Algerian Journal of Natural Products 3:3 **(2015)** 177-184

OPEN ACCESS

E-ISSN: 2353-0391

Algerian Journal of Natural Products
www.univ-bejaia.dz/ajnp

Type of the Paper (Original Article)

Effects of maternal linseed oil supplementation on oxidative stress markers in cafeteria diet induced obese rats

Nawel BENAISSA[1], Hafida MERZOUK [1,]*, Sid Ahmed MERZOUK [2], Michel NARCE[3]

[1]Laboratory of Physiology and Biochemistry of Nutrition, Department of Biology, University Abou-Bekr Belkaïd, Tlemcen 13000, Algeria, [2] Department of technical sciences, Faculty of Engineering, University Abou-BekrBelkaïd, Tlemcen 13000, Algeria, [3]INSERM UMR 866, "Lipids Nutrition Cancer", University of Burgundy, Faculty of Sciences, 6 Boulevard Gabriel, Dijon 21000, France.

*****Author to whom correspondence should be addressed; E-Mail: hafidamerzouk_2@hotmail.com; Tel.:* 00 213 778303645 *; Fax:* 00 213 40911082.

Received: 01/05/2015 Revised: 09/07/2015 /Accepted: 15/07/2015

Abstract: We investigated the role of dietary linseed oil in the modulation of biochemical parameters and oxidant/antioxidant markers in cafeteria-induced obese rats and their offspring. Female wistar rats were fed on control or cafeteria diet, supplemented or not with linseed oil (5%) for one month before and during the gestation. At parturition, the mothers and their offspring were killed. Weight gain, food intake, serum biochemical and oxidant/antioxidant markers were determined. Cafeteria diet induced a significant increase in body weight, food intake and adverse alterations in biochemical parameters such as an increase in serum glucose, triglyceride, cholesterol and oxidant markers. Linseed oil supplementation induced a reduction in weight gain, serum lipids and a modulation of oxidative stress, improving metabolic status. In conclusions, linseed oil displayed remarkable health benefits by decreasing plasma and oxidant/antioxidant markers in both obese mothers and their newborns.
Keywords: *cafeteria; linseed oil; maternal obesity; lipid metabolism; newborn; Oxidant/antioxidant markers.*

I. Introduction

During pregnancy, the availability of nutrients to the fetus depends on placental supply and maternal nutrition. Adverse nutritional environments during early life have long-lasting consequences and may change some physiological parameters at adulthood. Obesity is one of the most common health problems for pregnant women. Animal studies also indicate that maternal obesity programs offspring predisposition to a wide variety of chronic, later-life diseases [1]. Maternal obesity program changes in adiposity, hepatic metabolism, glucose homeostasis, and lipid profile, and are important in promoting obesity in offspring [2,3].

Obesity is an important metabolic disorder characterized by reduced insulin sensitivity and lipid metabolism abnormalities, in both animal models and humans [4].Obesity is characterized by insulin resistance, alterations in carbohydrate and fat metabolism and by increased adipose tissue mass as well as plasma and tissue cholesterol and triglyceride accumulation [4,5].

As the prevalence of obesity is reaching epidemic proportions, understanding the effect of maternal overnutrition on fetal growth and its long-term consequences on health are of particular importance. A cafeteria diet is known to induce obesity in animals [6]. Cafeteria-diet in pregnant rats induced maternal obesity with long-term metabolic consequences in the offspring, including an increase in

lipogenic capacity in adipose tissue, impaired glucose homoeostasis, and altered body composition and metabolism [7,8]. Indeed, maternal cafeteria feeding affected liver and adipose tissue metabolism leading to permanent changes in hepatic and adipose enzyme activities and fatty acid composition in the offspring [9].

Obesity is one of the most common health problems for pregnant women. Maternal obesity is associated to several complications, such as high blood pressure, eclampsia, gestational diabetes and macrosomia [10,11]. Obesity is associated with glucose and lipid metabolism abnormalities, increased cardiovascular risk and oxidative stress [12,13].

Oxidative stress is highly correlated with a wide variety of inflammatory and metabolic disease states, including obesity [14] and with cumulative damage in the body done by free radicals inadequately neutralized by antioxidants [15].

In obese patients, the increase in oxidative damage may be a consequence of hyperglycaemia, hyperlipidaemia, increased tissue lipid levels, inadequate antioxidant defenses, increased rates of free radical formation and chronic inflammation [16].

Previous findings have documented that not only quantity, but also fat type used in the diets will affect the rate of weight gain. Saturated fatty acids (SFAs) have been shown to produce weight gain and obesity compared with other types of fatty acids [17]. In contrast, n-3 polyunsaturated fatty acids (n-3 PUFA) generally reduce the rate of weight gain compared with other fatty acids, and play an important role in the prevention of metabolic diseases [18,19]. In fact, *n*-3 PUFA lower both plasma cholesterol and triglycerides and are useful in improving insulin sensitivity and treating dyslipidemia and metabolic syndrome [20,21].Therefore, both the amount of fats used in the diet and the fatty acid profile of these fats affect body-weight and metabolism regulation. Modification of dietary fat composition may influence metabolic disorders associated with obesity. However, although it is well documented that the consumption of diets high in n-3 PUFA can improve metabolic alterations, their beneficial effects on maternal and neonate obesity have not been elucidated.

Flaxseed or linseed (*Linum usitatissimum* L.) has been a focus of interest in the field of functional food because of its potential health benefits, such as the improvement of lipid profile, glycemia, and cardiovascular function [22-24]. Indeed, linseed oil is an important source of dietary n-3 PUFA as α-linolenic acid (ALA, 18:3 n-3). Previous studies have shown that linseed oil supplementation during pregnancy has beneficial effects on neonate metabolic parameters and health [25,26]. In addition, linseed oil induces epigenetic changes in maternal and offspring livers [27]. To the best of our knowledge there are no reports in the literature on the effect of linseed oil supplementation on metabolic status during maternal obesity and their repercussions on the offspring.

Because linseed oil may influence maternal and fetal metabolisms and because maternal obesity has profound effects on neonate metabolism in humans and also in animals, our aim was to evaluate the consequences of linseed oil supplementation in the diet before and during gestation on maternal and neonate disturbances induced by cafeteria diet.

II. Experimental Section

II.1. Animals and experimental protocol

Healthy adult wistar rats (aged 2 months) were obtained from Pasteur institute (Algiers, Algeria). The use of the animals according to our experimental design was approved by the Regional Ethical Committee. The study was conducted in accordance with the national guidelines for the care and use of laboratory animals. Forty female rats were randomly assigned into four feeding groups for 30 days prior to gestation. The feeding control group 1 (n = 10 females) received a control (C) diet (ONAB, Algeria). The feeding group 2, control linseed oil 5% (CL 5%) fed a control commercial diet enriched with linseed oil at 5%. The feeding group 3, cafeteria group (OB) fed a fat-rich hypercaloric diet.The feeding group 4, cafeteria linseed oil 5% (OB L 5%) fed a fat-rich hypercaloric diet enriched with linseed oil at 5%. Pure linseed oil was obtained from INRA (INRA, Algeria).The composition of the four diets was given in Table 1. After 30 days, females were mated, and each female was placed in an individual cage with free access to water and its specific diet until the birth of the offspring. Food intake and body weights were recorded. Female rats and their offspring were killed at birth.

At the end of the experimental period, the female rats were anaesthetized with intraperitoneal injection of sodium pentobarbital. The blood was drawn into heparinized tubes from the abdominal aorta. Newborn rats in each group were killed by decapitation, and blood was collected. Plasma was

used for glucose, lipid and vitamin C determinations. The remaining erythrocytes were washed and hemolyzed. Aflter centrifugation (2000 g for 15 min), the hemolysates were assayed for oxidative markers.

Table 1. Composition of experimental Diets

	Control Diet (C)	Control Linseed oil (CL) 5%	Cafeteria Diet (OB)	Cafeteria Linseed oil (OBL) 5%
Energy sources (% energy)				
Protein	20	20	16	16
Carbohydrate	60	60	24	24
Fat	10	10	50	50
Sunfloweroil	10	5	10	5
Linseedoil	-	5	-	5
Vitamin E (mg/100g)	5	5	5.5	5.5
Energy (Kcal/100g)	386	386	523	523
(% fatty acids)				
SFA	29	23	44	37
C18:1 n-9	21	22	28	29
C18:2 n-6	46	38	27	23
C18:3 n-3	3	16	1	11
C20:4 n-6	1	1	0	0

SFA: saturated fatty acids. Fatty acid composition was analyzed by gas liquid chromatography.

II.2. Chemical analysis

Plasma glucose was measured using the Trinder glucose kit (Sigma, St. Louis, MO). Plasma triglyceride, cholesterol, LDL and HDL- cholesterol were measured using colorimetric enzymatic kits (Sigma, St. Louis, MO).

II.3. Determination of oxidant / antioxidant status

The malondialdehyde (MDA) levels, a marker of lipid peroxidation, were determined in erythrocyte lysates by the method of Draper and Hadley [28] based on the reaction of MDA with thiobarbituric acid (TBA) at 95 °C.
Vitamin C levels were determined in plasma using dinitrophenylhydrazine, thiourea and copper sulfate according to the method of Roe and Kuether [29].
Erythrocyte carbonyl proteins (markers of protein oxidation) were assayed by 2,4-dinitrophenylhydrazine reaction as described previously [30].
Erythrocyte Catalase (EC 1.11.1.6) activity was measured by spectrophotometric analysis of the rate of H_2O_2 decomposition at 240 nm [31].
Erythrocyte reduced glutathione (GSH) levels were assayed by a colorimetric method based on the reduction of 5,5-dithiobis- (2-nitrobenzoic) acid by GSH to generate 2-nitro-5-thiobenzoic acid, according a Sigma Aldrich kit.

II.4. Statistical analysis

Results are expressed as means ± standard deviation (SD). The results were tested for normal distribution using the Shapiro–Wilk test. Data not normally distributed were logarithmically transformed. Significant differences among the groups were analyzed statistically by a one-way analysis of variance (ANOVA). The individual effects of cafeteria diet and oil supplementation were distinguished by two-way ANOVA. When significant changes were observed in ANOVA tests, Fisher least significant difference tests were applied to locate the source of significant difference. The significance level was set at $P < 0.05$. These calculations were performed using STATISTICA version 4.1 (STATSOFT, Tulsa, OK)

III. Results

III.1. Body weight, food and energy intakes in the rats

The cafeteria diet was associated with increased body weight, weight gain, food and energy intakes in mothers compared to standard chow fed ones (Table 2). Supplementation with linseed oil at 5% induced a reduction in body weight, in weight gain, in Food and energy intakes in both control and obese female rats. Newborn weight was significantly increased in offspring of cafeteria-fed dams compared to control values. Oil supplementation significantly reduced newborn weight in OB group to reach control weights.

Table 2. Characteristics of the Study Rats

	Body weight (g)	Food intake (g/day/rat)	Weight gain (g)	Energy intake (Kcal/day/rat)	Newborn weight (g)
C	244.33±13.93^{b}	42.10 ±1.55^{b}	110.01±14.60^{b}	160±14.65^{c}	5.17±0.35^{b}
OB	376.16±12.04^{a}	48.50±2.01^{a}	230.33±15.45^{a}	253.57±13.17^{a}	7.24±0.57^{a}
CL	216.66±15.50^{c}	35.01±1.10^{d}	82.16±10.45^{c}	130.51±8.02^{d}	5.10±0.46^{b}
OBL	240.50±8.01^{b}	38.31±1.50^{c}	111.83±12.48^{b}	197.21±13.55^{b}	5.78±0.55^{b}
P (ANOVA)	0.006	0.008	0.006	0.007	0.010

Values are presented as means ± SD. C: control diet; CL: control diet enriched with linseed oil at 5%; OB: cafeteria diet; OBL: cafeteria diet enriched with linseed oil at 5%. Values with different superscript letters (a, b, c, d) are significantly different ($P < 0.05$).

III.2. Biochemical parameters in the rats

The cafeteria diet significantly increased plasma glucose, cholesterol and triglyceride levels in both obese mothers and their newborns compared to control values (Table 3). Linseed oil supplementation significantly reduced plasma glucose, cholesterol and triglyceride concentrations in OB group, but had no effects in control group. However, after linseed oil supplementation, plasma cholesterol and triglyceride amounts remained higher in OBL group than in control group.
LDL-C was enhanced in mothers fed the cafeteria diet and in their newborns compared to control mothers and their newborns (Table 3). Oil supplementation significantly reduced these lipoprotein concentrations in OB group. HDL-C levels were lower in obese mothers. Linseed oil induced a significant rise in HDL-C in OBL mothers.

III.3. Oxidant / antioxidant markers

Erythrocyte MDA levels were increased in obese mothers and their newborns compared to control values (Table 3). Linseed oil supplementation significantly reduced MDA levels in mothers and newborns. Carbonyl proteins were increased in obese mothers, and were reduced by linseed oil supplementation.
Erythrocyte GSH levels were low in obese groups and were increased by oil supplementation (Figure 1). Erythrocyte catalase activity was decreased in obese rats and their newborns compared to controls (Figure 1). Linseed oils supplementation induced an increase in catalase activity in obese group. Plasma vitamin C levels in obese rats and in their newborns were similar to that in controls.

Table 3: Biochemical parameters and oxidant markers of the Study Rats

	C	OB	CL	OBL	*P* (ANOVA)
Mothers					
Glucose (g/L)	1.42±0.49^{c}	3.49±0.41^{a}	1.42±0.35^{c}	1.50±0.32^{c}	0.010
TC (g/L)	0.76±0.06^{d}	1.27±0.06^{a}	0.75±0.04^{d}	0.85±0.03^{c}	0.004
TG (g/L)	1.15±0.04^{d}	1.85±0.12^{a}	0.96±0.06^{d}	1.29±0.06^{c}	0.006
LDL-C (g/L)	0.20±0.03^{b}	0.55±0.05^{a}	0.23±0.04^{b}	0.24±0.03^{b}	0.009
HDL-C (g/L)	0.42±0.03^{a}	0.33±0.02^{b}	0.39±0.03^{a}	0.38±0.03^{a}	0.010
MDA (µmol/l)	1.53±0.32^{c}	3.68±0.24^{a}	1.45±0.36^{c}	1.62±0.24^{c}	0.010
PC (µmol/l)	0.51±0.06^{c}	1.82±0.19^{a}	0.65±0.12^{c}	0.86±0.11^{b}	0.020
Newborns					
Glucose (g/L)	1.08±0.21^{b}	1.70±0.20^{a}	0.81±0.14^{b}	0.77±0.12^{b}	0.010
TC (g/L)	0.58±0.04^{b}	0.82±0.02^{a}	0.57±0.02^{b}	0.60±0.02^{b}	0.007
TG (g/L)	0.83±0.04^{c}	1.29±0.02^{a}	0.85±0.03^{c}	0.98±0.02^{b}	0.005
LDL-C (g/L)	0.25±0.04^{b}	0.34±0.02^{a}	0.26±0.02^{b}	0.24±0.04^{b}	0.020
HDL-C (g/L)	0.21±0.04	0.21±0.03	0.18±0.05	0.22±0.03	0.184
MDA (%µmol/l)	1.41±0.13^{c}	2.30±0.13^{a}	0.76±0.15^{d}	1.78±0.27^{b}	0.008

Values are presented as means ± SD. TC: total cholesterol; TG: triglycerides; LDL-C: cholesterol of low density lipoproteins; HDL-C: cholesterol of high density lipoproteins; Values with different superscript letters (a, b, c, d) are significantly different (P<0.05).

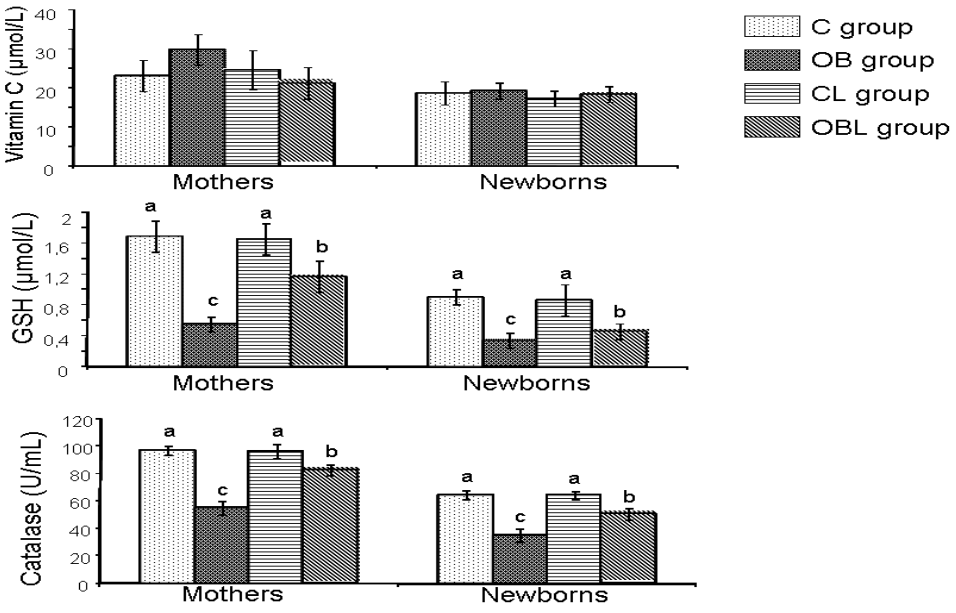

Figure 1. Antioxidant markers in the study rats. Values are presented as means ± SD. C: control diet; CL: control diet enriched with linseed oil at 5%; OB: cafeteria diet; OBL: cafeteria diet enriched with linseed oil at 5%. Values with different superscript letters (a, b, c, d) are significantly different (P < 0.05).

IV. Discussion and Conclusions

Maternal nutrition is a major modifiable environmental factor, which can affect fetal growth and development with potential long-term consequences. Previous studies have shown that maternal overnutrition and obesity program changes in neonate adiposity, leptin, glucose homeostasis and lipid profile [2,3,7-9]. Disease prevention should take place throughout life, starting with the mother's diet

during gestation. The search for functional foods, such as linseed oil, has been increasing because of their beneficial effects and their role in the prevention of diseases.

In the present study, the pregnant rats that received the cafeteria diet had an increase in total food and energy intakes that may explain the higher body weight, as described previously [2,3,7-9]. Offspring of these dams were heavier than offspring from dams fed control standard diet, in agreement with previous studies [2,3,7-9].

In our study, the pregnant rats fed cafeteria diet presented an increase in plasma glucose, cholesterol and triglyceride concentrations compared to pregnant rats fed standard diet. Our present finding also revealed lipoprotein abnormalities in obese pregnant rats, such as high LDL- and low HDL-cholesterol levels. Increased glucose, cholesterol, triglyceride and reduced HDL-C levels are the key characteristics of dyslipidemia in obesity [32]. We showed that offspring of cafeteria-fed dams had significantly higher glucose and lipid concentrations than offspring of control dams fed normal diet, in agreement with previous studies [9,33]. Maternal overnutrition, an enhancement in glucose and free fatty acid transfer from the mother, fetal hyperleptinemia and fetal hyperinsulinemia could explain fetal hyperglycemia and hyperlipidemia with increased fetal adiposity [1,2,7,8].

In the present investigation, a significantly lower of GSH levels and catalase activity were observed in both obese mothers and their newborns, reflecting reduced antioxidant defense as shown in obesity [12,15]. In addition, the elevated levels of erythrocyte MDA and of carbonyl proteins suggested an increased lipid peroxidation and protein oxidation in both mothers and newborns, in agreement with previous studies [8,9,16]. These findings showed an imbalanced oxidant/antioxidant system and enhanced oxidative stress in obese mothers and their offspring. Oxidative stress might be generated by maternal overnutrition, elevated circulating lipids, inflammation and insulin resistance [9]. Hyperenergetic, high-fat diets and exacerbated nutrient oxidation have been reported to increase oxidative stress and decrease antioxidant enzyme activity [34,35].

Linseed oil supplementation modulated metabolic parameters in obese pregnant rats, with beneficial effects including lower body weight, lower lipid accumulation and a reduction in oxidative stress. Indeed, after linseed oil supplementation, the mean weight of newborns in obese group was similar to that in control group, indicating a prevention of neonate obesity. It has been noted that linseed oil rich in linolenic acid prevented the excessive development of the adipose tissue [26], and induced an improvement in insulin sensitivity [23,24,26]. A diet rich in linoleic acid had a hypolipidemic effect with a reduction in adiposity [36].

In this study, the reduction in MDA and carbonyl protein levels after linseed oil supplementation could be due to an increase in the body's antioxidant capacity leading to reduced lipid and protein oxidation. It has been demonstrated that ALA-rich diets decrease the rate of peroxidation and production of free radicals with a concomitant increase in antioxidant enzyme activities [23-26]. Linseed oil attenuated maternal lipid and oxidative abnormalities which in turn had a beneficial effect on metabolic and redox parameters in the fetus.

In conclusion, our results clearly demonstrated that linseed oil displayed remarkable health benefits for prevention of obesity and associated metabolic disorders by decreasing plasma lipids and oxidative stress in both mothers and newborns.

V. Acknowledgments

This work was supported by the French Foreign Office [International Research Extension Grant TASSILI 08MDU723]; and by the financial support of the National Thematic Research Agency for Health Sciences (ATRSS, PNR2011).

The authors declare that they have no conflicts of interest.

VI. References

[1] Li M., Sloboda D.M., Vickers M.H. Maternal obesity and developmental Programming of metabolic disorders in offspring: Evidence from animal models. *Exp. Diabetes. Res.* 592408 (2011) 1-9

[2] Segovia S.A., Vickers M.H., Gray C., Reynolds C.M. Maternal obesity, inflammation, and developmental programming. *BioMed. Res. International.* 418975 (2014) 1-14

[3] Brenseke B., Prater M.R., Bahamonde J., Gutierrez J.C. Current thoughts on maternal nutrition and fetal programming of the metabolic syndrome. *J. Pregnancy.* 368461 (2013) 1-13

[4] Marinou K., Tousoulis D., Antonopoulos A.S., Stefanadi E., Stefanadis C. Obesity and cardiovascular disease: From pathophysiology to risk stratification. *Int. J. Cardiology.* 138 (2010) 3-8
[5] Attie A.D., Scherer P.E. Adipocyte metabolism and obesity. *J. Lipid. Res.* 50 (2009) 395–399
[6] Akyol A., Langley-Evans S.C., McMullen S. Obesity induced by cafeteria feeding and pregnancy outcome in the rat. *Br. J. Nutr.* 102 (2009) 1601–1610
[7] Lecoutre S., Breton C. Maternal nutritional manipulations program adipose tissue dysfunction in offspring. *Front. Physiol*. 6:158 (2015) 1-7
[8] Rkhzay-Jaf J., O'Dowd J.F., Stocker C.J. Maternal obesity and the fetal origins of the metabolic syndrome. *Curr. Cardiovasc. Risk. Rep.* 6 (2012) 487–495
[9] Rhianna C. Laker R.C., Wlodek M.E., Connelly J.J., Yan Z. Epigenetic origins of metabolic disease: The impact of the maternal condition to the offspring epigenome and later health consequences. Food*. Sci. Human. Wellness*. 2 (2013) 1–11
[10] Özdemir A. Maternal Obesity and Public Health. *Int. J. Caring. Sci.* 18 (2015 217-220
[11] Walsh S.W. Obesity: a risk factor for preeclampsia. *Trends. Endocrinol. Metab.* 18 (2007) 365–370
[12] Higdon J.V., Frei B. Obesity and oxidative stress: a direct link to CVD? *Arterioscler. Thromb. Vasc. Biol.* 23 (2003) 365-367
[13] Savini I., Catani M.V., Evangelista D., Gasperi V., Avigliano L. Obesity-associated oxidative stress: Strategies finalized to improve redox state. *Int. J. Mol. Sci.* 14 (2013) 10497-10538
[14] Sonta T., Inoguchi T., Tsubouchi H., Sekiguchi N., Kobayashi K., Matsumoto S., Utsumi H., Nawata H. Evidence for contribution of vascular NAD(P)H oxidase to increased oxidative stress in animal models of diabetes and obesity. *Free. Radic. Biol. Med.* 37 (2004) 115-116
[15] Abdali D., Samson S.E., Grover A.K. How effective are antioxidant supplements in obesity and diabetes? *Med. Princ. Pract.* 24 (2015) 201–215
[16] Vincent H.K., Taylor A.G. Biomarkers and potential mechanisms of obesity-induced oxidant stress in humans. *Int. J. Obes.* 30 (2006) 400–418
[17] Rosqvist F., Iggman D., Kullberg J., Cedernaes J., Johansson H.E., Larsson A., Johansson L., Ahlström H., Arner P., Dahlman I., Risérus U. Overfeeding polyunsaturated and saturated fat causes distinct effects on liver and visceral fat accumulation in humans. *Diabetes.* 63 (2014) 2356–2368
[18] Lawrence G.D. Dietary fats and health: Dietary recommendations in the context of scientific evidence. *Adv. Nutr.* 4 (2013) 294–302
[19] Breslow J. N-3 Fatty acids and cardiovascular disease. *Am. J. Clin. Nutr.* 83 (2006) 1477-1482
[20] Schwab U., Lauritzen L., Tholstrup T., Haldorsson T.I., Riserus U., Uusitupa M., Becker W. Effect of the amount and type of dietary fat on cardiometabolic risk factors and risk of developing type 2 diabetes, cardiovascular diseases, and cancer: a systematic review. *Food. Nut. Res.* 58 (2014) 1-26
[21] Nettleton J.A., Jebb S., Risérus U., Koletzko B., Fleming J. Role of dietary fats in the prevention and treatment of the metabolic syndrome. *Ann. Nutr. Metab.* 64 (2014) 167–178
[22] Haliga R., Mocanu V., Badescu M. Flaxseed have beneficial effects on endothelial dysfunction and oxidative stress in experimental atherosclerosis. *Annals of RSCB.* 2 (2013) 91-97
[23] Rasmy G.E. Protective effect of linseed oil on hyperlipidemia in experimental animals. *J. Gen. Eng. Biotech.* 5 (2007) 9-17
[24] Pacheco J.T., Daleprame J.B., Boaventura J.T. Impact of dietary flaxseed (*linum usitatissimum*) supplementation on biochemical profile in healthy rats. *Nutr. Hosp.* 26 (2011) 798-802
[25] Sankaran D., Eda banko V.N., Tu-Chenpeng C., Ogborn M.R., Aukema H.M. Dietary Flax Oil During Pregnancy and Lactation Retards Disease Progression in Rat Offspring With Inherited Kidney Disease. *Int. Pediat. Res. Foundation.* 60 (2006) 729-733
[26] Tripathi V., Abidi A.B., Markerb S., Bilal S. Linseed and linseed oil: health benefits- a review. *Int. J. Pharm. Biol. Sci.* 3 (2013) 434-442
[27] Niculescu, M.D., Lupu, D.S., Craciunescu, C.N. Perinatal manipulation of α-linolenic acid intake induces epigenetic changes in maternal and offspring livers. *FASEB J* 27 (2012) 350-358.
[28] Draper H., Hadley M. Malondialdehyde determination as index of lipid peroxidation. *Methods. Enzymol*. 186 (1990) 421-431
[29] Roe J.H., Kuether C.A. The determination of ascorbic acid in whole blood and urine through the 2,4 dinitrophenylhydrazine derivatives of dehydroascorbic acid. *J. Biol. Chem.* 147 (1943) 399–407
[30] Levine R.L., Garland D., Oliver C.N., Amici A., Climent I., Lenz A.G., Ahn B.W., Shaltiel S., Stadtman E.R. Determination of carbonyl content in oxidatively modified proteins. *Methods. Enzymol.* 186 (1990) 464–478
[31] Aebi H. Catalase. Methods of Enzymatic Analysis, 2nd ed. Bergmeyer,H.U., vol.2, Verlag Chemie, Weinheim, 1974, 673–684
[32] Wang H., Eckel R.H. Lipoprotein lipase: from gene to obesity. *Am. J. Physio. Endocri. Metabolism.* 297 (2009) 271–288

[33] Taylor P.D., McConnell J., Khan I.Y., Holemans K., Lawrence K.M., Asare-Anane H., Persaud S.J., Jones P.M., Petrie L., Hanson M.A., Poston L. Impaired glucose homeostasis and mitochondrial abnormalities in offspring of rats fed a fat-rich diet in pregnancy. *Am. J. Phys. Regul. Integr.* 88 (2005) 134-139
[34] Milagro F.I., Campion J., Martinez J.A. Weight gain induced by high-fat feeding involves increased liver oxidative stress. *Obesity.* 14 (2006) 1118–1123
[35] Unger R.H. Lipid overload and overflow: metabolic trauma and the metabolic syndrome. *Trends. Endocrinol. Metab.* 14 (2003) 398–403.
[36] Khalesi S., Jamaluddin R., Ismail A. Effect of raw and heated flaxseed (*Linum Usitatissimum L.*) on blood lipid profiles in rats. *Int. J. Appl. Sci. Tech.* 4 (2011) 84-89

Please cite this Article as:

Nawel BENAISSA, Hafida MERZOUK, Sid Ahmed MERZOUK, Michel NARCE, Effects of maternal linseed oil supplementation on oxidative stress markers in cafeteria diet induced obese rats, ***Algerian J. Nat. Products***, **3:3 (2015) 177-184**

www.ajnp.webs.com
www.univ-bejaia.dz/ajnp
Online ISSN: 2353-0391

Editor in chief: Prof. Kamel BELHAMEL