ORIGINAL ARTICLE

Effect of dietary fat type on plasma lipid profile and leptin concentration in rats fed on high-sucrose diets

Agata Krawczyńska, Katarzyna Okręglicka, Elżbieta Olczak, Joanna Gromadzka-Ostrowska

Division of Nutrition Physiology, Department of Dietetics, Faculty of Human Nutrition and Consumer Science,
Warsaw University of Life Sciences – SGGW, Warsaw, Poland

Abstract: The aim of the study was to investigate the effect of different dietary fat type on plasma leptin concentration and lipid profile in male Wistar rats fed normo-fat, normo-protein, high-sucrose (5, 19 and 35 % w/w, respectively) diets. The experiment was conducted on 21 adult male rats (260 ± 20g) fed diets with different fat sources: lard (L), grapeseed oil (G) and flaxseed oil (F). Radioimmunoassay was used to measure leptin concentration and enzymatic-colorimetric methods to estimate lipid profile. Total cholesterol (TC), high density lipoprotein cholesterol (HDL) and triglycerides (TG) plasma concentrations were higher in group L and G than F (ANOVA p≤0,01; p≤0,05 and p≤0,005, respectively), whereas low density lipoprotein cholesterol (LDL) level was higher in group L than G and F (ANOVA p≤0,05). Leptin concentration was significantly higher in group L in comparison to F (ANOVA p≤0,04). Significant positive correlations were found between plasma leptin concentration and final body weight, TC, HDL and TG (r = 0,64, p≤0,006; r=0,72, p≤0,002; r=0,69, p≤0,003; r=0,86, p≤0,00004 respectively). It can be observed that flaxseed oil rich in n-3 polyunsaturated fatty acids (PUFA) profitably influenced not only lipid profile lowering its parameters but also reduced leptin concentration which can suggest approximate lipidogenic potential of both grapeseed oil (rich in PUFA n-6) and lard (rich in monounsaturated and saturated fatty acids). The results provided evidence that dietary fat type can influence cardiovascular disease risk parameters when high-sucrose diet is consumed.

Key words: diet, flaxseed oil, grapeseed oil, lard, leptin, lipid profile, rat, sucrose

INTRODUCTION

When in 1994 Zhan and colleges discovered leptin, its role was limited to the regulation of food consumption and energy expenditure [1]. This 167-amino acid polypeptide produced mainly by adipocytes (white adipose tissue) in direct proportion to body fat stores was said to act on hypothalamic centres and peripheral organs, adipose tissue, liver, muscles and pancreas. But further investigations revealed the presence of leptin receptors (OB-R) also in tissues not connected with macronutrients metabolism or energy balance. They were found in reproductive as well as in cardiovascular systems [2]. Causing endothelial dysfunction, stimulating inflammatory reaction, platelet aggregation and provoking migration and proliferation of vascular smooth muscle cells, leptin is said to exert proaterogenic effects [2, 3]. Pathological actions of leptin in cardiovascular system ensue from leptin resistance which is associated with obesity. This highly increased plasma leptin concentration is said to be a new factor of cardiovascular pathology [4]. The connections between leptin and diet are generally known. Long consumption of high-fat diet seems to be one of the crucial causes of leptin resistance [5]. Also, the insulin-stimulating influence on leptin secretion shows a link between carbohydrates consumption and leptin gene (ob gene) expression [6, 7]. Moreover, dietary fatty acid profile as

well as dietary fat and carbohydrates levels was found to be a factor which affects endocrine function of adipose tissue [8]. However, there is a discrepancy between the results of effects exerted by saturated, monounsaturated and polyunsaturated fatty acids (SFA, MUFA, PUFA, respectively) on leptin secretion; therefore, more research is needed.

A second but better known and investigated diet-dependent factor of cardiovascular pathology is plasma lipid profile. It is well documented that high consumption of sucrose raises triglycerides (TG) plasma concentration [9]. Moreover, elevated plasma levels of total and in particular low-density lipoprotein (LDL) cholesterol which, like TG are associated with an increased risk of coronary events, are positively correlated with SFA intake. On the other hand, consumption of dietary fat rich in PUFA has a hypocholesterolemic effect, with differences between n-6 and 3, as s reviewed by Poli et al. [10]. It has been shown that long chain n-3 PUFAs (eicosapentaenoic and docosahexaenoic acids) found in fish oils or (α-linolenic acid) in flaxseed oil not only decrease plasma LDL and increase HDL levels, but also modulate the inflammation process preferring production of the less inflammatory series 3 eicosanoides and 5 series of leukotriens [11,12]. Endothelium relaxation is also intensified by stimulation of production of nitric oxide by omega-3 [13]. On the other hand, n-6 fatty acids are the precursors of pro-inflammation and pro-agreggation series 2 eicosanoides (such as thromboxane $2 - TXA_2$) which are mainly produced from arachidonic acid by enzyme cyclooxygenase 2 (COX-2) [14]. In the light of the said ratio of n-6 to n-3 fatty acids, this has been suggested by some authors to be particularly important [15].

As can be seen above there is a very close connection between leptin, plasma lipids and diet composition. Because there are many articles which focus on either carbohydrates or fat influences on leptin homeostasis, we decided to conduct experiments which would connect these parameters. We therefore proposed the hypothesis that any alterations in plasma leptin and lipids concentration would be observed as the effects of manipulations with dietary fat type in rats fed a high-sucrose diet.

MATERIALS AND METHODS

Animals, diets and experimental design. The experiment, approved by the Third Local Animal Care and Use Committee in Warsaw, was conducted on 21 male adult Wistar rats with initial body weights of 260 ± 20g. The initial body weights were measured after a week of acclimatization during which animals were fed a standard rodents' feed (Labofeed H, Andrzej Morawski Feed Production Plant, Kcynia, Poland). Rats were kept individually in polypropylene cages in stable environmental conditions (temperature 22°C; humidity 50%; 12:12 light:dark cycle). They were given free access to food and water.

After a one-week adaptation period, the animals were divided into three experimental groups receiving high-sucrose (35% w/w), normo-fat (5% w/w) and normo-protein (19% w/w) semi-synthetic diets based on data provided by Merat et al. [16] (Tab. 1), but with different types of dietary fat source: F – flaxseed oil (rich in n-3 PUFAs), G – grape seed oil (rich in n-6 PUFAs) and L – lard (rich in both MUFAs and SFAs). Fatty acids content in each dietary fat was assayed by GC analysis performed at the Analitical Centre of SGGW (Warsaw, Poland) (Tab. 2.). The animals stayed on the experimental diets for 3 weeks.

Table 1   Composition of diet.

Component	Amount [g/kg]
Sucrose	350
Wheat starch	307.3
Casein	190
Fat	50
Mineral Mixture[a]	50
Potato starch	36.7
Vitamin mixture[b]	10
DL-Methionine	3
Choline chloride	3

[a] Mineral mix composition (AIN-93M-MX Mineral Mix) according to Reeves P.G. [17] (in 100g of mix): $CaCO_3$ – 35,7g, K_2HPO_4 – 25g, NaCl – 7.4g, K_2SO_4 – 4.66g, $C_6H_5K_3O_7*H_2O$ – 2.8g, MgO – 2,4g, $C_6H_5FeO_7$ – 606mg, $ZnCO_3$ – 165mg, $Na_2SiO_3*9H_2O$ – 145mg, $MnCO_3$ – 63mg, $CuCO_3$ – 30mg, $CrK(SO_4)_2*12H_2O$ – 27.5mg, H_3BO_3 – 8.15mg, NaF – 6.35mg, $NiCO_3$ – 3.18mg, Li_2CO_3 – 1.74mg, Na_2SeO_4 – 1.025mg, KIO_3 – 1mg, $(NH_4)_6Mo_7O_{24}*4H_2O$ – 0.795mg, NH_4VO_3 – 0.66mg, powder sucrose up to 100g of mix.
[b] Vitamin mix composition (AIN-93-VX Vitamin Mix) according to certificate of producer MP Biomedicals (USA) (%): Nicotinic Acid – 3,00, D-Calcium Pantothenate – 1.60, Pyridoxine HCl – 0.70, Thiamine HCl – 0.60, Riboflavin – 0.60, Folic Acid – 0.20, D-Biotin – 0.02, Vitamin B_{12} (0.1% triturated in mannitol) – 2.50, Alpha Tocopherol Powder (250 U/gm) – 30,00, Vitamin A Palmitate (250,000 U/gm) – 1,60, Vitamin D_3 (400,000 U/gm) – 0.25, Phylloquinone – 0.075, Powdered Sucrose – 959,655

Food intake was quantified by monitoring the amount of consumed diet each day through the whole experimental period. Also, the animals' body weight was monitored once each week.

After 12 hours of food deprivation, the rats were anesthetized with Thiopental (120 mg/kg body weight) and completely bled

Table 2   Content of fatty acids in dietary fat sources.

Fatty AIDS		weight % of fatty acids in dietary fats		
Number of carbon atoms and double bounds	Type	Flaxseed Oil	Grapeseed Oil	Lard
C10:0	SFA			0.12
C12:0	SFA			0.11
C14:0	SFA	0.05	0.06	1.77
C14:1 (cis-9)	MUFA n-5			0.12
C15:0	SFA			0.1
C16:0	SFA	5.53	6.99	26.1
C16:1 (cis-9)	MUFA n-7	0.07	0.12	2.5
C17:0	SFA	0.06	0.07	0.39
C17:1 (cis-10)	MUFA n-7	0.06	0.06	0.3
C18:0	SFA	3.55	4.3	16.63
C18:1 (trans-9)	MUFA n-9		0.06	
C18:1 (cis-9)	MUFA n-9	14.87	18.79	37.64
C18:2 (all-trans-9,12)	PUFA n-6			0.06
C18:2 (all-cis-9,12)	PUFA n-6	11.43	66	7.32
C18:3 (all-cis-6,9,12)	PUFA n-6	0.25		
C18:3 (all-cis-9,12,15)	PUFA n-3	62.6	0.31	0.68
C20:0	SFA	0.18	0.19	0.23
C20:1 (cis-11)	MUFA n-9		0.18	
C20:2 (all-cis-11,14)	PUFA n-6		0.09	0.55
C20:3 (all-cis-8,11,14)	PUFA n-6			0.06
C20:3 (all-cis-11,14,17)	PUFA n-3			0.12
C20:4 (all-cis-5,8,11,14)	PUFA n-6			0.19
C22:0	SFA	0.13	0.13	
C24:0	SFA	0.1	0.06	
Total SFA		9.6	11.8	45.45
Total MUFA		15	19.21	40.56
Total PUFA		74.28	66.4	8.98
Total PUFA n-3		62.6	0.31	0.8
Total PUFA n-6		11.68	66.09	8.18

SFA – saturated fatty acids, MUFA – monounsaturated fatty acids, PUFA – polyunsaturated fatty acids.

by cardiac puncture. Blood was centrifuged and plasma stored at -20°C until further analysis. Bodies and organs were used in different analyses.

Leptin radioimmunological assay. Plasma leptin concentration was measured using Rat Leptin RIA Kit (Cat. # RL-83K, LINCO Research, USA). The intra- and inter-assay precision was 2.4% and 4.8%, respectively. Sensitivity of test was 0.5ng/ml. The assay was conducted according to the kit manual. Leptin concentration was expressed as ng per ml of plasma.

Plasma lipid profile colorimetric assays. Concentrations of plasma total cholesterol (TC), triglycerides (TG) and high-density lipoprotein cholesterol (HDL) were measured using enzymatic-colorimetric methods. Kits containing ready to use liquid reagents were purchased from PTH Hydrex (Warsaw, Poland). The analyses were conducted according to kit manuals. Concentrations were expressed as mg per 100ml of plasma.

Concentration of low-density lipoprotein cholesterol (LDL) was calculated from Friedewald's formula: LDL = TC − HDL − TG/5 [18].

Statistical analysis. Statistical analysis was undertaken by STATISTICA v. 8.00 (StatSoft Polska Sp z o.o., Cracow, Poland). Simple regression and one-way variance analysis ANOVA followed by *post-hoc* NIR Fisher's test were calculated. Differences with a value of p≤0.05 were considered significant. Before conducting ANOVA tests, two assumptions were tested: normal distribution and homogeneity of variances. All data are showed as means ± SD.

RESULTS

Final body weight and food intake. There were no significant differences in final body weight and body mass gain per day (ANOVA, NS) (Tab. 3.). Diet and total fat intake were also not statistically different. However, fatty acids consumption was highly differentiated between dietary groups (ANOVA for consumption of SFA, MUFA, PUFA, n-6 and n-3, p≤0,00 for each) (Tab. 4.) in accordance with fatty acids content in dietary fat source. As this stems from the fatty acids composition of used dietary fats, the highest consumption of SFA and MUFA was in group L (NIR, L *vs*. F and G – p≤0,00), PUFA n-3 – in group F (NIR, F *vs*. G and L – p≤0,00) and PUFA n-6 in group G (NIR, G *vs*. F and L – p≤0,00).

Table 3 Initial weight, final weight, body mass gain, and food intake (mean ± SD).

| Diet Group	n	Initial body weight, [g]	Final body weight, [g]	Body mass gain [g/d]	Food intake [g/d/100g body mass]
F	7	259.4 ± 24.1	303.3 ± 21.7	2.1 ± 0.5	6.3 ± 0.4
G	7	259.3 ± 21.4	304.6 ± 22.8	2.2 ± 0.2	6.4 ± 0.3
L	7	260.2 ± 15,1	308.3 ± 22.5	2.3 ± 0.5	6.5 ± 0.3

F – diet with flaxseed oil; G – diet with grapeseed oil; L – diet with lard; ANOVA NS for each parameter.

**Table 4** Intake of total fat, SFA, MUFA, PUFA, n-3, n-6 [g/d/100g body mass].

Diet Group	Total fat	SFA	MUFA	PUFA	n-3	n-6
F	0.316 ± 0.018	0.030 ± 0.002[a]	0.064± 0.004[b]	0.209± 0.012[b]	0.168± 0.010[b]	0.040± 0.002[a]
G	0.321 ± 0,016	0.031± 0.002[a]	0.052± 0.003[a]	0.225± 0.011[c]	0.000± 0.000[a]	0.224± 0.011[b]
L	0.324 ± 0.013	0.127± 0.005[b]	0.146± 0.006[c]	0.036± 0.001[a]	0.003± 0.000[a]	0.033± 0.001[a]

F – diet with flaxseed oil; G – diet with grapeseed oil; L – diet with lard; SFA - saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids; a,b,c - indicate values significantly different (p≤0.05) within columns.

Plasma lipid profile. Dietary fat type significantly affected TC, HDL, LDL and TG (ANOVA p≤0.01; p≤0.05; p≤0.02 and p≤0.005, respectively). As shown in Fig. 1, higher TC concentrations were found in groups G and L than in F (NIR, G *vs*. F – p≤0.01; L *vs*. F – p≤0.005). Similar results for plasma TG (NIR, G *vs*. F – p≤0.002; L *vs*. F – p≤0.02) and HDL (NIR, G *vs*. F – p≤0.02; L *vs*. F – p≤ 0.05) concentrations were shown.
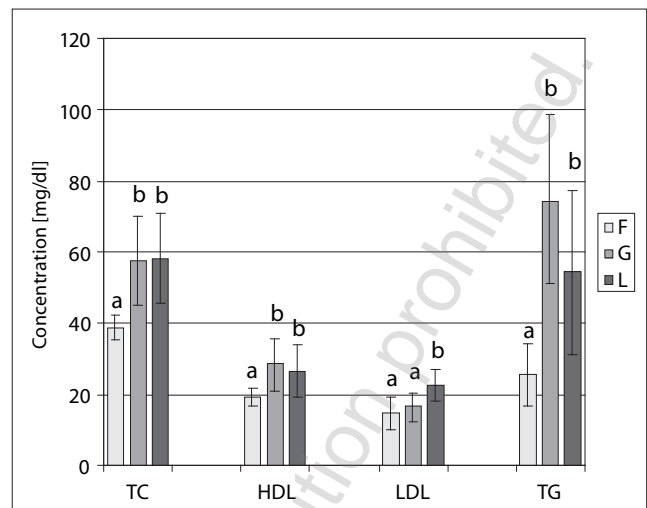
Figure 1 Plasma concentration of total cholesterol (TC), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), triglycerides (TG) [mg/dl]; F – diet with flaxseed oil; G – diet with grapeseed oil; L – diet with lard; a, b – indicate values significantly different (p≤0,05).
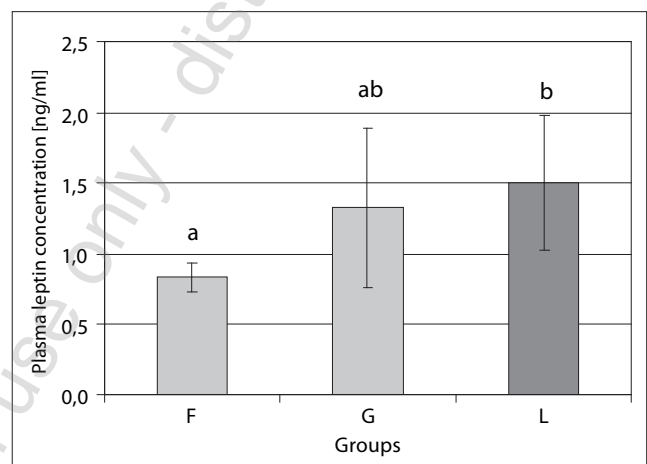
Figure 2 Plasma leptin concentration [ng/ml]; F – diet with flaxseed oil; G – diet with grapeseed oil; L – diet with lard; a,b – indicate values significantly different (p≤0,05).

LDL concentration was not significantly different in groups F and G, but we observed a statistically higher LDL value in group L (NIR, L *vs*. F – p≤0.01; L *vs*. G – p≤0.04).

Plasma leptin concentration. Plasma leptin concentration differed significantly between the groups fed different dietary fat sources (ANOVA p≤0.04) with a higher value in group L compared to group F (NIR, L *vs*. F – p≤0.02). There were no significant differences between group G and the other groups (Fig. 2.).

As calculated by regression analysis, positive correlations between plasma leptin concentration and final body weight, TC, HDL as well as TG (r = 0.64, p≤0.006; r=0.72, p≤0.002; r=0.69, p≤0.003; r=0.86, p≤0.00004, respectively) were found.

DISSCUSION

Diet is one of the most important factors which exerts an influence on body mass, lipid plasma profile and the circulation of hormones. It is generally known that a high-sucrose diet

not only increases body mass gain and plasma lipid profile, but also influences leptin secretion by insulin changes. The aim of this study was to confirm the hypothesis that different compositions of fatty acids in a diet can modulate alternations developed by high sucrose intake.

Body weight and diet consumption. As we observed, diets based on a high amount of sucrose (35% w/w) and the normal level (5% w/w) of all three types of dietary fat (lard, grapeseed oil, flaxseed oil) were well tolerated. The particular fat contained in a diet did not influence overall consumption, and the animals gained weight with no differences between groups. Similarly, no effect of the type of dietary fat on body weight gain was observed in the experiment conducted on male Wistar rats fed diets with 10% (w/w) of palm, rapeseed and sunflower oil and lard [19]. The same results were also obtained by Hynes et al. [20] who did not observe any effect of dietary fat type on these parameters in male Sprague-Dawley rats fed high-fat diets (20% w/w) containing fish oil, safflower oil or beef tallow as a fat source. However, Stachoń et al. [21] observed that rats fed a diet with 40% (w/w) of rapeseed oil (rich in MUFA) as a source of dietary fat, gained less weight than animals fed sunflower or palm oil, or even lard diets containing lard. It seems that the dietary fat type can affect body mass gain only in animals fed rich-fat diets which promote fat deposition and higher body weight gain.

Plasma lipid profile. It is generally known that a high-sucrose diet modifies triglycerides and cholesterol plasma concentrations. In the experiment conducted by Yang et al. [22], a high-sucrose diet caused increases in both stated parameters, as well as hepatic triglycerides content. The same observation, together with HDL increase, was seen by Ryu et al. [23]. Because in our experiment statistically important differences were observed between animals fed high-sucrose diets containing lard or grapeseed oil and flaxseed oil, we hypothesize that a sucrose-rich diet effects the plasma lipid profile, depending on the dietary fatty acids composition. This hypothesis might be confirmed by the results of Cintra et al. [24]. In their experiment, rats fed a diet with flaxseed had lower plasma cholesterol concentration in comparison to rats fed a diet rich in SFA from chicken skin. The same effect was found by Takeuchi et al. [25] who stated that triglycerides, HDL and total cholesterol levels were significantly lower in rats fed diets with flaxseed or sardine oil in comparison to rats fed a diet with tripalmitin, tristearin and corn oil mixture. The results of Murano et al. [26], conducted on Sprague-Dawley male rats, also indicated that an intake of lard enriched by linolenic acid suppresses the activity of hepatic fatty acids synthase (FAS), and this suppression may lead to the reduction of the plasma triglycerides concentration. Researchers have pointed out that PUFAs have influence on FAS gene expression by suppression of Sterol Regulatory Element-Binding (SREBP) – 1c [27]. On the other hand, it has been observed that PUFAs activate Peroxisome Proliferators-Activated Receptor α (PPARα), which leads to the induction of carnitine palmitoyltransferase which regulate fatty acids oxidation [28]. Thus, a higher activity of β-oxidation and the lower concentration of plasma triglycerides in animals with high linolenic acid intake can be stated. In another study, Morgado et al. [29] found that rats fed high amounts of n-3 PUFA from fish oil had significantly lower plasma total and HDL cholesterol. They stated that an n-3 PUFA-rich diet

significantly changed the hepatic membranes n-3/n-6 fatty acids ratio, which in turn caused plasma cholesterol reduction. They did not observe any modification in the expression levels of lecithin cholesterol acylotransferase, hepatic lipase, apo A-I and apo E mRNA, which may suggest that reverse cholesterol transport is not changed by n-3 PUFAs. However, n-3 PUFAs from fish (eicosapentaenoic and docosahexaenoic acid – EPA nad DHA) differ the from flaxseed oil PUFAs (α-linolenic acid - ALA) which was used in our study. It therefore seems important to refer to the study by Riediger et al. [30], the purpose of which was to investigate the cardiovascular benefits of both these oils. Researchers have stated that plasma total cholesterol levels were reduced in both fish and flax groups by 27% and 36%, respectively, compared to controls at the endpoint after 16 weeks of experiments. The mechanism of such actions has been proposed by Du et al. [31] who suggested a decrease in HMG-CoA reductase activity in high DHA and ALA groups. In our study, rats fed a diet rich in both n-6 PUFA (grapeseed oil) or SFA and MUFA (lard) showed higher concentration of cholesterol and triglycerides than animals fed a diet rich in α-linolenic acid (flaxseed oil). It seems appropriate to state that all mechanisms mentioned above could be involved in lowering the properties of flaxseed oil on plasma lipid profile, as also seen in our study. But we cannot omit the suggestion made by Cintra et al [24], that flaxseeds contain dietary fibre and lignans vital for the organism, which can also decrease the serum cholesterol level. However, comparing the oil obtained from flaxseeds in the cold-pressed process with its whole seeds, the content of lignans and fibres is much higher in the seeds [personal communication]. On the North American and New Zealand markets, cold-pressed flaxseed oil is commercially available in low and high-lignan forms [32], depending on whether the oil was produced from whole seeds or only from husks. In our case, the oil was produced from whole seeds and we can assume that the content of lignans was rather low. Taking this into consideration, this seems that mainly due to the n-3 fatty acids content and only slightly to lignans, and the presence of fibres in a diet rich in flaxseed oil is the most efficient in both decreasing blood cholesterol and triglycerides, or protecting the liver parenchyma.

Plasma leptin concentration. Meal composition and nutrients intake might affect plasma leptin concentration. This can be modified mainly by energy-yielding nutrients such as dietary carbohydrates or fat. Peyron-Caso et al. [33] found that both 3- and 6-week feeding with sucrose-rich diets (57.5%) induced a parallel increase in both plasma leptin level and adiposity. It has been demonstrated that glucose metabolism is the primary determinant of leptin secretion rather than insulin concentration [34]. Thus, the ability of a high-carbohydrate diet to induce an increase in the leptin peripheral level may be mediated by its insulin response, which promotes glucose uptake in adipose tissue [6]. Moreover, as reviewed by Orr and Davy [6], high-carbohydrate, low-fat meals produce higher a leptin concentration when compared to high-fat, low-carbohydrate meals.

Knowing that sucrose-enriched diet raised the leptin level, we wanted to check the effects of different dietary fatty acids on the plasma leptin level in rats consuming high-sucrose (35%) but normo-fat (5%) diet.

Generally, the studies focused on peripheral leptin level and dietary fat are based on high-fat diets with modification

of used fat source. It has been shown that a high-fat diet increased ob gene expression in adipose tissue of male Sprague-Dawley rats [35], causing the development of leptin resistance. However, further investigations demonstrated that different dietary fat sources can modulate this hormone concentration, depending on their fatty acids composition. A previous study conducted in our department by Stachoń et al. [21] showed that consumption of high-fat diet (40% w/w) with sunflower oil rich in n-6 fatty acids as a source of dietary fat caused the highest increase in plasma leptin concentration in Wistar male rats, in comparison with the consumption of diets with rapeseed oil, palm oil and lard. The influence of dietary fat type was also observed by Wang et al. [36], who found in mice that 7-week consumption of a diet rich in a mixture of safflower (n-6) and beef tallow (SPA and MUFA) resulted in a higher plasma leptin concentration, compared to consumption of diets rich in n-3 from fish oil or n-6 from safflower alone. Also, Ukropec et al. [37] found that a high-fat diet containing 10% n-3 PUFA and 18% SFA, in comparison with a high-fat diet containing 28% SFA, lowered plasma leptin level and leptin mRNA in adipose tissue.

Our results also showed that manipulation with fatty acids composition can modify leptin level, even if the diet is high-sucrose and normo-fat. We observed that increased n-3 fatty acids intake lowered leptin level in comparison to high SFA, but not n-6 intake. PPARs and SREBP-1 has been tried to explain the connection between fatty acids and leptin metabolism. Reseland et al. [38] observed that n-3 PUFAs decrease leptin gene expression by mechanisms associated with reduced PPARγ and SREBP-1 gene expression. The same issue was studied by De Vos et al. [39]. They also found that in rats, both ligands of PPARγ thiazolidinedione BRL 49653 and fatty acids (EPA and DHA given with diet enriched in fish oil) caused a decrease of ob mRNA expression by 40% and 33%, respectively. In view of the absence of consensus of PPRE (PPAR response element) in the ob gene, the authors find it important to identify the molecular mechanism of PPARγ which may be connected with positive modulators of ob transcription, such as C/EBPα (CCAAT/enhancer binding protein α) or Sp1 transcription factor. But much still remains unknown about the mechanisms involved in fatty acids control of leptin metabolism, and more research is needed.

In our study we also found significant positive correlations between the plasma leptin concentration and plasma lipid parameters. Especially interesting seems to be a correlation between leptin and TG concentrations. As stated by Banks et al. [40], there is strict relationship between TG and leptin transport across the blood-brain barrier (BBB). They showed that a diet with milk (as a source of TG) immediately inhibited leptin transport *in vivo, in vitro* and *in situ* models of the BBB. Thus, it can be stated that a diet rich in sucrose causing an increase of TG, slows down leptin transport through BBB and increases leptin serum concentration. Any factor which reverses the sucrose effects lowering TG (e. g. flaxseed oil used in our experiment), which would at the same time decrease the leptin level accelerating its transport to the brain.

To summarize, the results of our study indicate that plasma leptin and lipids concentration in rats fed high-sucrose diets were affected by the dietary fat type, with the lowest value in animals fed flaxseed oil as a dietary fat source.

CONCLUSIONS

1. The dietary fat type supplied is an important factor in plasma leptin and lipoprotein level regulation when a high-sucrose diet is consumed.
2. Obtained results suggest approximate lipidogenic potential of both lard (rich in SFA) and grapeseed oil (rich in PUFA n-6) and the normalizing potential of flaxseed oil (source of PUFA n-3) on both lipid profile and leptin concentration changes caused by high-sucrose diet consumption.
3. The dietary fat type can influence cardiovascular disease risk parameters when a high-sucrose diet is consumed.

ACKNOWLEDGMENT

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