

Summary

In the Czech republic, about 20% of women and 16% of men are obese.

The key role in the etiology of obesity plays the imbalance between energy intake and expenditure, which is influenced by many factors, including leptin and adiponectin.

Leptin is secreted mainly from adipocytes and its levels increase with cell fat content and body fat percentage. Leptin itself is supposed to lead to weight loss, both by decreasing appetite and increasing energy expenditure. But high levels of leptin can be a signal of so called leptin resistance, a state in which leptin is losing its effects.

Adiponectin is secreted exclusively from adipose tissue. In obese people, lower levels of adiponectin were proven (i.e. adiponectin levels are in a negative correlation with body fat percentage). That's consistent with the fact that adiponectin is affecting a range of metabolic processes and thus improves insulin sensitivity and metabolic profile.

For evaluation, we used a database of almost 750 subjects, that was build under the Nugenob study, conducted in 8 cities in 7 european countries. All the participants were measured their anthropometric and biochemical parameters at the baseline and after 10 weeks of a standardized weight reduction program. The czech center retested the patient once again 1 year after the initial study.

I found out, that higher baseline leptin and the ratio between leptin and fat mass (which we named leptin-resistance-index) is a risk factor for worse outcome after 10 weeks of low-calorie diet. This could be explained by the phenomenon of leptin resistance, in which leptin can not exert its potential and help in weight and fat mass reduction. Thus, I suggest that recognizing patients with high leptin or leptin-resistance-index at the beginning of weight reduction programmes that are part of secondary prevention of obesity (i.e. prevention of obesity complications), could be useful. These patients may need to receive extra attention and care in order to

achieve satisfactory results. A part of this care could be administration of a leptin sensitizing agent, e.g. amylin.

I also recognized a bigger decrease of leptin and leptin-sensitivity-index between baseline and 10th week as a risk factor for a worse outcome between 10th week and 1 year. Here the worse outcome was represented by higher increase in fat mass. This could be explained as a lack of leptin after leptin sensitivity was restored by 10 weeks of low-calorie diet. Also these patients could receive an extra care as a part of secondary prevention of obesity, and could be for example administered leptin.

I did not find any significant correlation between adiponectin and the anthropometrical measures investigated.