

# INFLAMMATION AS A BRIDGE BETWEEN OBESITY AND PSORIASIS

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## Summary

*Psoriasis is a chronic inflammatory skin disorder with a complex autoimmune basis, characterized by keratinocyte hyperproliferation and a dermo-epidermal inflammatory infiltrate. Conversely, obesity represents a metabolic condition defined by the excessive accumulation of adipose tissue and is increasingly acknowledged as a state of low-grade chronic inflammation. In recent years, a strong bidirectional association between these two entities has emerged, suggesting the existence of a vicious cycle wherein systemic inflammation associated with obesity exacerbates psoriasis, and vice versa.*

*Adipose tissue secretes a range of proinflammatory cytokines, collectively termed adipokines – including tumor necrosis factor-alpha (TNF- $\alpha$ ), interleukin-6 (IL-6), leptin, and resistin – which contribute to a systemic proinflammatory milieu that may potentiate autoimmune disorders such as psoriasis. Furthermore, psoriasis itself is associated with elevated levels of inflammatory mediators that can disrupt metabolic homeostasis and promote visceral adiposity, thereby further aggravating obesity. This intricate interplay bears significant clinical relevance, impacting therapeutic responses, disease prognosis, and the risk of cardiovascular comorbidities.*

*Psoriasis and obesity thus share a common inflammatory pathogenesis, and elucidating the molecular pathways linking these conditions could foster the development of more effective, personalized therapeutic strategies targeting systemic inflammation in an integrated manner.*

*This review aims to synthesize the current understanding of the pathogenic role of adipokines and to explore the potential mechanisms through which inflammatory processes mediate the association between obesity and psoriasis.*

**Keywords:** obesity, psoriasis, adipocyte, adipokines, inflammation.

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## Introduction

Obesity is a chronic disease affecting millions of adults and children worldwide. The prevalence of excessive weight gain continues to rise, exerting a negative impact on population health. Epidemiological studies over time have demonstrated that a high body mass index (BMI) constitutes a significant risk factor for numerous chronic diseases, including cardiovascular, renal,

musculoskeletal disorders, and diabetes mellitus [1,2]. The pathogenesis of obesity has been linked to behavioral, genetic, medical, sociodemographic, as well as inflammatory and immunological factors. Inflammation plays a pivotal role through adipose tissue remodeling and cytokine secretion, contributing to insulin resistance, atherosclerosis, and other associated conditions, including psoriasis [3].

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Psoriasis is a chronic, immune-mediated inflammatory skin disease that manifests at the cutaneous, articular, or both levels, affecting approximately 2–4% of the global population [4]. The disease is characterized by an imbalance between keratinocyte proliferation and differentiation, with patients presenting erythematous, scaly patches and plaques in various areas of the body [5].

Numerous studies have established an association between psoriasis and obesity, although the exact mechanism underlying this relationship remains unclear and warrants further investigation. Some researchers have proposed that the immunological component, specifically the role of adipokines, plays a critical role in linking the pathogenic processes of psoriasis and obesity [6-8].

## Materials and Methods

This work constitutes a narrative review of the existing scientific literature, aiming to explore and synthesize current data regarding the shared inflammatory mechanisms implicated in psoriasis and obesity. The study was designed to highlight the pathological connections between these two conditions, with a particular focus on the role of inflammatory mediators, adipokines, and systemic and local immunological alterations.

For this analysis, a systematic search of the scientific literature was conducted using major international medical databases, including PubMed, ScienceDirect, and Google Scholar. A series of keywords and their logical combinations in English – such as: obesity, psoriasis, inflammation, adipocyte, adipokines, cytokines, TNF-alpha, IL-6, leptin, systemic inflammation – were employed. Search terms were entered individually and combined using Boolean operators (AND/OR) to maximize the relevance and specificity of the results.

Inclusion criteria comprised: articles published in peer-reviewed journals, experimental and observational studies, systematic reviews, and meta-analyses investigating the relationship between obesity and psoriasis from the perspective of inflammatory changes.

During the selection phase, titles and abstracts were initially screened for relevance, followed by a full-text assessment of articles meeting the preliminary inclusion criteria. Particular attention was given to recent clinical and experimental studies that investigated the increased expression of specific cytokines and adipokines in both conditions, as well as studies analyzing the interaction between immune cells and adipose tissue within the context of chronic inflammation.

Through this methodological approach, the review aims to provide an integrated perspective on the common inflammatory mechanisms shared by psoriasis and obesity, with potential implications for future therapeutic strategies and multidisciplinary patient management.

## Adipose Tissue and Its Alterations in Obesity

The adipocyte plays a central role in the complex process of chronic inflammation observed in obesity. Primarily, adipose tissue serves as an energy reservoir by storing triglycerides; however, it is also recognized as a major endocrine organ, secreting a wide array of cytokines and adipokines [9]. The secretion of hormonal factors underlines the critical role of adipocytes in the inflammatory mechanisms associated with obesity: adipocyte hypertrophy leads to tissue hypoxia and the subsequent release of increased amounts of proinflammatory adipokines. To date, over 600 adipokines have been identified [10]. The principal alterations in adipocytes associated with obesity are briefly illustrated in *Figure 1*.

The altered production of proinflammatory molecules secreted by adipose tissue in obesity has been demonstrated to play a crucial role in the development of obesity-related metabolic complications. Particularly noteworthy are proteins whose secretion is increased in obesity and which promote inflammatory processes, including IL-6, TNF- $\alpha$ , inducible nitric oxide synthase (iNOS), transforming growth factor-beta 1 (TGF- $\beta$ 1), C-reactive protein (CRP), soluble intercellular adhesion molecule (sICAM), and

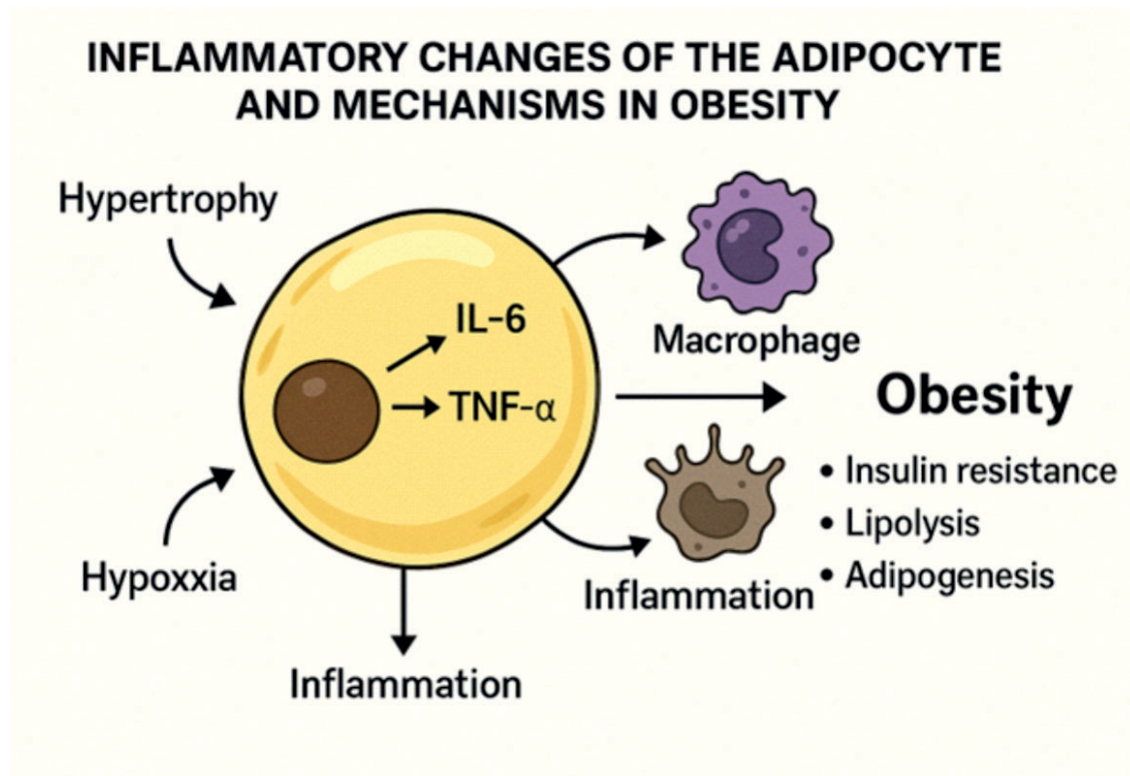


Figure 1. Adipocyte Alterations in Obesity – AI Generated

monocyte chemoattractant protein-1 (MCP-1) [11,12].

The cascade of inflammatory changes resulting from adipocyte hypertrophy in obesity also includes the recruitment of macrophages and immune cells mediated by the secretion of MCP-1. MCP-1 acts as a potent chemoattractant for macrophages, promoting their infiltration into adipose tissue where they undergo a phenotypic shift from an anti-inflammatory (M2) to a proinflammatory (M1) phenotype, characterized by the secretion of cytokines such as tumor necrosis factor-alpha (TNF- $\alpha$ ), interleukin-1 beta (IL-1 $\beta$ ), interleukin-6 (IL-6), and reactive oxygen species (ROS) [13-15].

Numerous studies have emphasized the pivotal role of adipose tissue macrophages, whose accumulation is proportional to body weight gain and is closely correlated with the development of insulin resistance through the active secretion of proinflammatory cytokines that impair insulin action in adipocytes [16,17].

### Pathogenic Factors Connecting Obesity and Psoriasis

The associations between obesity and psoriasis have been the subject of extensive research, with particular focus on the potential biological factors underpinning the development of both diseases. Some studies have acknowledged the critical role of adipokines in linking the pathogenic pathways of obesity and psoriasis [18].

As previously outlined, adipocytes are the principal cells of adipose tissue, and beyond their role in energy storage in the form of fat, they serve as an important source of hormonal factors involved in systemic inflammatory processes, such as adipokines [9]. These proinflammatory properties underscore their involvement in the pathogenesis of inflammatory diseases, including psoriasis. Research has demonstrated a relationship between psoriasis and other metabolic comorbidities, such as dyslipidemia and obesity, with a higher prevalence of obesity observed among patients with psoriasis compared to the

general population. Nonetheless, the underlying mechanisms remain incompletely understood [18,19]. The study of various adipokines implicated in the inflammatory process of psoriasis remains an active area of research.

Another important link between the inflammatory processes of psoriasis and obesity is represented by adiponectin levels. Adiponectin is an anti-inflammatory cytokine with a regulatory role in atherosclerosis. Lower adiponectin levels have been observed in patients with psoriasis compared to healthy individuals, and an inverse relationship between adiponectin levels and obesity has been consistently reported [20].

Additionally, elevated leptin levels are noteworthy in both conditions. Leptin is an adipokine primarily involved in regulating body weight; however, its increase is associated with arterial thrombosis. Leptin also exerts proinflammatory effects by activating the NF- $\kappa$ B (nuclear factor

kappa-light-chain-enhancer of activated B cells) pathway, which plays a key role in regulating immune and inflammatory responses, as well as by promoting the production of TNF- $\alpha$ , IL-6, and IL-17. NF- $\kappa$ B can be considered a central node in the obesity-psoriasis interaction, as this pathway can be activated both by proinflammatory cytokines secreted by adipocytes and by those produced by keratinocytes [22]. *Figure 2* briefly illustrates the major adipocyte alterations observed in psoriasis and their link to psoriatic inflammation.

While some authors suggest that obesity likely precedes or coexists with psoriasis, other studies advocate for an increased risk of newly developed obesity in patients already diagnosed with psoriasis. Several additional factors beyond immune mechanisms have been implicated. Among these, behavioral factors are notable, particularly the possible reluctance of psoriasis patients to engage in physical activities where

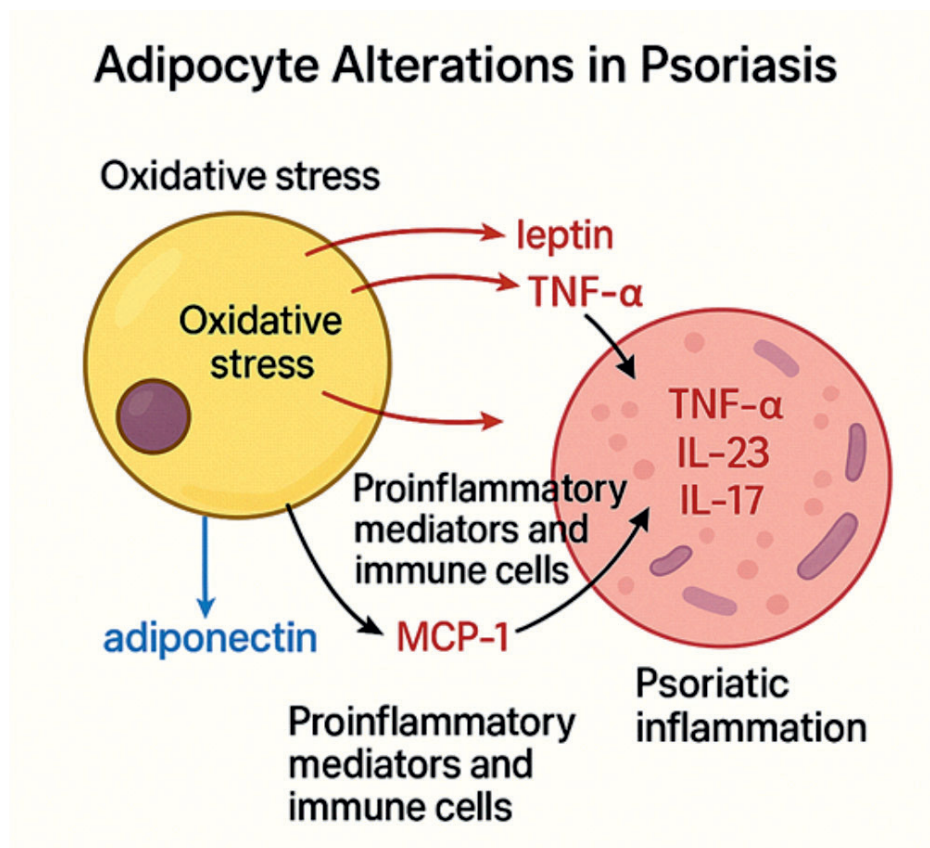


Figure 2. Adipocyte Alterations in Psoriasis – AI Generated

skin lesions may be visible to others. Furthermore, pharmacological treatments for psoriasis have been implicated in weight gain, although more complex studies are needed to fully determine the impact of systemic therapies on the development of obesity [23,24].

Considering the extensive inflammatory alterations observed in both obesity and psoriasis, it becomes crucial to further elucidate the role of adipokines, with the hypothesis that these molecules may explain the mechanistic link between the two conditions. A summary of the

key data regarding the major molecules involved in the inflammatory mechanisms common to both obesity and psoriasis is presented in *Table 1*.

### Adipokine-Targeted Therapy

Various studies have demonstrated that weight-loss interventions in patients suffering from obesity may also improve the pathogenesis of psoriasis. Moreover, behavioral modifications related to dietary control and increased physical activity in individuals affected by both obesity

**Table 1. Major Inflammatory Molecules Involved in Obesity and Psoriasis [11,18,25–27]**

Molecule	Localization/ Type	Role/ General Effects	Effects in Obesity	Effects in Psoriasis
<b>Leptin</b>	Adipocytes	Pro-inflammatory - Activates NF- $\kappa$ B pathway - Stimulates Th1 response	- Increased - Induces inflammation - Increases MCP-1, IL-6, TNF- $\alpha$ - Positively correlated with BMI	Increased - Stimulates Th1 cells and keratinocytes
<b>Adiponectin</b>	Adipocytes	Anti-inflammatory - Inhibits NF- $\kappa$ B - Decreases TNF- $\alpha$ , IL-6	- Decreased - Loss of anti-inflammatory protection	Decreased - Loss of anti-inflammatory protection
<b>Resistin</b>	Adipocytes Macrophages	Pro-inflammatory - Activates NF- $\kappa$ B pathway - Stimulates TNF- $\alpha$ , IL-6, MCP-1 production	- Increased - Sustains inflammation	- Increased - Enhances cytokine production
<b>Visfatin</b>	Visceral adipose tissue	Pro-inflammatory - Stimulates TNF- $\alpha$ , IL-6 - Activates T lymphocytes T	- Increased - Activates NF- $\kappa$ B pathway - Correlated with abdominal obesity - Induces dyslipidemia	- Increased - Contributes to local inflammation
<b>TNF-<math>\alpha</math></b>	Adipocytes Macrophages	Pro-inflammatory cytokine = Key mediator of inflammation - Activates NF- $\kappa$ B pathway - Stimulates IL-6 and iNOS production	- Increased - Positively correlated with BMI - Induces dyslipidemia	- Increased - Promotes inflammation and keratinocyte activation - Target for biological therapies
<b>IL-6</b>	Adipocytes Macrophages	Pro-inflammatory - Stimulates Th17 cells - Activates C-reactive protein (CRP) production	- Increased - Elevates CRP levels - Induces insulin resistance	- Increased - Sustains inflammatory response
<b>IL-17</b>	Th17 lymphocytes	- Stimulates production of IL-6 and TNF- $\alpha$ - Activates keratinocytes	- Activated by obesity-related inflammation	- Major contributor to epidermal inflammation
<b>NF-<math>\kappa</math>B</b>	Transcription factor	- Regulates the expression of inflammatory genes	- Activated by leptin and resistin - Promotes inflammation	- Sustains inflammatory processes
<b>MCP-1</b>	Adipocytes Endothelium	- Potent chemoattractant for macrophages	- Increased - Promotes macrophage infiltration	- Increased - Facilitates inflammatory cell recruitment to lesions
<b>PCR</b>	Liver	- Systemic inflammatory marker	- Elevated	- Elevated in severe forms of psoriasis
<b>iNOS</b>	Macrophages Keratinocytes	- Produces nitric oxide (NO), contributes to oxidative stress	- Increased - Heightened oxidative stress	- Increased in psoriatic lesions

and psoriasis have resulted in a significantly lower mean body weight compared to control groups. Additionally, a relevant observation for our review is that some authors have noted that low-calorie diets could decrease serum leptin levels while increasing adiponectin levels, suggesting that lifestyle changes may serve as valuable adjuncts to pharmacological therapy in obese patients with psoriasis [28,29].

Psoriasis requires lifelong therapeutic management. However, pharmacological treatments may not be suitable for all patients, leading to consideration of non-pharmacological alternatives. In this context, several studies have emphasized the beneficial effects of low-calorie diets. Results have been positive, indicating that dietary interventions, either alone or in combination with medications, may achieve improved clinical outcomes and could be considered successful adjuvant therapies [30]. For instance, a Mediterranean diet – rich in antioxidants, vitamins, and minerals – has been shown to positively influence the management and treatment of psoriasis in both adults and children [31,32]. Moreover, such diets may exert anti-inflammatory effects by modulating the gut microbiota and regulating levels of C-reactive protein (CRP), cytokines, TNF- $\alpha$  receptors, and chemokines [33].

It is also important to mention that numerous studies have suggested targeting adipokine levels, indicating that weight-loss strategies in patients with obesity and psoriasis should primarily focus on reducing inflammation and adipokine secretion. Recent research has shown that psoriasis therapy with methotrexate led to increased serum adiponectin levels. Similar findings have been reported with anti-TNF $\alpha$  agents, which additionally resulted in a significant reduction in serum IL-6 levels [24,34,35].

Obesity and psoriasis share a strong association in terms of therapeutic response, with inflammation playing a central role in this interplay. Notably, recent studies have suggested that obesity may reduce the efficacy of biological therapies and amplify adverse effects associated

with conventional treatments [36]. Thus, weight management in patients diagnosed with psoriasis becomes critically important, as the effectiveness of biological therapies decreases in direct proportion to the increase in BMI [37]. Studies have revealed that psoriasis patients with elevated BMI values achieved lower rates of PASI75 and PASI90 responses to therapy. Furthermore, behavioral factors such as alcohol consumption, smoking, family history of psoriasis, and previous use of biologics have also been shown to influence PASI scores [38-40]. Another study supporting the notion that obesity impacts the efficacy of biologic therapies in psoriasis is that of Pirro et al., who concluded that anti-interleukin therapies are more affected in patients with BMI  $\geq 30$  kg/m<sup>2</sup> compared to anti-TNF agents [41]. Conversely, the study by Hung et al. demonstrated that although obese patients initially exhibited a reduced therapeutic response, this effect diminished after four weeks of treatment with Guselkumab[42].

Based on the above, it is evident that targeting adipokines could support the management of both psoriasis and obesity; however, further extensive and in-depth studies are necessary to conclusively determine the therapeutic effects of such approaches.

## Conclusions

In conclusion, our review highlights a complex link between obesity and psoriasis, fundamentally mediated by adipokines. Study findings suggest that these two diseases may stem from a shared pathophysiological background. Future research should focus on this relationship to facilitate a more comprehensive approach to therapeutic and preventive strategies. Adipokines play a critical role in the inflammatory cascade observed in both obesity and psoriasis, and plasma level assessments, considering endocrine factors, are essential for a better understanding of these two interconnected diseases.

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Conflict of interest  
NONE DECLARED

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