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# EFFECT OF DIET AND PHYSICAL ACTIVITY ON PHYSIOLOGICAL AND BIOCHEMICAL PARAMETERS OF OBESE ADOLESCENTS

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**Background.** The main cause of the excessive deposition of fat is the destruction of the mechanisms controlling the expenditure of energy. Pathological increase of adipose tissue leads to disorders of the body, and lipid – carbohydrate parameters, promotes the development of vascular diseases and increases the risk of morbidity and mortality. The aim of the study is to demonstrate the impact of diet and physical activity changes in the parameters lipid-carbohydrate of adolescents.

**Material and methods.** The study included obese boys (n = 35), undergoing weight reduction. A low-energy diet and regular physical activity were applied. At the beginning and after four weeks were performed anthropometric measurements and indicators of the composition of venous blood was determined. In the venous blood was determined total cholesterol, HDL-cholesterol, triacylglycerols (TG), glucose and insulin. LDL-cholesterol was calculated.

**Results.** It was found that the applied treatment improved the lipid profile of blood. Only for triglyceride change was not statistically significant. Statistically significant was the reduction of the concentration of glucose.

**Conclusions.** Reduction of body mass resulted in positive changes in blood lipidogramme and reduction of waist hip ratio, which can reduce the risk of cardiovascular disease in the future. Reduction in serum insulin and glucose demonstrates improved carbohydrate metabolism and indicates a reduced risk for type II diabetes.

**Key words:** obesity, diet, physical activity, blood lipidograme

## INTRODUCTION

The increase of body mass, caused mainly by an accumulation of visceral fat, is common especially in Europe and North America. In these areas, the consumption of

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fat and caloric content of a daily food ration has increased in recent years. There are at least 300 million obese people and the number has been multiplied three times over the last 25 years [Ball 2005]. This problem affects not only adults, but also children and adolescents. The main cause of excessive deposition of fat is destruction of the mechanisms controlling the expenditure of energy [Lenart-Domka and Kwolek 2007]. According to the law of conservation of energy, the mass depends on the quantity supplied with food and consumed. If the balance is knocked down by excessive energy, the excess is stored as fat. Disorders of energy balance can be caused both by metabolic factors (genetic, some endokrynopathies). The second group is described as the causes of regulatory problems (environmental impacts, cultural, organic and functional disorders of the nervous system, emotional factors). Ingestion becomes more and more a form of leisure, which in combination with sedentary lifestyle leads to overweight.

Excess body fat is not just a matter of weight and aesthetics. From the standpoint of health consequences of the organism important is not only its quantity in the body, but its location. Pathological increase in body fat leads to an increase in the incidence of many diseases associated with it, and their occurrence at a much younger age [Goran et al. 2003]. Increasing the amount of body fat contributes to the endocrine lipid-carbohydrate disorder and consequently encourages the development of vascular diseases.

The aim of this work is to demonstrate the impact of diet and physical activity changes in the parameters lipid – carbohydrate among adolescents.

#### MATERIAL AND METHODS

The study was performed on the group of young boys at the age of  $15.6 \pm 1.40$  years with a substantial obesity (n = 35), subjected to reduce the excessive body mass. The degree of obesity was determined on the basis of the values of the body mass index (BMI). During the rehabilitation period (4 weeks) low-calorie diet and intense physical activity were applied. The diet delivered approximately 1300 kcal per day (Table 1). Daily nourishing ration was divided into 5 meals. The meals contained vegetables and fruits, the main source of vitamins, mineral salts and fiber. The proteins in the diet was derived from milk, dairy products, poultry and fish. The total amount of cholesterol in daily ration was not more than 300 mg. Essential fatty acid were derived mainly from vegetable oils.

Table 1. The average caloric diet and participation of proteins, fats and carbohydrates

Kcal	Protein		Fat		Carboh	Carbohydrate	
Kcai	% energy	g	% energy	g	% energy	g	g
1 291.3	21.1	66.5	24.0	33.3	54.9	176.9	31.18

Each day subjects were performing 2-hours walks and 1-hour sport activities (game sports, swimming) and three days a week they were performing 30-minutes exercise on a cycloergometer, each at an individually matched load, adequate to 70% of  $V_{\rm O2\ max}$ , at frequency of 60 per minute.

The physiological and biochemical tests were made twice – at the beginning (I term) and 4 weeks later (II term). The tests encompassed the measure of anthropometrical indices (BMI, WHR, body mass, height). The body content was determined (fatty mass – FM, fatty – free mass – FFM, total body water – TBW) by using the electrical bioimpedance method (Body Impedance Analyser – Akern, BIA-101), the results were calculated as the absolute values of mass (kg). The maximal oxygen absorption ( $V_{02\,max}$ ) was measured with an indirect method, by using the Ästrand-Rhyming nomogram [Ästrand and Rhyming 1954].

The parameters determined in a fraction of the venous blood serum, taken in the morning, using the tests by Cormay: total cholesterol, HDL-cholesterol fraction, triacy-loglycerol concentration (TG), glucose concentration. Insulin concentration, measured by radioimmunological method, using special sets produced by the Research and Development Radioisotope Center in Świerk, Poland. The concentration of LDL-cholesterol was calculated from the Friedewald's formula [Bobilewicz 1961]: LDL-cholesterol = total cholesterol – HDL-cholesterol – (TG/5).

The young boys were qualified to participate in the research by a doctor. The study was conducted with the consent of subjects and their parents as well as in agreement with Local Committee of Ethics in Scientific Research of the Karol Marcinkowski University School of Medical Sciences in Poznań, Poland.

#### STATIC ANALYSIS

The results of the study were prepared using the program STATISTICA (StatSoft). The Wilcoxon signed rank test was applied to make the comparison of the groups.

#### RESULTS AND DISCUSSION

The time and effect of body mass reduction among subjects with overweight and obesity depend on their energy balance. The augmentation of the energy expenditure during the process of reduction of the body mass is associated with more intense conversion of the nutritional substrates stored in adipose tissue, what is the effect of physical activity or smaller daily ration, or both. Therefore, the most important thing in the process of reduction of the body mass is still the appropriate daily ration and modification of the lifestyle.

The most successful results can be obtained by mixing the low-calorie diet and physical activity [Miller et al. 1990, Nazar and Kaciuba-Uściłko 1995, Kasprzak et al. 1995]. We can observe that obese people at all ages are less physically active and lead more sedentary lifestyle [Hardman 1999, Westerterp 1999] what mainly influences the increase of body mass. The method heading up to reduce body mass applied in this study relies on low-calorie diet with lower content of fatty acids (about 1300 kcal) and systematic, moderate aerobic physical activity. The result of this method is statistically important and significant decrease of body mass from  $107.7 \pm 21.43$  at the beginning to  $98.9 \pm 19.62$  at the end of the treatment (p  $\leq 0.01$ ). The reduction of the body mass of 8.8 kg was the effect of the decrease of quantity of adipose tissue (average 7.6 kg) that influenced also beneficial changes in visceral adipose tissue, proved by decrease of

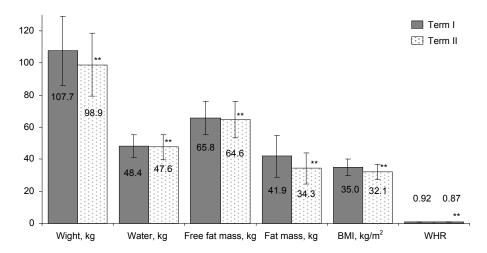


Fig. 1. Weight and body composition and BMI and WHR boys undergoing weight reduction in both periods of research at rest

WHR coefficient and also it minimizes the risk of threat to one's health (Fig. 1) [Cybulska and Szostak 1995, Gibney 1999].

Though, it is hard to predict, especially in further future, whether the benefits of the applied therapeutic procedures will be permanent. As it is written in the contemporary literature reports and from the authors' research, the "yo-yo" effect may be the consequence of rapid changes in body mass – alternately losing and gaining weight. Rapid filling and draining of adipose cells stimulate the progression of new adipose cells and as the result – increase of body mass [Niemiec et al. 2001].

Although, the possibility of metabolic disorders among young people with obesity is lower than among adults, it is necessary to reduce body mass concerning the potential metabolic complications in the grownup life.

Regular physical activity can evoke beneficial changes not only in terms of weight and body components, but can also lead to improve quality of life associated with better physical efficiency [Gutin et al. 1999]. The study reveals that systematic physical activity improves one's physical efficiency which is reflected in the increase of the relative

Table 2	Magn (ICD)	\ aamaantrations	aftha h	lood comme	limid and l	inammataina
rable 2.	Mean (±SD	concentrations	or the b	noou serum	npia ana i	ipopiotems

Parameter mg/dl	Term I $\overline{x} \pm SD$	Term II $\overline{x} \pm SD$
Total cholesterol	172.5 ±17.72	168.31 ±14.79*
LDL-cholesterol	$116.14 \pm 20.70$	$103.13 \pm 16.58*$
HDL-cholesterol	$51.90 \pm 16.12$	57.14 ±16.59*
Triglycerides	$79.02 \pm 16.54$	75.03 ±14.83

<sup>\*</sup>  $p \le 0.01$ .

coefficient of the maximal oxygen absorption and the rate of load. The coefficient  $V_{O2\ max}$  depends on the efficiency of the circulatory and respiratory systems and the metabolic processes occurring in the working muscle cells (Table 3).

	Table 3.	Biochemical	and	physiologic	al parameters
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Parameter	Term I $\overline{x} \pm SD$	Term II $\overline{x} \pm SD$
Insulin, μIU/ml	11.33 ±8.20	9.37 ±4.17*
Glucose, mg/dl	$85.40 \pm 11.07$	$80.55 \pm 6.95$
VO <sub>2</sub> max, ml/kg <sup>-1</sup> /min <sup>-1</sup>	$28.54 \pm 4.99$	32.81 ±6.25*
Load, W	129.43 ±35.27	142.86 ±36.85*

<sup>\*</sup>  $p \le 0.01$ .

It is proved that the accumulation of adipose tissue around the visceral organs induces the evolution of insulin resistance and hyperlipidemia [Ferrannini and Camastra 1998]. These processes are a consequence of specific metabolic properties of adipose tissue. The rising amount of abdominal adipose tissue causes not only an increase of insulin secretion from pancreatic beta cells, but also diminish the hepatic clearance [Peiris 1989].

Finally appear consequences – the disorder of homeostasis of the axis of glucose – insulin and the appearance of glucose intolerance, hyperinsulinemia, insulin resistance and dyslipidemia [Bray 1995]. The major metabolic outcome of insulin resistance is hyperglycemia, what is the result of hepatic glucose production and its reduced transport to the destined tissues.

The disorder in glucose – insulin homeostasis is caused by small number of membrane insulin receptors of the visceral adipose tissue [Taylor et al. 1984]. Due to this small number insulin receptors, adipose tissue is less sensitive to insulin antylipolytic action. Free fatty acids released in the process of lipolysis diffuse into the blood and thence to the liver. The increase of free fatty acids concentration in plasma influences pancreatic beta cell dysfunction. Visceral adipose tissue, therefore, is considered the main factor responsible for insulin resistance and development of diabetes type II [Ruan and Lodish 2003].

Obesity diagnosed at the time of adolescence is 3 to 4 times more dangerous because of higher risk of insulin dependent diabetes mellitus and coronary heart disease in mature life [Rybakowa 1993]. Hyperinsulinemia together with obesity lead to reduce the number of insulin receptors, mainly in muscle and lipid cells, both at young and adult subjects, furthermore, the parameters of visceral tissue of adolescents are correlated with some parameters of insulin resistance [Reaven 1988].

Physical activity is one of the main factors aimed to reduce hyperinsulinemia and insulin resistance [Rychlewski et al. 1997, Szcześniak et al. 1997]. It is known that body mass reduction evokes increase of insulin receptor affinity while reducing its secretion by pancreatic beta cells. This beneficial effect of physical activity to reduce hyperinsulinemia and insulin resistance is the result of insulin-independent glucose transport into muscle tissue, and suppression of its secretion by the increase of concentration of cate-

cholamines. Muscle tissue is responsible for 80-90% of insulin-stimulated glucose transport [DeFronzo et al. 1985]. Physical activity also increases the utilization of lipid energy sources and hence may induce body mass loss. The inhibitory effect of physical effort on insulin secretion showed Ferguson et al. [1999]. It's also confirmed by the presented study. Although the average insulin concentrations measured in rest were ranged in reference, in the second period of study they have declined, though on a statistically significant level (Table 3).

Obesity is a multifactorial disorder, and not every person with excessive adipose tissue is characterised by equally similar risk of lipid and carbohydrate disturbances. Severity of these disorders depends mainly on the distribution of body fat [Björntrop 1996], and its recognised regulatory factors are low-calorie diet and/or increased physical activity. Although the low-energy diet does not always provide a positive effect in terms of body mass reduction, due to the adaptive mechanism which reduces basic metabolism, but this type of diet usually leads to a reduction of cholesterol in blood due to its lower synthesis in liver cells and the lower consumption [Szczeklik-Kumala et al. 2002]. In addition, low-energy diet, it is usually rich in fiber diet based on low-calorie vegetables and fruits. Fiber, as shown by Rimma et al. [1996], has a direct impact on both, reducing total cholesterol and its fractions of low-density cholesterol (LDL--cholesterol). Soluble fiber fraction, ie, some gums and pectin presents hipolipemic action. Soluble fraction, by binding bile acids in the gastrointestinal tract, increases the excretion and thus contribute to lowering cholesterol in blood serum. Important role in reducing cholesterol levels play a beta-glucans. As substances hydrophile, they increase the viscosity of the contents of food in the intestine, thus impeding the absorption of lipids. By contrast tocotriene, especially D-tocotrianol by inhibiting action of HMG--CoA reductase (reductase 3-hydroxy-3-methyl-glutarolo-coenzyme A), reduces synthesis of cholesterol in the liver [Cybulska 2000].

Beneficial effects of dietary modifications, consisting of reducing consumption of saturated fatty acids and increase the consumption of unsaturated fatty acids in relation to lipid parameters, has been demonstrated in prevention programs conducted in Norway and Finland [Pietinen 1996]. The results of a prospective epidemiological 'Seven Countries' Study also demonstrated and revealed a close positive correlation between coronary heart disease and consumption of saturated fatty acids and cholesterol in the blood [Kromhout et al. 1995]. The research carried out by Levini et al. [1995] suggests that lowering total cholesterol in the blood by 1% reduces the risk of incidence of ischemic heart disease by 2%.

This study contributed to, significant at 1% level of confidence, lowering total cholesterol, LDL-cholesterol and increase HDL-cholesterol in blood serum (Table 2). HDL-cholesterol is known as "good cholesterol" that prevents oxidative modification of low density lipoprotein [Hasselwander 1999], due to the presence of paraoxonase (PON), an enzyme inhibiting lipid oxidation. Oxidative modification of LDL-cholesterol plays an essential role in the development of atherosclerosis [Inoue et al. 2001, Steinberg 1997], which is mainly due to its accumulation in macrophages, which leads to the transformation into foamy cells.

Antiatherosclerotic effect of HDL-cholesterol is not limited to the return transport of free cholesterol. Experimental results show that it has protective effects on endothelial cells, inhibits the activation and adhesion of leukocytes to the endothelium, affects blood coagulation and fibrinolysis [Hasselwander et al. 1999, Iskra and Pioruńska-Stolzman 2001].

It is known that TG may be an independent risk factor for coronary heart disease (CAD), not less important than hypercholesterolemia [Jeppesen et al. 1998]. Research of Hokanson and Austin [1996] conducted among women have shown that with increasing TG levels by 1 mmol risk of CAD increased by 32%. Attrogenne effect of elevated levels of TG may be manifested on the one hand in the form of proatherosclerotic changes in the lipoproteins fractions, on the other, induction proclotting threats. Mean measures of triglycerides, obtained in the studies, ranged reference values. Lowering the concentration of this parameter in the second period of study, although statistically insignificant, gives evidence of beneficial effects of therapy applied.

Glucose metabolism in the human body is strictly regulated both by the same blood glucose and hormones involved in glukostase, mainly by insulin. On the one hand, it stimulates the transport of glucose and other sugars on the spatial structure similar to glucose, on the other hand inhibits the process of synthesis of glucose through gluconeogenesis in the liver [Hughes et al. 1993].

The transport of glucose in the tissues takes place on two ways - dependent and independent of insulin. The first takes place in tissues insulin sensitive - in skeletal muscle, liver and adipose tissue and predominates when the concentration of insulin in the blood is high, such as after a meal. The second road transport takes place in tissues susceptible to the action of the hormone – brain, red blood cells, but also in those tissues sensitive to insulin. Both transport routes are functionally independent and governed by independent mechanisms [Baron et al. 1987]. The mechanism of insulin effect on glucose transport is very complex and not explained in every detail. A significant factor in increasing glucose transport into muscle is also a physical effort, and effort-induced increase in transport is proportional to the intensity of the effort [Katz et al. 1986]. In these studies statistically significant reduction was observed in serum glucose, indicating that physical activity positively affects the distribution of glucose (Table 3).

## CONCLUSIONS

Research results obtained allow to formulate the following conclusions:

- 1. The body mass loss, mainly by reducing the fat content points to the beneficial effect of the applied.
- 2. Dietary proceedings combined with physical activity resulted in positive lipidogramme changes in the blood and reduce the value of the ratio waist-hips what furthermore can decrease the risk of cardiovascular diseases in the future.
- 3. Reduction in serum insulin and glucose demonstrates improved carbohydrate metabolism and indicates a reduced risk for type II diabetes.

### REFERENCES

Ästrand P.O., Rhyming I., 1954. A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. J. Appl. Physiol. 9, 2-8.

Ball S., 2005. Zespół metaboliczny w otyłości i nadwadze [Metabolic syndrome in obesity and overweight]. Wyd. Medyk Warszawa [in Polish].

- Baron A., Wallace D., Brechtel G., 1987. In vivo regulation of non-insulin mediated and insulin mediated glucose uptake by cortisol. Diabets 36, 1230-1237.
- Björntorp P., 1996. The regulation of adipose tissue distribution in humans. Int. J. Obes. Relat. Metab. Dis. 20, 291-302.
- Bobilewicz D., 1961. Metody rozdziału frakcji lipoproteinowych [Methods separation of lipoprotein faction]. Cormay-Diagnostyka 1, 4-5 [in Polish].
- Bray G.A., 1995. Life insurance and overweight. Obes. Res. 3, 97-99.
- Cybulska B., 2000. Aterogenna dyslipidemia [Aterogenic dyslipidemia]. Medycyna po dyplomie 3-4, 39-43 [in Polish].
- Cybulska B., Szostak W., 1995. Otyłość wisceralna jako czynnik zagrożenia chorobą niedokrwienną serca [Visceral obesity as the factor of the Cardiovascular disease risk]. Pol. Tyg. Lek. Supl. 1, 43-47 [in Polish].
- DeFronzo R., Gunnarsson R., Bjorkman O., Olsson M., Wahren J., 1985. Effect of insulin on peripherial and splanchnic glucose metabolism in non-insulin-dependent (type II) diabetes mellitus. J. Clin. Invest. 76, 149-155.
- Ferguson M., Gutin B., Le N., Karp W., Litaker M., Humphries M., Okuyama T., Riggs S., Owens S., 1999. Effect of exercise training and its cessation on components of the insulin resistance syndrome in obese children. Int. J. Obes. Relat. Metab. Dis. 23, 889-895.
- Ferrannini E., Camastra S., 1998. Relationship between impaired glucose tolerance, non-insulin dependent diabetes mellitus and obesity. Eur. J. Clin. Invest. 28 (suppl 2), 3-7.
- Gibney M., 1999. Nutrition, physical activity and health status in Europe: an overview. Publ. Health Nutr. 2, 329-333.
- Goran M., Ball G., Cruz M., 2003. Obesity and risk of type 2diabetes and cardiovascular disease in children and adolescents. J. Clin. Endocrinol. Metab. 88, 417-1427.
- Gutin B., Owens S., Okuyama T., Riggs S., Ferguson N., Litaker M., 1999. Effect of physical training and its cessation upon percent fat and bone density of obese children. Obes. Res. 7, 208-214.
- Hardman A., 1999. Physical activity, obesity and blood lipids. Int. Obes. Relat. Metab. Dis. 23 (suppl 3), 64-71.
- Harris T., Gook E., Garrison R., Higgins M., Kannel W., Goldman L., 1988. Body mass index and mortality among nonsmoking older persons. The Framingham Heart Study. JAMA 259, 1520-1524.
- Hasselwander O., McEneny J., Mcmaster D., Fogarty D., Nicholls D., Maxwell A., Young I., 1999. HDL composition and HDL antioxidant capacity in patients on regular haemodialysis. Atherosclerosis 143, 125-133.
- Hokanson J., Austin M., 1996. Plasma triglyceride level is a risk factor for cardiovascular disease independent of high-density lipoprotein cholesterol level: a meta-analysis of population-based prospective studies. J. Cardiovasc. Risk 3, 213-229.
- Hughes V., Fiatarone M., Fielding R., Kaahn B., 1993. Exercis increase muscle GLUT-4 levels and insulin action in subjects with impaired glucose tolerance. Am. J. Physiol. 264, E855--E862.
- Inoue T., Uchida T., Kamishirado H., Takayanagi K., Hayashi T., Morooka S., 2001. Clinical significance of antibody against oxidized low density lipoprotein in patients with atherosclerotic coronary artery disease. J. Am. Coll. Cardiol. 37, 555-559.
- Iskra M., Pioruńska-Stolzman M., 2001. Przeciwutleniające właściwości lipoprotein o wysokiej gęstości i ich zmiana w reakcji ostrej fazy [Antioxidant properties of lipoprotein the high density and their change in reaction of the acute phase]. Czyn. Ryz. 3-4, 11-18 [in Polish].
- Jeppesen J., Hein H., Suadicani P., Gyntelberg F., 1998. Triglycerides concentration and ischemic heart disease. An eight-year follow up in the Copenhagen Male Study. Circulation 97, 1029--1036.
- Kasprzak Z., Szczęśniak Ł., Rychlewski T., 1995. Wpływ systematycznego wysiłku fizycznego oraz niskoenergetycznej diety na zachowanie się parametrów antropometrycznych i fizjologicznych u dzieci z otyłością prostą [Influence of a systematic physical exercise and low-

- energy diet on behaving of anthropometric and physiological parameters at children with the simple obesity]. Diabetol. Pol. 2, 42-48 [in Polish].
- Katz A., Broberg S., Sahlin K., Wahern J., 1986. Leg glucose uptake during maximal dynamic exercise in humans. Am. J. Physiol. 251, E65-E70.
- Kromhout D., Menotti A., Bloemberg B., Aravanis C., 1995. Dietary saturated and trans fatty acids and cholesterol and 25-years mortality from coronary heart disease: the Seven Country Study. Prev. Med. 24, 308-315.
- Lenart-Domka E., Kwolek A., 2007. Rehabilitacja dzieci otylych czy jest rzeczywiście potrzebna? [Rehabilitation of obese children – whether is really needed]. Przegl. Med. Uniw. Rzesz., 99-105 [in Polish].
- Levini G., Keaney J., Vita J., 1995. Cholesterol reduction in cardiovascular disease: clinical benefis and possible mechanism. N. Engl. J. Med. 332(8), 512-521.
- Miller W., Lindemann A., Wallace J., Niederpruem M., 1990. Diet composition, energy intake and exercise in relation to body fat in man and women. Am. J. Clin. Nutr. 52, 426-430.
- Nazar K., Kaciuba-Uściłko H., 1995. Aktywność ruchowa w zapobieganiu i leczeniu otyłości [Physical exercise in prevention and treatment obesity]. Pol. Tyg. Lek. 50, Supl. 1, 68-69 [in Polish].
- Niemiec A., Franek E., Kokot F., 2001. Wpływ odchudzania na oś leptyna-neuropeptyd Y u osób otylych z cukrzyca i bez cukrzycy [Influence of reduction of body mass on the axis leptin--neuropeptide Y at obese persons with diabetes and without diabetes]. Med. Metabol. 1, 18-26 [in Polish].
- Peiris A., Sothmann M., Hennes M., Lee M., Wilson C., Gustafson A., Kissebah A., 1989. Relative contribution of obesity and body fat distribution to alterations in glucose insulin homeostasis: predictive values of selected indices in premenopausal women. Am. J. Clin. Nutr. 49, 758-760.
- Pietinen P., Vartiainen E., Seppanen R., 1996. Changes in diet in Finland from 1972 to 1992: impact of coronary heart disease risk. Prev. Med. 25, 243-250.
- Reaven G., 1988. Role of insulin resistance in human disease. Diabetes 37, 1595-1607.
- Rimm E., Ascherio A., Giovannucci E., Spiegelman D., Stampfer M., Willett W., 1996. Vegetable, fruit, and cereal fiber intake and risk of coronary heart disease among men. JAMA 275, 447-541.
- Ruan H., Lodish H., 2003. Insulin resistance in adipose tissue: direct and indirect effects of tumor necrosis factor-alpha. Cytokine Growth Fact. Rev. 14, 447-455.
- Rybakowa M., 1993. Zaburzenia w odżywianiu. Otyłość [Disorders of feeding. Obesity]. In: Zaburzenia hormonalne u dzieci i młodzieży. Ed. T.E. Romer. Omnitech Press Warszawa 353-355 [in Polish].
- Rychlewski T., Szczęśniak Ł., Dylewicz P., Deskur E., Przywarska I., Kasprzak Z., Karolkiewicz J., Konys L., 1997. The influence of oral glucose intake on binding and degradation of <sup>125</sup>I--insulin by receptors on erythrocytes as well as on insulin and C-peptide serum levels in patients after myocardial infarction and healthy individuals. J. Physiol. Pharmacol. 48, 4, 839-
- Steinberg D., 1997. Low density lipoprotein oxidation and its pathological significance. J. Biol. Chem. 272, 20963-20966.
- Szczeklik-Kumala Z., Czech A., Łaz R., Jagielinska-Kalinowska E., Bernas M., Tatoń J., 2002. Epidemiologia aterogennych zaburzeń składu lipidów krwi w zdefiniowanej populacji [Epidemiology of aterogenic disorders of the composition of lipides in blood of the defined population]. Med. Metabol. 6, 1, 12-20 [in Polish].
- Szcześniak L., Rychlewski T., Kasprzak Z., Banaszak F., 1997. Insulinemia and insulin resistance in obesity-the influence of systematic physical effort. Ann. Diagn. Pediatr. Pathol. 1 (3), 220.
- Taylor R., Husband D., Marshal S., et al., 1984. Adipocyte insulin binding and insulin sensivity in "britte" diabetes. Diabetol. 27, 441-446.
- Westerterp K., 1999. Obesity and physical activity. Int. Obes. Relat Metab. Disord. 23 (suppl. 1), 59-64.

# WPŁYW DIETY I AKTYWNOŚCI FIZYCZNEJ NA POPRAWĘ PARAMETRÓW BIOCHEMICZNYCH I FIZJOLOGICZNYCH U OTYŁYCH CHŁOPCÓW

**Wstęp.** Główną przyczyną nadmiernego odkładania się tkanki tłuszczowej są zburzenia mechanizmów kontrolujących wydatkowanie energii. Patologiczne zwiększenie tkanki tłuszczowej prowadzi do upośledzenia czynności organizmu, zaburzenia gospodarki lipidowo-węglowodanowej, sprzyja rozwojowi chorób naczyniowych oraz zwiększa ryzyko chorobowości i śmiertelności. Celem pracy jest wykazanie wpływu diety i aktywności fizycznej na zmiany parametrów lipidowo-węglowodanowych u młodzieży.

**Materiał i metody.** Badaniami objęto chłopców z otyłością (n = 35), poddanych redukcji masy ciała. Zastosowano niskoenergetyczną dietę i systematyczny wysiłek fizyczny. Na początku i po czterech tygodniach wykonano pomiary wskaźników antropometrycznych oraz składu ciała. W krwi żylnej oznaczono stężenie cholesterolu całkowitego, frakcji HDL-cholesterolu, triacylogliceroli (TG), glukozy oraz insuliny. Wyliczono stężenie LDL-cholesterolu.

**Wyniki.** Stwierdzono, że zastosowane postępowanie terapeutyczne przyczyniło się do poprawy profilu lipidowego krwi. Tylko w przypadku trójglicerydów zmiana nie była istotna statystycznie. Stężenie glukozy zmniejszyło się istotnie statystycznie.

Wnioski. Redukcja masy ciała wpłynęła na pozytywne zmiany w lipidogramie krwi oraz zmniejszenie wartości ilorazu talia biodra, co może zmniejszyć ryzyko wystąpienia chorób sercowo-naczyniowych w przyszłości. Obniżenie stężenia insuliny i glukozy świadczy o poprawie metabolizmu węglowodanów i wskazuje na zmniejszenie ryzyka wystąpienia cukrzycy typu II.

Słowa kluczowe: otyłość, dieta, wysiłek fizyczny, lipidogram krwi, insulina

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