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OBESITY AS A CHRONIC INFLAMMATORY CONDITION

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ABSTRACT

Introduction: Obesity is defined as a disease of civilization, i.e., a non-communicable disease related to lifestyle. Its development is influenced by numerous factors, including environmental, genetic, and psychological factors. It results from disorders of weight control at the central level and can develop as early as childhood, predisposing individuals to obesity during adolescence or adulthood. (1)
Objective: The aim of this review was to present obesity as a cause of low-grade chronic inflammation in the body.
Method: In order to present obesity as a cause of low-grade chronic inflammation in the body, a systematic review was conducted between 2020 and 2025, covering the PubMed database.
Conclusions: Obesity causes chronic low-grade inflammation in the body through various mechanisms, known as meta-inflammation. Excessively developed adipose tissue becomes hypoxic, and the immune cells that flow into it take on an inflammatory phenotype and secrete numerous pro-inflammatory cytokines. Mitochondria, which are essential for proper metabolism, are damaged, ATP production mechanisms are disrupted, and numerous ROS are produced. Cells and tissues become resistant to circulating insulin, leading to the development of insulin resistance. Adipokines, which are essential for maintaining normal body weight, regulating appetite, and acting against atherosclerosis and inflammation, become dysregulated. In the gastrointestinal tract, the intestinal epithelium is damaged and the diversity of intestinal bacteria is reduced. The intestinal endothelium is damaged, peripheral blood flow in the vessels decreases, which promotes the development of cardiovascular diseases. The changes occurring in cells and tissues sustain and intensify inflammatory processes, which promote the persistence of inflammation. The data collected indicates that metaplasia is an important mechanism linking obesity to the development of cardiovascular diseases, cancers, fertility disorders, and autoimmune diseases. Obesity should be treated not only as a metabolic disease, but also as a condition of chronic inflammation with broad systemic consequences. Hence, we should put emphasis on the importance of early prevention and weight reduction in order to avoid complications connected with being obese.

KEYWORDS

Obesity, Metaflammation, Adipose Tissue, Inflammatory Macrophages

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Introduction

Obesity is defined by the World Health Organization (WHO) as an abnormal, excessive accumulation of body fat that poses a threat to health. The diagnosis of obesity is based on the BMI (body mass index), according to which obesity is diagnosed at a value of ≥ 30 kg/m². (2) Obesity is an increasingly common health problem, and studies show that it affects younger and younger age groups. Higher body weight in childhood increases the risk of obesity and its complications during adolescence and adulthood, making it crucial to prevent the development of excess body fat from an early age. (3) Studies show that 42% of the US population is obese, and since 1980, obesity rates have more than doubled in over 70 countries. Obesity is a significant risk factor for the development of insulin resistance, type II diabetes, cardiovascular disease, and certain cancers. Moreover, it also causes a chronic inflammatory process in the body called meta-inflammation. (4) This leads to reduced insulin sensitivity, especially in adipose tissue and the liver, and also causes inflammatory reactions in the pancreatic islets. (5)

The concept of metaflammation

The concept of metaflammation emerged in 2006 and referred to chronic low-grade inflammation caused by metabolic factors, primarily an excessive supply of calories in relation to demand, maintained over a long period of time. An infiltration of immune system cells, activation of signaling pathways and molecules characteristic of a typical inflammatory process were found in areas affected by inflammation. (6)

The relationship between inflammation and metabolic disorders is often referred to as immunometabolism, which includes the influence of immune system cells on the body's metabolism. (5)

The development of metaflammation

Obesity in adipose tissue leads to increased lipolysis (the breakdown of triglycerides into free fatty acids and glycerol), decreased glucose uptake, slowed metabolism, and adipokine dysregulation. In the liver, lipogenesis intensifies, insulin resistance and the production of glucose and the acute phase protein fetuin A increase (5), monocytes infiltrate, the number of macrophages increases, and NAFLD develops. (6) In skeletal muscles, insulin resistance increases and glucose uptake decreases, while in pancreatic beta cells, these cells proliferate and glucose-stimulated insulin secretion decreases. In the gastrointestinal tract, the production of N-formylated peptides increases, leading to a decrease in GLP-1 secretion and lipopolysaccharide endotoxemia. (5) Inflammatory reactions in the vascular endothelium contribute to the development of atherosclerotic plaques, which promotes the occurrence of cardiovascular diseases.

Adipose tissue

The pathophysiological adaptation of adipose tissue to obesity is its hypoxia. Macrophages present in hypoxic tissue exhibit a more inflammatory phenotype compared to macrophages from a normally oxygenated environment and release significant amounts of pro-inflammatory cytokines. (7) These pro-inflammatory phenotype-polarized macrophages (M1), in contrast to anti-inflammatory macrophages (M2), play a significant role in the development of metabolic diseases. In addition, significant amounts of B and T lymphocytes flow into hypertrophied adipose tissue, contributing to increased release of inflammatory cytokines. (8) Enlarged adipocytes release increased amounts of inflammatory mediators: TNF-α, MCP-1, (6), IL-1β, IL-6, (9) hormones and fatty acids, which disrupt the metabolic functioning of the entire body. (6) Local hypoxia in adipose tissue leads to the activation of HIF, which increases the transcription of inflammatory genes and intensifies fibrosis. Excessive accumulation of fatty acids activates Toll-like receptors, which contribute to increased NF-κB synthesis, which in turn increases chemokine secretion and intensifies macrophage infiltration. Inflammatory cytokines further disrupt the normal functions of insulin. (9) Along with an increase in inflammatory cytokines, there is an increase in CRP concentration – one of the acute phase proteins, whose elevated concentration indicates persistent inflammation. It has been observed that as body weight decreases, CRP levels decrease, with this relationship being more pronounced in women than in men. (11)

Mitochondria

Mitochondria play a key role in regulating metabolism. In the course of obesity, mitophagy intensifies and the content of mitochondria in adipose tissue macrophages decreases. (7) Due to the impairment of their normal functioning as a result of the inhibition of the formation of their fusion proteins and increased mitochondrial fragmentation, a decrease in respiratory efficiency, ATP production, and ROS formation occurs. Excess ROS activate NLRP3, which increases IL-1β production, activating microglia and intensifying neuroinflammation, as well as disrupting neuronal regulation of metabolism, lipolysis, and thermogenesis in adipose tissue. (9)

Adiponectin and leptin

Adiponectin and leptin are the most important hormones produced by adipose tissue. These are responsible for regulating appetite and metabolism.

Adiponectin is an adipokine that sensitizes liver and muscle tissue to insulin. It also has anti-inflammatory and anti-atherosclerotic effects. As obesity develops, the level of adiponectin in blood decreases, which further causes the development of inflammation. Leptin, known as the satiety hormone, suppresses appetite and accelerates fat metabolism. Its levels increase in obesity, but at the same time, resistance to its action increases, which results in weight gain. (10) In addition, leptin plays a crucial role in stimulating hematopoietic stem cells of the granulocyte and macrophage lines, so an increase in its levels in the course of obesity contributes to the occurrence of leukocytosis.

The digestive tract and gut microbiota

As body weight increases as a result of a high-fat diet, the intestinal epithelium becomes damaged. Mucus production decreases, villi become damaged, tight junctions between cells weaken, and inflammation develops in the intestines. Disorders of the intestinal environment cause the activation and proliferation of adipocytes, which in turn leads to increased secretion of inflammatory cells and disorders in the secretion and action of adipokines. (12)

The gut microbiota plays an important role in weight regulation. Studies have shown that the diversity and quantity of gut bacteria is significantly reduced in obese individuals. (13) It has been proven that the use of probiotics such as Bifidobacterium and Lactobacillus reduces the concentration of IL-1β, TNF-α, and CRP in serum and increases IL-6 and IL-10. A low-energy diet has been shown to have a positive effect on weight loss and the reduction of inflammation in the body. (14)

Vascular endothelium

High levels of free fatty acids observed in obesity cause inflammation of the endothelium, which in turn increases its permeability. In addition, a reduction in insulin-dependent NO synthesis occurs. A decrease in peripheral blood flow in the blood vessels is also observed. Fatty acids induce apoptosis of progenitor cells and proper endothelial cells, exacerbating its dysfunction. These changes contribute to the development of cardiovascular diseases: hypertension, atherosclerosis, heart failure, and coronary artery disease. (15)

Cancer

Genetic, epidemiological, and experimental studies show that elevated insulin levels characteristic of overweight individuals may precede the development of obesity and metabolic disorders. Hyperinsulinemia affects the functioning of the immune system and promotes chronic inflammation. It also activates key signaling pathways that promote proliferation, cell survival, and malignant transformation. Particularly strong evidence exists for the role of hyperinsulinemia in the pathogenesis of pancreatic cancer, as well as breast, endometrial, and colorectal cancers. (16) Obesity is one of the main carcinogenic factors. Due to the fact that in obesity, adipose tissue becomes an inflammatory and hormonally disturbed organ, there is an increased secretion of adipokines, inflammatory cytokines (TNF-α, IL-6), free fatty acids, and growth factors in the body. As a result, changes in the tumor microenvironment occur, leading to tumor growth, angiogenesis, and metastasis. (17) Leptin (increased in obesity) stimulates the proliferation and migration pathways of cancer cells (PI3K/Akt, JAK/STAT, MAPK), intensifying tumor growth, especially in breast cancer. Leptin and the leptin receptor are usually overexpressed in cancer patients, stimulating the ability to form stem cells in many types of cancer. (18)

In contrast, adiponectin is an adipokine that induces apoptosis and acts as an insulin-sensitizing hormone. However, adiponectin production is reduced in obese individuals, contributing to a state of “hypoadiponectinemia” and increased carcinogenesis. Furthermore, in the context of chronic inflammation, pro-inflammatory cytokines, including interleukin-6 (IL-6), IL-1β, and TNF-α, released by adipocytes increase the production of C-reactive protein (CRP) and serum amyloid A (SAA) and may contribute to the development of cancer (19)

Furthermore, obesity-related insulin resistance and type 2 diabetes cause increased levels of insulin and insulin-like growth factors, which activate the PI3K/Akt/mTO pathways. Moreover, as a result of chronic inflammation, pro-inflammatory cytokines, including interleukin-6 (IL-6), IL-1β, and TNF-α, released by adipocytes increase the production of C-reactive protein (CRP) and serum amyloid A (SAA) and may contribute to the development of cancer (19)

In addition, obesity-related insulin resistance and type 2 diabetes cause increased levels of insulin and insulin-like growth factors, which activate PI3K/Akt/mTOR and MAPK pathways. These also increase proliferation and survival of cancer cells, and enhance angiogenesis and resistance to treatment. (17)

Obesity is one of the most important modifiable risk factors for pancreatic cancer, and its increasing prevalence worldwide contributes significantly to the observed increase in the incidence of this cancer. Numerous epidemiological studies have shown that obese individuals have a 1.5–1.6 times higher risk of developing pancreatic cancer compared to individuals of normal weight, with the risk increasing with the degree of obesity. This relationship persists even after taking into account other risk factors such as smoking and diabetes. (20)

Adipose tissue in the mammary gland plays a key role in the pathogenesis of postmenopausal breast cancer. Under the influence of obesity-related factors (leptin, PGE2, IL-6, TNF), adipose tissue stromal cells show increased aromatase expression, leading to a local increase in estrogen concentration.This process is closely linked to metabolic regulation, in particular to the inhibition of the LKB1–AMPK pathway and the activation of HIF1α and CRTC proteins, which enhance the transcription of the CYP19A1 gene. (21)

Disturbances in the gut microbiota also are seen as a health drawback of obesity. The dysbiosis associated with obesity, characterized by a decrease in the number of bacteria producing short-chain fatty acids and an increase in pathogenic microorganisms, promotes chronic intestinal inflammation and carcinogenesis. Unhealthy eating patterns, typical of the Western diet, further exacerbate these processes. (22)

Biologically, obesity affects tumor metabolism, angiogenesis, and the inflammatory and immune environment, which is also particularly important in clear cell carcinoma, characterized by lipid metabolism disorders and VHL/HIF-dependent pseudohypoxia. Researchers suggest that obesity may modulate fatty acid metabolism, immune infiltration of the peritumoral tissue, and response to molecularly targeted therapy and immunotherapy. (23)

Infertility

Obesity significantly reduces female fertility through numerous hormonal and metabolic disorders. Excessive amounts of adipose tissue affect the hypothalamic-pituitary-ovarian axis, disrupt menstrual cycles, reduce ovulation frequency, and impair egg quality, embryo development, and endometrial receptivity. Obesity also exacerbates the symptoms of PCOS — it increases insulin resistance, hyperinsulinemia, and hyperandrogenism — which further reduce the chances of pregnancy. In assisted reproductive technology (ART) procedures, obese women require higher doses of gonadotropins, have poorer ovarian response, lower fertilization and implantation rates, more frequent cycle cancellations, and lower live birth rates. For this reason, weight reduction is recommended before starting infertility treatment. (24)

A study conducted between 2013 and 2020 in the United States observed 3,542 women, including 429 (i.e., 12.11%) who reported ever having been infertile. The results of the study confirmed that both general obesity (calculated on the basis of BMI) and abdominal obesity (determined by waist circumference) have a negative impact on fertility. Biological mechanisms occurring in connection with obesity also include hormonal changes, androgen aromatization, decreased SHBG, inflammation, and free fatty acid toxicity to reproductive cells. The authors emphasize that weight reduction prior to pregnancy or infertility treatment can significantly improve reproductive outcomes and reduce the risk of complications for both mother and child. (25)

Excessive body weight also contributes to infertility in men. Obese males also experience disturbances in the hypothalamic-pituitary-testicular axis, which lead to decreased production of testosterone and other sex hormones. It also leads to hypogonadism and changes in sperm production. Excess body fat is associated with higher estrogen levels, lower testosterone concentrations, and reduced sex hormone-binding globulin (SHBG) concentrations, which negatively affect spermatogenesis. In addition, adipokines and leptin produced by adipose tissue can disrupt hormonal balance and the local microenvironment in the testicles, increase inflammation and levels of reactive oxygen species, which in turn contribute to sperm DNA damage. Last but not least, there are also non-hormonal mechanisms, such as increased scrotal temperature caused by greater amounts of adipose tissue, which can disrupt the process of spermatogenesis. Changes in the gut microbiome and metabolic signaling which negatively affect sperm quality also occur. (26)

It should also be mentioned that maternal obesity can have a negative impact on the health of the child. In a study of a high-fat diet-induced obesity model, it was shown that maternal obesity disrupts meiosis in fetal oocytes. Delayed progression of prophase I, abnormal homologous chromosome synapsis, and defects in meiotic recombination were observed, accompanied by persistent unresolved double-strand DNA breaks. Importantly, intervention in the form of time-restricted feeding improved fetal development and meiosis in oocytes. It also reversed epigenetic changes in selected gene loci. (27)

Autoimmune diseases

Having all the mechanisms considered, it can be undeniably concluded that excess adipose tissue initiates an inflammatory response in the human body. More and more data indicate that obesity and metabolic diseases significantly modulate the functioning of the immune system. It has been shown that obesity fundamentally changes the nature of the immune response. Obesity not only increases the severity of the disease, but also leads to radically different responses to biological therapies targeting TH2 cytokines. (28)

The global obesity epidemic is one of the main factors contributing to the development of chronic diseases and disability. A key element in the pathophysiology of obesity is adipose tissue dysfunction, in particular the disruption of adipokine secretion. (29)

As obesity is associated with hyperleptinemia and chronic inflammation, it may increase the risk of developing autoimmune thyroiditis (Hashimoto's disease) and thus, overt or subclinical hypothyroidism. Both metabolic syndrome and subclinical hypothyroidism are independently associated with an increased risk of atherosclerosis and both liver and kidney diseases, and their coexistence may exacerbate adverse metabolic and cardiovascular effects.Subclinical hypothyroidism should be identified in obese patients and, where appropriate, treatment with levothyroxine should be initiated, adjusting the dose according to lean body mass and total body weight. (30)

Adipokine dysregulation in obesity may exacerbate Th1 and Th17 responses, inhibit regulatory T cell function, and modulate antigen-presenting cell activity, promoting the development and progression of autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, and type 1 diabetes. In summary, adipokines play a key role at the interface between metabolism and immunity, and their dysregulation in obesity may create an environment conducive to the development of autoimmune diseases. (29)

Summary

Obesity is a chronic metabolic disease that contributes to the development and maintenance of chronic inflammation in the body. Excess adipose tissue causes numerous adverse changes in many organs. Immune system cells in adipose tissue take on an inflammatory phenotype, insulin resistance develops in the body, and the functioning of mitochondria and hormones called adipokines is disrupted. The gut microbiota becomes depleted and the vascular endothelium increases its permeability, which promotes the development of cardiovascular diseases. Dysfunctions of adipose tissue, including hyperinsulinemia, altered adipokine secretion, and activation of proliferative signaling pathways, create a microenvironment that favors carcinogenesis, impaired reproductive function, and autoimmunity. Therefore, prevention and treatment of obesity represent crucial strategies for reducing the risk of multiple chronic diseases and improving long-term health outcomes.

Abbreviations:

NAFLD - non-alcoholic fatty liver disease
TNF-α - tumor necrosis factor α
MCP-1 - monocyte chemotactic protein-1
HIF - hypoxia-inducible factors
IL-1β - interleukin-1β
IL-6 - interleukin-6
ROS - reactive oxygen species
NLRP3 - NLRP3 inflammasome
NO - nitric oxide
SAA - serum amyloid A
PGE2 - prostaglandin E2
CRTC protein - gene transcription regulatory protein
PCOS - polycystic ovary syndrome
ART - assisted reproductive technology
SHBG - sex hormone-binding globulin

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