1% cholesterol diet, which is used for feeding atherosclerotic rats, while the diet without adding cholesterol (ordinary diet) was used for feeding healthy rats. Diets were moistened separately with distilled water and formed in the shape of pellets, dried in an electric oven at 55 °C, then stored in polyethylene bags in a dry cool place. Composition of diets and analysis are presented in Table 1.

Table 1. The ordinary diet composition (g/kg) and chemical analysis on dry weight basis

Diet composition		Diet chemical analysis	
Ingredients	%	Constituents	%
Corn meal flour	40.25	Protein	12.98
Wheat flour	20.00	Lipid (ether extract) [1]	5.56
Soybean flour	16.50	Fibre	4.18
Barley	10.50	Carbohydrate [2]	69.97
Wheat bran	6.75	Ash	7.31
Vitamins mixture	1.40	Metabolized energy [3]	381.84 Kcal/100g
Sodium chloride	1.00		
Calcium carbonate	1.00		
Sodium monophosphate	1.60		
Mineral mixture	1.00		

[1] Diet of atherosclerotic rats contain 1% cholesterol additional component to the ordinary diet.
[2] Calculated from the difference between the sum of protein, lipid, fibre, ash percentages and 100%.
[3] Calculated on the basis of protein and carbohydrate = 4 Kcal and lipid=9 Kcal metabolized energy.

Kuub and sunflower oil extract. Crude lipids were extracted from dry seeds of Kuub or sunflower (ether extract) using soxhlet apparatus and solvent mixture of equal volumes of diethyl ether and 95% ethanol according to the procedure of American Official Analytical Chemists (ANONYM., 1980).

Hydrogen peroxide drinking water. Daily prepared fresh water containing 0.5% hydrogen peroxide was used for drinking by atherosclerotic rats *ad libitum*.

Collection and preparation of blood samples. Blood samples were collected from the eye retro-ocular vein (TIMM, 1979) by using heparenized PCV tube. Plasma was separated by using anticoagulant 1mg EDTA/ml blood according to (FRIEDWALD et al., 1972). Obtained plasma was used for determination of TL, TC, and HDL. Liver tissue was separated and used for liver TC estimation.

Biochemical and clinical determinations.

1-Plasma TL was determined according to TORO and ACKERMANN (1975) using phohosphovanillin reagent. Light absorption of samples was measured in a spectrophotometer at a wave length of 540 nm.

2-Plasma TC was determined using BioMerieux kit according to the producer's procedural manual. Light absorption for the purple-red intermediate (quinoneimine) complex was measured at 500 nm, using a spectrophotometer.

3-Plasma HDL–C was determined using a BioMerieux Kit. Light absorption was measured spectrophotometrically at a wave length of 500 nm.

4-Plasma VLDL-C + LDL-C was calculated according to the Morita equation (MORITA et al., 1997) as follows: (VLDL-C + LDL-c) = TC – (HDL-C).

5-First atherogenic index = TC/HDL-C calculated according to YOUSIF (2000).

6-Second atherogenic index = VLDL-C + LDL-C / HDL-C calculated according to TAKUNAGA et al. (1982).

Nutritional rat status was estimated throughout the apparent digestibility and body mass factors. Apparent digestibility and dietary lipid absorption percentages determined according to SHARAF (1998), SHARAF and ALI (2003), respectively, as:

$$\text{Apparent digestibility \%} = \frac{\text{g total diet intake - g faeces}}{\text{g total diet intake}} \times 100$$

$$\text{Lipid absorption \%} = \frac{\text{g dietary lipid intake - g faeces lipid}}{\text{g dietary lipid intake}} \times 100$$

Chemical analysis: Moisture and ash were determined according to the (ANONYM., 1980). Protein, crude oil and fibre of the Kuub and sunflower seeds were determined according to PEARSON (1976), while seed carbohydrate was

calculated from the difference between the sum of protein, lipid, fibre, ash and moisture percentages and 100%.

Liver total cholesterol determination: Rats were anesthetized with diethyl ether. Liver was homogenized in a mixture of chloroform:methanol (2:1) and liver TC determined using the method of FOLCH et al. (1957).

Statistical analysis: Data were analyzed by using factorial experimental design according to STEEL and TORRIE (1980), using the analysis of variance and Duncan multiple range test (SAS program) at probability $P<0.05$.

Results and discussion

Table 1. shows composition and percentage of diet compounds (ordinary diet ingredients and their percentages) which produced sufficient metabolic energy to meet the nutritional and physiological requirements of the albino rats (ANONYM., 1978).

Table 2. shows the chemical analysis of Kuup and sunflower seeds and their oils. Kuub seeds contained higher oil and lower protein percentages than did sunflower seeds. The pH value of Kuub-seed oil was lower than the pH of sunflower oil, while the saponification No., saponifiable and unsaponifiable matters were higher than those of sunflower oil. Sunflower oil was used as a diluter for clofibrate oil drug and Kuub oil.

Table 2. Chemical analysis of Kuub, sunflower seeds and their oils on dry basis

	Constituents	Kuub	Sunflower
Seeds	Moisture %	8.58	7.82
	Oil %	34.30	31.10
	Protein %	16.49	19.38
	Ash %	4.38	4.67
Seed's oil	pH	6.60	7.10
	Saponification No	169	160
	Saponifiable matter %	51.11	53.63
	Unsaponifiable %	38.37	35.43
	Others (unknown) %	10.52	10.93

Table 3. Effect of Kuub seed's oil and clofibrate on the nutritional status of healthy and atherosclerosis-induced rats (mean[1] ± SE) [2]

Physiological status of rats	Rat groups	Body mass (g)		Ingested diet (g)		Eliminated feces (g)		Apparent digestibility %	Lipid absorption %
		initial	gain	Mass	Lipid content	Weight	Lipid content		
Healthy	Control	186.30 ±10.98a	34.98 ±3.12a	174.98 ± 7.53a	9.73 ± 0.73b	31.68 ± 1.09a	2.15 ± 0.21c	99.82 ± 1.82a	77.90 ±0.85a
Atherosclerosis	Control	167.04 ± 12.06b	3.30 ± 0.27e	103.44 ± 5.11b	6.79 ± 0.47c	22.22 ± 1.14c	4.54 ± 0.37b	78.52 ± 1.21c	33.14 ± 0.27c
	Clofibrate 300mg/kg B.M.	246.28 ± 16.62a	4.40 ± 2.33e	186.78 ± 10.38a	12.25 ± 0.68a	35.58 ± 3.26a	7.89 ± 1.23a	80.95 ± 2.01b	43.76 ± 1.58b
	Kuub oil extract 60 mg/kg B.M.	220.61 ± 7.98a	14.64 ± 0.93c	182.27 ± 8.15b	11.96 ± 0.92a	28.30 ± 2.15b	7.57 ± 1.62a	84.47 ± 1.69b	36.71 ± 1.92c
	Kuub oil extract 90 mg/kg B.M.	199.23 ± 5.46a	24.45 ± 1.42b	214.62 ± 7.91a	14.08 ± 0.61a	29.22 ± 1.90a	8.28 ± 1.14a	86.39 ± 2.11b	41.19 ± 1.23b
	Kuub oil extract 120 mg/kg B.M.	217.11 ± 10.27a	7.90 ± 0.85d	198.45 ± 10.11a	13.02 ± 0.83a	26.98 ± 2.04b	8.54 ± 0.99a	86.40 ± 1.49b	34.41 ± 0.98c

[1]mean of five rats.

[2]different letters in the same column are significantly different at $P<0.05$

Table 4. Effect of Kuub seed's oil and clofibrate on the plasma lipids, lipoproteins, atherosclerosis indices and liver total cholesterol in the healthy and atherosclerosis–induced rats (mean[1] ± SE)[2]

Pysiological status of rats	Rat groups	Plasma lipids and lipoproteins								Atherogenic indices				Liver TC mg/100g
		TL mg/dl		TC mg/dl		HDL-C mg/dl		VLDL-C +LDL-C mg/dl		TC/HDL-C		VLDL-C+LDL-C /HDL-C		
		1day	15days	1day	15days	1day	15days	1day	15days	1day	15days	1day	15days	15days
Healthy	Control	326.84 ± 61.12b	424.00 ± 72.1d	57.06 ± 3.99b	80.18 ± 4.17b	48.56 ± 2.11a	61.58 ± 2.61a	8.50 ± 1.62b	18.70 ± 3.77d	1.18 ± 0.52b	1.30 ± 0.74d	0.18 ± 0.09b	0.30 ± 0.04e	4.64 ± 0.19c
Atherosclerosis	Control	676.28 ± 79.37b	724.10 ± 84.1a	106.68 ± 3.15a	118.17 ± 4.66a	36.50 ± 3.00b	30.72 ± 3.42c	71.18 ± 4.66a	77.45 ± 5.17a	3.00 ± 0.17a	3.52 ± 0.12a	2.01 ± 0.10a	2.52 ± 0.32a	9.64 ± 0.34a
	Clofibrate 300 mg/kg B.M.	708.40 ± 56.24a	544.92 ± 34.5c	91.48 ± 2.62a	100.38 ± 5.81a	37.80 ± 5.30b	47.30 ± 3.55b	53.68 ± 4.84a	53.08 ± 4.69c	2.42 ± 0.66a	2.12 ± .08b	1.42 ± 0.67a	1.12 ± 0.07d	4.07 ± 0.07c
	Kuub oil 60 mg/kg B.M.	686.13 ± 56.82a	592.17 ± 46.8b	98.29 ± 2.91a	106.28 ± 6.03a	38.82 ± 3.44b	42.94 ± 2.91a	59.47 ± 3.99a	63.34 ± 5.14b	2.53 ± 0.45a	2.48 ± 0.40c	1.53 ± 0.37a	1.48 ± 0.42b	5.42 ± 0.28b
	Kuub oil 90 mg/kg B.M.	732.11 ± 56.32a	609.26 ± 62.5b	101.25 ± 62.5a	107.14 ± 6.52a	36.92 ± 4.63b	45.67 ± 4.62b	64.33 ± 4.24a	61.47 ± 4.32b	2.74 ± 0.61a	2.35 ± 0.34b	1.74 ± 0.45a	1.35 ± 0.22c	5.12 ± 0.41b
	Kuub oil 120 mg/kg B.M.	692.15 ± 71.25a	606.15 ± 42.2b	93.95 ± 3.66a	104.38 ± 4.81a	39.16 ± 6.29b	41.86 ± 3.57b	54.79 ± 2.86a	62.52 ± 5.15b	2.40 ± 0.53a	2.49 ± 0.51b	1.40 ± 0.29a	1.49 ± 0.37b	6.09 ± 0.33b

[1]mean of five rats

[2]different letters in the same column are significantly different at $P<0.05$.

Table 3. shows the nutritional status of healthy and atherosclerotic rats. There was a significant difference ($P<0.05$) in body mass (BM), ingested diet weight, lipid content of diet, eliminated faeces weight and faeces lipid content, apparent digestibility and dietary lipid absorption between the atherosclerotic rat groups orally treated with Kuub oil and clofibrate compared with atherosclerotic rat control and healthy rat control groups. The BM of the healthy rats was higher than the atherosclerotic rat groups. This result was in agreement with the result obtained by ALI (2001), SHARAF and ALI (2003). The BM gains of the atherosclerotic rats orally treated with Kuub oil were higher than the gain of the clofibrate treated rat group. The BM gains of the atherosclerotic rats treated with Kuub oil, increased with the elevated Kuub oil dose from 60 to 90 mg/kg but it decreased with an increase to 120 mg/kg. Therefore, the Kuub oil caused an improvement in the rats' nutritional and physiological status. Apparent digestibility of the healthy rats was higher than the digestibility of atherosclerotic rats. The Kuub oil and clofibrate improved the digestibility of the atherosclerotic rats, compared with the atherosclerotic rats control group. The apparent lipid absorption of the atherosclerotic rat groups was lower than the absorption of the healthy rat group.

Table 4. shows significant differences ($P<0.05$) between plasma TL, TC, HDL-C, VLDL-C + LDL-C levels of the atherosclerotic rat groups treated orally with Kuub oil and clofibrate compared with the atherosclerotic and healthy rat controls. Also, there were significant differences ($P<0.05$) between atherogenic indices values and liver (TC) level of atherosclerotic rat groups treated with Kuub oil and clofibrate compared with the atherosclerotic and healthy rat controls. Plasma TL, TC, VLDL-C + LDL-C levels, atherogenic indices and liver TC of the atherosclerotic rats treated with Kuub oil and clofibrate were lower than the atherosclerotic and healthy rats control groups. On the other hand, the highest HDL-C level (for the 15-day experimental period) was noted in the healthy rat control group. Kuub oil produced an even lower hypolipidemic effect than clofibrate in plasma lipids profile and liver TC.

In conclusion, Kuub oil may be regarded a hypolipemic agent. It improves nutritional and physiological status in rats, lowers atherogenic indices and liver TC, but its effect is still lower than the effect of the hypolipemic clofibrate compound.

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SAŽETAK

U istraživanju su korištene dvije skupine odraslih mužjaka albino štakora kao biološki model. U prvu skupinu bilo je uključeno 5 zdravih štakora (kontrolna skupina), a druga skupina se sastojala od 25 štakora s aterosklerozom koja je bila izazvana vodik peroksidom i kolesterolom pa su bili podijeljeni u pet jednakobrojnih podskupina. Prvoj podskupini bilo je oralno dano ulje suncokreta (aterosklerotična kontrolna skupina štakora). Drugoj podskupini bilo je dano 300 mg klofibrata na kilogram tjelesne mase (hipolipidemična referentna skupina). Trećoj, četvrtoj i petoj podskupini štakora bilo je *per os* dano 60, 90 i 120 mg kuub (Kanger) ulja na kg tjelesne mase svakoga. Krvni uzorci su skupljeni iz retro-okularne vene korištenjem heparinziranih PVC epruveta. U plazmi su određivani ukupni lipidi (UL), ukupni kolesterol (UK), kolesterol visoke gustoće (LKVG) i ukupni jetreni kolesterol (UJK). Procjenjivan je i nutritivni status štakora. Rezultati pokazuju da je ulje dovelo do poboljšanja nutritivnog statusa štakora s hipolipidemičnim učinkom u plazmi na UL, UK, kolesterol vrlo niske gustoće i na kolesterol niske gustoće te opadanje aterogenih znakova. S druge strane, došlo je do porasta kolesterola visoke gustoće u plazmi i opadanja razine ukupnih jetrenih lipida u aterosklerotičnih štakora. Uljni ekstrakt sjemenki s koncentracijom od 90 mg/kg tjelesne mase pokazao se najdjelotvornijim. Ipak, njegov hipolipemični učinak je bio slabiji od klofibrata.

Ključne riječi: štakor, ateroskleroza, hipolipemija, lipidni profil, kolesterol, klofibrat, vodik peroksid, *Gundelia tourefotti* A.