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Endocrine-Immune Mechanisms Linking Obesity, Diabetes, and Anaemia of Chronic Disease

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ABSTRACT

Obesity, type 2 diabetes mellitus, and anaemia of chronic disease are increasingly recognized as interconnected disorders driven by persistent endocrine-immune dysregulation. Obesity is not merely excess fat accumulation but a chronic inflammatory and hormonal state characterized by adipocyte hypertrophy, adipose tissue hypoxia, macrophage infiltration, insulin resistance, altered adipokine secretion, oxidative stress, and low-grade systemic inflammation. These changes promote the development of type 2 diabetes by impairing insulin signalling in adipose tissue, skeletal muscle, liver, pancreatic β-cells, vascular endothelium, and immune cells. At the same time, chronic inflammation alters iron metabolism and erythropoiesis, creating the biological basis for anaemia of chronic disease. Central to this process is the hepcidin-ferroportin axis. Inflammatory cytokines, particularly interleukin-6, stimulate hepatic hepcidin production, leading to ferroportin degradation, iron sequestration within macrophages, reduced intestinal iron absorption, and restricted iron delivery to the bone marrow. In diabetes, this inflammatory iron restriction is compounded by kidney disease, reduced erythropoietin production, oxidative injury, mitochondrial dysfunction, nutritional deficiencies, and shortened red blood cell survival. This review examines the endocrine-immune mechanisms linking obesity, diabetes, and anaemia of chronic disease, with emphasis on adipokines, cytokines, insulin resistance, hepcidin, erythropoietin impairment, and therapeutic implications.

Keywords: Obesity; type 2 diabetes mellitus; anaemia of chronic disease; hepcidin; immunometabolism

INTRODUCTION

Obesity and type 2 diabetes mellitus are among the most important metabolic disorders worldwide. Their health impact extends beyond excess body weight and high blood glucose, affecting cardiovascular function, kidney health, immune competence, inflammation, endocrine regulation, and haematological status [1]. In recent years, obesity has been redefined as a chronic endocrine and inflammatory disorder rather than a simple problem of excess energy storage. Adipose tissue is now understood as an active endocrine-immune organ that secretes hormones, cytokines, chemokines, lipid mediators, and growth factors capable of influencing whole-body metabolism [2]. Anaemia of chronic disease, also called anaemia of inflammation, is a common anaemia pattern seen in chronic infections, inflammatory disorders, kidney disease, malignancy, obesity, and diabetes. It is characterized by impaired iron mobilization, reduced iron availability for erythropoiesis, inflammatory suppression of bone marrow activity, shortened red blood cell survival, and blunted erythropoietin responses [3]. The anaemia is usually mild to moderate, but it can significantly worsen fatigue, cardiovascular strain, exercise intolerance, tissue hypoxia, kidney disease progression, and quality of life. The link between obesity, diabetes, and anaemia of chronic disease is best understood through endocrine-immune mechanisms [4]. Obesity causes adipose tissue inflammation and alters adipokine secretion, while diabetes amplifies oxidative stress, endothelial injury, kidney dysfunction, and immune

imbalance. Current reviews describe obesity, diabetes, and their related complications as chronic inflammatory conditions in which nutrient excess activates immune signalling pathways, including pattern-recognition receptors and inflammasomes [5]. These inflammatory responses then influence iron metabolism through hepcidin, a key hormone that controls systemic iron distribution. The result is a metabolic-inflammatory state in which glucose regulation, immune activation, iron restriction, and erythropoiesis are closely connected.

Adipose Tissue as an Endocrine-Immune Organ

Adipose tissue is a dynamic organ with metabolic, endocrine, immune, and vascular functions. In healthy conditions, adipocytes store lipids safely, release fatty acids when needed, secrete adiponectin, and communicate with the brain, liver, skeletal muscle, pancreas, and immune system. However, in obesity, adipocytes enlarge beyond their optimal storage capacity [6]. Hypertrophic adipocytes become hypoxic, stressed, insulin-resistant, and prone to cell death. These changes trigger immune-cell recruitment, particularly macrophage infiltration. Macrophages in obese adipose tissue form crown-like structures around stressed or dying adipocytes [7]. They release inflammatory mediators such as tumour necrosis factor-alpha, interleukin-6, interleukin-1β, and monocyte chemoattractant protein-1. These cytokines interfere with insulin signalling and promote systemic inflammation. Adipose inflammation in obesity is now recognized as a distinct chronic inflammatory state that contributes directly to insulin resistance [8]. Adipose tissue also releases adipokines, including leptin, adiponectin, resistin, visfatin, and others. Adipokines are important regulators of metabolic inflammation and potential therapeutic targets in obesity-related metabolic disease [9]. Leptin usually increases with fat mass and has effects on appetite, energy expenditure, immunity, and inflammation. In obesity, leptin resistance may develop, reducing its metabolic effectiveness while preserving some pro-inflammatory actions [10]. Adiponectin, in contrast, is generally anti-inflammatory and insulin-sensitizing, but its levels often decline in obesity and type 2 diabetes. The combination of high leptin, low adiponectin, elevated free fatty acids, and increased inflammatory cytokines creates a hormonal-immune environment that promotes insulin resistance and chronic inflammation [11-16].

Endocrine Mechanisms Linking Obesity to Diabetes

Obesity promotes type 2 diabetes mainly through insulin resistance and pancreatic β-cell stress. In insulin-sensitive tissues, insulin normally promotes glucose uptake, suppresses hepatic glucose production, and regulates lipid metabolism [17-23]. In obesity, elevated free fatty acids, inflammatory cytokines, ectopic lipid accumulation, mitochondrial dysfunction, and oxidative stress disrupt insulin signalling pathways. Skeletal muscle becomes less efficient at glucose uptake, the liver continues producing glucose despite insulin presence, and adipose tissue releases more free fatty acids [24-27]. Inflammatory mediators interfere with insulin receptor signalling by activating intracellular stress pathways such as c-Jun N-terminal kinase and nuclear factor-kappa B. These pathways impair insulin receptor substrate function and reduce downstream signalling [28-32]. Adipose-derived free fatty acids also accumulate in the liver and muscle as diacylglycerols and ceramides, which worsen insulin resistance. The endocrine pancreas initially compensates by increasing insulin secretion. Over time, chronic nutrient excess, glucotoxicity, lipotoxicity, endoplasmic reticulum stress, oxidative injury, inflammation, and islet amyloid deposition impair β-cell function. As β-cell compensation fails, blood glucose rises and type 2 diabetes becomes clinically established [33-37]. This endocrine disruption has immune consequences. Hyperglycaemia promotes advanced glycation end-products, oxidative stress, endothelial activation, and inflammatory cytokine release [38-43]. Diabetes therefore reinforces the inflammatory state initiated by obesity. The result is a self-perpetuating cycle in which obesity causes insulin resistance, insulin resistance promotes hyperglycaemia, hyperglycaemia worsens oxidative and inflammatory injury, and inflammation further worsens insulin resistance [44-48].

Immune Activation in Obesity and Diabetes

The immune system is deeply involved in obesity and type 2 diabetes. In obesity, innate immune cells such as macrophages, neutrophils, dendritic cells, and mast cells accumulate in metabolic tissues. Adaptive immune cells, including T cells and B cells, also contribute to inflammation and insulin resistance [18,49-54]. These cells are activated by nutrient excess, saturated fatty acids, cellular stress, mitochondrial damage, gut-derived endotoxins, and adipocyte injury. A key concept is immunometabolism, which describes how immune-cell function is shaped by metabolic pathways [55-58]. Pro-inflammatory immune cells often rely on glycolysis, while anti-inflammatory and regulatory immune states are more closely linked to oxidative phosphorylation and fatty acid oxidation. In obesity and diabetes, nutrient excess reprograms immune cells toward inflammatory phenotypes. This promotes production of cytokines such as interleukin-6, tumour necrosis factor-alpha, interleukin-1β, and C-reactive protein [59-62]. Inflammasome activation is especially important. Nutrient excess, cholesterol crystals, mitochondrial stress, and reactive oxygen species may activate the NLRP3 inflammasome, increasing interleukin-1β production [63-65]. Interleukin-1β contributes to β-cell dysfunction, insulin resistance, and vascular inflammation. This explains why

obesity and diabetes are increasingly considered inflammatory metabolic diseases rather than purely endocrine disorders [66-67].

Anaemia of Chronic Disease: Core Pathophysiology

Anaemia of chronic disease is caused primarily by inflammation-driven disruption of iron metabolism and erythropoiesis. Unlike simple iron-deficiency anaemia, where total body iron is depleted, anaemia of chronic disease often involves iron sequestration. Iron may be present in storage sites, especially macrophages, but it is not adequately released to support haemoglobin synthesis [23]. Hepcidin is the central regulator of this process. It is produced mainly by the liver and controls iron movement by binding to ferroportin, the main iron exporter on enterocytes, macrophages, and hepatocytes. When hepcidin levels rise, ferroportin is internalized and degraded. This reduces dietary iron absorption from the gut and prevents macrophages from releasing recycled iron from old red blood cells [24]. Consequently, circulating iron falls, transferrin saturation declines, and the bone marrow receives insufficient iron for erythropoiesis. High hepcidin is a defining mechanism in anaemia of inflammation. Inappropriately elevated hepcidin causes iron-restricted erythropoiesis and contributes to poor response to oral iron therapy in inflammatory anaemias [25]. Inflammatory cytokines also directly suppress erythroid progenitor cells and shorten red blood cell survival. Tumour necrosis factor-alpha, interleukin-1β, and interferon-related pathways can reduce erythroid colony formation; promote apoptosis, and blunt erythropoietin signalling [26].

Erythropoietin Impairment and Diabetic Kidney Disease

The kidney plays a central role in red blood cell production through erythropoietin secretion. Erythropoietin stimulates erythroid progenitor survival, proliferation, and maturation in the bone marrow. In diabetes, especially when diabetic kidney disease develops, erythropoietin production becomes impaired [27]. Diabetic kidney disease damages glomeruli, tubules, interstitial tissue, and renal microvasculature. The erythropoietin-producing cells in the renal interstitium are affected by fibrosis, hypoxia, oxidative stress, inflammation, and capillary rarefaction [28]. As a result, the kidney may fail to increase erythropoietin appropriately when haemoglobin falls. Reviews of anaemia in diabetes identify diabetic kidney disease, erythropoietin impairment, inflammation, haematinic deficiencies, and hypoxia-related pathways as major contributors [29]. Inflammation also reduces marrow responsiveness to erythropoietin. Even when erythropoietin is available, cytokines may impair erythroid progenitor signalling. Hepcidin-mediated iron restriction further limits the effectiveness of erythropoietin because iron is required for haemoglobin synthesis [30]. Therefore, anaemia in obesity-related diabetes often reflects a combined defect: reduced erythropoietin supply, reduced erythropoietin responsiveness, and restricted iron availability [31].

Oxidative Stress, Mitochondrial Dysfunction, and Red Blood Cell Survival

Oxidative stress is another mechanism linking obesity, diabetes, and anaemia of chronic disease. Obesity increases oxidative stress through adipose inflammation, excess free fatty acids, mitochondrial overload, and endothelial dysfunction [32]. Diabetes increases oxidative stress through hyperglycaemia, advanced glycation end-products, mitochondrial reactive oxygen species, and impaired antioxidant defences [33]. Red blood cells are highly vulnerable to oxidative damage because they transport oxygen and contain haemoglobin, which can undergo oxidative reactions. In diabetes, haemoglobin glycation, lipid peroxidation, and membrane protein damage reduce red blood cell deformability and increase clearance by macrophages [34]. This shortens erythrocyte lifespan and increases the demand for new red blood cell production. Developing erythroid cells in the bone marrow also require healthy mitochondrial function for haem synthesis and maturation [35]. Mitochondrial dysfunction can impair iron utilization, haem formation, and erythroid differentiation. In inflammatory diabetes, oxidative stress and cytokines may damage bone marrow progenitors, reducing the ability to compensate for increased red blood cell destruction [36]. Taken together, the convergence of oxidative injury, inflammatory suppression, hepcidin-mediated iron restriction, and erythropoietin deficiency explains why anaemia in this setting is persistent, poorly responsive to single interventions, and carries significant implications for long-term clinical outcomes [37].

CONCLUSION

Obesity, type 2 diabetes, and anaemia of chronic disease are linked by a shared endocrine-immune network. Obesity transforms adipose tissue into an inflammatory endocrine organ that releases adipokines, cytokines, free fatty acids, and immune signals. These changes promote insulin resistance, pancreatic β-cell stress, hyperglycaemia, oxidative injury, and type 2 diabetes. Diabetes then amplifies inflammation through glucotoxicity, lipotoxicity, endothelial damage, mitochondrial dysfunction, and kidney injury. Chronic inflammation disrupts iron metabolism mainly through interleukin-6-driven hepcidin elevation, which degrades ferroportin, reduces iron absorption, traps iron in macrophages, and limits iron supply to the bone marrow. At the same time, cytokines suppress erythroid progenitors, diabetic kidney disease reduces erythropoietin production, and oxidative stress shortens red blood cell survival. Anaemia of chronic disease in obesity and diabetes should therefore be understood not as an isolated blood disorder but as a systemic manifestation of endocrine-immune dysregulation. Effective management requires

integrated attention to weight, glucose control, inflammation, kidney function, iron status, nutrition, and erythropoietic health.

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