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Review Article

OBESITY: A CHRONIC LOW-GRADE INFLAMMATION AND ITS SYSTEMIC BIO-MARKERS

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ABSTRACT

As the prevalence of obesity continues to rise, the world is facing a major public health concern. Obesity is a complex disease associated with an increase in several inflammatory markers, leading to chronic low-grade inflammation. There remains much unknown regarding the association between obesity and inflammation. This review seeks to compile scientific literature on obesity and its associated inflammatory markers in chronic disease and further discusses the role of adipose tissue, macrophages, fatty acids, amino acids, adipokines, and hormones in obesity. Data were obtained using PubMed and Google Scholar. Obesity, inflammation, immune cells, hormones, and others were search words used to acquire relevant articles. Researchers also found the adipose tissue of lean individuals predominantly secretes anti- inflammatory markers, while in obese individuals more pro-inflammatory markers are secreted. Many studies found that adipose tissue in obese individuals showed a shift in immune cells from anti- inflammatory M2 macrophages to pro-inflammatory M1 macrophages, which was also correlated with insulin resistance. Obese individuals generally present with higher levels of hormones such as leptin, visfatin, and resistin. With obesity on the rise globally, it is predicted that severe obesity will become most common amongst low-income adults, black individuals, and women by 2030, making the need for intervention urgent.

INTRODUCTION

Numerous metabolic disorders and Cardiovascular Disease (CVD) have been shown to be regulated by inflammation in both healthy and pathological settings.[1] A significant contributor to chronic illnesses, obesity is also regarded as a persistent low-grade inflammatory state. In addition to attracting macrophages, the over-abundance of fat in adipose tissue causes the generation of numerous pro-inflammatory cytokines and chemokines, including as interleukin-6, tumor necrosis factor-a, and monocyte-chemoattractant protein 1, which can draw inflammatory cells.[2] Lastly, metabolic dysfunction, including insulin resistance[3], and obesity-related systemic illnesses are brought on by this obesity-mediated adipose tissue remodelling.[4] To reduce the burden of chronic diseases in this population and predict obesity-related health issues, it may be useful to assess an individual's inflammatory condition.

Numerous inflammatory biomarkers have been used to predict the risk of coronary heart disease and other age-related degenerative disorders, including white blood cell (WBC) count and high-sensitivity C-reactive protein (hs CRP).[5,6] These indicators[7,8] are also linked to the level of obesity as measured by body mass index (BMI), waist circumference (WC), waist hip-ratio, [10,11,12] and these body composition factors are inadequate for predicting CVD because they are unable to differentiate between visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT).[13] Compared to SAT area, visceral adipose tissue area is more strongly associated with cardiometabolic risk [14]. While the pathological mechanisms relating to VAT and its comorbidities are complex, altered pro-inflammatory adipokine secretion and a series of inflammatory processes in VAT are thought to be the main causes of CVD.[13]

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An important inflammatory component of obesity is linked to changes in insulin resistance, hypertension, atherosclerosis, and some types of cancer [11]. Tumor necrosis factor-alpha (TNF-), C-reactive protein (CRP), interleukins (IL-6, IL-18), resistin, and visfatin are among the inflammatory cytokines whose serum levels are changed in overweight and obese individuals [6,7]. Compared to BMI, there is a stronger correlation between body fat measurements and certain inflammatory markers.

Methods of literature review

Techniques for doing a literature review, the databases PubMed and Google Scholar were used to find English-language publications on the subjects of obesity and inflammatory markers that were published between 1993 and 2025. "Obesity," "fatty acids," "adipose tissue," "inflammatory markers," "resistin," and "visfatin" were among the search terms. The review produced publications that focused on the presence and impact of fatty acids on inflammation, as well as the description of obesity as an immunological disease or chronic inflammation and its inflammatory markers, including IL-6, TNF-ALFA, resistin, and visfatin.

Inflammation and its Markers

The signs of inflammation, an organized series of actions designed to preserve tissue and organ homeostasis is called inflammation. Completing the program and restoring tissues to their initial state requires the prompt release of mediators and receptor expression[14]. Furthermore, inflammation is a protective tissue reaction to tissue damage or destruction, which works to eliminate or lessen the harmful substance as well as the damaged tissues [15]. There are two kinds of inflammation: acute inflammation, which is characterized by leukocyte migration and edema and lasts for a brief period of time.

Leptin, interleukin (IL-6), tumor necrosis factor-α (TNF-α), monocyte chemoattractant protein-1 (MCP-1), and resistin are examples of inflammatory adipokines that are often secreted by adipose tissue and are thought to be a hallmark of metabolic syndrome [17,5]. Chronic inflammation has been linked to obesity, a characteristic of metabolic syndrome, in obese subjects [18]. The relationship between obesity and certain inflammatory indicators is examined in this review, which also clarifies the related health issues. C-reactive protein (CRP) and IL-6 are examples of inflammatory markers, while adiponectin is an example of an anti-inflammatory marker.

Interleukin-6

Interleukin 6 is a cytokine that mediates inflammatory reactions and is produced by a variety of cell types, including adipose tissue and immune cells [19]. In addition, the IL-6 receptor is present in a number of brain areas, including the hypothalamus, which regulates energy intake and appetite and plays a part in maintaining energy homeostasis by inhibiting lipoprotein lipase activity [20].

In contrast to other cytokines, IL-6 is unique in that its main effects occur at locations that are different from its source and are dependent on the levels of the cytokine in the blood. It is known as the endocrine cytokine as a result [21]. IL-6 primarily acts by attaching itself to the membrane-bound α receptor, also known as the IL-6 receptor (IL-6R). The IL-6/IL-6R complex then binds to glycoprotein 130 (gp130), resulting in the production of gp130-homodimers and signal initiation [22].

Obesity and IL-6

The metabolic syndrome is thought to be characterized by obesity[17]. Inflammation has been identified as the cause of their connection [6]. In Iran, for example, obesity has become a problem among both rural and urban populations [23]. An imbalance between energy intake and expenditure leads to obesity, indicating a positive energy balance[24]. Adipose tissue is classified into two main categories: white and brown[25]. Through thermogenesis, which is facilitated by the production of uncoupling protein-1 (UCP1), brown adipose tissue in newborn humans aids in controlling energy expenditure [26].

However, white adipose tissue is becoming recognized as an active participant in controlling physiologic and pathologic processes, such as immunology and inflammation, rather than an inert tissue primarily used for energy storage [27]. Adipose tissue contains macrophages, which actively take part in its functions. Immune modulation can also result from the interaction of lymphocytes and adipocytes [28]. The adipokines leptin, adiponectin, and resistin, as well as cytokines and chemokines including TNF-α, IL-6, and MCP-1, are among the pro- and anti-inflammatory substances that are produced and released by adipose tissue [18].

It has been reported that plasma IL-6 levels and various measures of obesity are positively correlated [29]. According to calculations, adipose tissue is the source of one-third of the total circulating concentrations of IL-6 [30]. Obesity is thought to be associated with inflammation because of the overexpression of pro-inflammatory cytokines [31]. In response to the stimulation of more nutrients, adipocytes undergo hyperplasia and hypertrophy in

adipose tissue. Adipocytes, immune cells, and endothelium are among the several types of adipose tissue [32]. Hypoxia may result from a decrease in the blood supply to adipocytes due to progressive adipocyte growth and obesity [33].

It has been suggested that hypoxia is a trigger for necrosis and macrophage infiltration into adipose tissue, which results in an excess of pro-inflammatory mediators. This causes adipose tissue to become inflamed locally, which spreads systemic inflammation and leads to the emergence of comorbidities linked to obesity [34]. Three inflammatory mediators- TNF-α, IL-6, and adiponectin- are produced by macrophages [27]. Additionally, IL-6 substantially induces hepatocytes to release and generate CRP, which is indicative of inflammation[35]. By using the inflammatory process, Ellulu et al.[36]

Additionally, the buildup of free fatty acids in obesity induces pro-inflammatory serine kinase cascades, including c-Jun N-terminal kinase and IkB kinase, which in turn encourages adipose tissue to release IL-6, which in turn causes hepatocytes to synthesize and secrete CRP [37].

The inflammatory mediators in morbidly obese patients following bariatric surgery were assessed by Illán-Gómez et al [38]. In addition to having a strong correlation with BMI and being linked to elevated CRP, IL-6 levels dramatically dropped after a year of surgery. The relationship between obesity and obstructive sleep apnea in IL-6 persons aged +50 years was evaluated by Arnardottir et al [39]. Patients were divided into three groups according to their BMI (less than 30, thirty to thirty-five, and more than thirty-five kg/m^2), and the distribution of IL-6 was 1.3, 1.6, and 2.2 pg/ml, respectively. This suggests that IL-6 and BMI are strongly correlated.

C-Reactive Protein

The body produces C-reactive protein, which is a sensitive indicator of systemic inflammation. Traditionally, acute injury, infection, and inflammation have been detected using C-reactive protein, a nonspecific acute-phase reactant [40].

Obesity and CRP

Each standard deviation increase in CRP was linked to a 60% increase in vascular risk, according to a 2010 meta-analysis conducted by the Emerging Risk Factors Collaboration. Obesity has been associated with CRP in numerous cross-sectional studies[41]. CRP was directly correlated with each degree of obesity, independent of sex and ethnicity[42]. The meta-regression study also found a high association between CRP and obesity in both adults and children.

The pathophysiological process provides a clear explanation for the connection between obesity and higher serum levels of CRP. Since the liver eliminates free fatty acids and circulating triacylglycerol encourages adipose tissue to generate cytokines (IL-6), which in turn causes hepatocyte expression and the production of CRP, the liver is thought to play a key role in inflammation[81]. The association between CRP and obesity was examined by the cross-sectional data. CRP and metabolic indicators were assessed by Klisic et al[43] in Montenegro among postmenopausal women of normal weight and those who were overweight.

An important inflammatory component of obesity is linked to changes in insulin resistance, hypertension, atherosclerosis, and some types of cancer [11]. Tumor necrosis factor-alpha (TNF-), C-reactive protein (CRP), interleukins (IL-6, IL-18), resistin, and visfatin are among the inflammatory cytokines whose serum levels are changed in overweight and obese individuals [6,7]. Compared to BMI, there is a stronger correlation between body fat measurements and certain inflammatory markers.

When comparing the blood from the radial artery and portal vein, IL-6 was approximately 50% higher in the former (p=0.007), and there was a direct correlation between portal vein IL-6 and systemic CRP (r=0.544, p=0.005). among conclusion, Ajani et al. [46] determined the prevalence of elevated CRP (≥ 3mg/l) among persons with free diabetes, cardiac disease, and non-lipidaemia in the United States. CRP was directly correlated with BMI, ranging from 31.4 to 35.1% for people who were overweight and from 52.5 to 60.9% for those who were obese. When those with CRP >10mg/l were excluded, the adjusted prevalence of high CRP (≥3mg/l) was 36.0% for obese people, 31.6% for overweight people, and 14.4% for normal weight people.

The thickness of the subcutaneous abdominal fat (SCFT) is a significant factor in the risk of cardiovascular and metabolic disorders. The metabolic alterations and maternal SCFT were assessed by Köşüş in pregnant women between 24 and 28 weeks of gestation [62]. The findings showed that SCFT and glycated hemoglobin (HbA1c) were significantly associated. On the other hand, 47.9% of those with SCFT ≥ 15mm had higher CRP levels.

However, there are a number of characteristics that have a strong correlation with CRP and systemic cytokine concentrations, including age, gender, smoking, and a sedentary lifestyle[54,47,48]. As a signal for more serious illnesses, elevated CRP levels may also be linked to certain pathological disorders; for example, high CRP levels in myocardial infarction patients may result in left ventricular systolic dysfunction [49]. Because it was thought to lower CRP,

sleep restriction was also linked to cardiovascular diseases (CVD) [50]. Similarly, the development of CVDs has been directly connected to obstructive sleep apnea, which has been strongly correlated with elevated CRP and BMI.[27]

Chronic stress in the key areas of life (relationships, employment, sympathetic caregiving, and finances) has been linked to worsening CRP in both men and women over 45, according to Shivpuri et al[51]. Furthermore, compared to men, women displayed a somewhat higher level of stress. Kwon et al[52] also discovered that patients with pulmonary hypertension (defined as "systolic pulmonary artery pressure ≥ 35mmHg") had a considerably higher plasma level of CRP than those without hypertension.

Adiponectin

The protein hormone adiponectin, which is produced from adipocytes, has become very important because of its beneficial effects on insulin resistance, atherosclerosis, inflammation, and type 2 diabetes mellitus (T2DM) [53–55]. As a result, it establishes a direct connection between adipose tissue and the causes of metabolic disorders [56]. Identified in 1995[57], adiponectin is a 247-amino acid peptide that circulates at relatively high (2–20μg/ml) blood concentrations [58], while Ouchi et al. [59] assessed values that generally range from 3–30μg/ml in healthy adult subjects. In healthy, non-metabolic people, adiponectin was associated with a lower risk of atherosclerosis and anti-inflammatory effects of type 2 diabetes [60]. Plaque start is inhibited and endothelial dysfunction is prevented by increasing local nitric oxide generation.

Consequently, it reduces oxidative stress and functions as a vasoprotective agent [62]. Adiponectin enhances insulin sensitivity, lowers gluconeogenic activity, boosts oxidation, and inhibits the absorption of non-esterified fatty acids in the liver [63]. In terms of controlling weight gain and raising energy expenditure, it is very effective [64]. There is a two-fold lower risk of myocardial infarction [65] and a seven-fold lower risk of coronary artery calcification progression [66] in healthy persons whose adiponectin levels fall within the top 20% range. Adiponectin's effects on peripheral tissues and insulin sensitivity are depicted

Obesity and Adiponectin

By overexpressing the Adipoq gene, which increases fat tissue bulk by increasing the number of adipocytes rather than their size, adiponectin plays a significant autocrine or paracrine role with "local effects" that influence adipose tissue activities [67]. Numerous investigations have demonstrated that weight increase and obesity considerably lower serum levels of adiponectin [68]. Although the exact cause of the lower adiponectin levels in obesity is still unknown, studies in humans have identified the role of visceral adipocytes in secreting more adiponectin and other adipokines than subcutaneous adipocytes.

However, it is assumed that the elevated levels of inflammatory mediators, such as TNF-α and IL-6, secreted by adipocytes, are what inhibit and reduce the synthesis and secretion of adiponectin. As a result, endothelial health and its cardio-protective function will also be depressed. Adipokines and inflammatory markers were compared in obese insulin-sensitive and insulin-resistant patients based on the homeostasis model assessment of insulin resistance (HOMA-IR) by Stępień et al. [69]. The trial comprised 64 out clinic adult patients having a BMI of > 30 kg/m^2, 25 men and 39 women, with or without hypertension.

In patients with insulin resistance, there was a high correlation between the BAI and adiponectin. A cross-sectional study was carried out by Eglit et al. [70] to assess the relationship between obesity and metabolic risk variables in a healthy population in Estonia that was at least 20 years old. In both males and females, adiponectin was negatively linked to obesity because it had a negative correlation with fasting blood glucose (FBG), total cholesterol (TC), and TG and a positive correlation with high-density lipoprotein cholesterol (HDL-c). An investigation on the connection between arterial stiffness, adiponectin, and obesity was carried out by Snijder et al. [71]. 456 participants from the Hoorn Study, ages 60 to 86, were included using a cross-sectional design

Adiponectin was negatively correlated with total and abdominal fat, and it was also linked to a reduction in peripheral arterial stiffness. In the Hoorn Study, Dekker et al. [72] investigated the role of adiponectin in predicting mortality and CVDs in a cohort of people aged 50–75. Significant trends were found in both men and women when adiponectin levels were categorized into quartiles, with the lowest quartile being linked to a higher BMI and the highest quartile to a lower BMI. Jaleel et al. [73] examined the levels of adiponectin in postmenopausal women of normal weight and those who were obese. Compared to subjects of normal weight, obese subjects had a considerably lower amount of adiponectin. T.

The study also demonstrated that aberrant lipid profiles and leptin levels are linked to obesity.

Visfatin with Obesity

Visfatin and obesity Fukuhara et al. discovered visfatin, a new cytokine derived from adipose tissue [74]. Viscotinamide phosphoribosyl transferase (NAMPT), an enzyme involved in the NAD+ salvage pathway, functions similarly to visfatin, which is highly expressed in visceral fat cells, once it is produced. First discovered to be a pre-B cell colony boosting factor

(PBEF), visfatin is released by human peripheral blood lymphocytes [75]. Visfatin mostly mimics insulin in the liver, bone marrow, and skeletal muscle, where it stimulates the development of B-lymphocyte precursors [76,77].

WAT accumulation is tightly associated with visfatin levels in the body, as visfatin mRNA levels rise during adipocyte maturation. Growth hormone, glucocorticoids, TNF, and IL-6 are some of the many variables that control visfatin production. Endotoxin-challenged neutrophils and WAT are the main producers of visfatin. The procedure by which these neutrophils stop cells from dying is then mediated by caspase 3 and 8 [78]. Patients with inflammatory bowel disorders have higher amounts of visfatin mRNA in their intestinal epithelium as well as higher levels of circulating visfatin. [79]

Additionally, it has been observed that visfatin can improve CD14+ monocytes' capacity to produce alloproliferative responses mediated intracellularly by MEKI and p38 in lymphocytes, as well as increase their chemotaxis and production of TNF, IL-1b, IL-6, and other costimulatory molecules [80]. Visfatin levels in the blood have been found to be higher in patients with acute lung damage[82] and rheumatoid arthritis [81]. Inflammatory bowel illness was also associated with significantly greater expression of visfatin mRNA [83].

Visfatin/PBEF/Nampt has a comparable binding affinity to the insulin receptor to that of insulin [84]. Higher levels of visfatin have been linked to conditions including diabetes mellitus, according to earlier study [85–87]. However, no link between PBEF and visfatin was found when an experiment was conducted on a group of obese people.

There is ongoing debate regarding visfatin's ability to bind to insulin receptors and its insulin-mimetic properties. But according to new study, systemic NAD+ production, which is mediated by Nampt/visfatin, is necessary for healthy β cell activity [89]. The function of visfatin in controlling glucose homeostasis is further supported by these results [89]. The implication of this novel adipokine in the inflammatory processes of obesity starting in early infancy is demonstrated by the greater visfatin levels in children with higher BMIs [90].

Females with visceral obesity and severely obese patients had greater visfatin levels following gastric banding [74]. On the other hand, visfatin levels in the blood decreased in morbidly obese women who had reduced their BMI by more than 20%. According to these studies, visfatin concentrations tend to decrease during weight loss, and the higher the BMI (obesity), the higher the concentration of visfatin.

Resistin with Obesity

Fighting off obesity in both in vitro and in vivo settings, Steppan and Lazar investigated the function of resistin as an antagonist to insulin [91]. According to their findings, mice with diabetes and obesity had higher levels of resistin. It has been shown that giving rodents resistin exogenously raises both their endogenous synthesis of the protein and their plasma glucose levels [92,93]. Resistin is produced by adipocytes and macrophages in rats as well as mononuclear cells in humans, such as macrophages [94,95].

Activated macrophages and other immune cells, as well as adipocytes, express additional chemokines like IL-6, TNF-α, visfatin, MCP-1, and PAI-1. It is currently unknown how much adipocytes and macrophages produce in relation to one another in adipose tissue. A review of resistin's possible function as a mediator between adipose tissue, inflammation, immunity, and adipose tissue has been conducted more recently [92]. Additionally, there is evidence that resistin functions as a pro-inflammatory adipocytokine, is linked to cartilage degradation markers, and is elevated after intense exercise like marathon running [94]. According to a number of studies, mononuclear cells secrete resistin, which may contribute to inflammatory situations [95–99].

Resistin levels in obstructive sleep apnea patients are believed to be positively connected with other inflammatory markers like lipoprotein-associated phospholipase and soluble TNF-R type II in patients with atherosclerotic plaques, as well as correlated with cell adhesion molecules (ICAM1) [95,96].

Lipopolysaccharide has also been shown to cause human and animal macrophages to produce resistin. TNF triggers resistin through the NFκB pathway, a cascade including pro-inflammatory cytokine secretion, which can further trigger the production of IL-6 in human peripheral blood mononuclear cells [97,98]. Furthermore, visfatin and IL-6 levels are substantially connected with resistin alterations [97].

CONCLUSION

Based on this review, we can conclude that multiple organ systems, especially those of adipose tissue and muscle, are responsible for metabolic homeostasis. Adipocytes secrete adipokines, which are hormones that regulate metabolism by acting on multiple cells and organs. The amounts of these hormones present in children, adolescents, and adults, along with the role they play in obesity, diabetes, hypertension, metabolic syndrome, atherosclerotic diseases, etc., are of significance. Thus, in order to gain a deeper insight into the regulation and pathology of these adipokines/hormones, it is important to further

explore how these factors affect lifestyles such as regular exercise, dietary intervention, supplementation, or how a combination of these may influence their concentrations in the body. Although the roles of resistin and visfatin in homeostasis/ metabolism are not yet completely understood; the interest in further researching these hormones should remain high. Several research studies have shown there is an association present between abdominal and total obesity and various inflammatory markers. However, this association has not been fully comprehended. Obesity is a chronic low-grade inflammation associated with an increase in inflammatory markers. Future research will provide insights into the correlation of different inflammatory markers with one another in different populations, to aid in the further understanding of the underlying mechanism between obesity and inflammation.

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