RELATION BETWEEN HUMAN PLASMA CONCENTRATION OF LEPTIN AND ADIPOSIS

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Leptin is a protein produced by matured, full fat cells which considerably influence the energy metabolism of the body cells and has probably one of the decisive functions in reduction of body weight. The leptin concentration in serum can be considered as an indicator of the amount of fatty tissue in the cells.

The aim of this study was to find and verify anthropological, age and functional relations to the leptin serum level in a wide population. A complex of 139 probands (sportsmen, sedentary healthy individuals and diabetics) of age from 18 to 82 years (56 men and 83 women) was examined. The recorded and statistically processed values of serum leptin, BMI, body fat (%) and cardiovascular efficiency (indices of CHR-test) were used for calculation of mutual relations to these parameters. Leptin correlated positively with all anthropological indicators and on the contrary, a relation was not established between serum leptin and the indices of cardiovascular system efficiency, both in men and women; in addition a positive relation was also established between leptin and age.

Physical activity together with diet belongs to the most efficient methods in a rational reduction of the superfluous amount of somatic fat. Controlling leptin can help in the identification of efficiency of this method. A return to optimal dynamics for leptin level based on behavioural interventions can in future become an indicator of the success of prevention or therapy of obesity.

Keywords: leptin, obesity, physical load, age.

INTRODUCTION

With the isolation of the obesity gene (Ob gene) in 1994, there was renewed interest in body weight regulation and pathophysiology of obesity. The human Ob gene is located on the 7th chromosome and its product – protein made up of 167 amino acids – was named leptin (from the Greek word "leptos" = thin – Hrnčiar, 1997).

Leptin is produced by mature, full fat cells and works as a signal informing the hypothalamic centres about the fatty–tissue quantity in the body (Hrnčiar, 1997; Lönnqvist et al., 1997; Maffei et al., 1995; McDougland et al., 1995; Saladin et al., 1995). The leptin concentration in serum can be considered as a marker of fatty-tissue quantity in the body (Haluzík et al., 1998; Stejskal et al., 1998a). Its synthesis is provable in brown and white fat (Drbalová et al., 1998).

The function of leptin is mainly body weight regulation. It achieves this by appetite reduction and intense energy disposal by the body. The ingestion center is inhibited, satiety is reached and the sympathetic system is stimulated (Maffei et al., 1995; McDougland et al., 1995; Saladin et al., 1995, 1996).

High serum leptin induces a receptor – or postreceptor resistance to this neurotransmitter which leads to the reduction in energy expenditure and a concurrent increase in appetite, followed by obesity (Hrnčiar, 1997; Muscelli et al., 1996; Stejskal et al., 1997).

Experimental studies proved the significant influence of leptin on the hematological, the immune, reproductive and the endocrine system and on the general progress and maturation of the individual (Drbalová et al., 1998; Haluzík et al., 1998; Hrnčiar, 1997; Malmstrom, 1996; Wabitsh et al., 1996; Stejskal et al., 1998a, 1998b, 1998c; Ryan et al., 1996).

The aim of this study was to verify, in particular, the age and anthropological relations to the leptin level in the plasma in a wide population pattern.

METHODOLOGY

There were 139 probands (56 of men, 83 of women) included in the study (53 university graduates, 27 patients of the exercise laboratory, and 59 patients of the metabolic and diabetological outdoor patients department). The subjects were fasting and the

examinations were carried out between 8 and 10 a. m. under standard conditions of the exercise laboratory.

The study comprised basic anthropological examination (weight, height, BMI, body fat % calculated from 10 skinfolds) and venous blood collection for serum leptin analysis (sandwich ELISA method of the Biovendor company with an accuracy of 0,1 ng/ml). After taking the blood samples, the patients of the exercise laboratory exercised on a bicycle ergometer (CHR-test according to Stejskal et Hejnová, 1994) for 30 minutes at 70 % of the maximum heart rate reserve.

The measured and calculated values were processed by using the programme Statgraphics version 5.0. Since the serum leptin values showed a lognormal pattern of distribution (the normal distribution was verified by the Kolmogorov – Smirnov test), follow-up correlations were made between the particular parameters and the simple logarithm of serum leptin values.

RESULTS

The examined complex consisted of probands of age between 18 and 82 years (the men and women groups did not vary significantly), the BMI between 17 kg/m^2 and 39 kg/m^2 and body fat from 2,7 % to 56,1 %. The average of serum leptin was 8, 1 ng/ml (minimum 0, 2 ng/ml, maximum 85,7 ng/ml). In women, the leptin levels and body fat values were significantly higher as compared to men ($p < 0,01$, resp. $p < 0, 05$) (TABLE 1, 2).

Those with BMI < 25 kg/m^2 had an average leptin level of 4, 3 ng/ml (women 5, 4 ng/ml, men 1, 9 ng/ml), in obese individuals (BMI > 30) 19,2 ng/ml (women 24,5 ng/ml, men 14,8 ng/ml) (Fig. 1).

Serum leptin was found to be significantly related to the body fat (correlation coefficient $r = 0,58$), BMI ($r = 0,44$), and age ($r = 0,46$). Relations between body fat, BMI and serum leptin were, according to correlation coefficients, closer in women than in men (TABLE 3).

We found out by partial correlation analysis that the body fat had a 22 % share in variations of the serum leptin values while BMI, age and sex did not significantly participate in serum leptin variations (Fig. 2).

The values of serum leptin and body fat were significantly lower in physically active persons than in sedentary individuals (patients of the exercise laboratory and metabolic out-patient department), in men and women alike (TABLE 2).

After dividing the examined complex into age subgroups, we ascertained that leptin levels increased with age until 70; likewise all other indices characterising obesity significantly increased with age (Fig. 3). Serum leptin did not increase with age after a statistical adjustment of leptin to the body fat (Fig. 4).

Relations between indices of cardiovascular system efficiency and leptin levels were not statistically significant, both in men and women.

DISCUSSION

The function of leptin is body weight regulation based on the principle of stimulation of the sympathetic system (increase in metabolic rate) and inhibition of the satiety center in the hypothalamus. The exact mechanism of this inhibition is not completely known. The leptin receptor gene probably induces more forms of receptors (for example 5 types of receptors are known in humans so far). Type A is located in plexus chorioideus and is responsible for picking up a circulating leptin and its transport through the hematoencephalic barrier. B-type receptors, located right in the hypothalamus, transferring the leptin influence to the hypothalamic nuclei. Here leptin inhibits expression of mRNA for neuropeptide Y, which positively influences appetite and lowers energy expenditure. Leptin level, both positively and negatively, influences the expression of other hypothalamic peptides, namely galanine, propio-melanocortine, neurotensine, melanocytes stimulating hormone, corticotropin – trigger hormone and cholecystokinin, which through the modulation of neuroendocrine activity participate in the regulation of ingestion. Leptin can directly influence even the insulin receptors by reducing their sensitivity and thus glucose utilization (Guan et al., 1998; Drbalová et al., 1998).

While searching for relations between leptin concentration and anthropological indicators we found a significant relation between body fat and leptin values. In terms of nature of sex, it was found out that in women this relation was closer. In majority of published studies the positive relation between somatic fat percentage and leptin plasma concentration, which was significantly higher in women than in men of the same weight, was proven; it is caused both by higher relative proportion of fat in women than in men and by difference in sex hormones (Haluzík et al., 1998; Hrnčiar J., 1997; Stejskal et al., 1997; Muscelli et al., 1996; Pasman et al., 1998; Ronnemaa et al., 1997; Vettor et al., 1997). The average values of serum leptin obtained by the same biochemical method vary in men of normal body weight by 1 to 11 ng/ml, in women these values are 3–4 times higher (Stejskal et al., 1997).

Serum leptin level was significantly lower in individuals of optimal weight than in the obese. In the latter, deficiency of leptin is not usually shown but surprisingly its abundance is. That is why a receptor resistance in obese individuals to the endogenous leptin influence is considered a fact. Explanation of this resistance is based on the fact, that leptin is transported through the hematoencephalic barrier by a saturable transport system. After reaching a certain serous concentration of leptin this transport to the central nervous system stops (Schwartz et al., 1996). This saturation can be the cause of development of leptin resistance and the following hyperphagia in obese individuals with high serous concentrations of leptin.

The levels of leptin in the neural tissues of obese people are about 30 % higher than non-obese, in serum up to 300 %. In individuals of a normal constitution the serous levels of leptin correlate with levels in cerebrospinal fluid (Drbalová et al., 1998).

It was found that together with age, indices characterizing obesity significantly increased; leptin level increased with age up to 70 years (Fig. 3). According to some of the authors, age does not correlate significantly with serum leptin, whereas according to the others it does (Hua-Li et al., 1997; Kamal, 1997; Matsuda et al., 1997; Ryan et al., 1996). Since leptin levels fluently increase from one's birth and keeps sex difference (Ryan et al., 1996), the possibility cannot be excluded that the age dependence of leptin is caused by an increasing quantity of the fatty tissue and advancing age. We confirmed this assumption by carrying out the statistical adjustment of leptin level to the quantity of fat tissue (Fig. 4).

In women, in the course of life, leptin values are considerably variable, probably in connection with the activity of sex hormones. A function of leptin as a puberty initiator can be considered (accumulation of sufficient fat reserves for the initiation of reproductive functions) (Drbalová et al., 1998).

There is not sufficient data dealing with the influence of physical activity on the serum leptin nowadays. In our study, we did not manage to confirm the relation between indices of cardiovascular efficiency and leptin level, both in men and women. Also the relation between cardiovascular system efficiency and body fat was not proven and that is why the leptin values and values of cardiovascular efficiency indicators did not correlate, nor after the statistical adjustment to the fatty tissue quantity. However, the leptin levels were lower in physically active persons in comparison with sedentary subjects. The latter also had significantly higher amount of body fat than those engaged in exercise or sports (TABLE 2).

Kohrt et al. (1996) studied the effects of a nine month exercise programme (walking, jogging, steps climbing) and hormonal substitutional therapy on the serum leptin level in older women. In persons who exercised, the changes of body fat significantly correlated with changes in the leptin serum concentration. The effect of exercise therefore seems to be dependent on changes of the amount of body fat. On the contrary, Pasman et al. (1998) described the lowering of leptin level independent of the body fat changes and also focused on changes of plasmatic insulin through long-term training for a period of 16 months (3–4 per week, 1 hour per day, medium intensity).

Hickey et al. (1997) followed a long-term effect of an aerobic exercise on leptin level. After a 12 week exercise (4 days per week, 30–40 minutes per day), the serum leptin concentration dropped significantly only in women which, in the first instance, had higher concentration of leptin than men in spite of lower quantity of fat. On the contrary, Pérusse et al. (1997) found out that a 20 week exercise regime for older men and women caused a reduction in the serum concentration of leptin, but only in men.

Hickey et al. (1996) dealt with the question of influence of a single exercise bout on the leptin level and the relation between serum leptin and body fat in case of relatively thin long-term trained sportsmen. In these trained individuals with a low leptin concentration no immediate effect on the circulating concentration of leptin was found after a 20 mile run (70 % VO2 max).

On the contrary Landt et al. (1997) described a significant decrease in leptin concentration during an extremely hard or long exercise which causes negative energy balance. After two hours of hard ride on the bicycle, the average leptin level dropped significantly by 8,3 % and at the same time, resulted in considerably increased levels of free fatty acids, which correlated negatively with the leptin decrease. After 6 hours of rest and taking food, leptin level returned to initial values.

Leal Cerro et al. (1998) focussed on a question whether leptin is regulated by an acute energy output. It was discovered that leptin levels dropped after finishing a marathon race parallelly with reduction of the body fat (the leptin level was reduced after an output of 2800 kcal). Hence it can be inferred that the leptin level can be regulated by a large energy output.

Racette et al. (1997) studied a leptin production in vivo in the abdominal fatty tissue, during the rest and after 60 minutes of exercise on the bicycle ergometer (50 % of maximal heart rate reserve). Blood samples were taken every 10 minutes during the exercise. The concentrations of plasma leptin did not change during the exercise and were identical to the values found during rest conditions.

Koistinen et al. (1998) described the influence of an exercise, both fasting and after taking food, upon the concentration of circulating leptin in healthy men and in diabetics type I with normal body weight and well compensated diabetes mellitus. During a 3 hour ride on the bicycle ergometer in fasting subjects the leptin level decreased by 42 % in healthy men and by 23 % in diabetics. If the exercise was carried out during postprandial conditions and related to a significant growth of serum level of cortisol, the leptin concentrations remained unchanged. These results show that although the circulating leptin can be reduced by exercise, this effect can be compensated with food or with enhancement of cortisol concentration in serum.

The influence of diet and exercise on the level of serum leptin and its relation to the metabolic syndrome X was, in case of postmenopausal women with excess weight, studied by Christensen et al. (1998). No influence on serum leptin in relation to diet was found. It seems that leptin is of no significance to the metabolic syndrome X but rather to the quantity of body fat.

However tests on rats show that the expression of leptin can be influenced by exercise and that these

changes (reduction of expression and secretion of protein) can become independent of changes in insulin sensitivity that leads to obesity (Zachwieja et al., 1997).

According to Tuominen et al. (1997) the serum leptin concentration drops during exercise induced expenditure of glycogen and increases during hyperinsulinemic clamp. Serum leptin level correlates positively with serum insulin, cortisol and triglycerides and inversely with concentration of growth hormone.

Lower concentrations of serum leptin were in general described in sportsmen in comparison with common medium values, namely in long-distance runners but also in rugby players. These low values correlated with the proportionally decreased amount of body fat (Haluzík et al., 1998; Hickey et al., 1996).

Despite differences in the results of the aforementioned experiments, it can be stated that both the single endurance intensive exercise bout and regular persistent training decrease the leptin level in serum. While in case of single exercise bout no leptin level reducing mechanism was definitely established, the drop of leptin level after long – term training and its lower level in sporting population alike are probably closely connected with the lower amount of body fat.

CONCLUSION

Serum leptin can be regarded as a very significant indicator of the quantity of body fat. Many studies show that the leptin level decrease surpasses considerably the drop in the BMI. It was even described that the leptin level decrease is accompanied by lowered reduction in body fat while BMI values remain unchanged. That is the reason why monitoring leptin level could be a more correct indicator of the body fat reserve than the mentioned anthropological indices (Haluzík et al., 1998; Stejskal et al., 1998a).

A long-term research is currently underway concerning changes of leptin level as a result of changes in behavioural habits within physical activity in relatively healthy individuals. Physical activity together with diet is among the most effective methods of rational reduction in the superfluous amount of somatic fats. The return of the optimal dynamics of leptin level based on behavioural interventions can in future become the aim and the indicator of success in prevention or therapy of obesity.

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TABLE 1
Average, standard deviation, minimum and maximum values of plasma leptin levels, BMI, % of body fat, and age.

(n)	**whole group (139)**				**men (56)**		**women (83)**	
	$\bar{x}$	**SD**	**min**	**max**	$\bar{x}$	**SD**	$\bar{x}$	**SD**
Age (years)	41,20	20,03	18	82	43,05	18,84	39,89	20,81
BMI (kg/m^2)	25,10	4,38	17,20	38,96	26,61	3,87	24,08	4,42
Fat (%)	25,50	13,14	2,70	56,07	22,46	12,00	27,59	13,53
Leptin (ng/ml)	8,08	11,58	0,20	85,70	4,97	11,69	10,18	11,10

TABLE 2
Average and standard deviation of serum leptin, age, weight, BMI and % of body fat in individual groups. Statistic significance of the differences between the groups.

$\overline{X}$ = average, **SD** = standard deviation, **S** = statistic significance of the difference, **ST** = students, **ELP** = exercise laboratory patients, **OPMD** = outdoor patients of metabolic department, **M** = men, **W** = women

A	ST-M (16)		ST-W (37)		ELP-M (21)		ELP-W (6)		OPMD-M (19)		OPMD-W (40)
	$\overline{X}$ (SD)	S	$\overline{X}$ (SD)	S	$\overline{X}$ (SD)	S	$\overline{X}$ (SD)	S	$\overline{X}$ (SD)	S	$\overline{X}$ (SD)
Age (years)	19,38 (1,36)	*NS **p<0,001 *** p<0,001 **** p<0,001 ***** p<0,001	19,11 (0,84)	° p<0,001 °° p<0,001 °°° p<0,001 °°°° p<0,001	45,71 (10,84)	♠ NS ♠♠ 0,01 ♠♠♠ 0,001	42,17 (7,52)	•0,01 ••0,001	56,78 (12,23)	+ NS	58,76 (11,25)
BMI (kg/m^2)	23,49 (1,99)	*p<0,001 **p<0,01 *** NS **** p<0,01 ***** p<0,001	20,72 (1,62)	° p<0,001 °° p<0,001 °°° p<0,001 °°°° p<0,001	26,74 (4,27)	♠ NS ♠♠ NS ♠♠♠ NS	26,08 (4,28)	• NS •• NS	28,19 (3,10)	+ NS	26,90 (4,11)
Fat (%)	10,26 (2,73)	*p<0,001 **p<0,001 ***p<0,001 **** p<0,01 ***** p<0,001	14,57 (4,26)	° p<0,001 °° p<0,001 °°° p<0,001 °°°° p<0,001	19,56 (5,78)	♠ NS ♠♠ p<0,001 ♠♠♠ p<0,001	24,3 (4,86)	•p<0,01 ••p<0,001	33,49 (8,40)	+ p<0,001	40,13 (6,46)
Leptin (ng/ml)	1,03 (0,50)	*p<0,001 **p<0,001 ***p<0,001 **** p<0,01 ***** p<0,001	4,41 (4,76)	° NS °° p<0,01 °°° NS °°°° p<0,001	4,21 (4,43)	♠ p<0,05 ♠♠ NS ♠♠♠ p<0,001	10,22 (6,45)	• NS •• NS	6,53 (16,37)	+ p<0,01	15,52 (13,11)

* = ST-M:ST-W, ** = ST-M:ELP-M, *** = ST-M: ELP-W, **** ST-M:OPMD-M, ***** = ST-M:OPMD-W, ° = ST-W:ELP-W, °° = ST-W:ELP-W, °°° = ST-W:OPMD-M, °°°° = ST-W:OPMD-W, ♠ = ELP-M:ELP-W, ♠♠ = ELP-M: OPMD-M, ♠♠♠ = ELP-M:OPMD-W
• = ELP-W:OPMD-M, •• = ELP-W:OPMD-W, + = OPMD-M:OPMD-W

TABLE 3
Correlations of the serum leptin values, anthropological indices and age
(significance ***p< 0.001, **p< 0.01, *p<0.05).

(n)	whole group (139)	men (56)	women (83)
Fat (%)	0,58***	0,47***	0,61***
BMI (kg/m^2)	0,44***	0,56***	0,65***
Age (years)	0,46***	0,56***	0,53***

Fig. 1
Comparison of the serum leptin levels in obese and non-obese subjects.

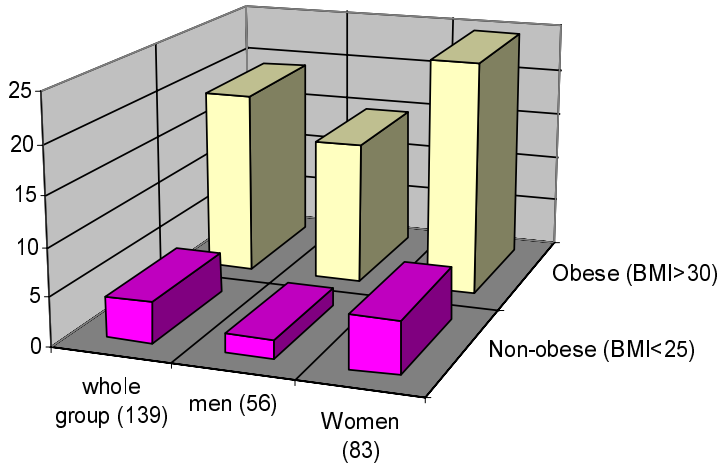


Fig. 2
Contribution of the separated parameters to the leptin level variations.

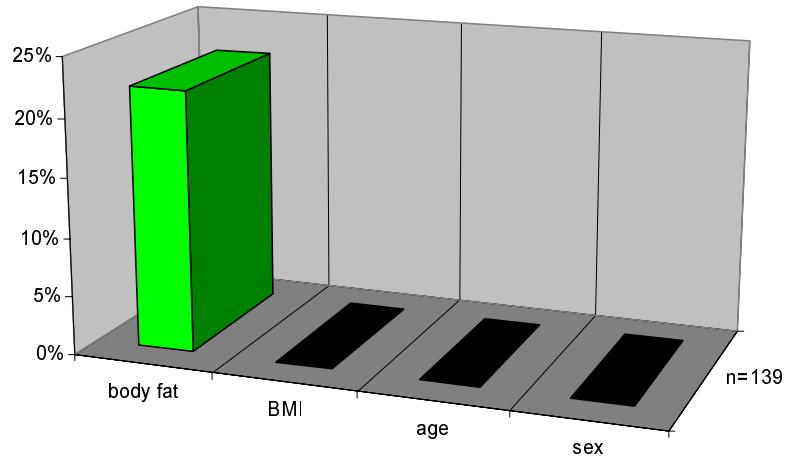


Fig. 3
Averages of the BMI, body fat, and serum leptin concentration in separated age categories.

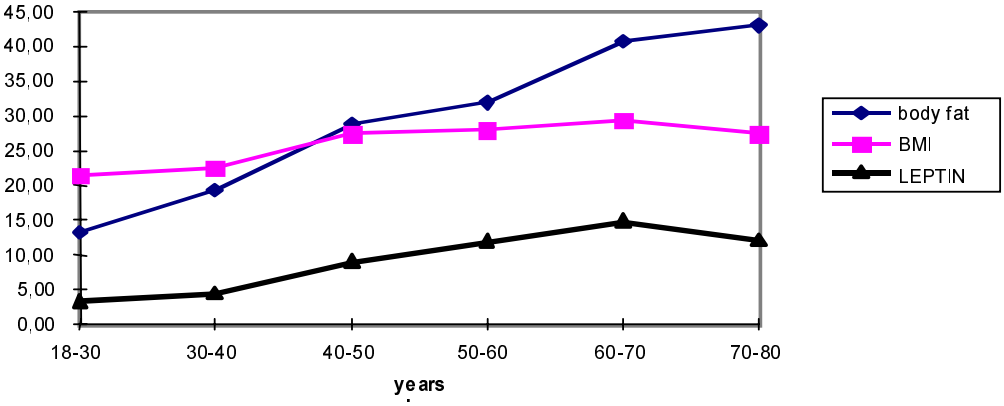
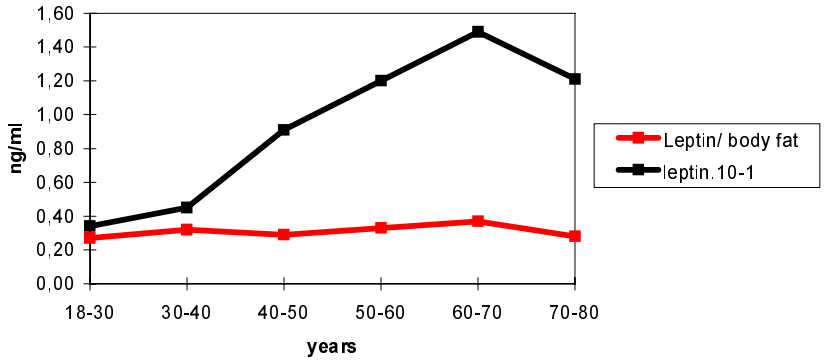


Fig. 4
Average values of the plasma leptin concentration and plasma leptin concentration to body fat ratio in the age groups.



ZUSAMMENHANG ZWISCHEN LEPTINKONZENTRATION IM SERUM UND FETTSUCHT BEI DEM MENSCH

(Zusammenfassung des englichen Textes)

Leptin ist ein von der ausgereiften Fettzellen produzierter Eiweißstoff, der auf grundsätzliche Weise den energetischen Stoffwechsel des Organismus beeinflußt. Die Leptinkonzentration im Serum ist für einen Anzeiger der Fettgewebemenge im Organismus zu halten.

Ziel der Studie war, bei einer breiten Populationsgruppe die anthropologischen, Alters- und funktionellen Zusammenhänge mit dem Leptinniveau im Blutwasser zu finden und zu überprüfen. Es waren 139 Probanden (Sportler, hypokinetisch gesunde Personen und Diabetiker) im Alter von 18–82 Jahre (davon 56 Männer und 83 Frauen) untersucht. Aus den gemessenen und statistisch bearbeiteten Werten der Leptinemie, BMI, relativer Vertretung der Fettkomponente (%) und der Kreislaufleistung (Anzeiger CHR-Test) wurden beiderseitige Zusammenhänge dieser Parameter berechnet. Leptin korrelierte positiv mit allen anthropologischen Anzeigern, und im Gegenteil wurde kein Zusammenhang zwischen Leptinemie und den Anzeigern der Leistungsfähigkeit des kardiovaskularen Systems bewiesen, sowohl bei Männern, als auch bei Frauen, bis auf den Zusammenhang zwischen der Kreislauleistungsfähigkeit und der Leptinemie; eine positive Beziehung wurde ebenfalls zwischen Leptinemie und Alter festgestellt.

Bewegung und Diät gehören zu den bedeutendsten Verfahren der rationalen Reduktion von überflüssiger Menge des körperlichen Fetts. Die Leptinemie-verfolgung kann der Erkenntnis der Wirksamkeit dieses Verfahrens beitragen. Rückkehr der optimalen Leptinemiedynamik aufgrund der behavorialen Interventionen kann künftig ein Anzeiger einer erfolgreichen Vorbeugung oder Fettsuchttherapie sein.

Schlüsselwörter: Leptin, Fettsucht, physische Belastung, Alter.

VZTAH MEZI PLAZMATICKOU KONCENTRACÍ LEPTINU A ADIPOZITOU U ČLOVĚKA

(Souhrn anglického textu)

Leptin je bílkovina produkovaná zralými tukovými buňkami, která ovlivňuje zásadním způsobem energetický metabolismus organismu a má pravděpodobně jednu z rozhodujících funkcí v redukci tělesné hmotnosti. Koncentraci leptinu v séru lze považovat za ukazatel množství tukové tkáně v organismu.

Cílem studie bylo nalézt a ověřit antropologické, věkové a funkční souvislosti s hladinou leptinu v krevní plazmě u široké populační skupiny. Byl vyšetřen soubor 139 probandů (sportovci, hypokinetičtí zdraví jedinci a diabetici) ve věku od 18 do 82 let (z toho 56 mužů a 83 žen). Z naměřených a statisticky zpracovaných hodnot leptinémie, BMI, relativní zastoupení tukové komponenty (%) a výkonnosti oběhu (ukazatele CHR-testu) byly vypočítány vzájemné souvislosti těchto parametrů. Leptin koreloval pozitivně se všemi antropologickými ukazateli a naopak souvislost nebyla prokázána mezi leptinémií a ukazateli výkonnosti kardiovaskulárního systému a to jak u mužů, tak i u žen, až na vztah mezi výkonností oběhu a leptinémií; pozitivní vztah byl zjištěn rovněž mezi leptinémií a věkem.

Pohybová aktivita spolu s dietními opatřeními patří mezi nejúčinnější metodu racionální redukce nadbytečného množství tělesných tuků. Sledování leptinémie může pomoci při poznání účinnosti této metody. Návrat optimální dynamiky leptinémie na základě behaviorálních intervencí může být v budoucnu ukazatelem úspěšnosti prevence nebo terapie obezity.

Klíčová slova: leptin, obezita, fyzická zátěž, věk.