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Modern Aspects of Obesity Pathophysiology

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Annotation: Obesity is a major contributor to metabolic dysfunction involving lipids and glucose, but on a broader scale, it affects organ dysfunction involving the heart, liver, intestines, lungs, endocrine and reproductive functions. Obesity promotes immune dysfunction from the consequences of its inflammatory adipokine secretion and is a major risk factor for many cancers, including hepatocellular, esophageal and colon. This review examines the main pathophysiological processes of obesity.

Key words: obesity, pathophysiology, metabolic syndrome, leptin, relin.

Relevance. Obesity is central to the pathophysiology of diabetes mellitus, insulin resistance, dyslipidemia, hypertension and atherosclerosis, largely due to excess secretion of adipokines. Obesity is a major contributor to metabolic dysfunction involving lipids and glucose, but on a broader scale, it affects organ dysfunction involving the heart, liver, intestines, lungs, endocrine and reproductive functions. Adiponectin protects against liver fibrosis due to its anti-inflammatory effect, while inflammatory cytokines such as tumor necrosis factor-α not only reduce liver function but also impair pancreatic excretory function. Obesity promotes immune dysfunction from the consequences of its inflammatory adipokine secretion and is a major risk factor for many cancers, including hepatocellular, esophageal and colon. This review examined the main pathophysiological processes of obesity.

The increasing prevalence of obesity worldwide is a source of concern for disease surveillance units, health monitoring agencies, and health care personnel worldwide. The allocation of health resources must be tailored to combat this global epidemic. However, the key to success in solving this problem lies in prevention, and this in itself requires a thorough understanding of the physiology of weight control and the pathogenesis of obesity [5]. The development of obesity is undoubtedly associated with the underlying causes of inappropriate diet and lifestyle. Genetic factors have been shown to be responsible for the phenotypic expression of obesity. Medications such as steroids and endocrine diseases (eg, hypothyroidism) may also lead to disruptions in the normal physiology of weight management [3]. However, in the vast majority of weight and obesity problems, the main etiological factor is a mismatch between food intake and energy expenditure [6,8]. Physiological Basis of Obesity A complex feedback control system consists of a central processing structure that receives afferent signals and produces appropriate efferent stimuli. As a result, the level of food intake, satiety and weight are controlled [10]. Age and gender differences in food intake were found with increasing adolescence, peaking in the second decade, after which it declines. Men tend to consume more food, unlike women. At the same time, a decrease in the concentrations of sex steroid hormones during perimenopause leads to an increase in visceral fat and is an increased risk of developing metabolic syndrome [1,4].

Metabolic syndrome is a set of manifestations originally described by Gerald Raven in 1993, including obesity, insulin resistance and increased atherosclerotic risk in diabetes, hypertension and hyperlipidemia [2,9]. Regulatory System The feedback system that regulates body weight and appetite is the subject of ongoing intensive research to evaluate the complexity of this system [7]. Afferent signals Gastric distension, together with vagal afferent impulses, determines satiety, and through gastric contractions, signals hunger. Nutrients, neural impulses, and hormones themselves act as afferent signals in regulating energy intake and expenditure. For example, the absorption of glucose initiates a feeling of fullness, while a drop in glucose levels increases hunger. This effect is mediated through various neurotransmitters, hormones and peptides [7]. Leptin is a peptide produced by adipocytes that correlates well with fat mass. The level of secretion of this peptide increases depending on the level of body fat. Its action is aimed at reducing food consumption. This peptide began to be used to treat people who were found to be deficient in the leptin gene [5]. Another important growth hormone peptide is relin, which is produced in the stomach and duodenum and stimulates the secretion of growth hormone. It is an endogenous ligand for the GH receptor. Relin increases the absorption of food, and the secretion of growth hormone, in turn, decreases when eating. Serum concentration increases with anticipation of food [5]. Regulatory Center Afferent impulses are sent to a center located at the back of the hypothalamus for integration and processing. In vivo studies, typically involving destruction of a specified region, have targeted several specific anatomical sites [5]. The arcuate nucleus of the hypothalamus receives signals from leptin and in turn increases the production and secretion of neuropeptide Y (NPY) and agouti-related peptide (AgRP), thereby increasing food intake. On the other hand, proopiomelanocortin (POMC) reduces food intake [6]. The paraventricular nucleus of the hypothalamus is stimulated by peptides from the arcuate nucleus and transmits signals further. It has been shown that destruction of the ventromedial portion of the hypothalamus leads to an increase in food consumption and subsequently to obesity in experimental animals. The lateral hypothalamic nucleus, in turn, has the opposite effects, such as reducing the amount of food eaten and reducing body weight. In addition, certain regions of the amygdala may influence feeding in part through the ventromedial region of the hypothalamus [6,7]. Efferent mediators The peripheral nervous system plays a role in stimulating thermogenesis in adipose tissue through activation of beta-3 adrenergic receptors, which leads to a decrease in food intake. The sympathetic nervous system stimulates energy expenditure processes. There are hormones, such as glucocorticoids, that act on the efferent endings of the regulatory system. In addition, they are thought to play an important role in mediating effects mediated through the sympathetic nervous system. For example, in the absence of glucocorticoids, it has been noted that leptin deficiency does not lead to obesity [5, 7]. Obesity is a multifactorial and complex disorder that has significant consequences for the people affected and for the health services that must deal with the consequences of the disorder. It is hoped that further research into the physiology and pathophysiology of obesity will lead to the development of preventive and therapeutic strategies to curb the obesity epidemic [6].

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