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EFFECT OF SOME POLYUNSATURATED FATTY ACIDS RICH-OILS ON PLASMA LIPID PROFILES USING NORMAL RATS

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ABSTRACT

The purpose of this study was to examine whether using small amount (8%) of flaxseed oil and small amount of evening primrose oil (8%) may affect the level of blood lipid profiles in rats. Twenty one male albino rats were divided randomly into three groups each of seven rats. One group received a basal diet as a control group; the other two groups were fed on diet with flaxseed oil (FO) or evening primrose oil (EPO). After 6 weeks, rats were fasted over night and blood samples were collected. The evolution of nutritional value depended on determination of total food intake, body weight gain and food efficiency ratio, the nutritional status of rats fed different diets was also evaluated through determination of certain biochemical parameters such as total lipids, total cholesterol, high and low density lipoproteins, the ratio of high density lipoprotein / total cholesterol (TC) and triglycerides in plasma were determined. The results showed that the adding of flaxseed oil to rat's diet lowers plasma cholesterol, triglycerides, and LDL while the adding of evening primrose in the rat's diet significantly lowers cholesterol and triglycerides. The group of rats fed on both oils cause an increase in HDL and HDL/TC ratio. No significant effect ($p<0.05$) using both oils was observed on the nutritional evaluation. These data indicate that n-3 and n-6 fatty acids found in flaxseed oil and evening primrose oil respectively, participate in the plasma lipids metabolism of rats. The present study examines the effect of using small amount of flaxseed and evening primrose oils (8 %) for the therapy of hyper-lipidemia.

Keywords: Flaxseed Oil, Evening Primrose Oil, Polyunsaturated fatty acids, plasma lipids, Rats.

Abbreviations:
PUFAs; Polyunsaturated Fatty acids
ALA; Alpha Linolenic acid
LA; Linoleic acid
FO; Flaxseed oil
EPO; Evening Primrose oil
HDL; High-Density Lipoprotein
LDL; Low-Density Lipoprotein
VLDL; Very Low-Density Lipoprotein
EPA; Eicosapentaenoic acid
DHA; docoshexaenoic acid

INTRODUCTION

Increasing fat intake in diet can cause an excessive increase in white adipose tissue (Obesity) which may cause diseases such as diabetes mellitus, hyperlipidemia and arteriosclerosis (Rothstein, 2006). Accumulation of triacylglycerol causes changes in liver function and is strongly associated with the development of pathological conditions such as fatty liver,

hyperlipidemia. Therefore, preventing fat accumulation in white adipose tissue and liver is an approach to preventing these diseases (Tsuzukia *et al.*, 2006).

The diet, containing polyunsaturated fatty acids (PUFAs), can play an important role in the control of lipid profile and cholesterol homeostasis (Cintra *et al.*, 2006 &Vijaimoha *et al.*, 2006).The ingestion of PUFAs (n-3 and n-6), is related to the lowering of the incidence of heart disease by decreasing cholesterol and triacylglycerol plasmatic levels (Simopoulos , 1999). Mammalian cells do not contain enzymes capable of adding double bonds (desaturate) to fatty acids after the ninth carbon from the carboxyl end of the molecule. As such, n-3 fatty acids can not be synthesized and must be provided in the diet (Whelan and Rust, 2006).

The main fatty acid components of flaxseed and evening primrose oils are presented in table (1). Flaxseed oil (FO) contains saturated fatty acids such as palmitic (16:0) and stearic (18:0), mono-unsaturated fatty acid as oleic (18:1, n-9) and rich with PUFAs such as α-linolenic acid (18 :3 n-3) a precursor to eicosapentanoic acid (EPA), (Vijaimoha *et al.*, 2006). Many authors showed the importance of FO in lowering hyperlipidemia and cholesterol in blood (Cunnane *et al.*, 1993 and 1994). Flaxseed also lowered serum total cholesterol and low-density-lipoprotein (LDL) - cholesterol. Pellizzon *et al.*, (2007) showed that flaxseed reduced plasma cholesterol levels in hypercholesterolemic mouse models. PUFAs, including ALA, have been reported to suppress the development of obesity and hepatic fat accumulation, as well as lowering serum lipid levels (Ikemoto *et al.*, 1996).

Table (1): Fatty acids contents (%) of evening primrose and flaxseed oils.

Fatty acids	Evening primrose oil*	Flaxseed oil**
Linoleic, 18:2 (n-6)	65-80	17
α- Linoleic, 18:3 (n-3)	-	51-55
γ- Linoleic, 18:3 (n-6)	8-14	-
oleic, 18:1 (n-9)	11-16	18
Stearic, 18:0	1.5-3.5	4
Palmitic, 16:0	7-10	6

*** according to Christie, 1999.**
**** according to Vijaimoha *et al.*, 2006.**

As presented in table (1), evening primrose oil contains saturated fatty acids such as palmitic (16:0) and stearic (18:0), mono-unsaturated fatty acid as oleic (18:1, n-9) oleic and rich in PUFAs such as linoleic 18:2(*n*-6) and γ-linolenic (18:3(*n*-6)) acids (Christie, 1999). EPO, enriched with γ-linolenic acid, produces significant changes in tissue fatty acid composition. Tissue incorporation of the n-6 PUFAs may restore the lipid environment which influences a number of cellular properties such as membrane fluidity, ion transport, receptor interactions and prostanoid synthesis (Engler and Engler, 1998) The hypocholesterolemic effect of γ-linolenic acid as evening primrose oil was proved (Sugano *et al.*, 1986, Balasinska, 1998, Fukushima *et al.*, 2001 and Lee, 2007). All the previous studies used PUFAs with content more

than 10% in the tested diet. So, The object of this study that using the flaxseed oil (8%) (4.24 g α-linolenic acid) and even primrose oil (8%) (5.9 g linoleic acid and 0.9 g ω-linolenic acid) would control the level of plasma lipid profiles in rats fed normal diet. In this case, FO and EPO can be used with such small amounts in the dietary diet beside the usual oils and still effective in lowering plasma lipid profiles. The objective of this study is to investigate the effect of FO and EPO (used with small amounts), rich in n-3 and n-6 PUFAs, on growth parameters and serum lipids of rats fed with normal diets.

MATERIALS AND METHODS

Experimental diets:

Diets were based on the AIN-93 diet (Reeves *et al.*, 1993). All diets were balanced to contain 12.0% protein, and 8% fat, with adequate vitamins and minerals provided by the AIN-93 formulation. The oil soluble vitamin were given weekly to rats separate form the diet, the diets were competed by corn starch to 100 % .The diets were prepared and stored frozen for the duration of the experiment (6 weeks). One group received a control diet. The other two groups were fed on tested .the composition of different experimental diets are shown in table (2).

Table (2): Composition of the experimental diets (g /100g).

Ingredients	Diet 1 Control	Diet 2 Flaxseed	Diet 3 Primrose
Casein*	15	15	15
Sucrose	10	10	10
Evening primrose oil	-	-	8
Flaxseed oil	-	8	-
Corn oil	8	-	-
Salt mixture*	3.5	3.5	3.5
Vitamin mixture*	1	1	1
Fiber (cellulose)	5	5	5
L-cystine	0.18	0.18	0.18
Choline bitartrate	0.25	0.25	0.25
Corn starch	57.07	57.07	57.07
Total	100	100	100

*** Casein was analyzed to be 80.0% protein (N= 6.38).(Reeves *et al.*, 1993).**
All diets containing 12% protein
**** Vitamin and Salt mix. (AIN-93, Reeves *et al.*, 1993).**

Experimental rats:

Twenty one male albino rats with an average weight of 100 g were obtained from the Animal House of the National Research Center. All animals were housed individually in stainless steel cages and fed on a basal diet for one week and then were divided randomly into three groups each of seven rats. During the period of experiment, diets and water were fed ad-libitum and food intake was calculated, body weights of rats were measured twice

weekly. After the end of experimental period, biological changes including food intake, body weight gain, food efficiency ratio (body weight gain (g) / total food intake (g)) was calculated according to Smith and Circle (1971). At the end of the experiment, rats were fasted 16-18 hours and the blood samples were collected.

Plasma was separated from heparinized blood by centrifugation (3000 rpm for 15 min) for lipid profiles determinations. Total lipids were determined according to the method described by Draven and Schmite (1964). Total plasma cholesterol was determined according to Richmond (1973), plasma high-density lipoprotein (HDL) -Cholesterol was determined according to the method used by Lopes-Virella (1997); plasma LDL Cholesterol was determined according to the method of Assmann *et al.* (1984) and triglycerides were determined according to the method used by Fossati & Prencipe (1982).

Statistical Analysis

Results are expressed as means ± standard errors of means (SEM). Comparison between the means (of diet effects) was accomplished using a one-way ANOVA, followed by Duncan Multiple Range Tests for all variables (Duncan, 1955). Differences between groups were considered significant at $p< 0.05$.

RESULTS AND DISCUSSION

Evening primrose and flaxseed oils considered a rich source of poly unsaturated fatty acids (n-3, n-6 and n-9). These oils provided a great control in plasma lipid profiles (Cintra *et al.*, 2006).This study tested the effect of adding small amount of these oils (8%) on levels of plasma lipid profiles in normal rats.

Food intake and the growth parameters:

All diets were well accepted by rats. There were no treatment- related changes in the appearance or behavior of rats during the experiment. All rats remained healthy during the experimented period. Table (3) showed that there are no significant differences ($p<0.05$) between the tested groups (EPO and FO) and control group for total food intake (657.6, 636 and 630g /42 days, respectively), food efficiency ratio which recorded 0.224, 0.227and 0.237, respectively, liver weight values were 7.3, 6.8 and 7.0 g, respectively, and liver / body weight ratio (2.7, 2.5 and 2.6 g/100g, respectively). There is a significant decrease for body weight gain in the EPO and FO groups in comparison with the control group (147, 149 and 164.2 g/42 days, respectively, $p<0.05$). This effect may be due to the influence of n-3 and n-6 fatty acids contained in the FO and EPO respectively on the β-oxidation of fatty acid in the liver which may contribute to lowering of the body lipids (Kabir and Ide, 1996).

Table (3): Effect of flaxseed and evening primrose oils on growth parameters.

Parameters	Control	Flax seed oil	Evening primrose oil
Food intake, *g/42 d*	630.4 ± 28.5 [a]	636 ± 36.0 [a]	657.6 ± 18.0 [a]
Food efficient ratio	0.237 ± 0.03 [a]	0.227 ± 0.01 [a]	0.224 ± 0.02 [a]
Body weight gain, *g/42 d*	164.2 ± 10.9 [a]	149 ± 13.3 [b]	147 ± 11.4 [b]
Liver weight, *g*	7.0 ± 1.0 [a]	6.8 ± 0.6 [a]	7.3 ± 0.2 [a]
Liver/body weight, *g/100 g*	2.6 ± 0.1 [a]	2.5 ± 0.2 [a]	2.7 ± 0. 2 [a]

Mean values with different letters in each testing parameter were significantly different (P<0.05).

Plasma lipid parameters.

Table (4) showed the plasma analysis including total lipids, total cholesterol, HDL, LDL, and the ratio of HDL / total cholesterol, triglycerides. For total lipids values, the rat group fed on the control diet has the highest value (272.4 mg/dl) while the FO group was the lowest value (227.4 mg/dl). EPO group showed a medium decrease (251.5 mg/dl). For total cholesterol values of the two tested groups fed with FO or EPO were lower than the control group (41.7, 52.6 and 55.9 mg/dl, respectively), but in case FO group showed a significant decrease ($p<0.05$). The HDL results showed that the tested groups which fed on (FO and EPO) have higher significant HDL than control group (36.0, 39.9 and 30.1 mg/dl, respectively, $p<0.05$) and with slightly significant increase in the EPO group. The ratio of HDL / Total Cholesterol showed significant increase ($p<0.05$) in the oils fed rat groups (FO or EPO) than the control group (1.298, 1.209 and 0.833, respectively). For the LDL, only the group of rats fed on FO showed low value (19.2 mg/dl) but this value was non-significant ($p<0.05$). The triglycerides values for oils groups (FO or EPO) showed tendency to be lower than the control group (56.1, 58.5 and 63.8 mg/dl, respectively).

Table (4): Effect of flaxseed and evening primrose oils on plasma lipid profiles of rats.

	Control	Flaxseed oil	Evening primrose oil
Total lipids (mg/dl)	272.4 ± 42.4 [a]	227.4 ± 68.2 [a]	251.5 ± 45.5 [a]
Total Cholesterol (mg/dl)	55.9 ± 8.8 [a]	41.7 ± 6.1 [b]	52.6 ± 5.6 [a]
Triglycerides(mg/dl)	63.8 ± 11.9 [a]	56.1 ± 10.5 [a]	58.5 ± 11.7 [a]
HDL- Cholesterol (mg/dl)	30.1 ± 8.3 [a]	36.0 ± 10.2 [a]	39.9 ± 6.0 [a]
LDL-Cholesterol (mg/dl)	26.9± 9.7 [a]	19.2 ± 4.9 [a]	26.2 ± 5.1 [a]
HDL-Ch /Total Cholesterol ratio	0.833 ± 0.3 [b]	1.298 ± 0.3 [a]	1.209 ± 0.2 [a]

Mean values with different letters in each testing parameter were significantly different (P<0.05).

The action of feeding FO resulted in a significant decrease of total cholesterol, this is an indicative of modifications in cholesterol metabolism but the action of feeding EPO decreased total cholesterol without significant difference this is because of FO contains mainly ALA as PUFA while EPO

contains LA 65-80 % and γ-linolenic acid 8-14 % as PUFA (table (1)). The effect of these oils on cholesterol and other lipid metabolism may be due to the PUFAs that found in these oils on the metabolism of lipids.

It was showed that dietary ALA increased both peroxisomal and mitochondrial β-oxidation of fatty acid in the liver which may contribute to lowering of the serum lipid (Kabir and Ide, 1996). Also, it was demonstrated that long-chain n-3 PUFAs such as eicosapentaenoic (EPA) and docoshexaenoic acids (DHA), which are metabolites of ALA, increase both peroxisomal and mitochondrial fatty acid oxidation in the liver (Yamazaki *et al.*, 1987 and Willumsen *et al.*,. 1993). Dietary ALA increases the proportion of both EPA and DHA in liver phospholipid, suggesting that dietary ALA is efficiently elongated and desaturated in the liver (Kabir and Ide, 1996). It is apparent that ALA-rich oils increase the gene expression of fatty acid oxidation enzymes and consequently enhance β-oxidation activity in the liver (Ide *et al.*, 2000).

Dietary γ-linolenic acid, like ALA, has a physiological activity to enhance hepatic β-oxidation in the rat liver. It was indicated that differences existed between n-3 and n-6 octadecatrienoic acids in the physiological activity affecting hepatic β-oxidation enzyme. Accordingly, dietary fat rich in ALA enhanced both the mitochondrial and peroxisomal fatty acid oxidation rate whereas fat rich in γ-linolenic acid enhanced the peroxisomal, but not the mitochondrial, fatty acid oxidation rate. Dietary ALA reduced 3-hydroxyacyl-CoA dehydrogenase activity in the rat liver while γ-linolenic acid, however, did not reduce this enzyme activity. In addition, ALA enhanced mitochondrial acyl-CoA dehydrogenase activity, but γ-linolenic acid profoundly reduced it. This reduced enzyme activity may account for the failure of dietary γ-linolenic acid to increase the mitochondrial β-oxidation rate in the rat liver (Kumamoto and Ide , 1998). Gormaz *et al.*, (2010) reviewed the effect of long-chain polyunsaturated fatty acids in non-alcoholic fatty liver disease which is characterized by steatosis, the abnormally high accumulation of triacylglycerols
in liver tissue (Anderson and Borlak, 2008), which is associated with an impairment in the bioavailability of LCPUFAs (Araya *et al.*, 2004). commonly presents along with mainly obesity that share with non-alcoholic fatty liver metabolic and inflammatory components. These conditions, particularly non-alcoholic fatty liver, are associated with alterations in the bioavailability of long-chain polyunsaturated fatty acids (LCPUFAs). In the human,the bioavailability of LCPUFAs depends both on endogenous biosynthesis and diet amount of preformed LCPUFAs.

Morise *et al.*, (2004) reported that in response to the ALA-rich diet, there was a lower accumulation of hepatic cholesteryl esters and higher activities of HMGCoA reductase and CYP7A1. So, it can be hypothesized that the higher activity of CYP7A1 results in a higher cholesterol secretion into bile, leading to a depletion of the interahepatic pool of cholesterol and thus to an increase in cholesterol synthesis and turnover.

It was reported that incubation of human hepatoma cell line, the Hep G2 cell, with EPA resulted in reducing the secretion of VLDL triacylglycerol and apoprotein B. Incubation with LA reduced VLDL triacylglycerol but not

apoprotein B (Wongl and Nestel , 1987). Other workers also concluded that dietary n-3 fatty acids lower plasma LDL levels in normal human subjects by reducing the rate of synthesis of apoprotein B (Illingworth *et al.*, 1984). Wong, *et al.* (1984) investigated the effects of n-3 and n-6 fatty acids-rich oils on lipid and lipoprotein metabolism in perfused rat liver. The results indicate that the lowering of plasma triacylglycerols by n-3 fatty acid reflects: (a) diminished lipogenesis; (b) increased fatty acid oxidation possibly in peroxisomes; and (c) diminished secretion of triacylglycerols by the liver. The effect of n-6 fatty acid was intermediate between the control group and the n-3 group.

The effects of dietary alpha or γ-linolenic acid on levels and fatty acid composition of serum and hepatic lipids in rats were investigated by Ihara-Watanabe *et al.*, (1999). They indicated that ALA exhibits a larger hypocholesterolemic effect than γ-linolenic acid and it may be displayed mainly through the repression of the activity and mRNA expression of HMG-CoA reductase. However, the hypocholesterolemic effect of γ-linolenic acid was proved by many authors (Sugano *et al.*, 1986, Balasinska, 1998, Fukushima *et al.*, 2001 and Lee, 2007).

The present results revealed that FO containing ALA lowers cholesterol significantly, triglycerides, and LDL lipids, while EPO, containing LA and γ-linolenic acids, lowers cholesterol and triglycerides, with less degree, but has no effect on LDL lipids. This difference may be due to the different mode of action of ALA, LA and γ-linolenic acids on lipid metabolism as mentioned in the previous discussion. The unaffected LDL levels in case of using EPO may be due to that LA has no effect on reducing the rate of synthesis of apoprotein B (Wongl and Nestel , 1987 and Illingworth *et al.*, 1984). The present study indicate that FO containing ALA lowers cholesterol significantly, triglycerides, and LDL lipids, while EPO, containing LA and γ-linolenic acids, lowers cholesterol and triglycerides, with less degree, but has no effect on LDL lipids. This difference may be due to the different mode of action of ALA, LA and γ-linolenic acids on lipid metabolism as mentioned in the previous discussion. The unaffected LDL levels in case of using EPO may be due to that LA has no effect on reducing the rate of synthesis of apoprotein B (Wongl and Nestel , 1987 and Illingworth *et al.*, 1984).

The tendency of increase in HDL concentration and the significant increase of HDL/Total Cholesterol ratio between groups fed PUFAs-containing oils and control group can be interpreted by the modulating action of PUFAs on the activities of lecithin: cholesterol acyltransferase (Applebaum-Bowdesn, 1995, Homma, 1989 and Fragoso and Skinner, 1992), which converts cholesterol into cholesteryl ester, which is then sequestered into the core of a lipoprotein particle, eventually making the newly synthesized HDL. Also, the modulating action of PUFAs on lipoprotein lipase plays an important role in lipoprotein metabolism (Saxena and Goldberg, 1990). The small difference in the increase of HDL between groups fed on FO and EPO may depend on the difference between the action of n-3 and n-6 fatty acids found in these oils on lecithin: cholesterol acyltransferase and lipoprotein lipase. Also, the amount of cholesterol measured in FO group was

less than found in EPO group. In this case, the action of lecithin: cholesterol acyltransferase was greater in EPO group than in FO group.

Conclusion

It could be concluded that the rat diets contain small amount of FO (8%) has a decreasing effect on plasma cholesterol, triglycerides, and LDL but the EPO has a moderate effect on lowering cholesterol and triglycerides in comparison to FO but has no effect on plasma LDL. Plasma HDL content was significantly increased in groups fed on FO and EPO. The clear effects of these oils were assigned to their content of n-3 and n-6 fatty acids and their effect on lipid metabolism. The results of nutritional evaluation and plasma lipid profiles indicate that these oils can be used safely in the treatment of hyperlipidemia with diet content (8%) of the tested oils.

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تأثير بعض الزيوت الغنية بمحتواها من الاحماض الدهنية عديدة عدم التشبع علي ليبيدات البلازما لفئران مغذاه علي علائق متزنة.

ماجدة سليمان محمد و عبير امين عفيفي
قسم علوم الأطعمة والتغذية- المركز القومي للبحوث- دقي- القاهرة- جمهورية مصر العربية

تهدف الدراسة الحالية الي اختبار تاثير اضافة الزيوت الغنية في محتواها من الآحماض الدهنية عديدة عدم التشبع مثل زيت الكتان وزيت البريميروز بنسبة ٨ % علي تركيز ليبيدات الدم وتشمل الليبيدات الكلية – الكوليسترول الكلي- التراي جليسريدات- الليبوبروتينات عالية و منخفضة الكثافة والنسبة بينهما وكذلك دراسة تأثيرهذه الزيوت علي كفاءة الاستفادة من الغذاء والزيادة في وزن الجسم.

في الدراسة الحالية تم استخدام عدد ٢١ فار(ذكور- الوزن ١٠٠ جم) علي ثلاث مجموعات: المجموعة الأولي تم تغذيتها علي عليقة متزنة بها زيت الذرة (مجموعة ضابطة) – المجموعة الثانية تغذت علي عليقة متزنة مع استبدال مصدر الزيت باضافة زيت الكتان بنسبة ٨ % – المجموعة الثالثة تغذت علي عليقة متزنة مع استبدال مصدر الزيت باضافة زيت البريميروز بنسبة ٨ % وكانت مدة التجربة ستة اسابيع. في نهاية التجربة تم تخدير الفئران وجمع عينات الدم واستخدام الطرق القياسية لإجراء التقديرات موضع الدراسة.

وكانت النتائج المتحصل عليها هي انخفاض مستوي الكوليسترول الكلي والجليسريدات الثلاثية والليبوبروتينات منخفضة الكثافة نتيجة لاضافة الزيوت موضع الدراسة مقارنة بالمجموعة الضابطة.

ولم يحدث تغير معنوي في تركيز الليبيدات الكلية وكفاءة الاستفادة من الغذاء حيث كانت هذه التقديرات في زيادة او نقص غير معنوي مقارنة بالمجموعة الضابطة.

نستخلص من هذه الدراسة انع يمكن استخدام زيت الكتان وزيت البريميروز بتركيز صغير (٨ %) لخفض تركيز ليبيدات الدم الضارة.

قام بتحكيم البحث

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