UDC: 611.016.26:613.25

Obesity and serum adiponectin levels in different age groups

Bexheti- Hamzai E[1], Beadini N[1], Beadini Sh[1]

[1]University of Tetovo, Faculty of Medical Sciences, Department of Biomedicine, Republic of North Macedonia
E-mail of the corresponding author: ermira.bexheti@gmail.com

Abstract:
Obesity is a major public-health problem worldwide. It has been associated with the development of metabolic syndrome (MS) which is an interrelated cluster of risk factors for cardiovascular disease (CVD) and type 2 diabetes such as hyperglycemia, raised blood pressure, elevated triglyceride levels, low high-density lipoprotein cholesterol levels, and central obesity
The growing rates of this obesity-related syndrome have spurred the search for greater insight about mechanisms contributing to the development of MS, especially those reflecting a dysfunction of adipose tissue, which probably plays a major role in its development.
Adiponectin, a hormone expressed in adipose tissue and encoded by the ADIPOQ gene (chromosome 3q27), plays an important role in regulating insulin sensitivity, glucose and lipid metabolism besides anti-inflammatory and anti atherogenic properties. Its high molecular weight (HMW) isoform is the major responsible for these functions. In the presence of obesity, adiponectin release is down regulated resulting in reduced circulating levels.
Key words: Metabolic syndrome, Obesity, Adiponectin.

Introduction

If a person has more than three health risk factors, it is said to have metabolic syndrome. In simple clinical practice, this syndrome is diagnosed simply by measuring the waist circumference according to the values set by the World Health Organization. For women this value is over 85 cm and for men over 102 cm. "And if high values are found, then triglyceride, hyperglycemia, arterial hypertension, etc.," say the doctors. Metabolic syndrome has become a worldwide health problem due to the pandemic size it has taken. According to doctors, this syndrome is quite problematic because it causes a risk for cardiovascular disease and type 2 diabetes. There are still no accurate figures on this syndrome in our country; it is believed that there is a tendency to increase its prevalence, there is such a tendency to obesity and overweight. This is because the main component of this syndrome is obesity, the cause of which is the changing lifestyle and nutrition approaching the Western style. Prevalence also depends on age. With the aging of the population, prevalence increases more, but physicians draw attention to this as well as at childhood ages. Albania is still considered a relatively young population, but the tendency for aging population is expected to bring and tend to overall health. Taking measures to avoid this pathology should start from childhood in order to control obesity and overweight, which is a very worrying world problem. According to doctors, for the sake of metabolic syndrome, attention should be paid to improving our lifestyle in our country, which means that people are educated to change the way they eat and live. Also, doctors draw attention to increasing physical activity, limiting fats of animal origin, sugars, because it affects the prevalence of metabolic syndrome. On the other hand, since metabolic syndrome poses a threat to health, doctors have taken steps to recognize it and to prevent its risk factors. Self-syndrome is a pile of risk factors, which are arterial hypertension, high triglycerides, hyperglycaemia, central or abdominal retinopathy, which increases the perimeter of the waist. The prevalence of metabolic syndrome in the world accounts for up to 35%. In Europe, this syndrome is estimated at 25%, in Latin America at 24-25% and in Asia around 20%. Metabolic syndrome has been described more than a decade ago and is known as Riven Syndrome; syndromes "X"; cardio-metabolic syndrome; etc.

Metabolic syndrome is the disorder of receiving energy material (food) and depositing it in the body. The main defect in that process is when insulin is not used efficiently, as a result of increased fat in the abdomen.

At first, the body arrives with increased insulin secretion to maintain the level of sugar and fat in the normal bloodstream. But, over time, pancreas cells get tired and this leads to increased levels of sugar and fat in the bloodstream. This condition is toxic to the blood vessels and accelerates the process of atherosclerosis. If we say more simply, metabolic syndrome presents a set of risk factors for the occurrence of diabetes and cardiovascular disease.

A large amount of adiponectin flows with the blood stream inside of vascular walls. It would be interesting to know whether adiponectin can enter into vascular walls. Immunohistochemical examination using anti-adiponectin antibody demonstrated that there is no existence of adiponectin in the untreated normal vascular walls in rabbit. However, markedly positive immunohistochemical stain was detected in the balloon-injured vascular walls. Because adiponectin has been shown to have an ability to bind subendothelial collagen, such as collagen V, VIII, and X, endothelial injury may induce the entering of adiponectin into subendothelial space by binding to these collagens.

Materials and methods

In this research are involved 50 patients where was 30 females and 20 males. In this patients has determinate the level of adiponectin and BMI in different age groups.

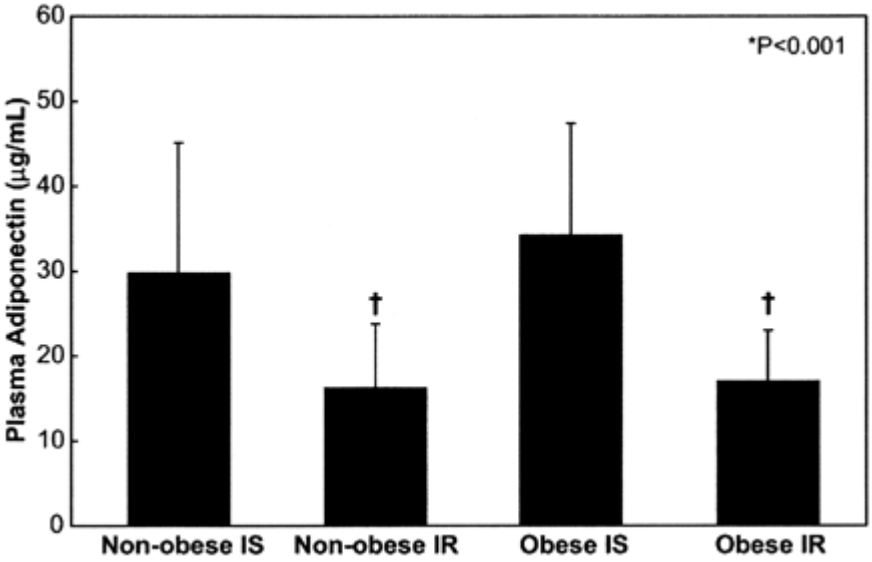
The research will be done with the colorimetric, spectrophotometric and fluorescent immunoassay method with the Vidas apparatus.

Tab.1. Determination of metabolic syndrome in male and female based on abdominal perimeter

Gender	**Metabolic syndrome based on abdominal perimeter**
Male	Over 102 cm
Female	Over 88 cm

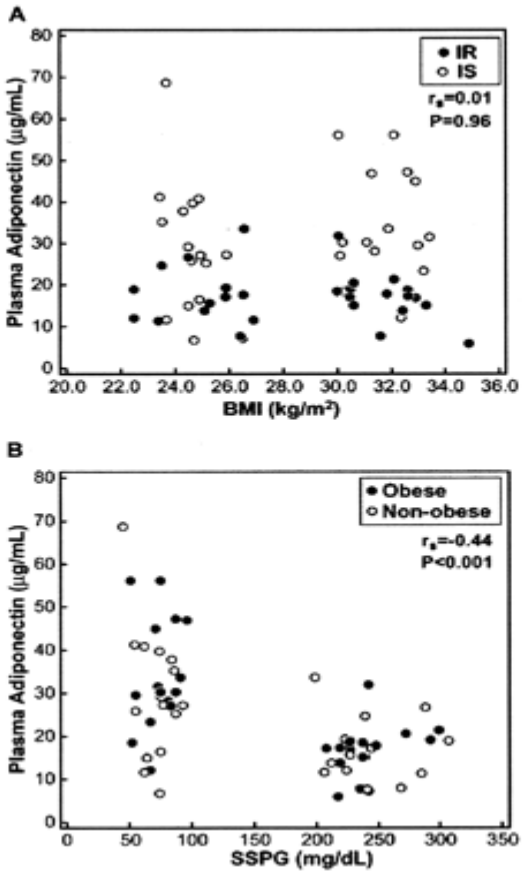
Results

From total 50 patients were determinate the plasma adiponectin in patients with metabolic syndrome.



Graph.1. Comparison of plasma adiponectin concentrations in the four experimental groups

Vertical bars represent the mean adiponectin concentrations, and error bars represent the SD. IS, insulin sensitive; IR, insulin resistant. *Mean adiponectin levels were not similar across the four groups as compared by one-way anova ($P < 0.001$); post hoc pairwise comparisons showed that adiponectin levels were significantly different ($P \leq 0.01$) between insulin-sensitive and insulin-resistant groups within each obesity category, whereas there were no significant differences ($P = 1.0$) between the nonobese and obese groups within each insulin action category

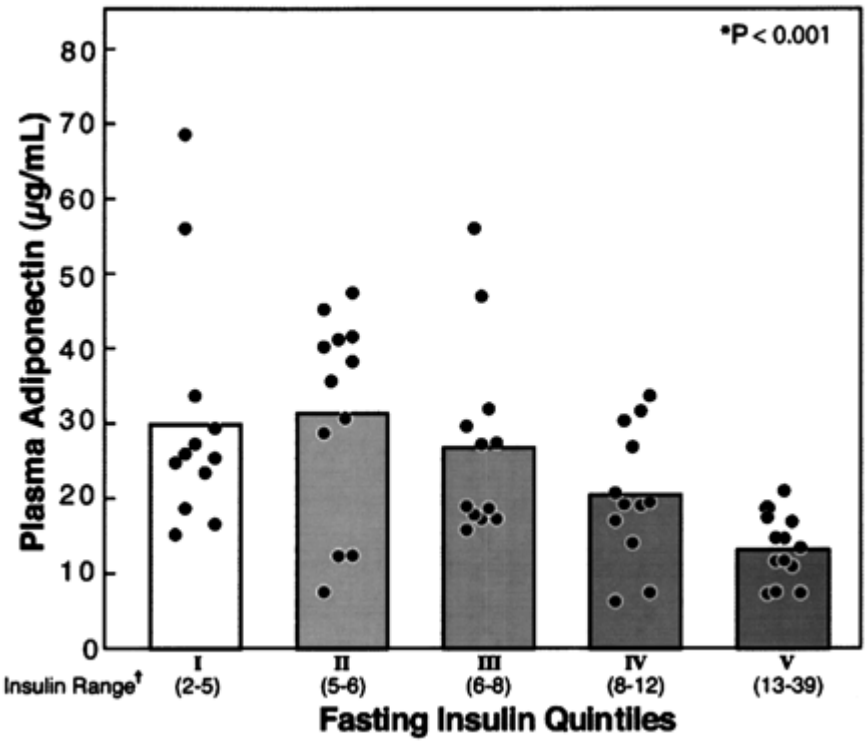


Graph.2. Prevalence of adiponectin in obese and non-obese patients

Relationship between plasma adiponectin concentrations, BMI, and SSPG concentrations in 60 nondiabetic volunteers.

A: The relationship between plasma adiponectin levels and BMI is shown, where subjects are identified by their insulin resistance status, insulin sensitive (IS, ○) and insulin resistant (IR,).

B: The relationship between plasma adiponectin levels and SSPG concentrations is shown, where subjects are identified by their obesity status, either nonobese. or obese. The Spearman correlation coefficient (rs) describes the strength of association between the graphed variables



Graph.3. Prevalence of adiponectin in correlation with fasting insulin

Relationship between fasting plasma adiponectin and quintiles of fasting insulin concentrations. Vertical bars represent the mean adiponectin concentration for each fasting insulin quintile. *Mean adiponectin levels were not similar across quintiles as compared by one-way anova (P = 0.001). Individual data points are also shown. Insulin concentrations are given in microunits per milliliter.

Discussion

The results show that exposure to 60 mg PEG-OB weekly for 8 weeks did not influence weight loss in obese subjects on a mildly hypoenergetic diet despite the high serum levels of PEG-OB achieved at the end of this study. Thus, augmentation of serum leptin concentration using a long-acting PEG-OB failed to promote additional weight loss over caloric restriction. The outcome of this study is, however, consistent with the alternative view of the physiological role of leptin proposed by the group of Flier.23 These investigators suggest that evolution would favour a leptin dose–response curve that functions briskly as a switch between the fed and fasted state, but would fail to limit further energy storage as levels rose with increased energy stores. The latter state could be described as ‘leptin resistance’. In addition, they speculate that the shape of this biological dose–response curve may depend on the conditions in which a certain species evolved. This hypothesis provides a possible explanation for the ineffectiveness of high-dose PEG-OB in our obese individuals on a mildly hypoenergetic diet as well for the fairly moderate results of the Heymsfield trial.5According to this view, PEG-OB might cause additional weight loss when administerzed during severe energy restriction or total leptin deficiency. The observation that recombinant human met-leptin treatment of a young hyperphagic very obese girl with a mutated *ob*gene resulted in weight loss by sustained reductions in appetite is consistent with this view. The fact that supraphysiological levels of PEG-OB cause weight loss in rodents, but not in humans suggests that the leptin dose–response curves of these species are different.

However, the possibility that the small number of subjects studied and the relative short duration of treatment might explain the lack of an effect of PEG-OB treatment on weight loss in this study cannot

be excluded .Also other biological effects of leptin in animals studies were not observed in this study. We failed to demonstrate any treatment effects on the levels of glucose, insulin (including estimated insulin resistance) and triglycerides. These results are supported by data obtained from the limited number of human patients with leptin deficiency or non-functional leptin receptors studied up to now who also lack substantial impairments in glucose homeostasis and lipids (unlike, respectively, the *ob*/*ob* and *db*/*db* mice) suggesting that leptin is not directly involved in the regulation of these systems in man. Our previous study suggested that PEG-OB treatment might have an additional effect on triglycerides in obese subjects consistent with similar changes repeatedly observed in animal studies. In the present study with a higher dose of PEG-OB no added effect on triglycerides was observed.

Conclusion

In conclusion, total and HMW adiponectin levels not only are lower in the presence of MS, but it also decreases by increasing number of MS criteria. These levels are partly determined by their relationship with HDL cholesterol, triglycerides and abdominal adiposity. Furthermore, chronic inflammation and insulin resistance may contribute to the decrease in adiponectin levels. Longitudinal data of prospective population based studies might be used to understand the role of adiponectin in the development of MS.

References

1. Abate, N, Garg, A, Peshock, RM, Adams-Huet, B & Grundy, SM (1996) Relationship of generalized and regional adiposity to insulin sensitivity in men with NIDDM. Diabetes 45, 1684–1693.CrossRef | Google Scholar | PubMed
2. Albu, JB, Murphy, L, Frager, DH, Johnson, JA & Pi-Sunyer, FX (1997) Visceral fat and race-dependent health risks in obese nondiabetic premenopausal women. Diabetes 46, 456–462.CrossRef | Google Scholar | PubMed
3. Allison, DB, Mentore, JM, Heo, M, Chandler, LP, Cappelleri, JC, Infante, MC & Weiden, PJ (1999 a) Meta-analysis of the effects of anti-psychotic medication on weight gain. American Journal of Psychiatry 156, 1686–1696.Google Scholar
4. Allison, DB, Zannolli, R, Faith, MS, Heo, M, Pietrobelli, A, VanItallie, TB, Pi-Sunyer, FX & Heymsfield, SB (1999 b) Weight loss increases and fat loss decreases all-cause mortality rate: results from two independent cohort studies. International Journal of Obesity and Related Metabolic Disorders 23, 603–611.CrossRef | Google Scholar | PubMed
5. Argyropoulos, G, Brown, AM, Willi, SM, Zhu, J, Reitman, M, Gevao, AM, Spruill, I & Garvey, WT (1998) Effects of mutations in the human uncoupling protein 3 gene on the respiratory quotient and fat oxidation in severe obesity and type 2 diabetes. Journal of Clinical Investigation 102, 1345–1351.CrossRef | Google Scholar | PubMed
6. Björntorp, P (1993) Visceral obesity: a 'civilization' syndrome. Obesity Research 1, 206–222.CrossRef | Google Scholar | PubMed
7. Bouchard, C (editor) (1994) Genetics of obesity: overview and research direction. In Genetics of Obesity, pp. 223–233. Boca Raton, FL:CRC Press.Google Scholar
8. Bray, GA & Gallagher, TF Jr (1975) Manifestations of hypothalamic obesity in man: a comprehensive investigation of eight patients and a review of the literature. Medicine 54, 301–330.CrossRef | Google Scholar
9. Bray, GA (1998) Contemporary Diagnosis and Management of Obesity. Newton, PA: Handbooks in Health Care.Google Scholar
10. Clément, K, Vaisse, C, Lahlou, N, Cabrol, S, Pelloux, V, Cassuto, D, Gourmelen, M, Dina, C, Chambaz, J, Lacorte, JM, Basdevant, A,Bougneres, P, Lebouc, Y, Froguel, P & Guy-Grand, B (1998)

A mutation in the human leptin receptor gene causes obesity and pituitary dysfunction. Nature 392, 398–401.CrossRef | Google Scholar | PubMed

11. Després, JP, Nadeau, A, Tremblay, A, Ferland, M, Moorjani, S, Lupien, PJ, Theriault, G, Pinault, S & Bouchard, C (1989) Role of deep abdominal fat in the association between regional adipose tissue distribution and glucose tolerance in obese women. Diabetes 38,304–309.CrossRef | Google Scholar | PubMed

12. Dilmanian, FA, Weber, DA, Yasumura, S, Kamen, Y, Lidofsky, L, Heymsfield, SB, Pierson, RN Jr, Wang, J, Kehayias, JJ & Ellis, KJ (1990) The performance of the delayed- and promptgamma neutron activation systems at Brookhaven National Laboratory. In In Vivo Body Composition Studies. Recent Advances, pp. 309–316[Yasumura, S, Harrison, JE, NcNeill, KG, Woodhead, AD and Dilmanian, FA, editors\.New York: Plenum Press.CrossRef | Google Scholar

13. Durnin, JV & Womersley, J (1974) Body fat assessed from total body density and its estimation from skinfold thickness measurements on 481 men and women aged from 16 to 72 years. British Journal of Nutrition 32, 77–97.CrossRef | Google Scholar | PubMed

14. Flegal, KM, Troiano, RP, Pamuk, ER, Kuczmarski, RJ & Campbell, SM (1995) The influence of smoking cessation on the prevalence of overweight in the United States. New England Journal of Medicine 333, 1165–1170.CrossRef | Google Scholar | PubMed

15. Fujimoto, WY, Bergstrom, RW, Boyko, EJ, Leonetti, DL, Newell-Morris, LL & Wahl, PW (1995) Susceptibility to development of central adiposity among populations. Obesity Research 3, 179s–186s.CrossRef | Google Scholar | PubMed

16. Gallagher, D, Visser, M, Sepulveda, D, Pierson, RN, Harris, T & Heymsfield, SB (1996) Body mass index as an estimate of fatness across gender, age, sex, and ethnic groups. American Journal of Epidemiology 143, 228–239.CrossRef | Google Scholar