Leptin in Obesity – A Review

Vunnam Sri Sai Charan
Saveetha Dental College and Hospitals
Saveetha University
Chennai-600077

Abstract:
Aim:
To determine the Leptin's role in obesity.

Objective:
Leptin also plays a role in other physiological processes as evidenced by its multiple sites of synthesis other than fat cells.

Background:
Leptin hormone synthesized by adipose cells helps to regulate energy balance by inhibiting hunger. Leptin resistance occurs when leptin levels are high due to this; your brain is starved while the body gets obese. Some researchers have referred to this as brain starvation.Lepotin is produced primarily in the adipocytes of white adipose tissue. It also is produced by brown adipose tissue placenta(synctiotrophoblasts), ovaries, skeletal muscle, stomach (the lower part of the fundic glands), mammary epithelial cells, bone marrow. Thus leptin levels correlate with obesity of an individual.

Conclusion:
This review shows the relationship between leptin levels and obesity.

INTRODUCTION:
Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have a negative effect on health. People are generally considered obese when their body mass index (BMI), a measurement obtained by dividing a person's weight by the square of the person's height, is over 30 kg/m², with the range 25–30 kg/m² defined as overweight. Some East Asian countries use lower values. Obesity increases the likelihood of various diseases, particularly heart disease, type 2 diabetes, obstructive sleep apnea, certain types of cancer, and osteoarthritis. It is most commonly caused due to excess food intake and lack of physical activities. A few cases are caused by genes and mental disorders. On average, obese people have a greater energy expenditure than their thin counterparts due to the energy required to maintain an increased body mass. Through a combination of social changes and personal choices. Changes to diet and exercising are the main treatments. Diet quality can be improved by reducing the consumption of energy-dense foods, such as those high in fat and sugars, and by increasing the intake of dietary fibre. Medications may be taken, along with a suitable diet, to reduce appetite or decrease fat absorption. If diet, exercise, and medication are not effective, a gastric balloon or surgery may be performed to reduce stomach volume or bowel length, leading to feeling full earlier or a reduced ability to absorb nutrients from food.

Obesity is more common in women than men. Authorities view it as one of the most serious public health problems of the 21st century. Obesity is stigmatized in much of the modern world (particularly in the Western world), though it was seen as a symbol of wealth and fertility at other times in history and still is in some parts of the world. In 2013, the American Medical Association classified obesity as a disease.

LEPTIN IN OBESITY:
Leptin is thought to be blood borne signal which is from adipose tissue that informs brain about the fat mass. A gene has been identified that is responsible for producing several forms of leptin receptors by splicing different segments of the gene. In obesity, a decreased sensitivity to leptin occurs, resulting in an inability to detect satiety despite high energy stores. Although regulation of fat stores is seemed to be the primary function of leptin, it also plays a role in other physiological processes, as evidenced by its multiple sites of synthesis other than fat cells, and the multiple cell types beside hypothalamic cells that have leptin receptors. As was predicted, leptin treatment, can be done by direct injection of leptin into the cerebral ventricle or hypothalamus, profoundly inhibited food intake and decreased weight and fat in animals lacking leptin. Leptin levels fall rapidly in response to fasting and evoke profound changes in energy balance and hormone levels. Low leptin levels induce overfeeding and suppress energy expenditure, thyroid and reproductive hormones, and immunity. CSF levels provide evidence for reduction in Leptin crossing the BBB and reaching obesity-relevant targets, such as hypothalamus, in obese people. The ratio of leptin in CSF compared to blood is lower in obese people compared to the normal people.

LEPTIN DIET:
There are some rules which you should not take snacks after the dinner and take your dinner before seven before you go to bed. Eat three times a day, but no snacks. Do not take large meal when you are quite full. The Leptin diet encourages you to eat fresh, organic foods whenever possible. On this diet, you neither religiously count calories nor totally ignore them. Coming to the breakfast. Breakfast on the Leptin diet. This meal should include 20 to 30 grams of protein. To fulfill this hefty requirement, you could eat 3 ounces of top round supplies 20 grams. Or try something lighter like oatmeal with almond milk and whey protein powder. For a breakfast-to-go, whip up one of the approved smoothies, like Lemon Basil, Berry Green or Coco Spice. For lunch salad is the best for the Leptin diet.
ROLE OF LEPTIN IN DISEASES:
Leptin levels and expressions are associated with the body adiposity and body mass index in both experimental animals and humans. Leptin provides important feedback mechanisms, which is necessary for the precise regulation of long-term energy balance. Leptin levels do not increase rapidly; Leptin is mainly responsible for large decreases in food intake that accompanies satiety. Leptin has a variety of important central and peripheral actions to regulate energy balance and metabolism, fertility, and bone metabolism that are mediated by specific cell surface leptin receptors. Importantly, leptin may also exert actions related to cardiovascular homeostasis that are potentially atherogenic, thrombotic, and angiogenic. Leptin has peripheral actions to stimulate vascular inflammation, oxidative stress, and vascular smooth muscle hypertrophy that may contribute to pathogenesis of type 2 diabetes mellitus, hypertension, atherosclerosis, and coronary heart disease.

LEPTIN AND INSULIN:
Leptin increases insulin sensitivity in rats and may improve vascular responses to insulin in states of insulin resistance. Leptin increases free fatty acid oxidation in isolated mouse soleus muscle by 42%, whereas insulin decreases this by 40%. When both hormones are administered, leptin is reduced in both the antioxidative and lipogenic effects of insulin by 50%. Leptin attenuates the antioxidative, lipogenic actions of insulin on muscle free fatty acid metabolism via a peripheral mechanism, whereas the effects of leptin in modulating insulin-stimulated glucose disposal appear to occur via a central mechanism. Recombinant mouse leptin inhibits glycogen synthesis in soleus muscle of ob/ob mice in the presence of insulin. By contrast, leptin increases glycogen synthesis in cultured C2C12 muscle cells.

LEPTIN IN HYPERTENSION:
Correlations between leptin and blood pressure are influenced by gender. Despite higher serum leptin levels in women, leptin and blood pressure associations have been reported more frequently in men than in women, regardless of hypertension and adiposity. Ethnic and racial background may also influence the relationship between leptin and blood pressure. We did not observe significant correlations between plasma leptin levels and blood pressure before efonidipine therapy.

CONCLUSION:
Leptin concentration and body composition were previously observed to be previously observed and Prepubertal children. The important finding is the energy expenditure in children and physical activity in correlation with plasma leptin concentration. Leptin plays an important role in the energy expenditure in humans and, more specifically, physical activity, perhaps by activation of the sympathetic nervous system. Leptin may therefore be important in regulating energy balance, not only by controlling food intake, but also by increasing total daily energy expenditure.

The association between leptin concentrations and energy expenditure in children does not prove a causal relationship whereby leptin produces an increase in energy expenditure. However, it is unlikely that energy expenditure actually elevates leptin concentrations since it is known that elevated levels of physical activity increase sympathetic nervous system activity. Leptin seems to increase energy expenditure and physical activity in children; leptin resistance in obese people is a normal part of mammalian physiology and possibly, could confer a survival advantage. Leptin resistance (in combination with insulin resistance and weight gain) is seen in rats after they are given unlimited access to palatable, energy-dense foods. This effect is reversed when the animals are put back on a low-energy diet. This also may have an evolutionary advantage: allowing energy to be stored efficiently when food is plentiful would be advantageous in populations where food frequently may be scarce.

REFERENCES:


