Medicina Sportiva, Vol. 8 (1): 21–30, 2004

LIPID AND LIPOPROTEIN PROFILE IN REGULARLY MENSTRUATING PHYSICALLY ACTIVE FEMALE OC-NON-USERS AND USERS AS COMPARED TO MEN OF MATCHED AGE AND PHYSICAL ACTIVITY

Grażyna Lutosławska[1], Marzena Niedbalska[1], Ewa Byszewska-Szpocińska[2] Elżbieta Skierska[3], Anna Kęska[3]

[1] Department of Biochemistry, Academy of Physical Education, Warsaw, Poland
[2] Department of Radioimmunology, Radioisotope Centre POLATOM, Swierk, Poland,
[3] Department of Biology, Academy of Physical Education, Warsaw, Poland

Abstract

Introduction: Vigorous physical activity depresses ovary function, circulating sex hormones and alters plasma lipid and lipoprotein profile which in amenorrheic females is less favorable than in their regularly menstruating counterparts. However, data concerning plasma lipid and lipoprotein profile in physically active, regularly menstruating females using and non-using oral contraceptive (OC) in comparison with males of similar physical activity are limited.

Aim of the study: This study was aimed at evaluation of plasma lipid and lipoprotein, glucose and insulin levels in regularly menstruating female physical education students (OC-users and non-users) in comparison with males.

Methods: A total of 113 subjects volunteered to participate in the study. Biochemical variables (total cholesterol (TC), HDL- and LDL-cholesterol (HDL-C and LDL-C), triacylglycerols (TG), free fatty acid (FFA), glycerol, glucose) were determined in fasting blood plasma using colorimetric methods and commercial kits (Randox Laboratories, United Kingdom). Insulin was measured by a standard radioimmunoassay and monoclonal antibody against insulin (IRMA-POLATOM. Poland).

Results: There were no between-group differences in insulin, FFA and glycerol plasma levels and Quantitative Insulin Check Index (QUICKI). However, plasma lipoprotein profiles in female OC-non-users were less favorable than in males due to higher TC and LDL-C concentrations. This difference was even more evident when the TC/HDL-C ratio was taken into consideration. In contrast, in female OC-users plasma HDL-C was significantly higher than in OC-non-users and in males. Despite slightly higher TC in OC-users. The TC/HDL-C ratio was lower than in both other groups. In males there was a significant correlation between plasma glycerol and TG levels ($r=0.376$, $p<0.01$). In contrast, in female OC-non-users such a correlation was found between plasma glycerol and LDL-C concentrations ($r=0.437$, $p<0.001$). In female OC-users plasma glycerol mostly affected plasma HDL-C levels without an effect on plasma TG and LDL-C levels.

Conclusions: Since our subjects did not differ with respect to dietary habits it is postulated that vigorous physical activity in premenopausal females promotes undesirable changes in pla-

sma lipoprotein profile and slightly, but significantly affects adipose tissue contribution to lipoprotein synthesis.

Key words: physical activity, lipids, lipoproteins, males, female OC-non-users, female OC-users

Introduction

Numerous reports from cross-sectional studies over the last decades provide compelling evidence for the positive effect of physical activity on blood lipid and lipoprotein concentrations in plasma (1-3). In general plasma lipid and lipoprotein profiles of physically active groups are more favorable than in sedentary ones irrespectively of gender, especially with respect to prebeta 1- HDL- C mediating cholesterol removal from peripheral tissues (4, 5).

However, it should be noted that some lipid and lipoprotein levels in plasma such as HDL-cholesterol (HDL-C) and triacylglycerols (TG) are more amenable to physical activity than LDL-cholesterol (LDL-C) and total cholesterol (TC) (6).

On the other hand, many cross-sectional studies do not account for confounding factors such as group differences in body weight and body fat, caloric intake, nutrient composition of diets and other potentially lipid-altering lifestyle characteristics. When these factors are controlled, the group differences in TC and LDL-C decrease and often are no longer significant (7, 8).

Additionally, it is well known that lipid and lipoprotein plasma levels are under control of genetic and hormonal factors affecting lipoprotein and apolipoprotein synthesis and interactions and in consequence their plasma concentrations (9, 10).

It is well recognized that estrogen has beneficial effects on lipoprotein metabolism enhancing HDL-C antioxidant potential, up-regulating LDL-C receptors in the liver and in this way decreasing circulating LDL-C levels (11-13). On the contrary, deficiency in estrogen secretion (e.g. in menopause) has been found to disturb lipoprotein profile increasing TC and LDL-C plasma levels, LDL-C oxidation and decreasing HDL-C concentrations (14).

Numerous studies have indicated that strenuous physical activity in high performance female athletes depresses ovarian hormone secretion causing luteal phase deficiency, anovulation, oligomenorrhea and even amenorrhea (15).

Data concerning the effect of ovarian hormone deficiency on plasma lipid and lipoprotein in female athletes are scarce and contradictory. Kaiserauer et al. (16) have demonstrated that in amenorrheic runners plasma LDL-C levels were higher than in regularly menstruating athletes and similar to those found in sedentary, regularly menstruating women. However, plasma HDL-C in both menstruating and amenorrheic athletes were higher than in sedentary women. In addition, LDL-C to HDL-C ratio in amenorrheic runners (1.4) was higher than in regularly menstruating runners (1.2) (17). Moreover, Lamon-Fava et al. (18) have shown slightly higher plasma TC and TG levels but significantly lower apo A-I and apo-B concentrations in amenorrheic than in eumenorrheic runners. In addition, apo-A-I and apo-B levels in ammenorrheic runners were lower than in their eumenorrheic counterpart, but in eumenorrheic runners were higher than in sedentary women. These results suggested that the disturbances in ovary hormone secretion reversed the beneficial effects of physical activity on apoprotein concentrations in plasma.

On the contrary, Thompson et al. (19) have noted that women runners regardless of menstrual status exhibit higher HDL-C levels in plasma than sedentary controls.

Physical education students even not engaged in any specific sport are highly

active due to the obligatory curiculum including different sports (e.g. games. gymnastics, track and field, martial arts, fencing). However, data concerning lipid and lipoprotein profiles in this group with respect to gender, hormonal status and dietary habits are limited.

Hence, this study was undertaken to compare lipid and lipoprotein plasma concentrations in female and male physical education students. Additionally, in order to examine the effects of hormonal status on lipid metabolism female participants were recruited among oral contraceptive users and non-users.

Materials and Methods

Subjects

A total of 113 students (42 males and 71 females) volunteered to participate in the study. Among females 47 were oral contraceptive (OC) non-users and 24 were OC users for at least 3 month prior to the study. Oral contraceptives used were monophasic and contained ethinylestradiol (0.02-0.05 mg) and different gestagens (desogestrel, gestogen, levonogestrel, norgestrel). All the participants were healthy non-smokers not taking any vitamin and mineral supplements on a regular basis. All OC-non-users were regularly menstruating with cycle length 27-31 days. Cycle length was calculated by the participants from the first day of bleeding to the first day of the next bleeding during three consecutive months prior to the study. The experimental protocol was accepted by the Ethics Commission at the Academy of Physical Education. All the subjects were informed about procedures involved in the study and gave their written consent prior to participation. None of the participants was engaged in high performance sport and their mean weekly physical activity (7.3-7.5 h) was due the obligatory physical education programme.

Blood samples

Blood was withdrawn after overnight fast under aseptic conditions from the antecubital vein into polypropylene tubes containing EDTA and centrifuged at -4^{o} C (3000 rpm/15 min). The plasma was stored at -70^{O} C until analyzed.

Biochemical analyses

Plasma levels of TG, glycerol, FFA, TC and HDL-C were determined colorimetrically using Randox commercial kits (Randox Laboratories, United Kingdom). Plasma VLDL levels were calculated using the Friedewald equation (20). Plasma LDL-C concentrations were calculated by subtracting plasma HDL-C and VLDL concentrations from TC levels. Plasma insulin was assayed using standard immunoassay method and commercial kits with monoclonal antibodies against insulin (IRMA. OBRI. Poland). Intra- and inter-coefficients of variations for insulin measurements were 6.8% and 9.3%, respectively.

Calculations of Quantitative Insulin Sensitivity Check Index (QUICKI)

QUICKI was calculated using the formula $QUICKI = 1/[\log_{10} I_0 + \log_{10} G_0]$ where I_0 and G_0 were fasting plasma insulin and glucose levels, respectively (21).

Dietary records

Daily intake of energy and macronutrients was briefly assessed from 24 h food records collected over 4 days preceding blood collection and analyzed using a computer programme. A set of pictures of meals and foods were shown to the subjects by an experienced interviewer. The household measures of food intake were converted into gram weights. An interviewer assigned codes to the foods reported by the participants and performed computer analysis using the Food 2 programme purchased from the Institute of Food and Nutrition in Warsaw.

Anthropometric measurements

Three skinfolds (abdominal, supscapular, triceps) were measured using Harpenden caliper (United Kingdom). Each measurement was repeated twice and the mean values were used for calculation of body fat (22).

Statistical analysis

Data distribution was evaluated using the Shapiro-Wilk test. Friedman ANOVA and Mann-Whitney tests were used for data comparison. Spearman rank correlations between fat mass and biochemical variables and between plasma lipoproteins and adipose tissue lipolysis expressed as plasma glycerol concentrations were also calculated. Significance was set at $p<0.05$.

Results

Characteristics of the subjects are presented in Table 1. There were no major differences in anthropometric variables between female OC-users and non-users, however, female OC-non-users were slightly but significantly younger than OC-users and males.

TC plasma levels in female OC-users and non-users were higher than in males ($p<0.001$) (Table 2). However, in female OC-users plasma TC concentrations were higher in comparison with OC-non-users ($p<0.04$). Only in 2 men, 2 female OC-non-users and 3 female OC-users did the plasma TC exceed the accepted physiological limit of 6.1 $mmol \bullet L^{-1}$.

In OC-users plasma HDL-C concentrations were significantly higher than in OC-non-users and in males ($p<0.004$). In all men and women OC-non-users plasma HDL-C levels were within physiological limits (0.8-1.8 $mmol \bullet L^{-1}$). On the contrary in 23 female OC-users (95.8% of all OC-using participants) plasma HDL-C concentrations exceeded the upper physiological limit.

Plasma LDL-C levels did not differ in males and OC non-users, but in OC-users were markedly lower than in OC-non-users and males ($p<0.001$). In addition, in 12 OC-non-users plasma LDL-C was higher than the upper physiological limit of 4.2 $mmol \bullet L^{-1}$. The incidence of high LDL-C levels in OC-users and males was negligible and was found in 1 man and 1 female OC-user.

The TC/HDL-C ratio did not differ in males and in female OC-non-users, but was markedly lower in OC-users ($p<0.02$). The LDL-C/ HDL-C ratio was markedly higher in OC-non-users than in males ($p<0.02$) and OC-users ($p<0.001$).

Plasma TG concentrations were lower in OC-non-users than in males ($p<0.02$) and OC-users ($p<0.03$).

Table 1. *Anthropometric characteristics of the subjects*

	Men (n = 42)	Female OC-non-users (n =47)	Female OC- users (n =24)
Age (years)	22.5 ± 1.2	20.6 ± 1.2[a]	21.7 ± 1.0
Body mass (kg)	78.6 ± 11.7	62.3 ± 7.8	59.9 ± 5.7
Body height (cm)	180.6 ± 5.2	170.7 ± 7.2	167.1 ± 5.8
Fat (kg)	12.4 ± 5.1	15.2 ± 2.9	14.6 ± 4.4
Fat (%)	15.4 ± 4.2	24.7 ± 5.7	24.0 ± 5.6
LBM* (kg)	66.2 ± 7.7	47.3 ± 5.0	45.3 ± 3.4
BMI ($kg \bullet m^{-2}$)	24.0 ± 2.7	21.3 ± 1.8	21.5 ± 3.4
Activity (h/week)	7.5 ±2.3	7.4 ± 2.6	7.3 ± 2.5

Data are means ± SD; LBM – lean body mass
a – $p < 0.001$ vs. males and female OC-users

Table 2. *Biochemical characteristics of the subjects*

	Men (n = 42)	Female OC-non-users (n =47)	Female OC- users (n =24)
TC (mmol•L^{-1})	4.7 ± 0.8	5.2 ± 0.9[a]	5.5 ± 1.2[a,b]
HDL-C (mmol•L^{-1})	1.7 ± 0.4	1.6 ±0.4	2.5 ± 0.6[c]
LDC-C (mmol•L^{-1})	2.8 ± 0.8	3.5 ± 0.8[d]	2.7 ± 1.1
TG (mmol•L^{-1})	0.91 ± 0.36	0.7 ± 0.25[e]	1.0 ± 0.41
TC /HDL-C	2.9 ± 0.8	3.4 ± 0.9	2.3 ± 0.5[f]
LDL-C/HDL-C	1.8 ± 0.8	2.3 ± 0.9[g]	1.2 ± 0.5
NEFA (µmol•L^{-1})	366.5 ± 237.8	320.7 ± 194.5	375.4 ± 199.4
Glycerol (µmol•L^{-1})	56.3 ± 25.9	72.2 ± 24.5	76.5 ± 26.6
Glucose (mmol•L^{-1})	4.9 ± 0.6	4.4 ± 0.6[h]	4.9 ± 0.5
Insulin (pmol•L^{-1})	48.7 ± 21.3	46.7 ± 21.2	48.2 ± 20.2
QUICKI	0.363 ± 0.028	0.365 ± 0.026	0.364 ± 0.027

Data are means ± SD
a – $p < 0.001$ vs. males; b – $p < 0.04$ vs. female OC-non-users; c – $p < 0.004$ vs. males and female OC-non-users; d– $p < 0.001$ vs. males; e – $p < 0.02$ vs. males and female OC-non-users; f – $p < 0.03$ vs. males and female OC-non-users; g – $p < 0.021$ vs. males and female OC-users; h– $p < 0.04$ vs. female OC-users and males

Table 3. *Spearman rank correlations between plasma glycerol and indices of lipid and lipoprotein metabolism*

	Men (n = 42)	Female OC-non-users (n =47)	Female OC- users (n =24)
LDL-C	0.086	0.437[a]	0.299
HDL-C	- 0.174	0.003	0.390[b]
TG	0.376[c]	0.001	0.040

a – $p < 0.001$; b – $p < 0.059$; c – $p < 0.01$

There were no between-group differences between plasma FFA and insulin concentrations. Plasma glucose levels in OC-non-users were slightly, but significantly lower than in OC-users ($p<0.04$). Plasma glycerol concentrations did not differ between males and both groups of females, however, there was a tendency to higher plasma glycerol in female than in male students (by 28% and 36% in female OC-non-users and OC-users, respectively).

Neither males, female OC-users nor OC- non-users differed with respect to QUICKI values.

In male students there was a significant and positive correlation between plasma glycerol and TG concentrations ($r=0.376$, $p<0.01$) (Table 3.). In contrast, in female OC-non-users plasma glycerol levels were significantly and positively correlated with plasma LDL-C concentrations ($r=0.437$, $p<0.001$). In female OC-users the correlation between plasma glycerol and HDL-C was close to the limit of significance ($r=0.393$, $p<0.059$).

Energy and macronutrient intake did not differ in females OC-users and non-users (Table 4.). There were no differences in the percent of energy derived from protein, fat and carbohydrates between males, female OC-non-users and OC-users.

Neither nutritional density nor the percent of energy derived from saturated, monounsaturated and polyunsaturated fat

Table 4. *Daily energy and macronutrient intake in male and female students*

	Men (n = 42)	Female OC-non-users (n =47)	Female OC- users (n =24)
Energy (kcal)	3083 ± 878	1721 ± 542	1674 ± 479
Protein (g)	103.8 ± 31.8	58.9 ± 15.4	57.1 ± 14.7
Fat (g)	122.3 ± 40.7	65.2 ± 29.7	66.2 ± 23.8
Carbohydrates (g)	413.9 ± 125.4	238.8 ± 78.9	225.8 ± 69.6
Protein (%)	14 ± 2	14 ± 3	14 ± 2
Fat (%)	35 ± 5	33 ± 7	35 ± 6
Carbohydrates (%)	51 ± 5	52 ± 7	51 ± 6

Values are means ± SD

Table 5. *Nutritional density of protein, fat and carbohydrate in male and female students*

Density (g/1000 kcal)	Men (n = 42)	Female OC-non-users (n =47)	Female OC- users (n =24)
Protein	33.9 ± 6.2	34.5 ± 6.8	34.8 ± 5.9
Fat	39.6 ± 5.9	37.7 ± 8.6	39.2 ± 6.6
Carbohydrates	134.0 ± 13.5	138.9 ± 20.8	135.1 ± 15.7

Data are means ± SD

Table 6. *Percent of total energy derived from fat in men and women*

Density (g/1000 kcal)	Men (n = 42)	Female OC-non-users (n =47)	Female OC- users (n =24)
SAT	13 ± 2	13 ± 2	13 ± 2
MUFA	16 ± 2	14 ± 2	15 ± 3
PUFA	7 ± 2	6 ± 2	7 ± 1

SAT – saturated fat; MUFA – monounsaturated fat; PUFA – polyunsaturated fat

differed with respect to gender and OC use (Table 5 and 6).

Discussion

The most important finding of the present study was that physically active female students OC-non-users were characterized by less favorable lipoprotein profile than their male counterparts. These findings were more evident when TC/HDL-C and LDL-C/HDL-C ratios were taken into consideration. It should be pointed out that in our female OC-non-users the lipoprotein profile was less favorable than in amenorrheic women with similar physical activity, especially in respect to LDL-C/HDL-C ratio higher in our participants (2.3 vs. 1.7) (23).

It is noteworthy that in the Heritage Family Study female subjects were characterized by lower fasting TG and LDL-C levels and reduced TC/HDL-C ratio as compared to males (24). In our study in female students fasting TG was lower but LDL-C level and TC/HDL-C ratio was higher than in their male counterparts.

Assuming similar weekly physical activity of the participants of the present study, it could be postulated that this factor was of minor importance as a possible reason of between-group difference in plasma lipoprotein profile. On the other hand,

it has been postulated that cardiorespiratory fitness expressed as $\dot{V}O_2$max. may affect lipoprotein profile in plasma and subjects with higher $\dot{V}O_2$max. are characterized by more favorable profile than those with lower $\dot{V}O_2$max. (25). This assumption may be true with respect to male participants of the present study since our unpublished data indicated that their $\dot{V}O_2$max. was significantly higher vs. female OC-users and non-users. However, similar $\dot{V}O_2$max. female OC-non-users and OC-users (Lutosławska- unpublished) did not explain the difference noted in lipoprotein profile between both female groups.

Another potential reason for the difference in lipoprotein profile noted in the present study was the dietary habits of the participants since the detrimental effect of high-fat diet on plasma lipids and lipoproteins, irrespectively of gender has been well documented (26).

However, the analysis of dietary macronutrient intakes in males and both groups of females revealed similar percent of energy derived from total fat as well as from saturated, monounsaturated and polyunsaturated fat. Additionally, there were no differences in nutritional density of macronutrients with respect to gender and OC-use.

On the other hand, it should be pointed out that a brief validation of dietary records based on body fat and fat free mass (in kg) indicated that approximately 60% females of our study, both OC-users and non-users underreported their energy intakes (27). These findings may be responsible for the differences in lipoprotein profiles between males and female OC-non-users, but do not explain the differences noted between female OC-users and non-users.

The probable reason for the differences in lipoprotein profile between males and female OC-non-users may be due to the effects of body fat content since it has been well documented that the elevation in body fat results in increased plasma LDL-C concentrations (28). However, there were no differences in percent of body fat between female OC-non-users and users, but they differ markedly with respect to plasma LDL-C levels.

Plasma lipoproteins undergo complicated regulation by the rate of very low density lipoprotein (VLDL) synthesis in the liver, its secretion into the blood stream, and modification into IDL, LDL and HDL-cholesterol (29). Additionally, it is well documented that apart from dietary fat adipose tissue-derived FFA seems to play an important role in stimulating hepatic VLDL production (30).

In the present study there were no differences in plasma FFA levels between male and female students. However, this did not exclude that FFA liver uptake may differ in males and females. It is well recognized that plasma FFA concentration reflects both adipose tissue lipolysis and FFA uptake by the peripheral tissues with the greatest contribution of the striated muscle (31). Therefore, the measurements of plasma FFA may not reflect subtle differences in FFA distribution due its supply to the liver.

On the other hand, plasma glycerol concentration is a reliable measure of lipolytic rate (32). In the present study there were no significant differences in plasma glycerol levels between males and females, however in females plasma glycerol tended to be higher indicating a greater lipolytic rate than in males.

In the present study marked differences were found in the relationships between lipolysis and circulating lipids and lipoproteins. In males adipose tissue lipolysis was significantly correlated with plasma TG levels, what was in agreement with literature data (30). However, it should be pointed out that adipose tissue lipolysis

accounted only for about 14% of the variance in plasma TG levels in male students.

In female OC-non-users adipose tissue lipolysis mostly affected plasma LDL-C levels with no effects on plasma TG concentrations being responsible for about 19% of the variance in plasma LDL-C levels. On the contrary, in OC-users there was a minor effect of glycerol provision on circulating LDL-C, with prevalence of its action on plasma HDL-C levels.

The above data suggested that physically active males and females differed with respect to adipose tissue contribution to the lipoprotein profile. Additionally, our data indicated that OC use markedly affected adipose tissue action on plasma lipoprotein profile. The reason for above findings remains elusive. However, it could not be excluded that less beneficial plasma lipoprotein profile in OC-non-users could be due to depressed ovarian hormone secretion in highly active female students.

It is well known that female sex hormones play an important role in lipid and lipoprotein metabolism. In animal studies *in vitro* it has been found that 17ß-estradiol induces VLDL synthesis in the liver (33). On the other hand, Wakatsuki and Sagara (34) have demonstrated that in postmenopausal and surgically menopausal women plasma LDL-C concentrations were inversely correlated with plasma 17ß-estradiol levels. Moreover, estrogen was found to up-regulate LDL-C receptors in the liver and LDL binding to human liver homogenates (35).

Data concerning OC and/or HRT action on plasma lipids and lipoproteins are equivocal, since detrimental, beneficial or no effects have been observed (36-38).

However, taking all the above together it is clear that ovarian hormones play a substantial role in the regulation of lipoprotein balance in the body. The results of the present study concerning beneficial effects of OC use on plasma HDL-C levels are in agreement with recently published data of Lamon-Fava et al. (39).

In the participants of the present study insulin sensitivity expressed as QUICKI did not differ with respect to gender and was similar to that reported in lean middle-aged men (40). Therefore, it could be presumed that the overall insulin sensitivity did not play an important role as a predictor of the differences in plasma lipoprotein profile in the participants of the present study (41).

Plasma TC and LDL-C and especially TG levels in our study were lower than those reported for middle-aged Polish men and women tested in 1993 and similar to reported for German men and women (42, 43). The favorable differences between our participants and Polish population were much more evident in males than in females OC-non-users and were probably due to the differences in age and physical activity as well as in dietary habits (44-46). Strikingly, in female OC-non-users TC/HDL-C ratio (3.4) was higher than reported in regularly active menopausal women living in Australia (2.9) (47). This undesirable difference may be due to numerous lifestyle and genetic factors (45, 48).

In conclusion, our study revealed that plasma lipoprotein profile in physically active regularly menstruating female students was less favorable than in their male counterparts due to slightly, but significantly higher TC and LDL-C concentrations. On the contrary, in female students using oral contraceptives an improved lipoprotein profile was found mostly due to elevation in plasma HDL-C levels. Additionally, in females using oral contraceptives adipose tissue lipolysis contributes more to the elevation in plasma HDL-C levels than in OC-non-users. In contrast, in female students not using oral contraceptives adipo-

se tissue lipolysis seems to adversely affect plasma LDL-C concentrations.

References

1. Halle M, Baumstark MW, Keul J. Association of physical fitness with LDL and HDL subfractions in young healthy men. *Int J Sports Med* 1999; 20: 464-9.
2. Kraus WE, Houmard JA, Duscha BD. et al. Effects of the amount and intensity of exercise on plasma lipoproteins. *N Engl J Me*d 2002; 347: 1483-92.
3. Twisk JW, Kemper HC, van Mechelen W. Tracking of activity and fitness and the relationship with cardiovascular disease risk factors. *Med Sci Sports Exerc* 2000; 32: 1455-6.
4. Jafari M, Leaf DA, MacRae H. et al. The effects of physical exercise on plasma prebeta-1 high-density lipoprotein. *Metabolism* 2003; 52: 437-42.
5. Skoumas J, Pitsavos C, Panagiotakos DB. et al. Physical activity, high density lipoprotein cholesterol and other lipids levels, in men and women from the ATTICA study. *Lipids Health Dis* 2003; 2: 1-7.
6. Durstine JL, Grandjean PW, Davis PG. et al. Blood lipid and lipoprotein adaptations to exercise. *Sports Med* 2001; 31: 1033-62.
7. Sternfeld B, Sidney S, Jacobs DR. Jr. et al. Seven-year changes in physical fitness, physical activity, and lipid profile in the CARDIA study, Coronary Artery Risk Development in Young Adults. *Ann Epidemiol* 1999; 9: 25-33.
8. Williams P. Relationship of heart disease risk factors to exercise quantity and intensity. *Arch Intern Med* 1998; 158: 237-45.
9. Larson IA, Ordovas JM, Sun Z. et al. Effects of apolipoprotein A-IV genotype on glucose and plasma lipoprotein levels. *Clin Genet* 2002; 61: 430-6.
10. Mahley RW, Palaoglu KE, Atak Z. et al. Turkish Heart Study: lipids, lipoproteins and apolipoproteins. *J Lipid Res* 1995; 36: 839-59.
11. Cheung AP. Acute effects of estradiol and progesterone on insulin, lipids and lipoproteins in postmenopausal women: a pilot study. *Maturitas* 2000; 35: 45-50.
12. Karjalainen A, Heikkinen J, Savolainen MJ. et al. Mechanisms regulating LDL metabolism in subjects on perorlal and transdermal estrogen replacement therapy. *Artheroscler Tromb Vasc Biol* 2000; 20: 1101-6.
13. Perrella J, Berco M, Cecutti A. et al. Potential role of the interaction between equine estrogens, low-density lipoprotein (LDL) and high-density lipoprotein (HDL) in the prevention of coronary heart and neurodegenerative diseases in postmenopausal women. *Lipids Health Dis* 2003; 2: 1-10.
14. Mosinger BJ. Human low-density lipoproteins: oxidative modification and its relation to age, gender, menopausal status and cholesterol concentrations. *Eur J Clin Chem Clin Biochem* 1997; 35: 207-14.
15. Cumming DC, Cumming CE. Estrogen replacement therapy and female athletes. *Sports Med* 2001; 31: 1025-31.
16. Kaiserauer S, Snyder AC, Sleeper M. et a., Nutritional, physiological, and menstrual status of distance runners. *Med Sci Sports Exerc* 1989; 21: 120-5.
17. Hetland MT, Haarbo J, Christiansen C. Body composition and serum lipids in female runners: influence of exercise level and menstrual bleeding pattern. *Eur J Clin Invest* 1995; 25: 553-8.
18. Lamon-Fava S, Fisher EC, Nelson ME. et al. Effect of exercise and menstrual cycle status on plasma lipids, low density lipoprotein particle size and apolipoproteins. *J Clin Endocrinol Metab* 1989; 68: 17-21.
19. Thompson DL, Snead DB, Seip Rl. et al. Serum lipid levels and steroidal hormones in women runners with irregular menses. *Can J Appl Physiol* 1997; 22: 66-7.
20. Friedewald WT, Levy R, Fredrickson D. Estimation on concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultra-centrifugation. *Clin Chem* 1972; 18: 499-504.
21. Katz A, Nambi SS, Mather K. et al. Quantitative insulin sensitivity check index: a simple, accurate method for assessing insulin sensitivity in human. *J Clin Endocrinol Metab* 2000; 85: 2402-10.
22. Huygnes W, Claessens AL, Thomis M. et al. Body composition estimations by BIA verus anthropometric equations in body builders and other power athletes. *J Sports Med Phys Fitness* 2002; 42: 45-55.
23. Friday KE, Drinkwater BL, Bruemmer B. et al. Elevated plasma low-density lipoprotein and high-density lipoprotein cholesterol levels in amenorrheic athletes: effect of endogenous hormone status and nutrient intake. *J Clin Endocrinol Metab* 1993; 77: 1605-9.
24. Despres JP, Gagnon J, Bergeron J. et al. Plasma post-heparin lipase activities in the Heritage Family Study: the reproducibility, gender differences, and associations with lipoprotein levels. *Clin Biochem* 1999; 32: 157-65.
25. Eisenmann JC. Blood lipids and peak oxygen consumption in young distance runners. *Sports Med* 2002; 32: 297-307.
26. Bravo E, Flora L, Cantafora . et al. The influence of dietary saturated and unsaturated fat on hepatic cholesterol metabolism and biliary excretion of chylomicron cholesterol in the rat. *Biochim Biophys Acta* 1998; 1390: 134-48.

27. Wallstrőm P, Wirfalt E, Lahmann PH, Gulberg B. et al. Serum concentrations of ß-carotene and α-tocopherol are associated with diet, smoking, and general and central adiposity. *Am J Clin Nutr* 2001; 73: 777-85.
28. Deschenes D, Couture P, Dupont P. et al. Subdivision of the subcutaneous adipose tissue compartment and lipid-lipoprotein levels in women. *Obes Res* 2003; 11: 469-76.
29. Diraison F, Beylot M. Role of human liver lipogenesis and reesterification in triglycerides secretion and in FFA reesterification. *Am J Physiol* 1998; 274:E 321-7.
30. Julius U. Influence of plasma free fatty acids on lipoprotein synthesis and diabetic dyslipidemia. *Exp Clin Endocrinol Diabetes* 2003; 111: 246-50.
31. Coyle EF. Fat oxidation during exercise: role of lipolysis, FFA availability, and glycolytic flux, In: Hargreaves M, Thompson M, eds, *Biochemistry of Exercise*, Champaign, Ill: Human Kinetics Books, 1999: 263-73.
32. Flechtner-Mors M, Ditschuneit HH, Yip I. et al. Sympathetic modulation of lipolysis in subcutaneous adipose tissue: effects of gender and energy restriction. *J Lab Clin Med* 1999; 134: 33-41.
33. Chan L, Jackson RL, Means AR. Female steroid hormones and lipoprotein synthesis in the cockerel: effects of progesterone and nafoxidine on the estrogenic stimulation of very low density lipoprotein (VLDL) synthesis, *Endocrinology* 1997; 100: 1636-43.
34. Wakatsuki A, Sagara Y. Lipoprotein metabolism in postmenopausal and oophorectomized women. *Obstet Gynecol* 1995; 85: 523-8.
35. Walsh BW, Schiff I, Rosner B. et al. Effects of postmenopausal estrogen replacement on the concentrations and metabolism of plasma lipoproteins. *N Eng J Med* 1991; 325: 1196-204.
36. Connelly PW, Stachenko S, MacLean DR. et al. The prevalence of hyperlipidemia in women and its association with use of oral contraceptive replacement therapy and nonlipid coronary arthery disease risk factors, Canadian Heart Health Group. *Can J Cardiol* 1999; 15: 419-27.
37. Knoop RH, Broyles FE, Cheung M. et al. Comparison of the lipoprotein, carbohydrate, and hemostatic effects of phasic oral contraceptives containing desogestrel or levonorgestrel. *Contraception* 2001; 63: 1-11.
38. Yong Rl, DelConte A. Effects of low-dose monophasic levonorgestrel with ethinyl estradiol preparation on serum lipids levels: A twenty-four months clinical trial. *Am J Obstet Gynecol* 1999; 181: 59-62.
39. Lamon-Fava S, Posfai B, Asztalos BF. et al. Effects of estrogen and medroxyprogesterone acetate on subpopulations of triglyceride rich lipoproteins and high-density lipoproteins. *Metabolism* 2003; 52: 1330-6.
40. Bastard JP, Robert JJ, Jardel C. et al. Is quantitative insulin sensitivity check index, a fair insulin sensitivity index in humans? *Diabetes Metab* 2001; 27: 69-70.
41. Sparks JD, Sparks CE. Insulin regulation of triacylglycerol-rich lipoprotein synthesis and secretion. *Biochim Biophys Acta* 1994; 1215: 9-32.
42. Chotkowska E, Jasiński B, Kupsc W. et al. Lipid risk factors of coronary heart disease during longterm observation of a Warsaw Pol-Monica population-based cohort. *Pol Arch Med Wewn* 1997; 97: 37-46.
43. Rehm J, Sempos C, Kohlmeier L. et al. A comparison of serum total cholesterol levels and their determinants between the Federal Republic of Germany and United States. *Eur J Epidemiol* 2000; 16: 669-75.
44. Ashton WD, Nanchahal K, Wood DA. Leisure-time physical activity and coronary risk factors in women. *Cardiovasc Risk* 2000; 7: 259-66.
45. Brunner EJ, Wunsch H, Marmot MG. What is an optimal diet? Relationship of macronutrient intake to obesity glucose tolerance, lipoprotein cholesterol levels and the metabolic syndrome in the Whitehall II study. *Int J Obes* 2001; 25: 45-53.
46. Warber J, Bazzarre T. A comparison between running and weight lifting on fasting plasma lipids of a well-conditioned hypercholesterolemic male. *Int J Sport Nutr* 1991; 1: 265-78.
47. Boutcher SH, Meyer BJ, Craig GA. et al. Plasma lipid and fibrinogen levels in aerobically trained and untrained postmenopausal women. *J Sports Med Phys Fitness* 2003; 43: 231-5.
48. Raitakari OT, Porkka KV, Rasanen L. et al. Relations of life-style with lipids, blood pressure and insulin in adolescent and young adults. The Cardiovascular Risk in Young Finns Study. *Atherosclerosis* 1994; 111: 237-46.

Received: December 12, 2003
Accepted: March 01, 2004

Address for correspondence:
Grażyna Lutosławska, PhD, DSc
Department of Biochemistry,
Academy of Physical Education
01-968 Warsaw 45
Box 55
Poland
E-mail: grazyna.lutoslawska@awf.edu.pl