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The Relationship Between Metabolic Syndrome Components (Central Obesity, Dyslipidemia, Hypertension, and Hyperglycemia) and Disease Severity in Psoriasis Vulgaris: A Literature Review

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ABSTRACT

Psoriasis vulgaris, a chronic immune-mediated inflammatory skin condition, is increasingly recognized as a systemic disorder with strong links to metabolic syndrome (MetS) and its individual elements: central obesity, dyslipidemia, hypertension, and hyperglycemia. Whether the magnitude of these metabolic derangements mirrors the extent of cutaneous involvement, more frequently quantified via the Psoriasis Area Severity Index (PASI), remains a clinically relevant yet unresolved question. Synthesizing recent literature published between 2021 and 2026, this narrative review investigates the specific relationship between each MetS component and psoriatic severity. The accumulated evidence demonstrates component-specific patterns rather than a uniform association. Impaired glucose homeostasis and insulin resistance demonstrate the most consistent positive correlation with PASI, characterized by a graded increase in fasting blood glucose across severity strata and an independent prognostic contribution of hyperglycemia to disease exacerbations. Dyslipidemia and central obesity/BMI are likewise positively associated with severity in most studies, with Mendelian randomization supporting a causal contribution of adiposity. Conversely, hypertension displays the weakest and least consistent relationship, with several large-scale investigations failing to observe a severity-dependent gradient. At the composite syndrome level, findings diverge regarding a direct severity gradient. Overlapping inflammatory pathways (TNF-α, IL-17, IL-6), adipokine dysregulation, and insulin resistance offer a plausible biological basis for these links. Consequently, routine cardiometabolic screening is warranted for all patients with psoriasis, since skin severity alone is an unreliable proxy for systemic metabolic risk.

Keywords: psoriasis vulgaris; metabolic syndrome; severity of psoriasis vulgaris

INTRODUCTION

Psoriasis vulgaris, as a chronic, immune-mediated inflammatory dermatosis affecting roughly 2–3% of the global population, is driven by T-helper (Th1/Th17) activation and pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin (IL)-17, and IL-23 that also contribute to systemic inflammation [1]. Once regarded as a disease limited to the skin and joints, psoriasis is now recognized as a systemic condition with multiple comorbidities, among which metabolic syndrome is the most frequent [1, 2].

Metabolic syndrome is a cluster of interrelated cardiovascular risk factors comprising central (abdominal) obesity, dyslipidemia, hypertension, and insulin resistance or hyperglycemia [3]. Reported prevalence among people with psoriasis ranges widely, from approximately 20% to 50%, depending on disease severity and the population studied [22]. Meta-analytic data confirm that psoriasis is significantly associated with dyslipidemia (odds ratio [OR] 1.63) and obesity (OR 1.70) compared with controls [4]. The association is widely attributed to shared inflammatory pathways, adipokine dysregulation, endothelial dysfunction, and common genetic and lifestyle risk factors [3, 25]. A central and clinically consequential question is whether the burden of these metabolic abnormalities scales with cutaneous disease severity, typically quantified by PASI or body surface area (BSA). If severity predicts metabolic risk, PASI could help triage patients for screening; if not, metabolic surveillance must be applied broadly regardless of skin findings. The literature, however, is heterogeneous: some components appear to track

closely with severity while others do not, and syndrome-level results conflict [6]. This review examines the evidence for each of the four core metabolic syndrome components central obesity, dyslipidemia, hypertension, and hyperglycemia- in relation to psoriasis vulgaris severity, drawing primarily on studies from 2021 to 2026.

METHOD

This is a narrative (non-systematic) literature review. Relevant English-language articles were identified through structured searches of biomedical literature using combinations of the terms "psoriasis," "psoriasis vulgaris," "metabolic syndrome," "central obesity," "waist circumference," "body mass index," "dyslipidemia," "lipid profile," "hypertension," "blood pressure," "hyperglycemia," "fasting glucose," "insulin resistance," "disease severity," and "PASI." Priority was given to primary observational studies (cross-sectional, case-control, cohort), systematic reviews, meta-analyses, Mendelian randomization studies, and mechanistic reviews.

Consistent with the requested scope, the synthesis emphasizes evidence published between 2021 and 2026, supplemented by the clinical articles supplied for this review. Studies were considered relevant if they examined one or more metabolic syndrome components, or metabolic syndrome as a whole, in patients with psoriasis (ideally psoriasis vulgaris or chronic plaque psoriasis) and reported, where possible, a relationship with a validated severity measure (PASI or BSA). Findings were organized thematically by metabolic component rather than by study, and both concordant and discordant results were retained to reflect the true state of the evidence. In the reference list, articles newly added beyond the supplied set are explicitly annotated as recent (2021–2026).

RESULT AND DISCUSSION

Central Obesity and Psoriasis Severity

Psoriasis and obesity exhibit a well-established, bidirectional epidemiological connection, with meta-analytical findings confirming a heightened risk of obesity among psoriatic individuals compared to non-affected controls (OR 1.70, 95% CI 1.43–2.02) [4]. Among the various metabolic markers, adiposity represents the most reproducibly documented anthropometric parameter linked to disease activity. According to a retrospective evaluation involving 208 patients diagnosed with psoriasis vulgaris, individuals classified as overweight or obese exhibited significantly elevated PASI scores relative to their normal-weight counterparts (median 18.90 vs. 15.90; $p = 0.032$), while body mass index (BMI) displayed a positive linear relationship with PASI across the entire cohort ($p = 0.016$) [11, 7]. In agreement with these observations, an observational study enrolling 147 patients with chronic plaque psoriasis demonstrated that higher BMI was positively correlated with both PASI and BSA metrics, specifically within the overweight and obese subset [8].

Emerging genomic data further corroborates a potential causal involvement. By integrating bidirectional Mendelian randomization across 366,776 subjects with regression modeling in 1,979 newly diagnosed cases, a multicenter investigation established that elevated BMI causally increased susceptibility to psoriasis (OR ~2.3). Additionally, every incremental unit increase in BMI yielded an approximate 6% rise in the odds of presenting severe skin involvement (PASI ≥ 10) alongside an average PASI elevation of 0.25 points ($p < 0.001$) [9]. These results were corroborated by a separate two-sample Mendelian randomization study reinforcing the causal link between BMI and psoriasis risk [10], whereas case-control analyses demonstrated that patients presenting with higher disease severity disproportionately exhibited greater waist circumference measurements [11].

Nevertheless, this severity correlation is not consistently observed across all clinical settings. Selected patient cohorts have shown no statistically significant association between PASI scores and either BMI or waist circumference, indicating that confounding variables or population-specific traits may modify the impact of adiposity [5]. On balance, central adiposity and body mass index remain positively, and likely causally, tied to enhanced cutaneous severity in the majority of literature, rendering adiposity the most reliable anthropometric marker of disease burden [7, 9]. From a pathophysiological standpoint, visceral adipose tissue serves as an active endocrine organ secreting key pro-inflammatory mediators such as TNF-α and IL-6. These cytokines directly reinforce the pathogenic inflammatory cascade of psoriasis, generating a self-sustaining pathological feed-forward loop between cutaneous disease and systemic metabolic dysfunction [9].

Dyslipidemia and Psoriasis Severity

Psoriasis displays an independent correlation with an atherogenic lipid phenotype, as meta-analytic evaluations demonstrate a marked link between psoriatic disease and dyslipidemia (OR 1.63, 95% CI 1.42–1.88) [4]. Beyond this baseline comorbidity, specific lipid aberrations, encompassing heightened triglyceride levels, elevated total and LDL cholesterol fractions, and diminished HDL concentrations, demonstrate a clear pattern of worsening in parallel with increased cutaneous disease activity. In a cross-sectional investigation involving 250 individuals diagnosed with psoriasis, total cholesterol, LDL, and triglyceride parameters were markedly elevated, whereas HDL levels were systematically reduced, showing more profound lipid disturbances in severe cases than in mild presentations [12].

Correspondingly, an analytical study published in 2025 identified a significantly higher frequency of dyslipidemia and hypertriglyceridemia among patients with severe cutaneous involvement [13], a finding echoed by case-control data indicating elevated serum triglycerides in those presenting with more advanced skin disease [11].

Reaffirming this severity gradient, a narrative synthesis documented increased odds of dyslipidemic alterations in severe psoriatic cases relative to milder presentations [14].

Nevertheless, the correlation between lipid parameters and disease severity displays variability across differing methodological frameworks. Although meta-analytic evidence confirms the systemic association between psoriasis and dyslipidemia (OR 1.63), it contextualizes this predominantly as a binary case-control phenomenon rather than a strictly PASI-dependent continuum [4]. Furthermore, a mechanistic appraisal of lipid dysregulation highlighted qualitative biochemical alterations, such as elevated circulating soluble LOX-1 levels correlating with lesion extent, over conventional lipid panels tracking PASI scores, while additionally observing clinical reductions in psoriatic severity following statin interventions [15]. Pro-inflammatory mediators shared between both conditions, notably TNF-α and IL-6, impair physiological lipid homeostasis; concurrently, weight reduction strategies have been shown to ameliorate cutaneous lesions, thereby connecting the dyslipidemia-severity axis back to systemic inflammatory burden and central adiposity [16].

Hypertension and Psoriasis Severity

From an epidemiological perspective, psoriasis exhibits a recognized co-occurrence with elevated blood pressure, showing a pooled hypertension prevalence of approximately 32% across 200 observational investigations that is largely attributed to shared vascular inflammatory processes [17]. Nevertheless, whether systemic blood pressure correlates proportionally with the extent of skin involvement remains considerably ambiguous. Among the four classic components of metabolic syndrome, hypertension displays the weakest and most discordant correlation with psoriasis severity scores. An extensive meta-analysis encompassing 200 observational studies conducted between 2006 and 2023 demonstrated virtually equivalent hypertension rates between mild and severe clinical subgroups (31.71% vs. 33.19%), demonstrating the absence of a distinct severity-dependent gradient [17]. A 2025 systematic review reached comparable conclusions regarding the mixed nature of current data, highlighting that while isolated studies suggest higher correlations in severe presentations, a major UK population-based study observed no statistically significant trend across severity strata, prompting recommendations for further prospective evaluation [18].

Conversely, contrasting observations have been documented in smaller clinical investigations. A prospective study reported a stepwise increase in hypertension frequency across advancing severity tiers (2% in mild, 12% in moderate, and 13% in severe cohorts) [19], an observation supported by a 2025 analytical inquiry identifying the highest hypertension rates in severe cases [7].

Additionally, a narrative synthesis highlighted meta-analytic findings indicating a progressive increase in hypertension risk relative to disease severity (OR 1.30 for mild vs. 1.49 for severe disease) [14]. In contrast, alternative cohort evaluations detected no meaningful disparity in hypertension prevalence when comparing overweight or obese psoriatic patients to normal-weight controls [7]. Synthesizing these findings, blood pressure data remain the most variable across the four metabolic parameters: large-scale pooled analyses reveal negligible severity gradients, whereas smaller single-center studies periodically report positive correlations [17, 18]. Irrespective of skin severity, psoriasis maintains a broader association with heightened overall hypertension risk, mediated primarily through endothelial dysfunction, systemic oxidative stress, and dysregulation of the renin–angiotensin cascade [20].

Hyperglycemia, Insulin Resistance, and Psoriasis Severity

Psoriasis is linked to an elevated risk of developing type 2 diabetes mellitus and altered glycemic control, a clinical relationship that becomes increasingly pronounced as skin disease severity advances [20]. In alignment with this observation, glucose dysregulation demonstrates arguably the most consistent positive association with psoriatic severity among all individual metabolic components. Fasting plasma glucose levels progressively increase alongside rising PASI scores, displaying marked statistical significance across severity strata ($p < 0.001$) in an investigation enrolling 250 patients [12]. Likewise, a 2025 analytical evaluation observed a higher frequency of impaired fasting glucose and overt hyperglycemia among individuals presenting with severe cutaneous involvement [13], a pattern further corroborated by case-control data indicating higher fasting glucose concentrations in more severe disease cohorts [11].

Insulin resistance appears to function as the fundamental mechanistic bridge connecting these conditions. A targeted review of psoriatic immunometabolism revealed that insulin resistance correlates with disease severity independently of systemic metabolic syndrome, with body mass index and waist circumference exerting no confounding effect on this relationship, suggesting that disease extent directly influences glucose homeostasis beyond the influence of adiposity [5]. From a prognostic standpoint, a 15-year retrospective cohort study tracking 201 psoriatic patients with co-existing type 2 diabetes or impaired fasting glucose demonstrated that unmanaged hyperglycemia ($HbA1c \geq 7\%$) served as an independent predictor of psoriasis exacerbations specifically within the severe subgroup (adjusted OR 1.905, 95% CI 1.328–2.731), whereas no such risk elevation was detected in mild presentations [21]. Across cross-sectional, pathophysiological, and longitudinal dimensions, altered glucose handling and insulin resistance represent the metabolic parameters most robustly and reproducibly linked to psoriatic disease activity,

including an independent impact on long-term disease flare-ups [5, 21]. Biologically, sustained hyperinsulinemia can stimulate keratinocyte and dermal fibroblast proliferation, whereas high levels of pro-inflammatory cytokines characteristic of severe psoriasis impair systemic insulin signaling cascades, establishing a bidirectional pathological framework [5].

Metabolic Syndrome and Psoriasis Severity

At the composite syndrome level, the clinical evidence is fundamentally divided. Several investigations demonstrate a clear dose-dependent association: an analytical study published in 2025 observed that psoriatic patients with metabolic syndrome presented with significantly higher PASI scores, while PASI metrics increased in a stepwise manner alongside the accumulation of individual metabolic components [13]. Similarly, a 2025 systematic review and meta-analysis identified a progressive escalation in metabolic syndrome risk parallel to disease severity, with severe psoriasis (PASI ≥ 10) conferring an odds ratio of 2.247 (95% CI 1.267–3.985) compared with controls, alongside a positive linear trend connecting PASI scores to metabolic syndrome incidence [22]. Supporting these findings, case-control evaluations indicate that more advanced cutaneous disease may function as a marker of pronounced systemic metabolic impairment [11].

Conversely, numerous investigations detect no equivalent severity-dependent continuum. A cross-sectional inquiry evaluating psoriatic individuals with and without metabolic syndrome revealed no statistically significant differences in PASI scores between the two cohorts [23]. In a similar vein, a 2025 cross-sectional study documented virtually identical meaning PASI values among psoriatic patients irrespective of metabolic syndrome status (10.34 vs. 11.76; p = 0.99), implying a potential decoupling between localized cutaneous inflammation and systemic metabolic burden [24]. Furthermore, a comprehensive multimorbidity clustering analysis identified a distinct cardiometabolic comorbidity pattern but failed to link its presence directly to PASI or BSA indices [6]. Reinforcing this perspective, a 2026 cohort study characterized by a high overall metabolic syndrome prevalence (54%) concluded that skin severity metrics alone provide an unreliable surrogate for underlying systemic risk [1].

The most scientifically robust synthesis suggests that the relationship between metabolic syndrome and psoriatic severity is component-dependent rather than uniform: parameters of glucose regulation, and to a lesser degree adiposity and lipid profiles, consistently mirror PASI variations, whereas hypertension status and composite-syndrome metrics frequently do not [13, 17, 24]. This observed heterogeneity likely stems from variations in clinical severity cutoffs (such as PASI ≥ 10), population-specific ethnic and body composition demographics, cross-sectional versus prospective study designs, and inconsistent adjustment for key confounding variables such as baseline BMI and systemic therapeutic interventions.

Shared Pathogenic Mechanism

The biological plausibility of a correlation with disease severity is anchored in shared systemic inflammatory cascades. Key pro-inflammatory cytokines central to psoriatic pathogenesis, specifically TNF-α, IL-17, IL-6, and IL-23, propel both localized cutaneous lesions and systemic metabolic derangements, thereby fostering insulin resistance, endothelial dysfunction, and atherogenic lipid profiles [3, 13]. Complementary pathophysiological mechanisms encompass adipokine imbalances (characterized by diminished adiponectin levels and elevated leptin concentrations, with PASI scores exhibiting an inverse relationship with circulating adiponectin), endoplasmic reticulum stress, heightened reactive oxygen species generation, and alterations in gut microbiota composition [5, 25]. Given that the activity of these shared biochemical pathways scales with inflammatory intensity, more severe skin manifestations could theoretically exert a greater metabolic burden, a concept that aligns with the stronger severity gradients observed for altered glucose metabolism relative to blood pressure metrics [25].

Conclusion and Suggestions

The relationship between metabolic syndrome and the severity of psoriasis vulgaris is fundamentally component-dependent rather than uniform. Altered glucose regulation and insulin resistance represent the metabolic parameters most reproducibly linked to elevated PASI metrics, with hyperglycemia exerting an independent prognostic influence on disease flare-ups among patients presenting with severe cutaneous involvement [21]. Dyslipidemic features, particularly hypertriglyceridemia and reduced HDL levels, alongside central obesity and elevated BMI show positive correlations with disease extent across the majority of literature, with Mendelian randomization analyses reinforcing a potential causal involvement of body adiposity [9]. Conversely, hypertension exhibits the least consistent severity-dependent relationship, while composite metabolic syndrome status aligns with PASI scores in select patient populations but fails to do so in others [17]. On balance, although psoriasis maintains a robust overall link with cardiometabolic comorbidities, cutaneous disease severity alone functions as an imprecise surrogate for underlying systemic metabolic risk.

These conclusions yield several important clinical and research considerations. First, comprehensive cardiometabolic surveillance (encompassing waist circumference measurements, lipid profiling, blood pressure monitoring, and fasting glucose or HbA1c testing) should be extended to all patients diagnosed with psoriasis vulgaris regardless of lesion coverage, as mild skin involvement does not preclude significant underlying metabolic burden [2, 1]. Second, glycemic control warrants prioritized clinical vigilance as the specific component most strongly tied to disease activity and exacerbation

risk, supporting the routine integration of glycemic monitoring into standard psoriatic care. Third, therapeutic interventions focusing on weight reduction and anti-inflammatory pathways may target shared pathophysiological cascades, offering dual benefits for both cutaneous manifestations and systemic metabolic health. Finally, future research requires adequately powered, multi-center prospective studies across diverse ethnic groups, utilizing standardized PASI stratifications and rigorous adjustments for body mass index and systemic treatments, to delineate causal directions and resolve current discrepancies at the composite syndrome level.

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