

## **Hormonal disbalance in non-alcoholic steatohepatosis: the role of leptin in fat metabolism disorders**

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Relevance. Non-alcoholic steatohepatitis (NASH) is associated with increased mortality and risk of complications, but is often asymptomatic and underrecognized. The disease can remain asymptomatic for many years or progress to liver cirrhosis and hepatocellular carcinoma [1,2]. The role of endocrine disorders is so great. The liver is a target organ for metabolic syndrome and endocrine disorders in particular [3,4,5]. These studies show the role of hormones as predictors in the development of NAFLD [6,7]. Hyperleptinemia and resistance to weight loss are two common characteristics of obesity [8]. Leptin regulates food intake, body weight, reproductive function and plays a vital role in fetal growth, proinflammatory immune responses, angiogenesis and lipolysis [9,10]. Until recently, emerging evidence pointed to new mechanisms of leptin resistance. Here, we summarized the advances and controversies in the field of leptin resistance and related diseases to better understand the physiology and pathophysiology of leptin, as well as new treatment strategies for obesity and metabolic disorders [11].

Purpose: To critically summarize the experimental and clinical data on leptin in the development of steatohepatosis.

Materials and Methods: The experiments were performed on 120 white rats weighing 160-200 g. Fatty liver disease is modeled by a high-fat diet. During the study, the animals were divided into 4 groups: 1 - intact (healthy), 2 - rats fed a high-fat diet and called a model of fatty hepatosis for 14, 18, 20 weeks. Blood ELISA was performed to determine leptin levels.

Results: The mean serum leptin levels ( $\pm$  SEM) were  $6.77 \pm 0.001$  and  $27.41 \pm 0.001$  ng / ml in the NAFL group and control group, respectively. The mean serum leptin level in the NAFL group was significantly higher than in the other tested groups. There was a significant correlation between obesity and serum leptin levels when all subjects were assessed together (NAFL and control group,  $r = 0.337$ ,  $p = 0.012$ ). Of the predisposing factors for NAFL, obesity was observed in 44% of rats and hyperlipidemia in 67%. Serum cholesterol and triglyceride levels were significantly higher in the NAFL group than in the control group ( $p < 0.05$ ). It was found that in the rats consuming a large amount of fat, it was significantly lower than in the control group.

Conclusions: Serum leptin levels were significantly lower in patients with NAFL.

This decrease was disproportionate to serum triglyceride, cholesterol levels and in most cases could be associated with hyperlipidemia. Thus, decreased serum leptin levels may contribute to the development of hepatic steatosis.

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