

SERUM LIPOPROTEIN (a) [Lp(a)] LEVELS IN OVERWEIGHT AND OBESE YOUTHS – A COMBINED EFFECT OF PHYSICAL ACTIVITY AND LOW-CALORIE DIET

K.Stankiewicz¹, L.Szcześniak¹, T.Rychlewski², E.Deskur-Śmielecka¹, Z.Kasprzak¹

¹*Dept. of Cardiac Rehabilitation, Academy of Physical Education, Poznań, Poland;* ²*Chair of Dept. of Physiology, Biochemistry and Hygiene, Academy of Physical Education, Poznań, Poland*

Abstract. The aim of the present study was to determine the effect of low-calorie diet alone and in combination with systematic aerobic exercise on serum lipoprotein (a) levels in obese youths. Obese youths (BMI>30.0 kg/m²) enrolled into the study were randomly divided into three groups. Patients in the Group I (n=20) trained daily on bicycle ergometer at intensity of aerobic threshold for 21 days. Subjects in the Group II (n=23) performed analogous training for 28 days. The ventilatory threshold was determined during bicycle ergometry (Ergo-Line, Germany) with CardioO₂ computer system (ECG Exercise System, Medical Graphic, USA). Youths in the Group III (n=16) did not participate in bicycle training. Subjects in all three groups were prescribed a low-calorie diet (average intake 1300 kcal) with restriction of saturated fats and monosaccharides. Prior to starting and after finishing the program, blood samples were taken from basilic vein after an overnight fast and serum lipoprotein (a) levels were measured by an immunoenzymatic assay (Cormay, Poland). Serum Lp(a) levels significantly decreased in obese youths subjected to 21 or 28-day therapy combining low-calorie diet and systematic exercise. Particularly important improvement in serum Lp(a) concentrations was observed in subjects who initially had elevated serum Lp(a) levels. Youths put on low-calorie diet alone did not demonstrate significant change in serum lipoprotein (a) concentrations. These results indicate that systematic aerobic exercise may have a potential to prevent atherogenic action of lipoprotein (a).
(Biol.Sport 21:171-179, 2004)

Key words: Lipoprotein (a) - Obese youths - Physical exercise - Low-calorie diet

Reprint request to: Prof. dr hab. Łucja Szcześniak, Chair of Dept. of Physiology, Biochemistry and Hygiene, Academy of Physical Education, Królowej Jadwigi 27/39; 61-871 Poznań (Poland) Tel.: (+4861) 8355177; Fax: (+4861) 8330087



Introduction

Obesity is a common civilization-related disease and an important social problem. It has been also recognized as risk factor of atherosclerosis, together with elevated serum cholesterol level, hypertension, cigarette smoking, low physical activity and genetic predisposition. Obesity prevalence depends on environmental factors, such as life-style and nutrition habits [2]. Obesity is associated with changes in metabolism of lipids and glucose, which result in increased levels of triglycerides and LDL-cholesterol, decreased HDL-cholesterol concentrations and hyperinsulinemia [3,13]. Metabolic abnormalities related to obesity can be effectively treated with non-pharmacological methods such as increased physical activity and low-calorie diet [20,22]. The reduction in body mass index due to increased physical activity was associated with improved HDL-cholesterol concentrations [27] and lowered total and LDL-cholesterol levels [6].

Lipoprotein (a) [Lp(a)] is a separate class of lipoproteins. Although its physiological function remains unknown, the correlation between raised level of Lp(a) with increased risk of ischaemic heart disease is well documented. Several attempts have been made to estimate the influence of diet and physical exercise on serum lipoprotein (a) levels, yet the results are conflicting and inconclusive [15].

The aim of the present study was to investigate the combined influence of low-calorie diet and regular physical activity on serum lipoprotein (a) levels in obese youths and to compare it with the effect of low-calorie diet alone.

Material and Methods

Overweight and obese youths aged between 14.5 and 17 years (average 15.9 ± 1.62 years) attending 21-day or 28-day sanatorium turns were asked to participate in the study. All subjects enrolled into the study performed a bicycle ergometry (Ergo Metrics 900, Ergo-Line, Germany) with determination of ventilatory threshold (CardioO₂ computer system, ECG Exercise System, Medical Graphic, USA).

Subjects in the Group I (n=20) trained on bicycle ergometer 30 min daily for 21 days. The intensity of training was adjusted so that the exercise heart rate was equal to that at which ventilatory threshold had been reached. Heart rate was controlled with type PE 3000 Sport Tester (Finland). Patients in the Group II (n=23) trained in analogous way for 28 days. Youths in the Group III (n=16) did not participate in bicycle training. All patients were prescribed a low-calorie diet composed of 18.9% protein, 26.1% fat and 54.9% carbohydrate. An average prescribed intake was 1300 kcal. According Ziemiński [28] this energy supply



covers 49% and 45% of the mean recommended daily demands for youths aged 13-15 and 16-18 years, respectively.

Prior to starting and after finishing the program, blood samples for lipoprotein (a) levels determination were taken from basilic vein after an overnight fast. Serum Lp(a) concentrations were measured by an immunoenzymatic assay exact to 1 mg/dl (Cormay, Poland).

Informed consent was obtained from each participant. The study protocol was approved by the local Ethics Committee for Research in Humans.

(All data are expressed as means± standard deviation. Differences in patient variables before and during the therapy were statistically evaluated by Wilcoxon test. A P value<0.01 was considered significant.)

Results

Anthropometric characteristics of the patients are presented in Table 1. Youths in all study groups were obese (body mass index exceeding 30 kg/m²). Significant reductions (P<0.01) in body mass index were achieved in all study groups (5.5 kg and 1.6 kg/m² in the Group I, 7.9 kg and 2.9 kg/m² in the Group II, 5.4 kg and 1.9 kg/m² in the Group III).

Table 1

Basic characteristics of study population (x ±SD)

Study group	N	Years	Body mass (kg)		BMI (kg/m ²)	
			I term	II term	I term	II term
Group I	20	15.6±1.38	92.8±22.25	87.3±20.34	30.8±4.89	29.2±4.42
Group II	23	15.7±1.44	99.5±22.76	91.6±20.13	33.0±5.45	30.1±4.99
Group III	16	16.5±1.96	85.2±12.64	79.8±12.00	30.6±4.70	28.7±4.49

A percentage share of study population with reference to lipoprotein (a) levels prior to enrollment into the study is presented in Fig. 1. In two of three study groups no subjects with undetectable Lp(a) levels were found. Lp(a) levels exceeding reference range (1-30 mg/dl) were recorded in 45% subjects in the Group I, 13% in the Group II and 38% in the Group III. In general, 31% obese youths had serum Lp(a) concentrations higher than the acceptable value of 30 mg/dl.



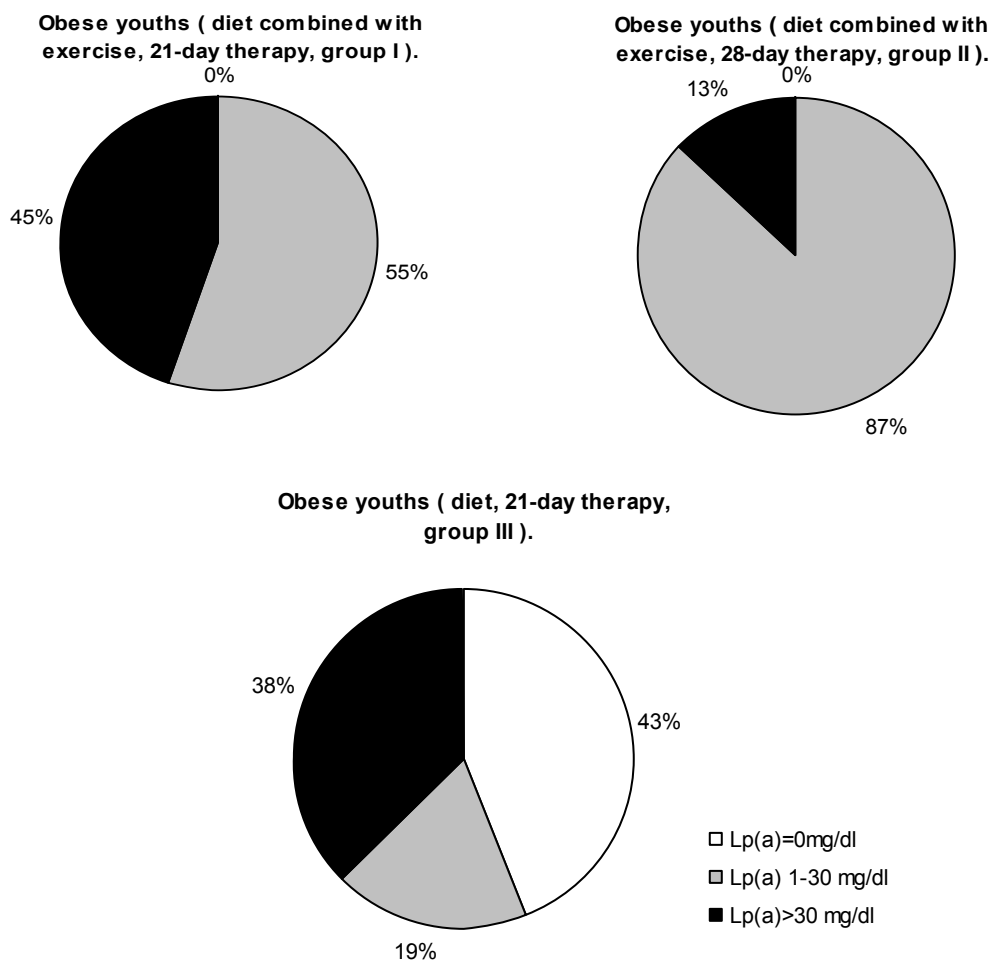


Fig. 1
Percentage share of study population with reference to baseline lipoprotein (a) levels



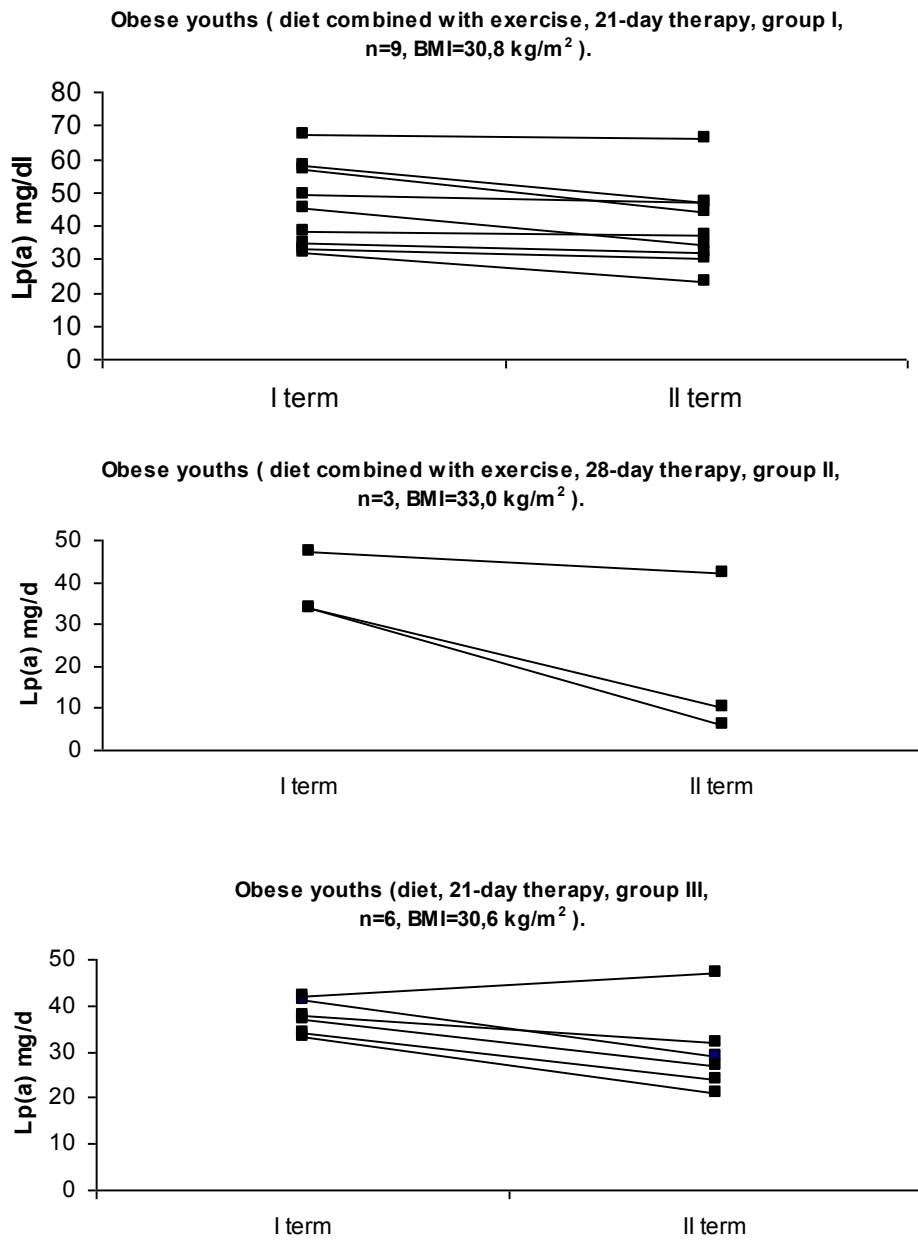


Fig. 2 Changes in serum lipoprotein (a) concentrations during therapy in individual obese youths with hyperlipoproteinemia (a) >30 mg/dl



Serum lipoprotein (a) levels prior to and after finishing the therapy are presented in Table 2. Serum Lp(a) levels significantly decreased in the Group I in subjects with hyperlipoproteinemia (a) ($P<0.01$) and in the Group II both in youths with elevated and normal Lp(a) levels ($P<0.01$). In Group III, in which subjects were not subjected to bicycle training, the serum Lp(a) levels remained unchanged. Particularly important improvement in serum Lp(a) concentrations was observed in subjects who initially had elevated serum Lp(a) levels. Changes in serum Lp(a) concentrations in individual patients are shown in Fig. 2.

Table 2

Serum lipoprotein (a) concentrations before and after therapy ($x \pm SD$)

Study group	Lp(a) (mg/dl)	N	I term ($x \pm SD$)	II term ($x \pm SD$)	Difference I-II	Wilcoxon Test
Group I	1-30	11	20.8±5.30	19.9±5.33	0.9±2.98	NS
	>30	9	46.0±12.57	40.0±12.68	6.0±4.89	$P<0.01$
Group II	1-30	20	10.2±4.61	7.3±6.52	2.9±5.72	$P<0.01$
	>30	3	38.3±7.50	19.3±19.73	19.0±12.28	$P<0.01$
Group III	0	7	0.0	0.0	0.0	-
	1-30	3	9.6±5.50	13.0±4.58	- 3.4±1.52	NS
	>30	6	37.5±3.61	30.0±9.16	7.5±6.50	NS

Discussion

The results of research work on the effect of physical activity on serum lipoprotein (a) levels are conflicting. Favorable influence of exercise on Lp(a) levels has been demonstrated in a few papers [7,8,16,21], while other authors reported increases in Lp(a) levels following prolonged, intense exercise in healthy subjects [9,11,17]. Transient increases in lipoprotein (a) levels directly after strenuous exercise were explained by the important role for lipoprotein (a) in repairing tissues damaged by free radicals generated during exercise [5]. Several investigators found no relationship between increased physical activity and Lp(a) concentrations. Hubinger *et al.* [10] observed no changes in Lp(a) concentrations in healthy subjects after 6-week physical training at intensity of 60-85% maximum heart rate. The lack of effect of training on Lp(a) levels may be due to the fact that its concentrations are genetically determined [25] and thus are hardly modifiable by external factors such as physical exercise, diet and medication [1]. In our study



we found a significant ($P < 0.01$) decrease in Lp(a) levels in obese youths after 21 or 28-day therapy combining low-calorie diet and physical training on bicycle ergometer of intensity of anaerobic threshold. The favorable effects of applied treatment were particularly apparent in subjects with hyperlipoproteinemia (a) (Table 2). In patients with serum lipoprotein (a) levels exceeding 30 mg/dl, Lp(a) concentrations decreased by 13.04% after 21-day treatment and by 49.61% after 28-day therapy. In subjects with lipoprotein (a) concentrations within normal range, its levels decreased by 28.5% only after 28-day therapy. No significant changes in Lp(a) concentrations were observed in obese youths treated with low-calorie diet alone.

The exact mechanisms responsible for reduction of serum lipoprotein (a) levels under influence of aerobic training have not been well elucidated. Activation of lipoprotein lipase observed during physical exercise may, at least in part, contribute to this phenomenon. Lipoprotein lipase facilitates binding of Lp(a) particles to proteoglycans of heparan sulphate on cell surface and thus enhances its catabolism [26]. Other possible explanation is an increase in LDL-receptor activity, induced by physical exercise. According to studies of Liu et al [14], 25% of lipoprotein (a) is degraded by the LDL-receptor.

The improvement in lipid metabolism and reduction in total and LDL-cholesterol levels may be achieved as a result of low-calorie diet with restricted intake of saturated fats and monosaccharides [18]. The replacement of saturated fatty acids with mono- and polyunsaturated fatty acids results in marked decrease in LDL-cholesterol levels. This effect is probably due to increased activity of receptor for LDL and, in consequence, augmented catabolism of low-density lipoproteins [2]. It is possible that dietary fat saturation affects also lipoprotein (a) concentrations, however the data concerning humans are lacking [4]. In present study, we did not observe significant changes in serum Lp(a) levels in obese youths treated with low-calorie diet with fat intake restriction, unless the diet was combined with systematic physical exercise. In subjects treated with diet alone (Group III), despite marked reduction in body mass, lipoprotein (a) concentrations tended to increase both in subjects with normal and elevated Lp(a) levels. These results are in keeping with studies of Perruse *et al.* [19].

The impact of physical activity on lipoprotein (a) levels may depend on the type of the exercise. In our study, training was entirely aerobic. Such training does not increase the generation of oxygen free radicals and thus it does not lead to radical-mediated tissue injury [12]. Moreover, during aerobic training fats are used as energetic substrate.

The results of our present and previous studies [23,24] prove that regular aerobic training may lead to reduction of atherogenous lipoprotein (a) levels. The favorable



influence of training on Lp(a) concentrations was observed even when exercise was not combined with low-calorie diet and not accompanied by reduction in body mass. Further research is necessary to clarify the mechanisms contributing to the impact of physical training on serum lipoprotein (a) levels.

References

1. Albers J.J., S.M.Marcovina, M.S.Lodge (1990) The unique lipoprotein (a): properties and immunochemical measurement. *Clin.Chem.* 36:2019-2026
2. Bailleul S., R.Couderc, C.Rossignol, J.Fermanian, F.Boutouchent, M.A.Farnier et al. (1995) Lipoprotein (a) in childhood: relation with other atherosclerosis risk factors and family history of atherosclerosis. *Clin.Chem.* 41:241-245
3. Björntrop P. (1988) Abdominal obesity and the development of non – insulin dependent Diabetes Mellitus. *Diabetes Metab.Rev.* 4:615-622
4. Brousseau M.E., J.M.Ordozas, R.J.Nikolasi, E.J.Scheafer (1994) Effects of dietary fat saturation on plasma lipoprotein (a) and hepatic apolipoprotein (a) mRNA concentrations in cynomolgus monkeys. *Atherosclerosis* 106:109-118
5. Cardoso G.C., C.Posadas, O.O.Orvananos, C.Peniche, J.Zamora et al. (1994) Long distance runners and body-builders exhibit elevated plasma levels of lipoprotein (a). *Chem. Phys.Lipids* 67/68:207-221
6. Haskell W.L. (1986) The influence of exercise training on plasma lipids and lipoproteins in health and disease. *Acta Med.Scand.* (Suppl. 711):25-37
7. Hellsten G., K.Boman, G.Hallmans, G.Dahlen (1989) Lipids and endurance physical activity. *Atherosclerosis* 75:93-94
8. Herrmann W., J.Biermann, H.G.Lindhofer, G.Kostner (1989) Beeinflussung des atherogenen Risikofaktors Lp(a) durch supplementäre Fischölaufnahme bei Patienten mit moderatem physischem Training. *Med.Klin.* 84:429-433
9. Hubinger L.M., L.T.Mackinnon, F.Lepre (1995) Lipoprotein (a) [Lp(a)] levels in middle – aged male runners and sedentary controls. *Med.Sci.Sports Exerc.* 27:490-496
10. Hubinger L.M., L.T.Mackinnon (1996) The effect of endurance training on lipoprotein (a) [Lp(a)] levels in middle – aged males. *Med.Sci.Sports Exerc.* 28:757-764
11. Israel R.G., M.J.Sullivan, R.H.L.Marks, R.S.Cayton, T.C.Chenier (1994) Relationship between cardiorespiratory fitness and lipoprotein(a) in men and women. *Med.Sci.Sports Exerc.* 26:425-431
12. Karolkiewicz J., Ł.Szczęśniak, Z.Kasprzak, T.Rychlewski, F.Banaszak (1998) Reduced glutathione concentration in red blood cells and lipid profile individuals subjects to systematic exercise and low calorie diet. *Diabetol.Pol.* 5(1):51-56 (in Polish, English abstract)
13. Kissebah A.H., A.N.Peiris (1988) Biology of regional body fat distribution relationship to non – insulin- dependent Diabetes Mellitus. *Diabetes Metab.Rev.* 5:83-109



14. Liu A., R.Lawn (1994) Lipoprotein (a) and atherogenesis. *Trends Cardiovasc.Med.* 4:40-44
15. Mackinnon L.T., L.M.Hubinger, F.Lepre (1997) Effects of physical activity and diet on lipoprotein (a). *Med.Sci.Sports Exerc.* 29:1429-1436
16. Mackinnon L.T., L.M.Hubinger (1999) Effects of exercise on lipoprotein (a). *Sports Med.* 28:11-24
17. Mankowitz K., R.Seip, C.F.Semenkovich, A.Daugherty, G.Schonfeld (1992) Short – term interruption of training effects both fasting and postprandial lipoproteins. *Atherosclerosis* 95:181-189
18. Murray R.K., D.K.Granner, P.A.Mayes, V.W.Rodwell (1995) *Biochemia Harpera*. PZWL Warszawa, pp. 721-733
19. Perruse L., C.Bouchard (1999) Role of genetic factors in childhood obesity and in susceptibility to dietary variations. *Ann.Med.* 31(Suppl):19-25
20. Rychlewski T., Ł.Szczeńniak, Z.Kasprzak, A.Nowak, F.Banaszak et al. (1996) An overall evaluation of the reaction in obese boys' organisms to systematic physical effort combined with a calorie restricted diet. *Pol.Arch.Med.Wewn.* 96:344-350 (in Polish. English abstract)
21. Sönnichsen A.C., W.O.Richter, P.Schwandt (1990) Reduction of lipoprotein (a) by weight loss. *Internal J.Obesity* 14:487-494
22. Stankiewicz K., Ł.Szczeńniak, A.Nowak, T.Rychlewski, Z.Kasprzak (1998) Some selected indices of impaired metabolism in obese people – influence of systematic physical activity and calorie restricted diet. *Endokr.Pol.* 49:295-301
23. Stankiewicz K., Ł.Szczeńniak, T.Rychlewski, P.Dylewicz, E.Deskur et al. (2001) The influence of rehabilitation training on serum concentrations of lipoprotein (a) in patients with ischaemic heart disease. *Med.Sportiva* 5(EE2):113-120
24. Stankiewicz K., Ł.Szczeńniak, T.Rychlewski, W.Romanowski (2000) Impact of increased physical activity and vegetable-fruit diet on blood lipid profile and lipoproteins in obese people. *Nowa Med.* 108:41-44 (in Polish, English abstract)
25. Uterman G. (1989) The mysteries of lipoprotein (a). *Science* 246:904-910
26. Williams K., G.Fless, K.Petrie, M.Snyder, R.Brocia et al. (1992) Mechanisms by which lipoprotein lipase alters cellular metabolism of lipoprotein (a), low density lipoprotein and nascent lipoproteins. *J.Biol.Chem.* 267:284-292
27. Wood P.D., M.L.Stefanick, D.M.Drean, B.Frey-Hewitt, S.C.Garay (1988) Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. *N.Engl.J.Med.* 319:1173-1179
28. Ziemiański Ś., B. Bułhak-Jachymczyk, J.Budzyńska-Topolowska, B.Panczenko-Kresowska, M.Wartanowicz (1998) Normy żywienia dla ludności w Polsce II (energia, białko, tłuszcze, witaminy i składniki mineralne). *Nowa Med.* 5(4):1-27

