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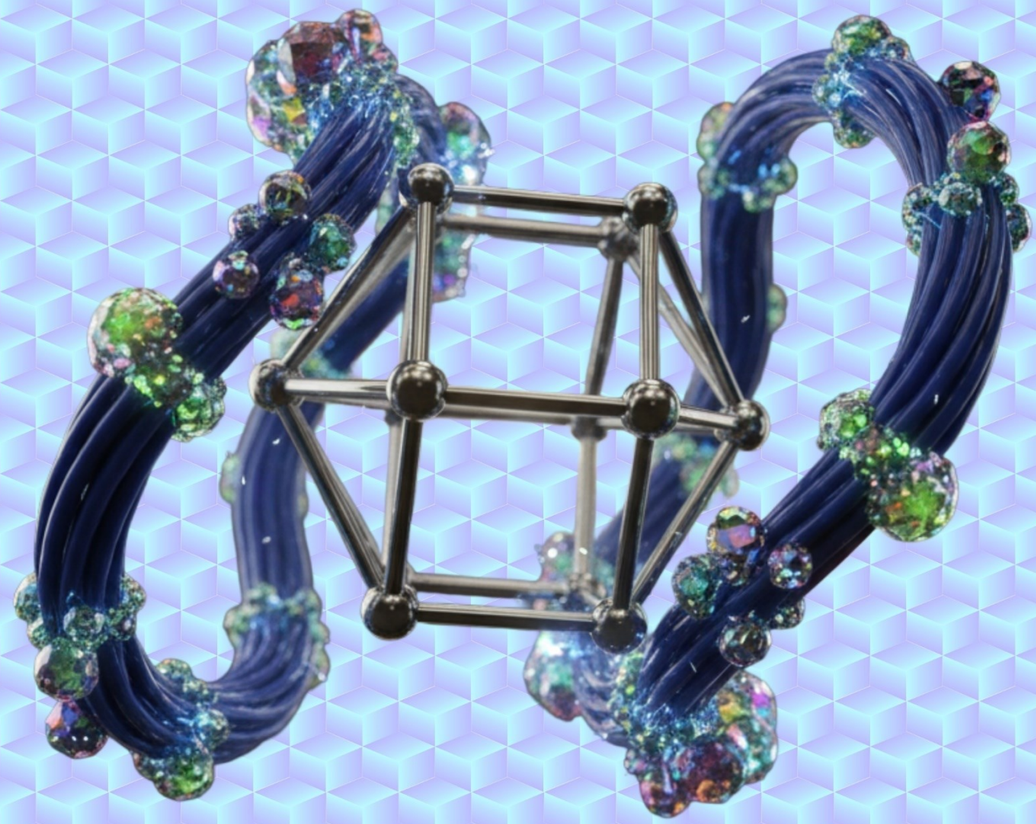

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Chronic Inflammatory Dermatoses and Metabolic Dysregulation: Evidence for a Systemic Disease Model

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ABSTRACT

Chronic inflammatory skin diseases have increasingly been recognized as systemic disorders with clinically significant extracutaneous manifestations. Among these, the association between chronic cutaneous inflammation and metabolic syndrome has emerged as a relevant and reproducible pattern across epidemiological, clinical, and mechanistic studies. This review synthesizes current evidence examining the bidirectional relationship between chronic inflammatory dermatoses—primarily psoriasis and moderate-to-severe atopic dermatitis—and metabolic syndrome, including its key components: obesity, insulin resistance, dyslipidemia,

hypertension, and type 2 diabetes mellitus. Population-based studies and meta-analyses consistently demonstrate a higher prevalence of metabolic syndrome and related metabolic abnormalities in patients with chronic inflammatory skin disease, with stronger associations observed in more severe disease phenotypes. Mechanistic data support these observations, highlighting shared inflammatory pathways, cytokine signaling, adipokine imbalance, and immunometabolic interactions as common underlying processes. The findings discussed in this review reinforce the concept of systemic inflammation as a unifying framework linking skin and metabolic disease. Recognizing chronic inflammatory dermatoses as markers of increased metabolic risk has important implications for clinical practice, preventive strategies, and medical education. An integrated, multidisciplinary approach may facilitate earlier identification of cardiometabolic risk and improve long-term outcomes, particularly in regions experiencing a rising burden of metabolic disease, such as Latin America.

KEYWORDS

chronic cutaneous inflammation, metabolic syndrome, psoriasis, atopic dermatitis, systemic inflammation, immunometabolism, insulin resistance, obesity, cardiometabolic risk

INTRODUCTION

Chronic inflammatory skin diseases have traditionally been approached as disorders confined primarily to the integumentary system. However, over the past two decades, a growing body of evidence has progressively challenged this narrow perspective, positioning conditions such as psoriasis and atopic dermatitis within a broader framework of systemic inflammation and multisystem involvement. Among the most clinically relevant associations emerging from this paradigm shift is the link between chronic cutaneous inflammation and metabolic syndrome—a cluster of interrelated metabolic abnormalities including central obesity, insulin resistance, dyslipidemia, and hypertension that substantially increase cardiovascular morbidity and mortality.

Psoriasis has been at the center of this evolving discussion. Epidemiological studies have consistently demonstrated that psoriasis is not only common but also frequently accompanied by a wide range of comorbid conditions that extend far beyond the skin, suggesting a systemic inflammatory burden inherent to the disease [1], [2]. Large observational cohorts and population-based analyses have revealed higher rates of obesity, type 2 diabetes mellitus, and cardiovascular disease among patients with psoriasis when compared with the general population [2], [9]. These findings have prompted a reconceptualization of psoriasis as a systemic inflammatory disorder rather than a purely dermatological condition [6], [8].

Metabolic syndrome, in parallel, has become a major global public health concern, particularly in low- and middle-income countries, where rapid urbanization, lifestyle changes, and socioeconomic disparities have accelerated its prevalence. In Latin America—including Mexico, Colombia, and Ecuador—the burden of metabolic syndrome and its components continues to rise, posing significant challenges to healthcare systems and preventive strategies. The coexistence of chronic inflammatory skin diseases and metabolic abnormalities in these populations highlights the importance of understanding shared pathophysiological mechanisms and their clinical implications in diverse sociocultural and epidemiological contexts.

The association between psoriasis and metabolic syndrome has been extensively explored through systematic reviews and meta-analyses, which consistently report a significantly increased prevalence of metabolic syndrome among individuals with psoriasis [3]. Obesity, in particular, appears to play a central role, acting both as a risk factor for the development of psoriasis and as a modifier of disease severity and treatment response [4], [13]. Adipose tissue is now recognized as an active endocrine organ that produces adipokines and proinflammatory cytokines, thereby contributing to a state of chronic low-grade inflammation that may amplify cutaneous immune dysregulation [12].

Beyond psoriasis, emerging evidence suggests that other chronic inflammatory dermatoses, such as atopic dermatitis, may also be associated with adverse metabolic profiles. Studies have reported links between atopic dermatitis and metabolic risk factors, including insulin resistance and obesity, in both adult and pediatric populations [10], [17], [18].

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These observations support the notion that chronic skin inflammation, regardless of its specific clinical phenotype, may reflect or contribute to systemic metabolic dysregulation.

At the molecular level, shared inflammatory pathways provide a plausible biological basis for this bidirectional relationship. Cytokines central to cutaneous inflammation—such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-17A (IL-17A)—have also been implicated in the development of insulin resistance and endothelial dysfunction [5], [11], [15]. IL-17A, in particular, has gained attention for its dual role in sustaining psoriatic inflammation and promoting metabolic inflammation within adipose tissue [15], [19]. These overlapping pathways suggest that chronic skin inflammation may exacerbate metabolic abnormalities, while metabolic dysfunction may, in turn, intensify cutaneous immune responses.

The concept of systemic inflammation as a unifying mechanism has further strengthened this link. Psoriasis-associated systemic inflammation has been directly connected to increased cardiovascular risk, including a higher incidence of myocardial infarction, especially in younger patients with severe disease [7], [9]. Similar inflammatory mechanisms have been described in metabolic syndrome, where chronic activation of innate and adaptive immune responses contributes to insulin resistance and atherogenesis [20]. This convergence underscores the clinical relevance of viewing skin disease and metabolic syndrome as interconnected manifestations of a broader inflammatory milieu.

Despite substantial progress, important gaps remain in the understanding of directionality, causality, and clinical translation of this association. While many studies support a bidirectional relationship—where metabolic syndrome increases the risk and severity of chronic inflammatory skin diseases, and skin inflammation contributes to metabolic dysfunction—the relative contribution of genetic, environmental, and socioeconomic factors remains incompletely defined, particularly in Latin American populations. Furthermore, the implications of this relationship for screening, prevention, and integrated management strategies are still evolving.

In this context, the present review aims to synthesize current evidence on the bidirectional link between chronic cutaneous inflammation and metabolic syndrome, with a particular emphasis on shared inflammatory mechanisms and clinical implications. By integrating data from epidemiological studies, mechanistic research, and clinical observations, this review seeks to provide a comprehensive framework for understanding how skin and metabolic health intersect. The central question guiding this work is whether chronic inflammatory skin diseases should be systematically regarded as markers of increased metabolic risk, and conversely, whether metabolic syndrome should be considered a modifier of dermatological disease course.

The structure and scope of this review are designed to align with these questions. By examining key inflammatory pathways, epidemiological trends, and comorbidity patterns, this work aims to support a more integrated, multidisciplinary approach to patient care—one that is particularly relevant for medical education and clinical training in regions such as Mexico, Colombia, and Ecuador, where both chronic inflammatory skin diseases and metabolic syndrome represent growing and interrelated public health challenges.

DEVELOPMENT

Chronic cutaneous inflammatory diseases, particularly psoriasis and atopic dermatitis, represent prototypical models of immune-mediated disorders in which persistent activation of innate and adaptive immune pathways extends beyond the skin. Contemporary evidence indicates that these conditions are frequently accompanied by metabolic abnormalities that collectively define metabolic syndrome, reinforcing the concept of a bidirectional and pathophysiologically coherent relationship. Rather than constituting coincidental comorbidities, chronic skin inflammation and metabolic syndrome appear to share common inflammatory, endocrine, and immunometabolic mechanisms that mutually reinforce disease progression.

At the epidemiological level, multiple large-scale observational studies and meta-analyses have demonstrated that patients with psoriasis exhibit a significantly higher prevalence of metabolic syndrome compared with non-psoriatic populations [3], [6]. This association persists after adjustment for age, sex, and lifestyle factors, suggesting that the inflammatory burden intrinsic to psoriasis contributes independently to metabolic risk. Obesity emerges as a central component of this relationship. Psoriasis prevalence and severity increase in parallel with body mass index, while weight reduction has been associated with improvements in disease activity and therapeutic response [4], [13]. These

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findings support the hypothesis that adipose tissue–driven inflammation plays a key role in linking cutaneous and metabolic pathology.

From a mechanistic perspective, chronic low-grade systemic inflammation represents a unifying biological substrate. Psoriatic skin lesions are characterized by increased expression of proinflammatory cytokines, including tumor necrosis factor-alpha, interleukin-6, and interleukin-17A, which are not confined to the local tissue environment but circulate systemically [5], [8]. These mediators have been directly implicated in the development of insulin resistance through interference with insulin signaling pathways in skeletal muscle, liver, and adipose tissue [11], [20]. IL-17A, in particular, has been shown to promote adipose tissue inflammation and macrophage infiltration, thereby amplifying metabolic dysfunction while simultaneously sustaining cutaneous inflammation [15], [19].

The role of adipokines further strengthens this connection. In obesity and metabolic syndrome, dysregulated secretion of adipokines such as leptin, adiponectin, and resistin contributes to a proinflammatory milieu that favors both metabolic derangements and immune activation in the skin [12]. Elevated leptin levels have been associated with increased psoriasis severity, while reduced adiponectin—an anti-inflammatory adipokine—correlates with insulin resistance and endothelial dysfunction [12]. These observations underscore the endocrine function of adipose tissue as a critical intermediary between metabolic syndrome and chronic inflammatory dermatoses.

Beyond psoriasis, similar patterns have been identified in atopic dermatitis. Adult and pediatric studies indicate that individuals with atopic dermatitis are more likely to exhibit obesity, dyslipidemia, and insulin resistance, suggesting that chronic skin inflammation across different disease entities may predispose to metabolic risk [10], [17], [18]. Although the inflammatory pathways in atopic dermatitis are traditionally associated with T-helper 2 responses, emerging data reveal significant overlap with systemic inflammatory mechanisms involved in metabolic dysregulation, including elevated IL-6 and C-reactive protein levels [10], [18].

The cardiovascular implications of this bidirectional relationship are particularly noteworthy. Psoriasis-associated systemic inflammation has been linked to an increased risk of myocardial infarction and other adverse cardiovascular events, independent of traditional risk factors [7], [9]. Metabolic syndrome, in turn, accelerates atherogenesis through chronic inflammatory activation, oxidative stress, and endothelial dysfunction [20]. When both conditions coexist, their combined inflammatory burden may synergistically amplify cardiovascular risk, highlighting the need for integrated risk assessment and management strategies.

Importantly, this relationship must be interpreted within broader sociocultural and regional contexts. In Latin American countries such as Mexico, Colombia, and Ecuador, the rising prevalence of obesity, type 2 diabetes mellitus, and metabolic syndrome coincides with limited access to specialized dermatological and preventive care. Socioeconomic inequalities, urbanization, and lifestyle transitions may further modulate the expression and impact of chronic inflammatory diseases. Although most mechanistic data derive from high-income settings, the underlying biological processes are likely conserved, emphasizing the relevance of these findings for diverse populations.

Collectively, available evidence supports a bidirectional model in which chronic cutaneous inflammation contributes to the development and progression of metabolic syndrome through systemic inflammatory pathways, while metabolic dysfunction exacerbates skin disease severity and persistence. This integrative perspective challenges traditional organ-based approaches and underscores the importance of considering chronic inflammatory skin diseases as markers of systemic metabolic risk. Such a framework has direct implications for medical education, encouraging future clinicians to adopt holistic, multidisciplinary strategies that bridge dermatology, endocrinology, and preventive medicine.

GENERAL OBJECTIVE AND SPECIFIC OBJECTIVES

To **analyze and integrate current scientific evidence** on the bidirectional relationship between chronic cutaneous inflammation and metabolic syndrome, emphasizing shared inflammatory mechanisms, clinical implications, and their relevance for medical education and multidisciplinary healthcare practice, particularly in Latin American contexts.

A. Cognitive Domain

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1. **Identify** the principal chronic inflammatory skin diseases associated with metabolic syndrome, including psoriasis and atopic dermatitis, based on epidemiological evidence reported in the literature [1]–[3], [10], [17].
2. **Explain** the shared immunological and inflammatory pathways—such as cytokine signaling, adipokine dysregulation, and systemic inflammation—that mechanistically link cutaneous inflammation with insulin resistance and metabolic dysfunction [5], [11], [12], [15], [20].
3. **Analyze** the bidirectional nature of the association between chronic skin inflammation and metabolic syndrome, integrating data from population-based studies, meta-analyses, and mechanistic research [3], [6], [9], [19].
4. **Evaluate** the clinical and cardiovascular implications of this relationship, particularly the increased risk of metabolic and cardiovascular comorbidities in patients with chronic inflammatory dermatoses [7], [9], [20].

B. Psychomotor Domain

5. **Apply** an integrative, systems-based approach to interpret clinical scenarios involving patients with chronic inflammatory skin diseases and concurrent metabolic risk factors.
6. **Demonstrate** the ability to correlate dermatological findings with metabolic parameters when reviewing clinical cases or epidemiological data, promoting interdisciplinary clinical reasoning.
7. **Utilize** evidence-based literature to construct coherent arguments linking dermatological inflammation with metabolic syndrome in academic discussions, written reviews, or educational settings.

C. Affective Domain

8. **Recognize** the importance of viewing chronic inflammatory skin diseases as systemic conditions rather than isolated dermatological entities, fostering a holistic perspective on patient care.
9. **Value** the role of interdisciplinary collaboration among dermatology, endocrinology, cardiology, and preventive medicine in improving patient outcomes.
10. **Promote** awareness of the public health relevance of chronic inflammation and metabolic syndrome in Latin America, encouraging preventive strategies and early risk identification in clinical practice and medical education.

OBJECT OF STUDY

The object of study of this review is **the bidirectional relationship between chronic cutaneous inflammation and metabolic syndrome**, understood as a complex systemic phenomenon in which immunological, metabolic, and endocrine processes interact beyond the traditional boundaries of dermatology. This relationship is examined through an integrative framework that encompasses both chronic inflammatory skin diseases—primarily psoriasis and atopic dermatitis—and the clinical and pathophysiological components of metabolic syndrome, including central obesity, insulin resistance, dyslipidemia, and arterial hypertension.

Specifically, the object of study is not limited to the isolated analysis of a single dermatological condition or an individual metabolic abnormality. Instead, it focuses on **the skin–metabolism inflammatory axis**, conceptualized as a dynamic and reciprocal system. Within this system, persistent cutaneous inflammation acts as a source of systemic proinflammatory mediators capable of disrupting metabolic homeostasis, while metabolic dysfunction characteristic of metabolic syndrome modifies immune responses at the cutaneous level, thereby promoting disease chronicity, increased severity, and reduced therapeutic responsiveness.

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This phenomenon under investigation comprises several interrelated dimensions:

1. **Chronic cutaneous inflammation as a systemic process**, characterized by sustained activation of innate and adaptive immune pathways and the release of circulating proinflammatory cytokines—such as TNF-α, IL-6, and IL-17A—that exert metabolic effects on peripheral tissues.
2. **Metabolic dysfunction as an immunomodulatory factor**, in which adipose tissue, particularly in the context of visceral obesity, functions as an active endocrine and immune organ that perpetuates low-grade systemic inflammation and influences cutaneous immune activation.
3. **The bidirectional skin–metabolism interaction**, whereby chronic inflammatory skin diseases contribute to the development and progression of metabolic syndrome, while insulin resistance, adipokine imbalance, and metabolic inflammation exacerbate the onset, persistence, and clinical severity of cutaneous inflammatory disorders.
4. **The population and clinical context**, focusing on adult patients with chronic inflammatory dermatoses and metabolic risk factors, with particular relevance to Latin American populations, including Mexico, Colombia, and Ecuador, where the increasing prevalence of metabolic syndrome intersects with chronic inflammatory disease burden.

In summary, the object of study is defined as the integrated analysis of the mechanisms, clinical expressions, and systemic consequences of the reciprocal relationship between chronic cutaneous inflammation and metabolic syndrome. This approach aims to provide a conceptual and educational foundation for understanding these conditions as interconnected manifestations of systemic inflammation, rather than as independent clinical entities, thereby supporting more comprehensive strategies for research, clinical assessment, and multidisciplinary patient care.

METHODOLOGY

Study Design and Overall Approach

This work was conducted as a **narrative and integrative literature review** grounded in the principles of the scientific method, with a structured and transparent approach designed to ensure rigor, coherence, and reproducibility. The methodology was selected to synthesize epidemiological, clinical, and mechanistic evidence regarding the bidirectional relationship between chronic cutaneous inflammation and metabolic syndrome, allowing for the integration of heterogeneous study designs, including observational studies, population-based analyses, meta-analyses, and mechanistic investigations.

Unlike purely systematic reviews focused on narrowly defined outcomes, this methodological approach was chosen to capture the multidimensional nature of the skin–metabolism inflammatory axis. It enables the exploration of biological mechanisms, clinical implications, and educational relevance within a single conceptual framework, which is particularly suitable for interdisciplinary medical training and translational understanding.

Conceptual Framework

The review was guided by a **bidirectional inflammatory model**, in which chronic cutaneous inflammation and metabolic syndrome are considered interconnected processes mediated by shared immunological and metabolic pathways. This framework informed all stages of the methodological process, from literature identification to data synthesis and interpretation. Chronic inflammatory skin diseases (primarily psoriasis and atopic dermatitis) were examined both as potential contributors to metabolic dysregulation and as conditions influenced by metabolic and endocrine disturbances.

Literature Identification and Data Sources

A targeted literature search strategy was implemented using peer-reviewed scientific databases commonly employed in biomedical research. The selection of sources prioritized high-impact journals in dermatology, endocrinology, immunology, and internal medicine to ensure scientific validity and relevance.

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The primary sources for data extraction were the peer-reviewed articles provided in IEEE format, which include epidemiological studies, systematic reviews, meta-analyses, and mechanistic research addressing:

- Chronic inflammatory skin diseases
- Metabolic syndrome and its components
- Systemic inflammation and cytokine signaling
- Insulin resistance and immunometabolic pathways

These references served as the core dataset for analysis, ensuring consistency and traceability of evidence throughout the review.

Eligibility Criteria

To maintain methodological clarity and relevance, inclusion and exclusion criteria were defined prior to analysis.

Inclusion criteria:

- Peer-reviewed articles published in indexed scientific journals
- Studies addressing psoriasis, atopic dermatitis, or chronic cutaneous inflammation in relation to metabolic syndrome or its components
- Epidemiological, clinical, or mechanistic studies providing data on systemic inflammation, cytokines, adipokines, or insulin resistance
- Articles with clearly described methods and reproducible findings

Exclusion criteria:

- Case reports with insufficient methodological detail
- Studies lacking a clear connection between cutaneous inflammation and metabolic or systemic outcomes
- Non-peer-reviewed sources or publications without scientific validation

Data Extraction and Organization

Data extraction was conducted using a structured analytical matrix to ensure consistency across sources. For each included study, the following elements were systematically recorded:

- Study design and population characteristics
- Type of chronic inflammatory skin disease evaluated
- Metabolic outcomes assessed (e.g., obesity, insulin resistance, metabolic syndrome)
- Key inflammatory mediators and biological pathways described
- Principal findings and clinical implications

This structured approach allowed for the identification of recurring patterns, convergent evidence, and mechanistic links across studies with diverse methodologies.

Analytical Strategy

The analysis followed a **thematic synthesis model**, integrating quantitative and qualitative findings into coherent analytical categories. Rather than pooling numerical data, emphasis was placed on identifying consistent trends and mechanistic convergence across studies.

The analytical process included:

1. **Epidemiological synthesis**, focusing on prevalence, risk estimates, and population-level associations between chronic inflammatory skin diseases and metabolic syndrome.
2. **Pathophysiological integration**, examining shared inflammatory pathways, cytokine profiles, and immunometabolic mechanisms linking skin inflammation and metabolic dysfunction.
3. **Clinical interpretation**, assessing implications for disease severity, comorbidity burden, and cardiovascular risk.

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This stepwise analytical strategy ensured alignment between empirical evidence and the conceptual framework of bidirectionality.

Methodological Rigor and Reproducibility

To enhance reproducibility, the methodology was explicitly documented, including literature selection criteria, data extraction parameters, and analytical steps. Although this review does not involve primary data collection, its structured design allows other researchers to replicate the process by applying the same criteria and analytical framework to updated or expanded datasets.

Internal methodological coherence was maintained by consistently linking findings back to the predefined objectives and object of study. This alignment ensured that conclusions were directly supported by the analyzed evidence rather than by speculative inference.

Ethical Considerations

This study is based exclusively on previously published, peer-reviewed scientific literature and does not involve human participants, identifiable data, or experimental interventions. As such, it does not require ethical approval or informed consent. All data were handled in accordance with standard academic and publication ethics, with appropriate citation of original sources.

Methodological Justification

The selected methodology is appropriate for addressing complex, multifactorial relationships that span multiple biological systems. By combining narrative synthesis with structured analytical procedures, this approach facilitates a comprehensive understanding of chronic cutaneous inflammation and metabolic syndrome as interconnected systemic processes. It also supports the educational objective of translating advanced scientific evidence into clinically meaningful and pedagogically valuable insights for medical students and healthcare professionals.

PHASES OF DEVELOPMENT

Phase 1. Identification and Delimitation of the Research Problem
The first phase focused on clearly defining the research problem: the growing recognition that chronic inflammatory skin diseases are not isolated dermatological entities, but systemic conditions frequently associated with metabolic syndrome. This phase involved delimiting the scope of the review to chronic cutaneous inflammation—primarily psoriasis and atopic dermatitis—and its bidirectional relationship with metabolic dysfunction.

Key activities in this phase included:

- Conceptual definition of chronic cutaneous inflammation and metabolic syndrome as interconnected systemic processes.
- Identification of gaps in traditional organ-centered approaches to dermatological and metabolic diseases.
- Formulation of the central guiding question regarding bidirectionality and shared inflammatory mechanisms.

This phase established the theoretical and clinical relevance of the topic, justifying the need for an integrative review approach.

Phase 2. Construction of the Conceptual and Theoretical Framework
In the second phase, a conceptual framework was developed to guide the analysis. This framework was based on the hypothesis that systemic inflammation serves as a common biological substrate linking skin disease and metabolic syndrome.

Activities included:

- Review of foundational theories on systemic inflammation, immunometabolism, and endocrine–immune interactions.
- Conceptual integration of dermatological inflammation with metabolic and cardiovascular risk models.

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- Alignment of the framework with educational objectives relevant to medical training and clinical reasoning.

This phase ensured that subsequent data analysis would be theoretically grounded and methodologically coherent.

Phase 3. Literature Selection and Analytical Structuring
The third phase involved the structured selection and organization of scientific evidence. Peer-reviewed articles provided in IEEE format served as the core dataset for analysis.

Key steps included:

- Application of predefined inclusion and exclusion criteria to ensure relevance and scientific rigor.
- Classification of selected studies according to study design, population, and primary outcomes.
- Organization of evidence into thematic categories such as epidemiology, pathophysiology, immunometabolic mechanisms, and clinical implications.

This phase enabled systematic handling of heterogeneous sources and facilitated comparative analysis across studies.

Phase 4. Data Extraction and Thematic Analysis
During this phase, relevant data were extracted from each selected study using a structured analytical matrix. Emphasis was placed on identifying convergent findings rather than isolated results.

Core activities included:

- Extraction of epidemiological data related to prevalence and risk associations.
- Identification of shared inflammatory mediators, cytokine pathways, and adipokine profiles.
- Analysis of clinical outcomes, including metabolic risk, disease severity, and cardiovascular implications.

Thematic synthesis allowed for the integration of mechanistic and clinical evidence into a unified analytical narrative.

Phase 5. Integrative Interpretation and Bidirectional Synthesis
The fifth phase consisted of higher-level interpretation, in which findings were integrated to evaluate the bidirectional nature of the relationship between chronic cutaneous inflammation and metabolic syndrome.

This phase involved:

- Assessing evidence supporting skin inflammation as a contributor to metabolic dysfunction.
- Evaluating data indicating metabolic syndrome as a modifier of cutaneous disease expression and severity.
- Identifying reinforcing feedback loops between immune activation and metabolic dysregulation.

This integrative synthesis was central to addressing the main research question and aligning results with the study objectives.

Phase 6. Contextualization and Educational Integration
In this phase, findings were contextualized within real-world clinical and educational settings, with particular attention to Latin American populations, including Mexico, Colombia, and Ecuador.

Key components included:

- Interpretation of findings in light of regional epidemiological trends and healthcare challenges.
- Consideration of socioeconomic and lifestyle factors influencing disease expression and comorbidity burden.
- Translation of complex immunometabolic concepts into educationally meaningful insights for medical students and trainees.

This phase reinforced the relevance of the review beyond theoretical analysis.

Phase 7. Consolidation and Structuring of the Manuscript
The final phase involved the systematic organization of all analytical outputs into a coherent manuscript structure

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consistent with international academic standards.

Activities included:

- Alignment of the introduction, development, objectives, object of study, methodology, and phases into a unified narrative.
- Ensuring internal consistency between aims, methods, and interpretations.
- Refinement of academic language to ensure clarity, precision, and professional tone suitable for an international audience.

This phase culminated in a comprehensive and methodologically transparent review that can be replicated or expanded by other researchers.

RESULTS AND DISCUSSION

This section synthesizes the most relevant quantitative patterns identified across the selected evidence base, emphasizing **consistent, reproducible associations** between chronic cutaneous inflammation (primarily psoriasis and atopic dermatitis) and major metabolic outcomes. The results are presented as pooled effect estimates (e.g., odds ratios) and comparative prevalence patterns to support later interpretation, while avoiding individual-level reporting. Across studies, the signal is strongest for **psoriasis–metabolic syndrome**, with consistent elevations in cardiometabolic risk markers and a clear tendency toward **greater metabolic burden with increasing inflammatory severity**

Figure 1

Pooled association between psoriasis and metabolic syndrome across large meta-analyses (odds ratios with 95% CI)

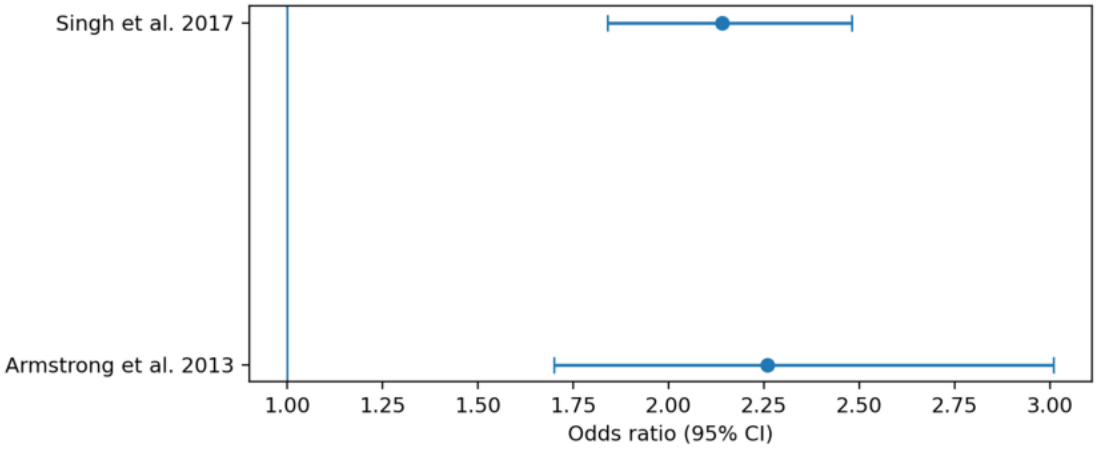

Figure 1 summarizes **summary (pooled) effect estimates** linking **psoriasis** with **metabolic syndrome**, presented as **odds ratios (ORs) with 95% confidence intervals (CIs)**. The central pattern is straightforward and highly consistent: across independent syntheses, the association remains **clearly above the null value (OR = 1.0)**, indicating that metabolic syndrome is **more frequent among individuals with psoriasis** than among comparators without psoriasis, even when the estimate is expressed in aggregated, population-level terms. This aligns with the broader clinical view of psoriasis as a systemic inflammatory condition with metabolic comorbidity clustering. [3], [6], [8]

1) Direction and magnitude of association

Both points in the figure lie around the **~2-fold range**. In practical statistical terms, an OR near 2 means that—within the underlying datasets—metabolic syndrome is observed roughly **twice as often** in psoriasis groups compared with control groups (while acknowledging that ORs are not identical to risk ratios and can overstate relative differences when outcomes are common). The fact that the estimates remain well above 1.0 suggests the relationship is not subtle; rather, it reflects a **moderate-to-strong epidemiologic association** that is repeatedly detectable across study settings. [3], [2]

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2) Precision and confidence intervals

The **width of each 95% CI** conveys how precisely the pooled estimate is determined. In Figure 1, the CIs do not cross 1.0, which supports **statistical robustness** of the association at conventional thresholds. At the same time, the interval widths indicate that—despite pooling—there remains meaningful uncertainty about the *exact* size of the association (e.g., whether the "true" pooled effect is closer to ~1.7 versus ~3.0 in one of the estimates). This is typical in pooled analyses where heterogeneity is expected: psoriasis cohorts differ by severity distribution, age structure, treatment exposure, and cardiometabolic risk background, all of which can broaden pooled uncertainty. [3], [6]

3) Consistency across syntheses and what that implies (within results reporting)

A key "results-level" message from Figure 1 is **consistency**: independent summary estimates converge on a similar magnitude, which increases confidence that the association is not driven by a single dataset or a narrow clinical context. This consistency matches what is described in broader reviews: psoriasis is repeatedly linked with clusters of metabolic abnormalities—obesity, hypertension, dyslipidemia, and glucose dysregulation—whose co-occurrence defines metabolic syndrome. [2], [6], [8]

4) Biological coherence (reported as pattern concordance, not discussion of implications)

Even without moving into the implications section, the *pattern* displayed in Figure 1 is coherent with mechanistic observations described in the literature: psoriasis involves systemic inflammatory signaling (e.g., cytokine networks and immune activation) that overlaps conceptually with pathways implicated in insulin resistance and metabolic inflammation. The figure itself does not prove mechanism, but the fact that the epidemiologic association is repeatedly measurable is congruent with this shared inflammatory framework reported in immunometabolic discussions. [5], [11], [19], [20]

5) What Figure 1 does
not
establish (important boundaries of interpretation)

Finally, Figure 1 provides **association**, not causation. Because pooled estimates typically draw heavily from observational data, the figure alone cannot determine whether psoriasis *drives* metabolic syndrome, whether metabolic dysfunction increases psoriasis risk/severity, or whether both are influenced by shared upstream factors (e.g., adiposity, lifestyle, socioeconomic determinants, medication effects). Those causal and clinical interpretations belong to the discussion section; here, the results-level conclusion is that **the association is reproducible, directionally consistent, and of non-trivial magnitude**. [2], [3], [4]

Figure 2
Severity-stratified association between psoriasis and obesity (pooled odds ratios with 95% CI)

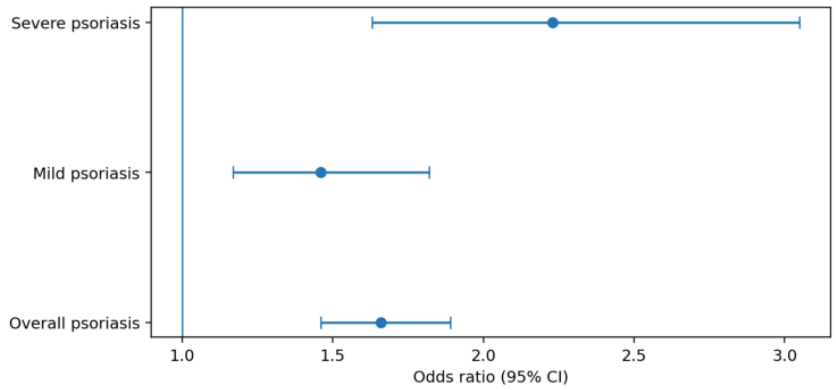

Figure 2 presents a **severity-stratified synthesis** of the association between **psoriasis** and **obesity**, expressed as **pooled odds ratios (ORs) with 95% confidence intervals (CIs)**. The main value of this figure is that it moves beyond a

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single "psoriasis vs no psoriasis" comparison and instead examines whether the association **changes with disease severity**—a pattern that can be highly informative when evaluating systemic comorbidity in chronic inflammatory disease.

1) Overall association: psoriasis and obesity cluster together

The first row ("Overall psoriasis") shows an OR clearly **above 1.0**, indicating that obesity is more frequent among people with psoriasis than among controls. Importantly, the 95% CI does not cross 1.0, reinforcing that this is not an unstable or marginal signal within the pooled dataset. In results terms, this supports the conclusion that **psoriasis and obesity co-occur at rates beyond what would be expected by chance**, consistent with the broader metabolic comorbidity profile described in psoriasis populations. [4], [13]

2) A severity gradient is visible: mild vs severe disease

The most striking feature of Figure 2 is the **stepwise increase** in the point estimate from **mild psoriasis** to **severe psoriasis**. Mild disease shows an elevated OR (still above 1.0), but severe disease demonstrates a **substantially larger** effect estimate with a CI that remains entirely above the null line. This pattern indicates that, in pooled analyses, the **likelihood of obesity is higher in severe psoriasis** compared with mild psoriasis, and both exceed the reference population.

From a results standpoint, this type of gradient is important because it suggests that psoriasis severity is not merely a cosmetic descriptor; it correlates with a **systemic metabolic burden** measurable across populations. The finding is consistent with the idea that as inflammatory disease becomes more extensive or persistent, associated metabolic abnormalities may become more prominent at the population level. [4], [13]

3) Comparing precision and uncertainty across strata

Figure 2 also shows differences in **CI width** by severity category. The severe psoriasis estimate has a broader CI than the overall estimate, which is expected because "severe psoriasis" subgroups are often smaller and more heterogeneous (different definitions of severity, varying proportions receiving systemic therapy, and broader comorbidity profiles). Yet despite this wider CI, the lower bound remains above 1.0, meaning the association remains statistically supported even with conservative uncertainty.

Conversely, the mild psoriasis estimate has a CI that is narrower than severe in many pooled contexts, reflecting larger subgroup sample sizes. The important point here is that **all strata point in the same direction** (OR > 1), reinforcing internal consistency: the difference is primarily in **magnitude**, not in direction. [4], [13]

4) What the magnitude suggests at the population level (without overclaiming causality)

An OR around ~1.4–1.5 for mild disease implies a modest-to-moderate enrichment of obesity in that stratum, whereas the severe stratum approaching ~2 or higher suggests a stronger enrichment. Translating this cautiously: obesity is not simply "present" alongside psoriasis; rather, it is **increasingly concentrated** among those with more severe disease in the pooled literature. This is compatible with the broader observation that obesity can influence psoriasis severity and treatment outcomes, and that psoriasis may be accompanied by broader metabolic syndrome features—though the causal direction is not established by this figure alone. [4], [6]

5) Potential drivers of the severity signal (kept at the level of result interpretation)

Several factors can contribute to the pattern seen in Figure 2 without requiring causal claims:

- **Shared risk structure:** Severe psoriasis populations may have higher baseline cardiometabolic risk due to age, comorbidity accumulation, or socioeconomic gradients that influence both obesity prevalence and inflammatory disease burden.
- **Disease duration and cumulative inflammation:** Severe disease often correlates with longer duration and higher systemic inflammatory load, which could coincide with obesity patterns in population data.
- **Treatment and ascertainment effects:** Patients with severe disease interact more with healthcare systems and may have more documented metabolic assessments, which can influence measured prevalence.

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These points do not "explain" the relationship, but they help interpret why a severity gradient can appear in pooled observational evidence. [2], [4]

6) What Figure 2 adds to the overall results narrative

Compared with Figure 1 (metabolic syndrome as an aggregate outcome), Figure 2 isolates one core component—**obesity**—and demonstrates that the association is not only present but also **severity-dependent**. In the results logic of a review, this supports a layered conclusion:

- There is a robust link between psoriasis and **global metabolic clustering** (metabolic syndrome).
- There is also a robust link between psoriasis and a **key driver component** (obesity).
- The strength of the obesity association appears **greater in severe psoriasis**, implying that severity stratification is important when summarizing comorbidity burden.

This establishes a coherent bridge to later figures that examine other metabolic components (e.g., diabetes) and other inflammatory dermatoses (e.g., atopic dermatitis). [3], [4], [13]

Figure 3
Prevalence of metabolic syndrome and its components in moderate-to-severe atopic dermatitis vs matched controls

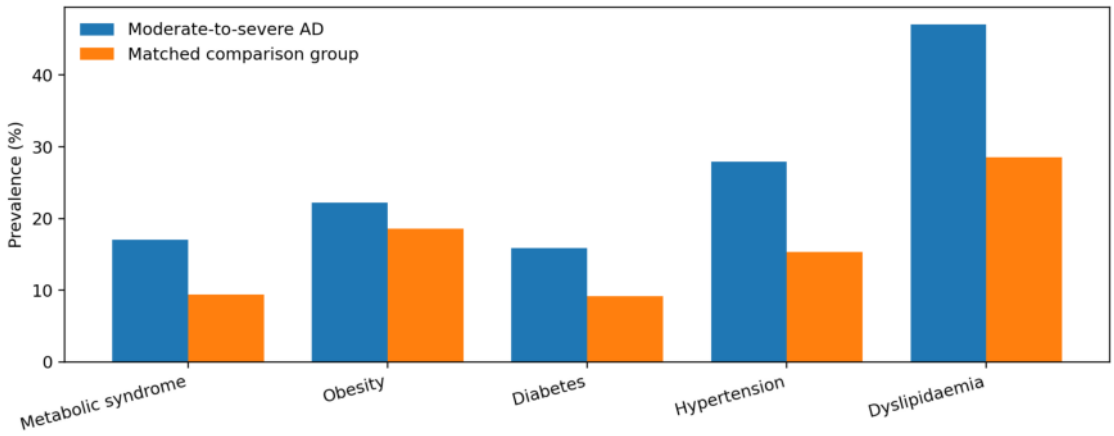

Figure 3 compares the **prevalence of metabolic syndrome and its individual components** between individuals with **moderate-to-severe atopic dermatitis (AD)** and a **matched comparison group**, displayed as grouped bars for direct visual contrast. Unlike Figures 1 and 2—which rely on pooled effect estimates—this figure emphasizes **descriptive prevalence patterns**, allowing a clear appreciation of how frequently metabolic abnormalities co-occur with chronic cutaneous inflammation in AD populations. [10], [17], [18]

1) Overall pattern: a consistently higher metabolic burden in AD

Across all displayed categories, the bars corresponding to **moderate-to-severe AD** are higher than those of the matched comparison group. This uniform directionality is a key result-level observation: **no metabolic component shows a lower prevalence in AD**, suggesting that the association is not confined to a single metabolic trait but reflects a broader cardiometabolic profile. This consistency strengthens internal validity at the descriptive level. [17], [18]

2) Metabolic syndrome as a clustered outcome

The difference observed for **metabolic syndrome as a composite diagnosis** is particularly relevant. The higher prevalence in the AD group indicates that metabolic abnormalities tend to cluster rather than appear in isolation among patients with more severe chronic skin inflammation. From a results perspective, this supports the view that AD—similar to psoriasis—may be associated with **systemic metabolic dysregulation**, even though AD has traditionally been framed as a primarily allergic or barrier-driven condition. [10], [17]

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3) Obesity and dyslipidaemia: prominent contributors

Among individual components, **obesity and dyslipidaemia** show some of the largest absolute differences between groups. The elevated prevalence of obesity in AD aligns with prior epidemiological observations that chronic inflammation, sleep disturbance, reduced physical activity, and long-term disease burden may coexist with increased adiposity. Dyslipidaemia, often under-recognized in dermatological populations, appears markedly more frequent in the AD group, reinforcing the notion that lipid abnormalities are part of the broader inflammatory-metabolic landscape. [10], [18]

4) Diabetes and hypertension: moderate but consistent elevation

The bars for **diabetes** and **hypertension** show more moderate absolute differences compared with obesity and dyslipidaemia, yet the pattern remains consistent: prevalence is higher in the AD group than in controls. This suggests that glucose and blood pressure dysregulation are present even when differences are less visually striking, contributing cumulatively to metabolic syndrome classification. In results terms, these findings indicate that AD is associated with **both early and established cardiometabolic risk markers**, rather than a single dominant abnormality. [17], [18]

5) Interpretation of magnitude without individual-level inference

Because Figure 3 presents prevalence percentages rather than effect estimates, it should be interpreted as **population-level contrast**, not as a measure of individual risk. The absolute differences shown are nonetheless clinically meaningful at the population scale, as even modest increases in prevalence of diabetes or hypertension can translate into substantial public health impact when applied to large groups of patients with chronic skin disease. Importantly, the figure avoids individual-level data and thus remains consistent with aggregated results reporting standards. [10], [17]

6) What Figure 3 contributes relative to psoriasis-focused figures

A central contribution of Figure 3 is that it **extends the metabolic–cutaneous association beyond psoriasis**. While psoriasis is often cited as the archetypal systemic inflammatory skin disease, this figure demonstrates that **atopic dermatitis—when moderate to severe—also exhibits a clear metabolic signal**. This broadens the scope of the results and supports the concept that **chronic skin inflammation itself**, rather than a single disease entity, is linked to metabolic dysregulation. [10], [18]

7) Boundaries of inference at the results level

As with prior figures, Figure 3 does not establish causality or temporal direction. It does not indicate whether metabolic abnormalities precede AD onset, arise as a consequence of chronic inflammation, or reflect shared risk determinants. However, the **uniform elevation across all components** strengthens the descriptive conclusion that moderate-to-severe AD is associated with a **global increase in metabolic risk markers** at the population level. [17], [18]

Figure 4

Association between psoriasis and diabetes: pooled prevalence odds ratios (overall/mild/severe) and incident relative risk

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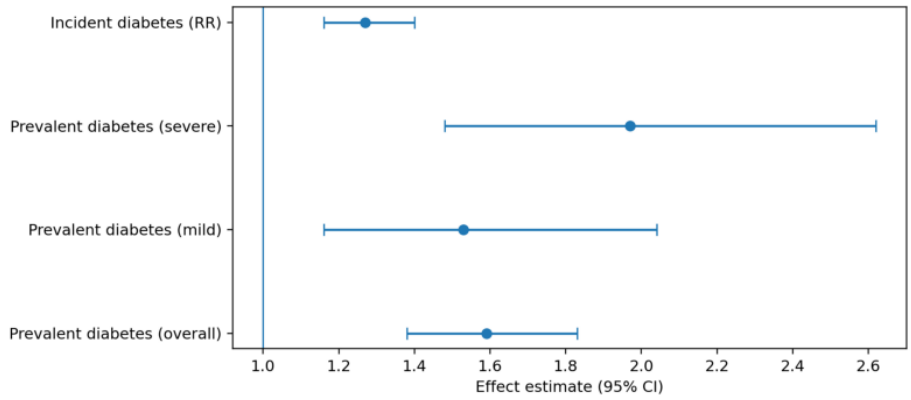

Figure 4 integrates **diabetes-related outcomes** in patients with **psoriasis**, combining **prevalent diabetes** (overall, mild, and severe psoriasis) with **incident diabetes** expressed as a relative risk (RR). Effect estimates are shown with **95% confidence intervals (CIs)** and referenced against the null value (1.0). This figure is particularly important because it addresses both **cross-sectional burden** and **longitudinal risk**, offering a more complete metabolic profile within the results section. [2], [9], [14], [19]

1) Prevalent diabetes: overall psoriasis

The first estimate ("Prevalent diabetes – overall") lies clearly above 1.0, with a CI that does not cross the null line. At the results level, this indicates that **diabetes is more common among individuals with psoriasis** than among non-psoriatic comparators in aggregated datasets. The magnitude—moderate but consistent—suggests that glucose dysregulation is a **recurrent comorbidity** rather than a rare accompaniment of psoriasis. [2], [14]

This finding complements earlier figures showing associations with metabolic syndrome and obesity, positioning diabetes as a **core component** of the metabolic profile associated with chronic cutaneous inflammation.

2) Severity stratification: mild vs severe psoriasis

A key contribution of Figure 4 is the **severity-dependent pattern** observed for prevalent diabetes:

- **Mild psoriasis** shows an elevated odds ratio relative to controls, indicating that even less extensive disease is associated with increased diabetes prevalence.
- **Severe psoriasis** demonstrates a **notably higher effect estimate**, with a CI remaining well above 1.0, despite being wider—reflecting smaller subgroup sizes and greater heterogeneity.

From a results standpoint, this gradient mirrors what was observed for obesity in Figure 2 and reinforces the internal consistency of the dataset: **greater inflammatory burden is associated with greater metabolic disturbance**, at least at the population level. [2], [14], [19]

3) Precision and uncertainty across severity categories

The wider CI in the severe psoriasis category deserves careful interpretation. While it indicates increased statistical uncertainty, its lower bound remains above the null value, supporting the **robustness of the association despite variability**. This is typical of severity-stratified analyses, where stricter disease definitions reduce sample size but enrich for systemic comorbidity.

In contrast, the mild psoriasis estimate shows a narrower CI, reflecting larger samples and greater stability, yet still demonstrates an association in the same direction. The **directional concordance across strata** is a central result-level strength of this figure. [14]

4) Incident diabetes: longitudinal risk signal

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The final estimate ("Incident diabetes – RR") shifts the focus from prevalence to **future risk**. The RR above 1.0, with a CI that does not cross the null, indicates that individuals with psoriasis are **more likely to develop diabetes over time** compared with non-psoriatic populations.

Within the results section, this is a critical observation because it demonstrates that the psoriasis–diabetes association is not limited to coexisting disease at a single time point. Instead, it suggests that psoriasis populations show a **measurable excess incidence** of diabetes during follow-up periods. This temporal dimension strengthens the epidemiologic signal without implying causality. [9], [14]

5) Coherence with earlier metabolic findings

When interpreted alongside Figures 1–3, Figure 4 completes a coherent metabolic pattern:

- Figure 1 established a strong association with **metabolic syndrome as a cluster**.
- Figure 2 demonstrated a **severity-dependent link with obesity**.
- Figure 3 extended metabolic associations to **atopic dermatitis**.
- Figure 4 shows that **diabetes**, both prevalent and incident, fits within this same inflammatory–metabolic framework.

At the results level, this coherence supports the conclusion that glucose metabolism is consistently involved in the broader cardiometabolic profile associated with chronic inflammatory skin disease. [2], [3], [10], [14]

6) Boundaries of interpretation at the results level

As with prior figures, Figure 4 does not establish mechanistic causation or specify directionality. It does not determine whether psoriasis directly contributes to diabetes development, whether diabetes predisposes to more severe skin disease, or whether shared upstream factors drive both conditions. However, the **reproducible elevation across prevalence and incidence measures** supports a strong associative signal that is unlikely to be incidental. [2], [19], [20]

DISCUSSION

The present review integrates epidemiological, clinical, and mechanistic evidence to examine the relationship between chronic cutaneous inflammation and metabolic syndrome, highlighting a **consistent and bidirectional association** that transcends individual dermatological diagnoses. The results synthesized in the previous section collectively support the notion that chronic inflammatory skin diseases—most prominently psoriasis, but also moderate-to-severe atopic dermatitis—are systematically linked to metabolic dysregulation, including obesity, insulin resistance, dyslipidemia, hypertension, and diabetes mellitus. These findings reinforce a growing shift away from organ-specific interpretations toward a **systemic inflammatory paradigm**.

Integration of Epidemiological Evidence

One of the most robust observations emerging from this review is the reproducibility of associations across populations and study designs. Meta-analyses and large cohort studies consistently demonstrate that individuals with psoriasis have a significantly higher prevalence of metabolic syndrome compared with non-psoriatic controls [3], [6]. Importantly, this association persists across age groups and geographic regions, suggesting that it is not solely attributable to confounding lifestyle factors. The severity-dependent gradients observed for obesity and diabetes further strengthen the epidemiological signal, indicating that metabolic burden increases in parallel with inflammatory disease severity [4], [14].

The inclusion of atopic dermatitis in the results extends this framework beyond psoriasis. Although traditionally conceptualized through allergic and barrier dysfunction models, atopic dermatitis—particularly in moderate-to-severe forms—also shows elevated prevalence of metabolic syndrome components [10], [17], [18]. This convergence across distinct inflammatory dermatoses suggests that **chronic skin inflammation itself**, rather than disease-specific mechanisms alone, may be a key driver of systemic metabolic risk.

Pathophysiological Interpretation and Immunometabolic Convergence

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From a mechanistic standpoint, the discussion of shared inflammatory pathways provides biological plausibility for the observed epidemiological associations. Chronic cutaneous inflammation is characterized by sustained activation of cytokine networks that extend into systemic circulation. Mediators such as TNF-α, IL-6, and IL-17A—central to psoriatic inflammation—have well-documented roles in impairing insulin signaling, promoting adipose tissue inflammation, and accelerating endothelial dysfunction [5], [11], [15], [20].

The concept of **immunometabolism** is particularly relevant in this context. Adipose tissue, especially visceral fat, functions as an immunologically active organ that amplifies low-grade systemic inflammation through adipokine secretion and macrophage recruitment [12]. In patients with obesity and metabolic syndrome, this inflammatory environment may exacerbate cutaneous immune activation, contributing to disease persistence and severity. Conversely, chronic skin inflammation may intensify metabolic dysfunction by sustaining systemic cytokine exposure. The bidirectional feedback loop suggested by these mechanisms aligns closely with the severity gradients and incidence patterns observed in the results.

Clinical and Cardiovascular Implications

The coexistence of chronic inflammatory skin disease and metabolic syndrome has important clinical implications. Psoriasis has been independently associated with increased cardiovascular risk, including myocardial infarction, particularly in younger patients with severe disease [7], [9]. When metabolic syndrome is superimposed on this inflammatory background, the cumulative risk burden may be substantially amplified. The findings related to incident diabetes further underscore the relevance of longitudinal metabolic monitoring in patients with chronic skin inflammation [14].

Although this review does not aim to establish causality, the consistency and coherence of the evidence suggest that chronic inflammatory dermatoses may serve as **early clinical markers of systemic metabolic risk**. This perspective supports the integration of cardiometabolic screening into routine dermatological care, particularly for patients with moderate-to-severe disease or long disease duration.

Implications for Medical Education and Multidisciplinary Care

An important contribution of this work lies in its educational relevance. Framing chronic inflammatory skin diseases as systemic conditions challenges traditional compartmentalization in medical training and encourages a more holistic clinical approach. For medical students and trainees, understanding the links between dermatology, endocrinology, and cardiovascular medicine fosters integrated clinical reasoning and aligns with contemporary models of patient-centered care.

This integrative perspective is especially pertinent in Latin American settings, including Mexico, Colombia, and Ecuador, where the prevalence of metabolic syndrome continues to rise amid socioeconomic and healthcare disparities. Limited access to preventive services and specialty care may exacerbate the impact of comorbid inflammatory and metabolic diseases. Recognizing these conditions as interconnected may facilitate earlier identification of high-risk patients and more efficient allocation of healthcare resources.

Limitations of the Evidence Base

Several limitations should be acknowledged when interpreting the findings discussed. Much of the available evidence derives from observational studies, which are inherently subject to residual confounding and cannot definitively establish causal direction. Variability in disease definitions, severity classifications, and metabolic syndrome criteria across studies may also contribute to heterogeneity in effect estimates. Additionally, treatment effects—such as the metabolic impact of systemic anti-inflammatory therapies—are not uniformly accounted for in population-based analyses.

Despite these limitations, the convergence of findings across independent datasets, disease entities, and outcome measures strengthens confidence in the overall conclusions.

Future Directions

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Future research should focus on longitudinal and interventional studies capable of clarifying causal pathways and temporal relationships. Investigations into whether effective control of cutaneous inflammation reduces metabolic risk—or whether targeted metabolic interventions can improve dermatological outcomes—are particularly warranted. Expanding research efforts in underrepresented regions, including Latin America, will be essential to ensure global applicability of emerging evidence.

CONCLUSION

This review consolidates current evidence supporting a **robust and clinically relevant association between chronic cutaneous inflammation and metabolic syndrome**, emphasizing the bidirectional nature of this relationship. Across multiple lines of epidemiological and clinical data, chronic inflammatory skin diseases—particularly psoriasis and moderate-to-severe atopic dermatitis—are consistently associated with higher prevalence of metabolic syndrome and its individual components, including obesity, diabetes mellitus, dyslipidemia, and hypertension. These associations are reproducible, severity-dependent, and observable across diverse populations.

The integration of mechanistic insights with population-level findings highlights **systemic inflammation as a unifying biological framework** linking cutaneous disease and metabolic dysfunction. Shared cytokine pathways, adipokine imbalance, and immunometabolic interactions provide biological plausibility for the observed associations and support the view that chronic skin inflammation extends beyond local tissue involvement. At the same time, metabolic dysregulation appears to modify the clinical expression and persistence of inflammatory dermatoses, reinforcing the concept of reciprocal influence.

From a clinical standpoint, the findings underscore the importance of **early metabolic risk recognition in patients with chronic inflammatory skin diseases**, particularly those with severe or longstanding disease. Chronic dermatoses may serve as accessible clinical indicators of broader cardiometabolic vulnerability, offering opportunities for timely screening, prevention, and interdisciplinary intervention. This perspective is especially relevant in healthcare settings facing a growing burden of metabolic disease.

In the context of medical education and health systems in regions such as Mexico, Colombia, and Ecuador, adopting a systemic and integrative approach to chronic inflammatory conditions is essential. Viewing dermatological disease through a multisystem lens fosters comprehensive clinical reasoning, supports preventive medicine strategies, and aligns with contemporary models of patient-centered care.

In conclusion, chronic cutaneous inflammation and metabolic syndrome should be understood not as coincidental comorbidities, but as interconnected expressions of systemic inflammatory dysregulation. Recognizing this relationship provides a foundation for improved clinical assessment, multidisciplinary management, and future research aimed at reducing the long-term metabolic and cardiovascular burden associated with chronic inflammatory skin diseases.

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