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EFFECT OF FLAXSEED ON SERUM LIPIDS AND ATHEROSCLEROSIS IN RABBITS

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EFFECT OF FLAXSEED ON SERUM LIPIDS AND ATHEROSCLEROSIS IN RABBITS

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ABSTRACT

*This study was carried out to investigate the beneficial effect of flaxseed-rich diet on atherosclerotic rabbits. Rabbits were divided to 4 groups: **group I**, control diet, **group II**,1%cholesterol diet, **group III**, 10% flaxseed diet, and **group IV**, 1% cholesterol+ 10% flaxseed diet. Blood and tissue samples were collected after 2 months for measurement of serum lipid profile :total lipids, total cholesterol (TC), triacylglycerol (TAG), HDL-cholesterol (HDL-C), LDL- cholesterol(LDL-C) ;blood and tissue antioxidants :reduced glutathione (GSH) ,superoxide dismutase (SOD) and catalase ;oxidative stress parameters :nitric oxide(NO) ; serum lipid peroxidation product : malondialdehyde (MDA) and histopatholgical examination of aorta. The results of the current work indicated that dietary cholesterol supplementation significally increased lipid profile parameters ;total lipids, total cholesterol(TC), triacylglycerol (TAG), HDL-cholesterol (HDL-C) and LDL-cholesterol (LDL-C),MDA and tissue SOD. On the other hand, cholesterol supplementation reduce NO, catalase, blood SOD and blood GSH but there was no change in tissue GSH .Also the result indicated that cholesterol and flaxseed supplementation on the same time lead to increase lipid profile parameters (total lipids, total cholesterol (TC), HDL-cholesterol (HDL-C) and LDL-cholesterol (LDL-C) but not affect triacylglycerol (TAG), also, increase MDA, NO, catalase and tissue SOD but not affect blood SOD and decrease blood GSH , but not changed in tissue. On the other hand flaxseed supplementation alone was resulted in increased total lipids ,slight increase in total cholesterol (TC) ,there was no change in triacylglycerol (TAG), decrease HDL-cholesterol (HDL-C) ,increase LDL- cholesterol(LDL-C), no change in MDA and NO, increase blood catalase, liver GSH, but decrease tissue catalase ,blood GSH. From these results it can be concluded that flaxseed assured no protection against atherosclerosis in cholesterol fed rabbits.*

INTRODUCTION

A highly nutritious diet, good health, and a reduced risk of chronic diseases relationship have been studied (**Kendall et al., 2008).** As a result, the searching for functional foods had great importance resulting in healthful benifits **(Anjo, 2004).** It has gotten a major boost, because of the great attention to nutrition **(Niva; Makela, 2007).** Flaxseed has a great role in the field of diet and disease investigation due to its beneficial effects to health and disease prevention **(Herci et al., 2011).** Flax (*Linumusitatissimum*) is one of the oldest plants with high importance in food production, healthcare, pharmaceutics and industry as it containes fiber and oil. Flaxseed considers as major source of phytochemicals in the field of nutrition. Flaxseed considers an

excellent source of protein with good-quality and soluble fiber and has considerable potential as a source of phenolic compounds, in addition to being member of the highest sources of $\omega-3$ fatty acids and lignans **(Jhala and Hall ,2010; Oomah, 2001).** The use of whole flaxseed (Linumusitatissimum) and its derivatives (ground flax, flax oil, defatted flax, flax fibre and lignan extracts) has growing interest as functional food or nutraceutical ingredients and adjuncts to a healthy diet, as a result of the growing body of evidence over the past 20 years, studying the flaxseed protection against a many chronic diseases and risk factors as breast and colon carcinogenesis, atherosclerosis, insulin dependent diabetes mellitus (IDDM) and hyperlipoproteinemias **(Daun et al., 2003; Thompson, 2003; Prasad, 2000)**.

This study was carried out to investigate the beneficial effect of flaxseed-rich diet on atherosclerotic rabbits.

MATERIAL and METHODS

Animals and sample collection

Forty male rabbits were randomly classified in four groups as the following: Group I: supplied with control diet for two months which was kept as a Control group. Group II: (cholesterol group): rabbits supplied with 1% cholesterol diet. Group III: (flaxseed treated group): rabbits supplied with 10% flaxseed diet and Group IV:(cholesterol and flaxseed group): rabbits supplied with1% cholesterol diet, and 10% flaxseed.

Each group was consisted of ten rabbits, supplied with clean source of water *adlibitum* for two months. Chemicals for lipid profile, antioxidant and oxidative stress parameters was supplied from (Biodiagnostic Company for chemicals and laboratory reagents, Egypt) for measuring total lipids according to **Zollner et al., (1962)**, total cholesterol level (TC) according to **Allain et al.,(1974)**, triglycerides level (TAG) according to **Fossati and Prencipe, (1982)**, HDL-cholesterol level (HDL-C) according to **Lopez – Virella et al., (1977)** ,LDL- cholesterol level (LDL-C) according to **Friedwald et al., (1972)**, blood and tissue antioxidants :reduced glutathione concentration (GSH) according to **Beutler, (1963)**, superoxide dismutase activity (SOD) by the method of **Nishikimi et al., (1972)** and catalase activity according to **Aebi , (1984) and Fossati, et.al . (1980)** ; oxidative stress parameter : nitric oxide concentration (NO)according to **Montgomery et al., (1961)** ; serum lipid peroxidation product : malondialdehyde concentration (MDA) according to **Ohkawa, et al., (1979).** Aorta of dissected rabbit of different groups were taken and fixed in 20% formalin for histopathological examination of aorta according to **Woods and Ellis, (1994).**

Statistical analyses

The results of this research were analyzed using SPSS (V.17) according to **Steel and Torrie, (1960).** The data was expressed by mean accompanied with its standard error and statistically analyzed using one way ANOVA.

Table (1) : The diet formulation according to **NRC,1977**.

Total protein	DE	Ca	P	Fiber
16%	2600 Kcal/Kg	1%	0.7%	14.2%

Table(2): Effect of different treatments on changes produced by cholesterol on serum lipid profile level.

Parameters / Groups	Total lipids (mg/dl)	TC (mg/dl)	TAG (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)
G I	1504.67±61.92^{d}	54.83±4.61^{c}	80.83±4.36^{c}	24.00±1.18^{c}	23.00±1.71^{c}
G II	3286.50±81.79^{b}	1138.83±30.46^{a}	277.00±3.18^{a}	250.67±1.48^{b}	884.83±3.44^{a}
G III	2449.33±101.18^{c}	309.83±17.32^{b}	84.50±4.79^{c}	12.83±1.25^{d}	278.50±23.72^{b}
G IV	3843.50±195.28^{a}	1186.67±11.16^{a}	100.50±2.08^{b}	255.00±.97^{a}	890.67±10.93^{a}

The values are represented by mean ±SEM at P≥0.05

Table (3): Effect of different treatments on changes produced by cholesterol on blood and tissue antioxidants activity and oxidative stress concentration

Parameters / Groups	Serum MDA (nmol/ml)	Liver MDA(nmol/g.tissue)	Liver NO (µmol/L)	Plasma Catalase (U/L)	Liver Catalase (U/g)	Blood SOD (U/ml)	Liver SOD (U/gm tissue)	Blood GSH (mg/dl)	Liver GSH (mg/g. tissue)
G I	24.33 ± 2.60^{b}	14.50 ± 1.12^{c}	4.08 ± .71^{ba}	799.67 ± 14.00^{b}	.92 ± .01^{a}	340.50 ± 3.77^{ab}	262.30 ± 51.77^{a}	5.37 ± .32^{a}	3.30 ± .06^{b}
G II	86.00 ± 2.37^{a}	41.33 ± 5.64^{b}	2.83 ± .33^{b}	701.67 ± 14.00^{c}	.89 ± .02^{a}	250.00 ± 6.58^{c}	282.17 ± 3.47^{a}	2.68 ± .11^{c}	3.41 ± .07^{b}
G III	28.33 ± 1.61^{b}	20.67 ± 2.26^{c}	4.67 ± .44^{a}	885.67 ± 9.17^{a}	.91 ± .02^{a}	308.00 ± 20.61^{b}	327.50 ± 1.71^{a}	3.98 ± .12^{b}	4.99 ± .12^{a}
G IV	79.67 ± 2.33^{a}	71.00 ± 1.37^{a}	4.90 ± .35^{a}	880.00 ± 25.56^{a}	.93 ± .01^{a}	342.33 ± 4.02^{a}	313.50 ± 3.20^{a}	3.87 ± .22^{b}	3.43 ± .08^{b}

The values are represented by mean ±SEM at P≥0.05

Histopathological examination

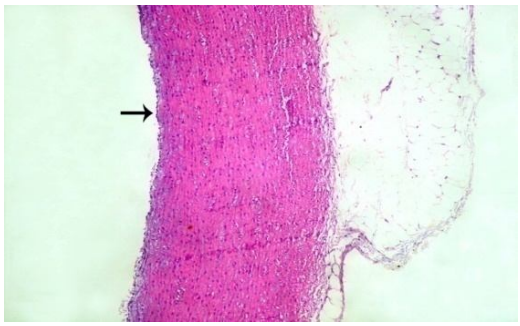

Figure (1): Aorta is showing normal tunica intima (arrow) with normal endothelium and normal tunica muscularis. (HE, 100x)**group I(control)**

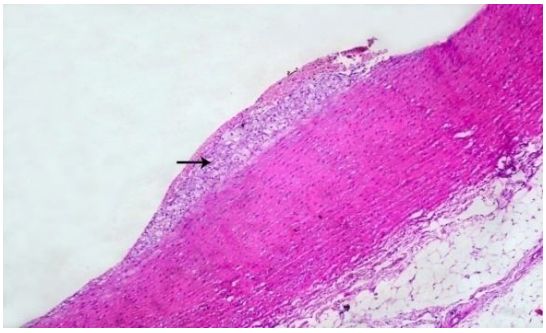

Figure (2): Aorta is showing atheromatous plague in tunica intima (arrow). (HE, 100x) **groupie (cholesterol)**

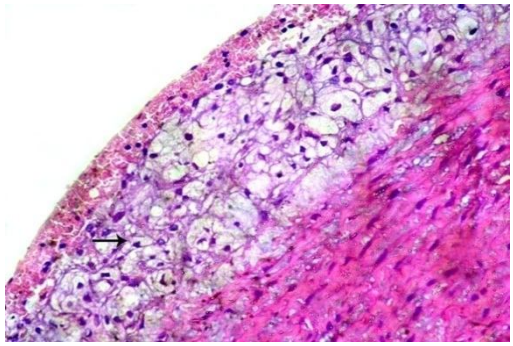

Figure (3): Aorta is showing atheromatous plague formed from aggregations foamy macrophages in sub-endothelial (arrow). (HE, 400x) **group II (cholesterol)**

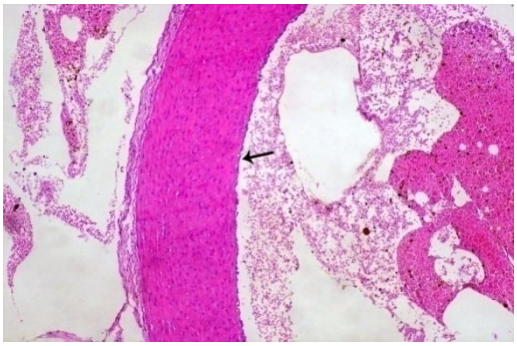

Figure (4): Aorta is showing normal tunica intima (arrow) with normal endothelium and normal tunica muscularis. (HE, 100x) **group III (flaxseed)**

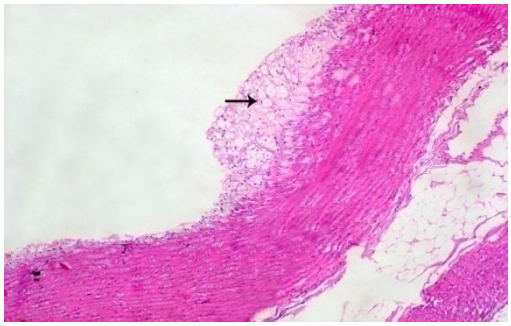

Figure (5): Aorta is showing atheromatous plague in tunica intima (arrow). (HE, 100x) **group IV (cholesterol + flaxseed)**

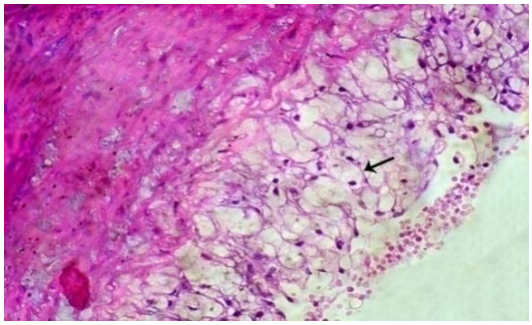

Figure (6): Aorta is showing atheromatous plague formed from aggregations foamy macrophages in sub-endothelial (arrow). (HE, 400x) **group IV (cholesterol + flaxseed)**

RESULTS & DISCUSSION

Atherosclerosis has several risk factors as; high concentration of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG) and a low concentration of high-density lipoprotein cholesterol (HLD-C) (**Program,1984; Criqui, 1991).** Flaxseed considers as anti-inflammatory and can affects glycemic control and oxidative stress positively. It has a great role in cardiovascular diseases (CVDs) control becaues it can decrease total cholesterol and low-density lipoproteins **(Bhathena et al., 2002; Rhee and Brunt, 2011).** The intestine can absorb oxidized fatty acids as linoleic acid (LA) from diet then converted it into lipoproteins and potentially impose oxidative stress and exacerbate atherogenesis **(Penumetcha et al., 2000).** FLC reduce the development of hypercholesterolemic atherosclerosis; in addition to decrease in oxidative stress and serum lipids (**Prasad, 2005**). Flax lignan complex (FLC) isolated from flaxseed contains 34% to 38% secoisolariciresinol diglucoside (SDG), 15% to 20% cinnamic acid glucoside, and 9.6% to 11.0% hydroxyl methylglutaric acid (**Westcott et al., 2008**). Secoisolariciresinol diglucoside **(Prasad, 2000)** and cinnamic acid **(Foti et al., 1996)** are antioxidants, and hydroxyl methylglutaric acid is a hypolipidemic agent **(LupienP et al., 1979).** Hypercholesterolemia lead to increase the release of platelet activating factor (PAF), **(Prasad, Kalra et al.,1994.)** which stimulate the synthesis and release of interleukin-1 **(Pignol et al., 1987)** and tumor necrosis factor (TNF) **(Bonavida, Mencia-Huerta et al.,1989)**, Leukotriene B4 (**Sumimoto et al., 1984**), PAF, **(Shaw et al., 1981)**, interleukin-1**(Braquet et al.,1989**)and tumor necrosis factor **(Paubert-Braquet et al., 1988)** are lead to stimulate PMNLs to produce OFRs, which would stimulate the development of hypercholesterolemic atherosclerosis. Aortic endothelial cells in the rabbit initiate NO release which are stimulated by oxidized low-density lipoprotein **(Fries DM et al ., 1995)** and NO may affect the synthesis and metabolism of lipoproteins . Lipoproteins considers as inhibitors of endothelium-dependent relaxation of rabbit aorta **(Takahashi et al ., 1990).** The results of the current work indicated that dietary cholesterol supplementation increased lipid profile parameters(total lipids, total cholesterol(TC),triacylglycerol(TAG), HDL-cholesterol (HDL-C) and LDL-cholesterol(LDL-C) as showed in table (2), increased MDA and tissue SOD as showed in table (3). On the other hand, we find that cholesterol supplementation reduce NO, catalase, blood SOD and blood GSH but no change in tissue GSH as showed in table (3) . These results were supported by histopathological examination which showed atheromatous plague in tunica intima of aorta as showed in figure (2) and figure (3) (high power). Also, the result indicated that cholesterol and flaxseed supplementation on the same time lead to increase lipid profile parameters (total lipids, total cholesterol (TC), HDL-cholesterol (HDL-C) and LDL-cholesterol (LDL-C) but not affect triacylglycerol (TAG)as showed in table (2), also, increase MDA, NO, catalase andtissue SOD but not affect blood SOD and decrease blood GSH , but not changed in tissue GSHas showed in table (3) . Also, these results were supported by histopathological examination which showed atheromatous plague in tunica intima of aortaas showed in figure (5) and figure (6) (high power) . On the other hand flaxseed supplementation alone is resulted in increased total lipids ,slight increase in total cholesterol (TC) ,no change in triacylglycerol (TAG), decrease HDL-cholesterol (HDL-

C),increase LDL- cholesterol (LDL-C)as showed in table (2) ,no change in MDA and NO, increase blood catalase, liver GSH, but decrease tissue catalase ,blood GSH as showed in table (3) .These results were supported by histopathological examination which showed normal tunica intima with normal endothelium and normal tunica muscularis of aorta as showed in figure (4).These results were in agreement with those of **Essam Fetal., 2012 ; Kailash Prasad ,1999 ;Andrew et al., 2013 ; Delgado Roche et al., 2010.** FLC prevent acceleration of atherosclerosis, stimulated by removal of a high-cholesterol diet, but it does not produce regression of it (**Prasad, 2007).**

CONCOLUSSION

From these results it can be concluded that flaxseed assured no protection against atherosclerosis in cholesterol fed rabbits.

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الملخص العربي

تأثير بذور الكتان علي دهون الدم وتصلب الشرايين في الأرانب

أ.د/السعيد الشربيني السعيد أ.د/جهاد رمضان السيد د/محمود جميل السباعي فاطمة حسن الغريب

قسم الكيمياء الحيوية وكيمياء التغذية . كلية الطب البيطري .جامعة المنصورة.مصر

أجريت هذه الدراسة لمعرفة تأثير إضافة بذور الكتان في العليقة إلى الأرانب المصابة تجريبيا بتصلب الشرايين. حيث قسمت الأرانب إلى ٤ مجموعات: المجموعة الأولى تتغذى على عليقه طبيعية ، المجموعة الثانية تتغذى على عليقة مضاف إليها كوليستيرول ١% ، المجموعة الثالثة تتغذى على عليقة مضاف إليها بذور الكتان ١٠% والمجموعة الرابعة تتغذى على عليقة مضاف إليها كوليستيرول ١% بالإضافة إلى بذور الكتان ١٠%.

تم تجميع عينات الدم بعد شهرين لقياس دهون الدم مثل الكوليستيرول والدهون عالية الكثافة النافعة والدهون الضارة والدهون الثلاثية والدهون الكلية ومضادات الاكسدة فى الدم والانسجة والفحص الباثولوجي للشريان الأورطى. أظهرت النتائج ان اضافة بذور الكتان أدت إلى زيادة دهون الدم مثل الدهون الكلية والكوليستيرول والدهون الثلاثية والدهون عالية الكثافة والدهون الضارة في الأرانب المضاف إلى عليقتها كوليستيرول . بينما إضافة كوليستيرول وبذور كتان فى نفس الوقت إلى العليقة أدى إلى زيادة دهون الدم مثل الدهون الكلية والكوليستيرول والدهون عالية الكثافة والدهون الضارة ولم يؤثر على الدهون الثلاثية. من ناحية أخري إضافة بذور الكتان فقط للعليقة أدت إلى زيادة دهون الدم مثل الدهون الكلية والكوليستيرول والدهون الضارة ولم يؤثر على الدهون الثلاثية لكنها قللت الدهون عالية الكثافة. أثبتت النتائج أن بذور الكتان لا تؤدى الى الوقاية من حدوث تصلب الشرايين في الأرانب المصابة تجريبيا.