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Comparative Study of Psoriasis and Atopic Dermatitis

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Abstract: This comprehensive comparative study aims to elucidate the distinctive and overlapping aspects of psoriasis and atopic dermatitis. These two prevalent chronic inflammatory skin diseases substantially impact global health. Employing a systematic literature review of peer-reviewed articles, clinical trials, and meta-analyses from 2010 to 2024, the research synthesises current evidence on epidemiology, pathophysiology, clinical manifestations, comorbidities, disease burden, and management approaches. Key findings highlight that while both conditions share immune dysregulation and impaired skin barrier function, psoriasis is primarily driven by the IL-23/Th17 axis with characteristic scaly plaques and significant cardiovascular and musculoskeletal comorbidities. In contrast, atopic dermatitis stems from filaggrin deficiency and Th2-biased immunity, presenting with pruritic, lichenified lesions and frequent atopic comorbidities such as asthma and allergies. Both diseases profoundly diminish quality of life through physical symptoms and psychological distress. The study emphasises the necessity of accurate differential diagnosis and advocates for patient-centred, multidisciplinary care strategies that encompass personalised therapeutic plans, psychosocial support, and education to manage these lifelong conditions effectively. Recommendations call for increased public awareness, enhanced diagnostic precision, comprehensive management incorporating mental health, and equitable access to advanced biologic treatments to improve patient outcomes and reduce systemic disease burden.

Keywords: Atopic dermatitis, Psoriasis, Patient-centred care, Th2 immunity

دراسة مقارنة بين مرض الصدفية والتهاب الجلد التأتبي

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المستخلص: تهدف هذه الدراسة المقارنة الشاملة إلى توضيح الجوانب المميزة والمتداخلة للصدفية والتهاب الجلد التأتبي، وهما مرضان جلديان التهابيان مزمنان شائعان يؤثران بشكل كبير على الصحة العالمية. باستخدام مراجعة منهجية للأدبيات العلمية، وتجارب سريرية، وتحليلات تلوية من عام ٢٠١٠ إلى عام ٢٠٢٤، يُجمّع البحث الأدلة الحالية حول علم الأوبئة، والفيزيولوجيا المرضية، والمظاهر السريرية، والأمراض المصاحبة، وعبء المرض، وأساليب إدارته. تُبرز النتائج الرئيسية أنه في حين تشترك كلتا الحالتين في اختلال مناعي وضعف وظيفة حاجز الجلد، فإن الصدفية تُعزى بشكل رئيسي إلى محور IL-23/Th17 مع لويحات متقشرة مميزة وأمراض قلبية وعائية وعضلية هيكلية مصاحبة كبيرة، بينما ينشأ التهاب الجلد التأتبي من نقص الفيلاجرين ومناعة منحازة لـ Th2، ويظهر على شكل آفات حاكة ومتحززة وأمراض تأتبية مصاحبة متكررة مثل الربو والحساسية. يُضعف كلا المرضين جودة الحياة بشكل كبير من خلال الأعراض الجسدية والضيق النفسي. تُشدد الدراسة على ضرورة التشخيص التفريقي الدقيق، وتدعو إلى استراتيجيات رعاية متعددة التخصصات تُركّز على المريض، وتشمل خططًا علاجية مُخصصة، ودعمًا نفسيًا واجتماعيًا، وتثقيفًا، لإدارة هذه الحالات المرضية المُزمنة بفعالية. وتدعو التوصيات إلى زيادة الوعي العام، وتحسين دقة التشخيص، وتوفير إدارة شاملة تُراعي الصحة النفسية، وتوفير فرص متساوية للحصول على العلاجات البيولوجية المُتقدمة لتحسين نتائج المرضى وتخفيف العبء المرضي الجهازي.

الكلمات المفتاحية: التهاب الجلد التأتبي، الصدفية، الرعاية التي تركز على المريض، مناعة Th2.

1. Introduction

Psoriasis and atopic dermatitis occupy a prominent place among chronic inflammatory dermatoses, affecting millions across varied populations and contributing substantially to the global noncommunicable disease burden. [1-2]. Though both conditions are characterised by aberrant immune activation and disruption of the epidermal barrier, they differ in the undercurrent of immunologic pathways, clinical manifestations, hereditary predisposition, and systemic disease associations. From a clinical vantage, psoriasis and atopic dermatitis may display overlapping features—erythema, scaling, and pruritus [3]—that, when considered in isolation, obfuscate the differential diagnosis and may jeopardise therapeutic outcomes, especially in settings outside speciality care. Accordingly, a systematic, comparative framework is essential; such a framework not only sharpens diagnostic precision but also informs therapeutic strategies that seek to address the multifaceted burden of disease while foregrounding the values and lived reality of each patient.

The rationale for a simultaneous inquiry into psoriasis and atopic dermatitis rests on their lifelong trajectory, their substantial psychosocial burden, and the reciprocal interplay of genetic susceptibility and diverse environmental triggers. Both diseases profoundly compromise health-related quality of life, inducing psychological distress, sleep interruption, social stigmatisation, and increased absenteeism [4-5]. In addition, their considerable systemic burden is evident: psoriasis is correlated with heightened risks for cardiovascular pathology, metabolic syndrome, and the inflammatory arthritis variant, whereas atopic dermatitis is frequently associated with the respiratory manifestations of the so-called atopic march, as well as food sensitisations and prevalent psychiatric comorbidities such as anxiety and affective disorders [6-8]. By delineating the distinctive and overlapping dimensions of these disorders, the present analysis seeks to inform a cohesive clinical approach that emphasises early recognition, multidisciplinary management, and proactive measures to mitigate the risk of enduring morbidity.

The global epidemiological impact of psoriasis and axial spondyloarthritis demonstrates consistent and steep upward trajectories [9]. The most recent estimates from the Global Burden of Disease Study published in 2016 reveal that psoriasis now afflicts well over 125 million persons globally and contributes approximately 5.6 million disability-adjusted life-years (DALYs) [10], a burden that already exceeds that of several other major immuno-inflammatory disorders, notably inflammatory bowel disease. Prevalence figures exhibit marked geographic variation, with higher estimates observed in countries progressively farther from the equator; for instance, surveyed rates ascend from 0.9% in the United States to 8.5% in Norway [11-12]. Additionally, a longitudinal investigation conducted in the United States has documented a near doubling of psoriasis incidence over the past three decades, the annual figures rising from 50.8 to 100.5 per 100,000 person-years between 1970 and 1999 [13]. Collectively, these data reinforce the growing importance of psoriasis as a pressing public health priority.

Atopic dermatitis, together with psoriasis, ranks as the most common chronic inflammatory dermatosis, with current global data revealing a prevalence of 15 to 20 % in children and 1 to 3 % in adults [14]. The past thirty years have witnessed a sustained rise in incident cases within industrialised nations, a phenomenon best explained by the interplay of inherited predisposition and shifting environmental factors, including growing urban density, elevated ambient pollutant levels, and altered lifestyle behaviours [15]. Long considered a condition confined to childhood, the disorder now presents a significant and growing cohort of cases with adult onset and late onset, the latter defined as onset at or after 40 years, prompting a critical reassessment of its chronologic categorisation. Longitudinal data indicate that nearly half of children presenting with atopic dermatitis will have persisting symptoms into adulthood [16], substantiating the view of the condition as a dermatologic and systemic trajectory with lifelong implications.

These population-based developments underscore the urgent need for improved public awareness, greater precision in differential diagnosis, and the design of coordinated, multidisciplinary management strategies. In 2014, the World Health Organisation classified psoriasis as a primary noncommunicable disease [17], a designation that galvanised global partners to confront the associated stigma and dismantle barriers to comprehensive treatment.

Concurrently, atopic dermatitis has elevated its prominence on the public health agenda as a disorder exhibiting early-life onset, substantial socioeconomic ramifications, and a persistent predisposition to a spectrum of concomitant atopic diseases of study.

This study is significant as it provides a comprehensive comparative analysis of psoriasis and atopic dermatitis, two widespread chronic inflammatory skin diseases with overlapping clinical features but distinct immunopathologies and comorbidities. By emphasising accurate diagnosis, standardised severity assessments like Psoriasis Area and Severity Index (PASI) Moreover, the Scoring Atopic Dermatitis (SCORAD), and a patient-centred approach that incorporates both objective signs and subjective symptoms, the study aims to improve clinical decision-making, enhance therapeutic outcomes, and address the substantial psychosocial and systemic burdens

these diseases impose. Furthermore, it highlights the necessity for multidisciplinary management and equitable access to emerging therapies, ultimately contributing to better disease control and quality of life for affected patients worldwide.

1.1 Problem Statement

The research problem focuses on the need for improved differentiation and management of chronic inflammatory skin disorders, specifically psoriasis and atopic dermatitis, which impose significant psychosocial burdens and exhibit distinct immunologic triggers, clinical features, and treatment responses. Despite advances in understanding the pathophysiology of these conditions, challenges remain in achieving accurate diagnosis, assessing disease severity comprehensively, and implementing patient-centred care strategies tailored to individual needs. Prior studies [14-16] emphasise the importance of standardised severity assessment tools like SCORAD and PASI for guiding therapeutic decisions and the integration of patient perspectives to optimise treatment outcomes. Additionally, the coexistence of comorbidities and the variability in clinical presentations underscore the complexity of management, necessitating multidisciplinary approaches supported by ongoing research. This problem is further highlighted by the global prevalence and public health impact of these diseases, as noted by the World Health Organisation's classification and epidemiological data.

The problem of psoriasis and atopic dermatitis is underscored by recent and reliable epidemiological data highlighting their substantial prevalence and public health impact:

1. Global Burden: Psoriasis affects well over 125 million people worldwide, contributing approximately 5.6 million disability-adjusted life-years (DALYs), a burden surpassing several other major immuno-inflammatory disorders such as inflammatory bowel disease.
2. Geographic Variation: The prevalence of psoriasis varies significantly by geography, with higher rates in countries farther from the equator. For example, prevalence reaches 8.5% in Norway compared to 0.9% in the United States.
3. Rising Incidence: Longitudinal studies in the United States have documented nearly a doubling of psoriasis incidence over the past three decades, increasing from 50.8 to 100.5 per 100,000 person-years between 1970 and 1999.
4. Rising Atopic Dermatitis Prevalence: Atopic dermatitis ranks alongside psoriasis as one of the most common chronic inflammatory dermatoses globally, affecting 15-20% of children and 1-3% of adults, with incidence rates rising steadily over the past 30 years.
5. Psychosocial and Systemic Impact: Both diseases significantly affect health-related quality of life, leading to psychological distress, sleep disturbances, social stigma, and increased work absenteeism. They also associate with systemic comorbidities; psoriasis with cardiovascular disease, metabolic syndrome, and inflammatory arthritis, and atopic dermatitis with the atopic march (including asthma and food allergies), as well as psychiatric disorders.
6. Public Health Recognition: Psoriasis has been classified by the World Health Organisation as a primary noncommunicable disease, emphasising the urgency to address stigma, improve diagnosis, and enhance access to comprehensive treatment globally.

These statistics emphasise not only the widespread nature of psoriasis and atopic dermatitis but also their growing prevalence and profound multifaceted impact, reinforcing the need for effective patient-centred management strategies.

1.2 Objectives

1. To systematically compare the clinical features, immunologic pathways, and epidemiology of psoriasis and atopic dermatitis to enhance differential diagnosis accuracy.
2. To evaluate and contrast standardised severity assessment tools such as the Psoriasis Area and Severity Index (PASI) and the SCORAD. Index in capturing both objective clinical signs and patient-reported symptoms.
3. To investigate the impact of psoriasis and atopic dermatitis on patients' quality of life, including psychosocial burden, sleep disturbance, and comorbidities, to promote patient-centred care models.
4. To explore current and emerging treatment modalities, including biologic therapies, and their alignment with personalised therapeutic strategies based on disease severity, comorbid conditions, and patient preferences.
5. To provide evidence-based recommendations for improving multidisciplinary management and resource allocation to address the individual and public health challenges posed by these chronic inflammatory skin diseases.

2. Methodology

The methodology section outlines the systematic approach employed to gather, evaluate, and synthesise current evidence on psoriasis and atopic dermatitis. Emphasising rigour and transparency, this section details the comprehensive literature search strategy, including database selection, search terms, and inclusion-exclusion criteria designed to capture high-quality, clinically relevant studies. It further describes the multi-step screening and selection process implemented to minimise bias and ensure reproducibility. Finally, it explains the standardised data extraction and synthesis procedures used to integrate diverse findings, thereby establishing a robust foundation for the ensuing comparative analysis and recommendations.

2.1. Study Setting

2.1.1 Literature Search Strategy

A comprehensive and systematic literature search was conducted across four major electronic databases—PubMed, Scopus, Web of Science, and Google Scholar—to identify relevant studies published between January 2010 and November 2024. Search terms combined keywords and Medical Subject Headings (MeSH) related to "psoriasis," "atopic dermatitis," "severity assessment," "clinical features," "treatment," and "patient-centred care." Boolean operators (AND, OR) were applied to refine the search scope. The search was limited to articles published in English and involving human subjects to ensure applicability to clinical practice. Reference lists of key articles were also screened to capture additional pertinent studies not retrieved in the initial search. This approach ensured a broad yet focused evidence base covering epidemiology, clinical characteristics, comorbidities, and management strategies for psoriasis and atopic dermatitis.

2.1.2 Inclusion and Exclusion Criteria

Studies were included if they were peer-reviewed original research articles, multicenter clinical trials, systematic reviews, or meta-analyses that provided substantial data on psoriasis and/or atopic dermatitis, focusing on disease characterisation, severity scoring tools, treatment outcomes, or associated comorbidities. Eligible studies needed to supply comprehensive clinical, epidemiological, or therapeutic dimensions and be published in English. Exclusion criteria encompassed non-English language papers, single case reports, conference abstracts lacking full-text availability, editorials, and studies deemed methodologically weak or peripheral to the main research questions. This rigorous selection ensured the inclusion of high-quality, relevant evidence to underpin the comparative analysis.

2.1.3 Screening and Selection Process

The initial search results underwent a two-step screening process to identify eligible studies systematically. First, two independent reviewers screened titles and abstracts to exclude duplicates and clearly irrelevant articles. Next, full texts of potentially relevant articles were retrieved and appraised against the predefined inclusion and exclusion criteria. Disagreements between reviewers were resolved through discussion or consultation with a third expert to minimise bias. A PRISMA flow diagram was maintained to document the number of records identified, screened, excluded, and included, thereby ensuring transparency and reproducibility of the selection process.

2.1.4 Data Extraction and Synthesis

Data were meticulously extracted from included studies using a standardised extraction form capturing key elements such as study design, population characteristics, clinical features, assessment tools used (e.g., PASI, SCORAD), treatment modalities, outcomes, and noted comorbidities. Extracted data were synthesised qualitatively to compare and contrast findings across studies. Where applicable, quantitative data such as severity scores and treatment efficacy measures were tabulated to facilitate comparative analysis. This integrated synthesis enabled a comprehensive understanding of the current landscape of psoriasis and atopic dermatitis management and highlighted gaps and consensus in clinical practice.

2.2 Psoriasis

Psoriasis is a chronic immune-mediated skin condition marked by well-defined, reddened plaques covered with silvery scales, commonly affecting the scalp, extensor surfaces of the elbows and knees, and the lumbosacral region [19]. It arises from a complex interaction of genetic predisposition and diverse environmental triggers, culminating in the activation of the IL-23/Th17 axis, which accelerates keratinocyte turnover and sustains a dysregulated inflammatory environment [19]. Clinicians confirm the diagnosis by noting characteristic features, such as the appearance of bleeding points after scale removal (the Auspitz phenomenon) [20], the development

of lesions at sites of trauma (the Koebner phenomenon) [21], and nail changes, including pitting and onycholysis [22]. The condition frequently coexists with inflammatory arthritis, metabolic syndrome, cardiovascular disease, and psychiatric morbidity. Despite etiological pathways that exclude transmissibility, the condition inflicts a significant psychosocial burden. The World Health Organisation classifies psoriasis as a severe noncommunicable disease, underscoring its relevance to global public health.

2.2.1 Epidemiology

Countries that are located further from the equator, particularly along the Northern European peninsula, tend to have a higher prevalence [23]. The clinical severity of the condition may be exacerbated, and immune dysregulation may be exacerbated by these geographic differences, which are thought to be the result of varying lifestyle patterns, lower temperature regimens, and reduced ultraviolet radiation [24].

The chronology of the first symptom expression in psoriasis is marked by significant variability, with two primary age clusters: the first peaks between the ages of 20 and 30 and the second between the ages of 50 and 60 [25]. The joint contributions of inherited susceptibility and differing ambient exposures are underscored by the wide range of prevalence figures among children, which range from 0% in Taiwan to 2.1% in Italy [26]. Studies indicate that the earliest-presenting phenotypes may be differentially influenced by sex-modulated hormonal or immunological factors, despite an overall sex ratio that is close to parity in adults [27].

Genetic predisposition has a profound impact on the aetiology of psoriasis, with an increasing number of susceptibility loci identified throughout the genome. The PSORS1 region, located on chromosome 6p21.3, is the most prominent of these loci. The HLA-C 06:02 allele is consistently associated with an earlier age at disease onset within this genomic enclave [28-29]. In genetically susceptible individuals, a constellation of environmental exposures—including infection by streptococci, cigarette smoking, excessive ethanol consumption, obesity, psychological stress, and specific pharmacologic agents (notably lithium, beta-adrenergic antagonists, and certain 4-aminoquinolines) [30-32]—can precipitate or exacerbate psoriasis. Nevertheless, heritable risk is neither fully adequate nor exclusive.

Although psoriasis was classified as a severe noncommunicable disease in the World Health Organisation's 2014 report, low- and middle-income countries continue to experience substantial disparities in the availability of treatment, diagnostic precision, and surveillance [33].

2.2.2 Clinical Features

Erythematous lesions, finely defined and frequently covered by silvery, lamellar scales, distinguish psoriasis. The symmetrical lesional distribution typically affects the elbows, knees, cranium, and lower back [20]. The increased stratum corneum is a result of the increased turnover of keratinocytes, which is facilitated by the dysregulation of the immune system, particularly through the activation of the T-helper 17 pathway and interleukin 23 [34]. Although patients report varying degrees of distress, such as pruritus, stinging, or tenderness, the intensity of pruritus is typically less severe than that observed in atopic dermatitis [35]. The disease is relapsing and chronic, with new exacerbations frequently attributable to specific environmental or systemic triggers.

During an examination, one may observe Auspitz's sign, which features pinpoint haemorrhage points that appear when the scale is removed, and the Koebner phenomenon, where new plaques emerge on previously traumatised skin [36]. Approximately 50% of patients have nail dystrophy, which can include longitudinal pitting, onycholysis, erythrodermic oil patches, and keratin congregation beneath the nail plate [37]. These modifications have the potential to deteriorate psychosocial well-being and impede fine motor function.

There are numerous significant clinical variants of psoriasis:

- Plaque psoriasis [38], the most common form (80-90%), is characterised by raised, erythematous plaques that preferentially cover extensor surfaces and exhibit characteristic silvery scaling.
- Guttate psoriasis [39] is characterised by well-circumscribed, oval lesions that resemble droplets and often occur following an episode of Group A beta-hemolytic streptococcal pharyngitis. Its incidence is highest in pediatric and adolescent populations.
- Inverse psoriasis [40] is characterised by smooth, glistening red lesions that exhibit little to no visible scale in skin fissures, including the axillae, groin, and inframammary areas.

- Pustular psoriasis [41] can manifest as confined pustules on the palms and soles or in a disseminated pattern across the trunk and proximal limbs. The latter form can trigger a widespread systemic inflammatory response and requires immediate inpatient management.
- Erythrodermic psoriasis [42] is distinguished by a continuous red surface that covers the majority of the skin, sheds scales, and poses acute risks of sepsis, electrolyte disturbance, and cardiocirculatory failure.

2.2.3 Comorbidities

Psoriasis is a systemic inflammatory disorder that is associated with a broad spectrum of comorbidities, transcending cutaneous involvement. The convergence of psoriasis with divergent pathogenic mechanisms suggests that its cutaneous symptoms are merely the most visible aspect of a pervasive disorder. The most critical comorbidities are as follows [43-45]:

- Psoriatic arthritis affects approximately 20–30% of patients.
- An increased risk of stroke and myocardial infarction demonstrates cardiovascular disease.
- Metabolic syndrome is characterised by central adiposity, insulin resistance, and dyslipidemia.
- Chronic kidney disease.
- NAFLD
- Inflammatory bowel disease (Crohn's disease)
- Mood disorders, most notably anxiety and depression

The persistent elevation of inflammatory cytokines, such as interleukin-17 and tumour necrosis factor-alpha, is considered a central mechanism that transmits endothelial dysfunction and converts cutaneous pathology into an atherogenic phenotype.

2.2.4 Pathogenesis

The development of psoriasis is a carefully controlled, immune-mediated process that arises from complex interactions between a variety of environmental stimuli, both arms of the immune system, and hereditary predisposition [46]. When endogenous nucleic acids attached to antimicrobial peptides produced from injured keratinocytes during mechanical trauma, microbial infection, or psychological stress are recognised by dermal dendritic cells, the illness is first triggered. Upon reaching draining lymph nodes, these activated dendritic cells produce a variety of cytokines, including interleukin-23, tumour necrosis factor-α, and type I interferons, which promote the differentiation and widespread growth of T helper 17 (Th17) cells [47-48]. Interleukin-17A, interleukin-17F, interleukin-22, and other tumour necrosis factor-α are the main cytokines released by activated Th17 cells [49]. These cytokines directly stimulate keratinocytes, causing them to proliferate more quickly, disrupting their normal differentiation, and triggering the synthesis of more inflammatory mediators and antimicrobial peptides [49].

Psoriatic plaque is characterised by the classic dermatopathological triad of thickened epidermis (acanthosis), the presence of uninucleated keratinocytes in the stratum corneum (parakeratosis), and the formation of small, intra-epidermal collections of neutrophils (microabscesses), which are all caused by the cytokine-mediated chain reaction [38].

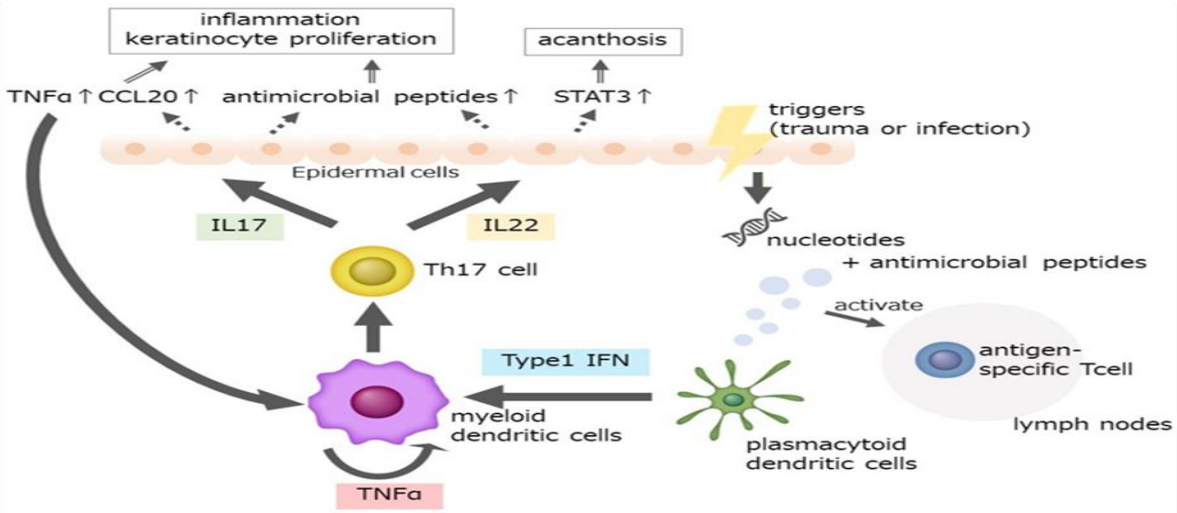

Figure 1: Psoriasis pathophysiology: Skin injury or infection triggers keratinocytes to release antimicrobial peptides and nucleotides, forming a complex detected by plasmacytoid dendritic cells. These cells produce type I interferons, activating myeloid dendritic cells to release TNF and IL-23. Together with IL-1, they stimulate Th17 cells to produce IL-17 and IL-22. IL-17 drives inflammation and keratinocyte proliferation by inducing TNF, CCL20, and more antimicrobial peptides [48].

The primary cause of psoriatic inflammation is now understood to be the IL-23/Th17 signalling pathway. Activated dendritic cells primarily release IL-23, which is crucial for Th17 cell persistence and clonal proliferation [19]. For this reason, blocking this cytokine is a strong approach to customised treatment (see figure 1). Numerous susceptibility loci have been found by genome-wide association studies; the most important of them is PSORS1 on chromosome 6p21.3, where the HLA-C 06:02 variation is highly correlated with an increased risk of early-onset illness [28-29]. For those who are genetically susceptible, environmental stimuli can either reveal or worsen the disorder: Guttate psoriasis is a common symptom of group A β-hemolytic streptococcal infection, and the risk is increased by stress, obesity, smoking, and several systemic medicines [39]. One noteworthy feature is "lesional memory," which occurs when previously afflicted skin has a propensity to recur even after clinical remission. This is attributed to long-lasting epigenetic and transcriptional alterations in the epidermis that prepare the tissue for a rapid inflammatory response [50].

2.2.5 Treatment Approaches

Reducing skin inflammation, restoring normal keratinocyte proliferation, and generally improving the patient's health-related quality of life continue to be the key treatment goals. The level of intervention is increased in proportion to the degree and severity of illness presentation, and treatment regimens are arranged hierarchically into topical treatments, phototherapeutic modalities, and systemic medications.

The initial therapeutic approach for patients with mild psoriasis, which is defined as less than 10% of the body surface area affected, usually consists of vitamin D analogues, most commonly calcipotriene, and highly effective topical corticosteroids, along with regimen combinations designed to maximise anti-inflammatory activity [51-52]. Patients may also use topical therapies containing salicylic acid or tar-based medications to minimise scale and subepidermal irritation further.

Therapists typically refer patients with moderate to severe illness, defined as those with high plaque, particularly in sensitive anatomical locations, to phototherapeutic methods. It has been demonstrated that both narrowband UVB and PUVA (psoralen plus UVA) slow down keratinocyte turnover and inhibit the faulty immune systems that cause psoriasis [53].

Therapists must, however, advise patients of the hidden danger of cumulative UV exposure that might cause cancer. Systemic treatments are necessary if psoriasis spreads widely or does not respond to topical and phototherapeutic treatments. Traditional non-biologic treatments for psoriasis include cyclosporine, which should only be used for short periods because it is nephrotoxic; methotrexate, which requires regular monitoring for liver toxicity; and acitretin, which works well for pustular phenotypes but should not be used during pregnancy [54].

Biologics that target specific immunologic cascades, including TNF-α, IL-17, IL-12/23, or IL-23, are highly effective; in most patients with moderate to severe illness, they typically achieve PASI 90 and PASI 100 responses. Etanercept, adalimumab, ustekinumab,

ixekizumab, and guselkumab are among the agents in this group [55]. Before starting therapy, a thorough test for latent infections, such as hepatitis B and TB, must be carried out.

Management teams must keep the patient at the centre while considering the patient's complete medical history, any concomitant chronic diseases, socioeconomic background, and

future reproductive goals. When topical treatments fail to provide enough response in children, systemic biologics, namely etanercept, adalimumab, ustekinumab, and ixekizumab, are authorised [56]. Achieving and maintaining a long-lasting remission with the least amount of risk and the highest level of tolerability is the main treatment objective.

2.3 Atopic Dermatitis (AD)

It is the most prevalent kind of eczema. It represents the "atopic march," often occurring before food sensitisation, asthma, and allergic rhinitis. Filaggrin loss-of-function mutations, impaired barrier integrity, Th2-skewed immunity, and a variety of environmental triggers, such as inhaled allergens, irritants, and Staphylococcus aureus colonisation, are all characteristics of this genetically conditioned illness [57]. New-onset adult instances do occur, and juvenile cases may continue, even though the majority of cases start in infancy or childhood. Clinical diagnosis is made, and established measures like Scoring Atopic Dermatitis (SCORAD) are used to measure severity [58].

2.3.1 Epidemiology

Atopic dermatitis (AD), which affects 1–3% of adults and 15%–20% of children, is still the most common chronic inflammatory skin condition [59]. Incidence rates have gradually increased over the last three decades, particularly in high-income nations. About half of afflicted children carry the disease into adulthood, although the sickness usually manifests by the age of five [60]. Late-onset disease, which manifests symptoms after age forty, has also gained more attention recently. The most significant occurrence is found in urban, industrialised areas, which may be related to modern lifestyle variables and airborne particulate pollution.

AD has a complex aetiology that combines environmental factors and genetic predisposition. When the stratum corneum barrier is compromised by mutations in the filaggrin (FLG) gene, exposure to allergens, irritants, psychological stresses, and severe weather conditions can trigger immunological inflammation [61].

2.2.2 Clinical Features

The first lesions appear as erythematous, exudative plaques on the cheeks, scalp, and extensor surfaces during infancy (Table 1). Lesions often localise to the flexural regions, primarily the antecubital and popliteal fossae, by preschool and school age and continue throughout adulthood. There, they become lichenified as a result of constant scratching. While serous exudation characterises flare episodes and tiny scales and fissures predominate during interflare times, the afflicted skin remains persistently xerotic [62]. As a result of the underlying barrier failure, AD lesions are less well defined than psoriatic plaques and are more vulnerable to Staphylococcus aureus colonisation [63].

The itching-scratch cycle exacerbates dermatitis and the breakdown of the epidermal barrier. At the same time, sleep disturbance brought on by nocturnal pruritus causes exhaustion and increased suffering for both patients and caretakers. In chronic instances, recurring conjunctival inflammation, preauricular fissures, and infraorbital hyperpigmentation are associated with extracutaneous symptoms. Instead of the usual erythematous scaling, lesions may manifest as hyperpigmented or hypopigmented macular regions in individuals with colored skin [64]. Asthma, food allergies, and allergic rhinitis frequently follow the onset of atopic dermatitis, which is thought to be the initial clinical manifestation of the atopic march [65].

Table 1: Comparison of Psoriasis and Atopic Dermatitis – Clinical Features and Epidemiology

Feature	Psoriasis	Atopic Dermatitis
Age of Onset	Can occur at any age; often adults	Typically begins in early childhood
Typical Lesion Appearance	Sharply defined, red plaques with silvery-white scales, symmetrical distribution mainly on scalp, elbows, knees, trunk,	Eczematous lesions with varying intensity, often on the flexural areas, face, and neck

Feature	Psoriasis	Atopic Dermatitis
Associated Symptoms	Itching, pain, stinging, bleeding upon scale removal (Auspitz's sign)	Intense itching, dryness, and skin thickening
Prevalence/Incidence	Variable globally; data from the Global Psoriasis Atlas is ongoing.	Common worldwide; prevalence data vary regionally
Genetic & Immunological Aspects	Multiple genetic loci identified; immune dysregulation involving T cells and cytokines,	Genetic predisposition and complex immune alterations involving barrier dysfunction and immune activation

2.2.3 Comorbidities

The burden of atopic dermatitis (AD) is increased by the significant physical and mental comorbidities it presents. The condition commonly triggers the well-known "atopic march" in children, which progresses to rhinitis, asthma, and food hypersensitivity [66]. Research shows that whereas asthma rates range from 14.2% to 52.7%, up to 75% of young children with severe AD go on to acquire allergic rhinitis [67]. The incidence of food allergies is higher than that of the general pediatric population, indicating similar enrichment.

When the epidermal barrier is compromised, people are more susceptible to recurring cutaneous infections, the most common of which are caused by Staphylococcus aureus colonisation and the herpes simplex virus, which results in dermatitis herpeticum. Other ocular sequelae, such as allergic conjunctivitis, keratoconus, and cataract development, may develop in chronic instances [68]. Though not yet as strong as the evidence for systemic inflammation in psoriasis, preliminary research suggests that inflammation may have broader ramifications beyond only the skin.

Due in large part to the constant itching, related sleep deprivation, and widespread social stigma, people with atopic dermatitis at different stages frequently suffer from comorbid psychological illnesses, including anxiety, depression, ADHD, and suicidal thoughts. Once started, the cycle of itching and scratching can become self-sustaining, deteriorating quality of life to the point that the condition's physical symptoms are subordinated to its psychological effects [69]. As a result, the methodical detection and management of these comorbidities continue to be crucial elements of comprehensive, patient-centred care.

2.2.5 Pathogenesis

Heritable characteristics, environmental exposures, and microbial populations all influence the pathophysiology of atopic dermatitis, which is characterised by an interaction between immune dysregulation and epidermal barrier impairment. Mutations in the filaggrin (FLG) gene that are homozygous or heterozygous impair lipid organisation and keratinocyte differentiation, leading to xerosis, transepidermal water loss, and an increased susceptibility to allergens and irritants [70]. The immune system's allergy arms are activated by reactivity to these antigens, and this is initially characterised by a distorted type 2 helper T (Th2) cytokine profile.

Therefore, overproduction of interleukin (IL)-4, IL-5, and IL-13, which collectively stimulate IgE synthesis, mast-cell activation, and eosinophilia, is a hallmark of clinically acute flares, whereas IL-31 is a significant cause of persistent pruritus [71]. The immune landscape changes to a mixed Th1/Th22 response in the chronic stage, causing acanthosis and lichenification due to the continuous production of interferon-γ, tumour necrosis factor-α, and IL-22 [72].

Reduced production of antimicrobial peptides, particularly human cathelicidin, also makes it easier for Staphylococcus aureus to colonise large areas; in more than 90% of acute flares, colonisation has been culture-proven [73]. At any stage of the illness, environmental variables such as allergens, air pollution, psychological stresses, and climate changes can exacerbate atopic dermatitis symptoms.

At the same time, changes in the gut microbiota that were discovered as early as three months of life have been strongly linked to a higher risk of developing atopic dermatitis and its progression through the atopic march to asthma and food allergy symptoms [74]. Atopic dermatitis must be recognised as a systemic condition rather than just a cutaneous one because of this early microbial disruption, which seems to start a long-lasting and self-replicating cycle marked by epithelial barrier dysfunction, continuous immune activation, and persistent cutaneous and systemic inflammation.

2.2.6 Treatment Approaches

The severity of the illness, the patient's age, and particular therapy objectives—such as barrier repair, inflammation control, pruritus relief, and flare prevention—must all be considered when developing treatment paradigms for atopic dermatitis. Frequent use of moisturisers is beneficial for patients with isolated mild illness. Topical corticosteroids are often used to treat flare-up episodes, with tacrolimus and pimecrolimus being saved for sensitive skin areas [75]. Children two years of age and older are recommended to use the topical phosphodiesterase four inhibitor Crisaborole. While systemic medications with immunosuppressive qualities, such as methotrexate and cyclosporine, may be used for shorter

durations under strict supervision, narrowband UVB phototherapy is effective for moderate-to-severe symptoms in adults and older children [76]. Biologics that specifically block the IL-4 and IL-13 signalling pathways are now part of the recent therapeutic evolution, and dupilumab has been approved by the FDA for use in patients six months of age and above [77].

Oral Janus kinase inhibitors, such as abrocitinib and upadacitinib, offer immediate alleviation of pruritus if dupilumab is unable to provide sufficient control; nevertheless, continued use of these medications necessitates strict safety monitoring [78]. Strategic management includes targeted microbial control, such as weekly diluted bleach baths to reduce Staphylococcus aureus colonisation, along with proactive avoidance of identified triggers, such as irritants, allergies, and emotional stress [79]. Ongoing psychological assistance is an essential part of care since inflammatory skin illnesses are known to coexist with anxiety, sleeplessness, and other psychosocial aftereffects. According to the reviewed research, the foundation for long-lasting therapeutic benefit continues to be a patient-centred, individualised framework in which medical intervention, mental health, and educational empowerment are all smoothly linked.

3. Literature Review

The following studies collectively advance understanding and management of psoriasis and atopic dermatitis through diverse methodological approaches. Study [55] developed and validated the Psoriasis Area and Severity Index (PASI) using observational and clinical trial data, effectively quantifying key clinical parameters such as erythema, thickness, scaling, and the extent of body surface area involvement. While PASI demonstrated reliability in assessing psoriasis severity and monitoring treatment response, its limitations include insufficient integration of patient-reported outcomes, particularly pruritus and the associated psychological impact.

Study [56] employed a longitudinal trial to evaluate biologic therapies using the PASI 75 benchmark, confirming substantial lesion clearance yet highlighting persistent residual symptoms and the necessity for enhanced patient-reported outcome metrics alongside objective assessments.

Study [58] introduced and validated the SCORAD index for atopic dermatitis, integrating objective clinical signs with subjective symptoms such as pruritus and sleep disturbance, though its complexity and lack of standardisation impede widespread clinical utility.

Study [65] utilised a cohort design to affirm nail manifestations—including pitting and onycholysis—as common in psoriasis, correlating with severity, while noting inconsistent reporting methods and scarce data on quality-of-life impacts. In a prospective cohort Study [66] linked early atopic dermatitis onset within the atopic march to later asthma and allergic rhinitis, though environmental and genetic heterogeneity complicates causal inferences. Study [67] conducted a meta-analysis identifying critical IL-23/Th17 pathway genes underlying susceptibility to both conditions but noted limitations in generalizability due to heterogeneous study populations and designs. Randomised controlled trials in Study [78] assessed oral JAK inhibitors like abrocitinib for pruritus relief in atopic dermatitis, evidencing rapid efficacy tempered by safety concerns warranting vigilant monitoring.

Study [79] examined weekly diluted bleach baths to reduce Staphylococcus aureus colonisation in atopic dermatitis via a prospective intervention, supporting microbial control as adjunct therapy while recognising uncertainties about optimal regimens and long-term benefits. Qualitative analyses in Study [80] underscored patient-centred care frameworks emphasising shared decision-making and individualised treatment plans to enhance adherence and satisfaction, though constrained by healthcare system limitations and variable clinician training. Observational research in Study [36] validated the Koebner phenomenon and Auspitz's sign as diagnostic indicators for psoriasis, calling for standardisation across populations and clinical education. Epidemiological mapping and molecular studies in Study [19] identified IL-23/Th17 axis dysregulation as central to psoriasis pathogenesis. However, they highlighted challenges in applying molecular insights to personalised treatment due to disease heterogeneity.

A systematic review in Study [14] summarised psoriasis-associated comorbidities, notably cardiovascular and metabolic risks, revealing gaps in longitudinal data and inconsistent screening practices. Study [17] described the WHO's classification of psoriasis as a primary noncommunicable disease, emphasising the imperative for public health campaigns, stigma reduction, and integrated dermatological services amid resource constraints. Registry data explored in Study [51] advocated biologic treatments beneficial for patients with comorbid psoriatic arthritis and cardiovascular disease, though access limitations restrict broader implementation. Survey research in Study [52] highlighted reproductive concerns affecting psoriasis treatment decisions, underscoring the need for personalised counselling and safer therapeutic options. Patient education programs for atopic dermatitis examined in Study [80] enhanced treatment adherence and psychological outcomes in multicenter trials but faced challenges in scalable delivery.

Study [81] stressed standardised outcome measures such as PASI 75 in biologic trials for consistent efficacy evaluation despite patient heterogeneity. Finally, controlled studies in Study [53] confirmed the effectiveness of topical corticosteroids in atopic dermatitis management, while noting adherence challenges and side effects, signalling the need for balanced therapeutic strategies. Together, these investigations attest to the multifaceted and evolving landscape of clinical research aiming to optimise diagnosis, treatment, and patient-centred care in psoriasis and atopic dermatitis.

4. Incorporate Patient-Centred Perspectives

Integrating patient-centred viewpoints is the crucial next step in expanding treatment benefits for psoriasis and atopic dermatitis. Unless operationalised as care suited to each patient's multifaceted reality, the substantial evidence shedding light on the causes of disease and therapeutic advancements would remain stagnant in the clinic. Advancements in translational dermatology have revealed the immunological and genetic makeup of these disorders; yet, the ongoing gap between bench and bedside indicates that success depends on more than just gaining information. A continuous, iterative process that requests and integrates the patient's values, daily experiences, and treatment objectives at every clinician contact is necessary to activate patient-centred care.

As seen in Figure 2 [80], a truly patient-centred approach emphasises the idea of shared decision-making, in which patients, their families, and medical professionals collaborate to create customised management plans. Given that both psoriasis and atopic dermatitis are chronic, lifelong illnesses characterised by erratic flare-ups, significant symptom loads (such as itching, pain, and visual lesions), and extensive effects on health-related quality of life, this procedure is essential. Healthcare professionals often fail to fully acknowledge patients' anguish, underscoring the importance of empathetic, in-person communication that fosters trust. Whether the patient wants the cleanest skin possible, the least disruptive side effects, a less time-consuming routine, or quantifiable gains in sleep and psychological well-being, this kind of conversation is crucial to aligning treatment goals with their primary priorities.

Patient-centred care for psoriasis management requires adjusting interventions to a comprehensive, multifaceted profile that includes the severity of the disease, related comorbidities (such as psoriatic arthritis and increased cardiovascular risk), personal lifestyle factors, treatment accessibility, and reproductive considerations [51-52]. A real-world example would be a woman of childbearing age who expressly refuses methotrexate due to teratogenicity concerns. At the same time, a person with obesity and metabolic syndrome is recommended to start a biologic medication whose reduction of cardiovascular risk contributes to therapeutic benefit. Similar to this, atopic dermatitis treatment needs to go beyond the conventional use of topical corticosteroids and moisturisers. It needs to include thorough training on avoiding allergens and irritants, psychosocial interventions for coexisting anxiety and sleep disorders, and organised assistance for caregivers, who frequently experience significant stress due to the rigorous and frequent nature of the necessary skin-care routine.

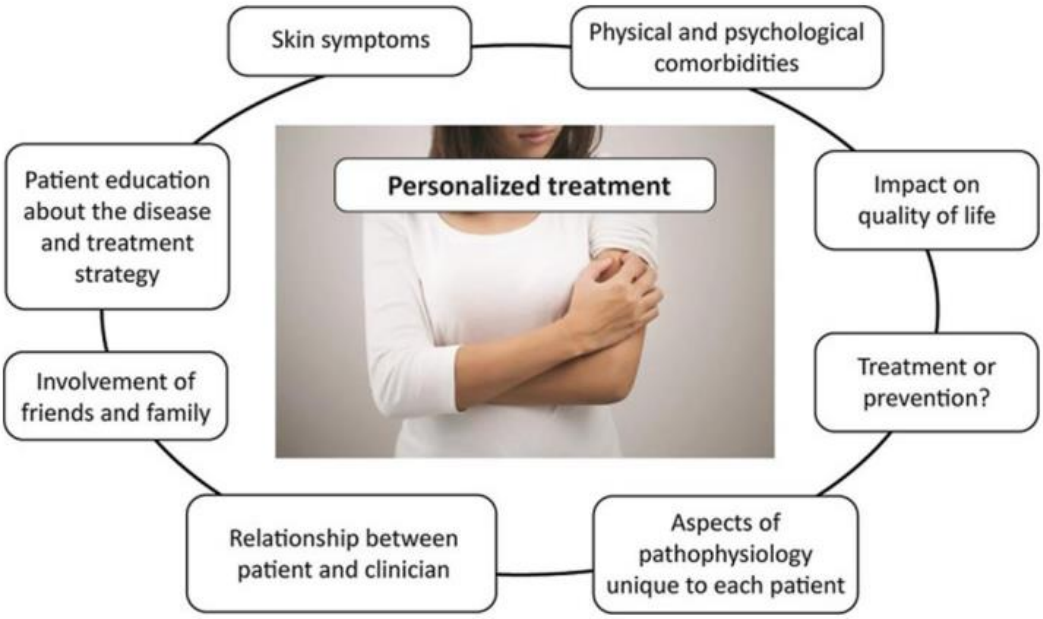

Figure 2. A personalised treatment strategy that incorporates a patient-centred approach for those with psoriasis or atopic dermatitis [80].

5. Comparative Review

Despite being categorised as chronic immune-mediated inflammatory skin diseases, psoriasis and atopic dermatitis (AD) differ significantly in their underlying pathophysiology, clinical presentations, associated comorbidities, and cumulative disease burden [19,57]. Although each condition has a substantial impact on quality of life, they vary in terms of specific immunologic triggers, systemic health outcomes, and the combination of pharmacological and non-pharmacological treatments that produce the best improvement. The main conclusions of the systematic literature review are summarised in this part, with a focus on their significance for informing clinical decision-making, creating patient-centred treatment plans, and creating evidence-based public health campaigns and resource allocation plans.

5.1 Standardised Severity Assessment in Psoriasis and Atopic Dermatitis

Precise assessment of illness severity is essential for guiding therapeutic decision-making, assessing treatment outcomes, and maintaining consistency in clinical practice and research investigation. Table 2 summarises two of the most often used, empirically validated instruments for this purpose: the SCORAD index [58] and the PASI index [55]. The PASI quantifies erythema, induration, and scaling, which are objective clinical criteria, based on their distribution in four anatomically defined locations. The severity of each site is altered based on the %age of the BSA that is affected. By providing a quantitative, repeatable measure of illness severity through an integrated combination of clinical symptoms, PASI has proven to be a valuable endpoint in clinical studies and a standard by which to measure the impact of systemic and biologic therapies [55-56]. Nowadays, the PASI 75 threshold—which represents a 75% decrease from baseline—is a commonly accepted criterion for determining treatment efficacy, especially in biologic drug studies [81]. Even though the PASI is reliable in assessing the severity of plaque psoriasis, it still has a limited ability to account for patient-reported aspects like pain, pruritus, and the psychological effects of visible plaques, which are crucial to a thorough understanding of disease burden.

Table 2: Scoring and Severity Assessment Tools

Tool	Disease	Description	Components
PASI	Psoriasis	Composite score measuring severity and coverage of lesions	Erythema, thickness, scaling, % body area affected
SCORAD	Atopic Dermatitis	Composite score assessing the extent, intensity, and symptoms	Area, intensity (redness, swelling, oozing), and subjective symptoms (itch, sleep loss),

On the other hand, the SCORAD index was purposefully designed to capture the complexity of atopic dermatitis [58]. It measures both subjective patient-reported symptoms, such as pruritus and disturbed sleep during the previous three days, and objective clinical indicators, such as the %age of body surface area affected, the degree of erythema, oedema, exudation or crusting, lichenification, excoriations, and xerosis. Through the integration of both patient-driven and observer-gauged factors, SCORAD directly tackles the detrimental effects of severe pruritus and the ensuing loss of sleep on general health. By emphasising the patient's experience, the score validates symptom reduction as a treatment objective in addition to cutaneous clearance. SCORAD and earlier indices agree on the critical significance of systematic, repeatable evaluation in chronic inflammatory skin disorders, notwithstanding this complex approach. Their ongoing application in registry studies, outpatient consultations, and clinical trials promotes meaningful epidemiological and interventional comparisons across various cohorts and therapeutic modalities, enhances clinician-patient communication, and helps optimise individualised treatment.

5.2 Comparative Clinical Features and Diagnostic Challenges

Based on the morphologic features listed in Table 3, practising dermatologists surveying psoriasis and atopic dermatitis may rapidly distinguish between the two conditions. Sharply defined, highly erythematous plaques with silvery-white lamellar scales on top are the typical presentation of psoriasis; lesions tend to form on extensor surfaces, such as the elbows, knees, scalp, and sacrum, and they are symmetrically distributed. Up to 50% of patients may also have various nail findings, such as pitting, onycholysis, and subungual hyperkeratosis [36,65]. The Koebner phenomenon, which causes new plaques to form at the locations of previous injuries, and Auspitz's sign, in which precise drops of blood emerge when scales are removed, are examples of confirmatory diagnostic findings. On the other hand, atopic dermatitis is typified by eczematous patches that are poorly defined and extremely itchy. While babies may exhibit red, exudative, and crusty patches on the face, head, and dorsal surfaces of the limbs, lesions in older children and adults primarily localise to flexural locations, such as the antecubital and popliteal fossae.

Severe itching is the defining clinical characteristic of atopic dermatitis (AD), and it is frequently more intense at night. This itching-scratch cycle exacerbates immunological inflammation and further deteriorates the epidermal barrier [62-63]. Unlike psoriasis, where increased keratinocyte turnover is reflected in circinate scaling, AD's scaling and dryness are primarily caused by a breakdown of barrier function, which is typically brought on by heritable mutations in the filaggrin (FLG) gene [34-35]. These alterations hinder the stratum corneum's terminal differentiation, which increases transepidermal water loss and allows microbial and environmental allergens—particularly Staphylococcus aureus, which colonises more than 90% of AD clinical flares—to penetrate the epidermis.

Table 3: Comparative Overview of Psoriasis and Atopic Dermatitis

Aspect	Psoriasis	Atopic Dermatitis
Epidemiology	**0.9–8.5% adults, genetic & environmental risk**	**Affects 15–20% children, 1–3% adults, with a rising incidence**
Clinical Features	**Scaly, red plaques, nail involvement**	**Pruritic eczematous lesions, skin barrier defect**
Comorbidities	**Cardiovascular, arthritis, metabolic syndromes**	**Asthma, allergies, and psychological disorders**
Disease Burden	**High DALYs, stigma, disability**	**Psychosocial burden, economic costs**
Pathogenesis	**Immune hyperactivation (Th1/Th17, cytokines)**	**Barrier dysfunction, Th2 dominance**
Treatment	**Topicals, phototherapy, biologics**	**Emollients, anti-inflammatories, systemic in severe cases**

Diagnostic ambiguity may persist even when the underlying processes are transparent, particularly in primary care settings with minimal dermatological competence. Erythema, scaling, and pruritus are overlapping clinical symptoms that can make it challenging to distinguish AD from psoriasis, especially in the early stages of the condition or when lesions localise in unexpected ways. Nummular eczema can show up as circinate patches that resemble psoriatic plaques, whereas inverse psoriasis can show up in flexural zones that are usually classified as chronic eczema. On the surface, lichenified lesions associated with persistent AD might be mistaken for psoriatic plaques, and vice versa. To receive an accurate and timely diagnosis, a comprehensive clinical assessment is necessary, incorporating the history of atopiforms in the person and family, age of start, anatomical distribution, and any associated systemic abnormalities.

5.3 Pathophysiological Divergence: Immune Pathways and Genetic Factors

The pattern of cytokine signalling that controls psoriasis and atopic dermatitis best illustrates the immunological differences between the two conditions. As a self-sustaining autoimmune program, psoriasis is caused by a chronic inflammatory circuit that is geared toward IL-23 and Th17. Naïve T cells are instructed to tilt toward a Th17 destiny by IL-23, which is released by dendritic cells triggered by tissue damage or microbial invasion [49]. The dedicated Th17 population then produces a range of cytokines, including IL-17A, IL-17F, IL-22, and TNF-α, which collectively increase keratinocyte proliferation, disrupt the terminal differentiation program, quicken epidermal turnover, and create an inflammatory feedback loop. Acanthosis, parakeratosis, and the development of microabscesses are the hallmark lesions that show a unified tissue reaction to the cytokine environment. The proportionate decrease in psoriatic plaques seen with biologic drugs targeting IL-23, IL-17, or TNF-α provides evidence for the importance of this circuit [48].

The underlying cause of atopic dermatitis (AD), which is mainly associated with loss-of-function mutations in the filaggrin gene, is a weakened epidermal barrier. A type 2 (Th2) immune response is triggered by this deficiency, which allows environmental allergens, irritants, and microbiological contaminants to penetrate the skin 57,72]. The Th2 effector pathways are dominant during acute exacerbations, leading to increased production of cytokines such as IL-4, IL-5, IL-13, and IL-31, the latter of which is crucial for pruritus perception. As the illness progresses to its chronic stage, there is a reciprocal shift towards the Th1 and Th22 pathways along with an increase in IFN-γ, TNF-α, and IL-22 production [55]. These factors collectively cause epidermal hyperplasia and prolong the inflammatory process. Reduced production of antimicrobial peptides exacerbates this dysregulated cytokine milieu, which not only allows Staphylococcus aureus to colonise but also encourages the reactivation of the herpes simplex virus, leading to the severe presentation of dermatitis herpeticum.

The higher-order methods used to classify psoriasis and atopic dermatitis in modern dermatological classification systems are informed by these pathogenetic features. Unlike atopic dermatitis, which is thought to be a barrier-compromised hypersensitivity state that contributes to the so-called "atopic march"—the sequential clinical evolution toward a constellation of IgE-mediated conditions like asthma, allergic rhinitis, and food allergy—psoriasis is currently understood to be a chronic autoimmune disease with systemic inflammation. Recent data have started to link the susceptibility to atopic dermatitis to early gut microbiome dysbiosis. Disturbances that occur within the first three months after giving birth seem to be associated with the subsequent development of atopic dermatitis and a more generalised allergic response, defining a time-limited window during which microbiome-focused therapies may be effective in preventing these outcomes

5.4 Comorbidities: Systemic Implications and Clinical Relevance

Because both psoriasis and atopic dermatitis rely on chronic, dysregulated immunological mechanisms, they are plagued by overlapping but different comorbidities. The kind and severity of the corresponding systemic disease, however, vary significantly between the two conditions.

The systemic effect is particularly noticeable in psoriasis. 20% to 30% of individuals with psoriatic arthritis experience complications [44]; if treatment and diagnosis are postponed, the resulting joint and entheseal degradation may become permanent. The cardiovascular consequences seem to be equally severe: people with psoriasis have higher rates of myocardial infarction, stroke, and resistant hypertension. This is probably because of the constant release of interleukin-17 and tumour necrosis factor-α [49], which harm endothelial cells and hasten the development of atherosclerotic plaques (Table 4). Visceral obesity, dysglycemia, dyslipidemia, and high blood pressure are the hallmarks of the metabolic syndrome, which is more common in psoriasis and increases the risk of cardiovascular disease. Non-alcoholic fatty liver disease, progressive renal disease, inflammatory bowel disease (primarily Crohn's disease) [43-45], and mental illnesses, particularly major depressive disorder and anxiety, are examples of additional comorbidities.

The identification of shared genetic loci, such as variations in the IL23R locus, connects Crohn's disease and psoriasis and suggests a pleiotropic immune dysregulation that transcends tissue boundaries, bringing together intestinal and cutaneous inflammatory phenotypes under a common pathophysiological umbrella.

The two main comorbidities associated with atopic dermatitis (AD) are allergies and mental health conditions. As the starting point of the progressive "atopic march," AD significantly raises the risk of developing asthma later on, with an estimated prevalence of 14.2% to 52.7%, and allergic rhinitis, with a burden of over 75% in the most severely affected pediatric populations [67]. Food allergies are also more common, and robust epidemiological evidence shows that sensitisation to specific dietary allergens—most notably

peanuts, cow's milk, and hen's eggs—is closely associated with early-onset, chronic AD. After years of illness, ocular problems such as keratoconus, allergic conjunctivitis, and cataract development may develop. These are probably caused by repeated rubbing and the long-term accumulation of inflammatory mediators. With high rates of anxiety, depressive disorders, attention-deficit/hyperactivity disorder, and recurrent suicidal thoughts, psychiatric comorbidities are common. These psychopathological outcomes are believed to be caused by a combination of recurrent sleep disruption, sociocultural stigma, and the psychosocial burden of visible skin lesions.

Although the prevalence of metabolic and cardiovascular comorbidities varies, an increasing number of longitudinal studies indicate that a low-grade, chronic systemic inflammatory state may encourage vascular dysregulation, increasing the risk of cardiovascular disease, particularly in severe and treatment-refractory AD subpopulations. However, the observed association appears to be smaller than that found in psoriasis, which likely reflects different cytokine production patterns and a usually shorter illness duration in persons with atopic dermatitis.

Table 4: Comorbidities Associated with Psoriasis and Atopic Dermatitis

Comorbidity	Psoriasis	Atopic Dermatitis
Cardiovascular Disease	Increased risk documented,	Less clearly defined, but there is a potential risk via inflammation
Psoriatic Arthritis	Common comorbidity,	Rare
Metabolic Syndrome	Frequently reported,	Not typically associated
Crohn's Disease	Associated with pleiotropic genetic loci,	Not typical
Psychological Disorders	Depression and anxiety are prevalent,	Also common, including anxiety and depression
Asthma and Allergic Rhinitis	Not typical	Common comorbid allergic conditions
Non-Alcoholic Fatty Liver Disease	Associated with some psoriasis patients	Limited data

5.5 Disease Burden: Physical, Emotional, and Economic Impact

Atopic dermatitis and psoriasis cause a worldwide burden that goes far beyond skin conditions, affecting people's physical and mental health as well as their financial stability. Psoriasis has 5.6 million disease-adjusted life years (DALYs), which is more than inflammatory bowel disease, according to the 2016 Global Burden of Disease Study [10]. These DALYs show the long-term psychosocial consequences of psoriatic plaques, including social isolation, stigma, and diminished life satisfaction, in addition to their persistence. Psoriasis, which has historically been mistaken for leprosy or other contagious diseases, still causes stigma that lowers self-esteem, damages relationships with others, and hinders job opportunities. Atopic dermatitis, which is sometimes written off as a benign pediatric ailment, has an equally significant impact. Persistent itching disrupts sleep, which results in daytime exhaustion, diminished focus, and decreased productivity in both work and educational environments. Particularly for parents of children with the condition, the daily pressure to use emollients, recognise and stay away from triggers, and alter environmental conditions has a negative psychological and financial impact. The SCORAD index demonstrates the need for a multifaceted framework to assess and treat disease severity comprehensively by integrating affected surface area, clinical intensity, and subjective symptoms of itching and sleep loss.

Psoriasis and atopic dermatitis both pose significant financial obstacles to healthcare systems and society at large. According to available data, the average American patient with atopic dermatitis spends between $100 and $2,000 per year on direct medical expenses [82]. This amount includes the cost of outpatient visits, topical and systemic medications, skin care products, and sleep aids, as well as the value of lost work productivity and caregiving time. However, the cost associated with psoriasis, and especially with treatments including biologics, sometimes exceeds this range. However, because biologic treatments have been shown to significantly improve health-related quality of life and lessen the long-term burden of treatment-related comorbid disorders, the incremental cost-effectiveness literature is increasingly supporting biologic therapy for patients with moderate or severe psoriasis.

Table 5: Disease Burden and Economic Impact

Aspect	Psoriasis	Atopic Dermatitis
Economic Burden	Annual cost per patient ranges from $100 to 2,000 $	Significant emotional and medical costs, similar or higher due to early onset and chronic course
Psychosocial Impact	Stigma, depression, and anxiety were reported.	Considerable burden on patients and families
Quality of Life Impact	Impact on work, social interactions due to visibility and symptoms,	Disrupted sleep and social limitations; caregiver burden

5.6 Treatment Approaches: From Symptom Control to Disease Modification

A paradigm change from a simple symptomatic strategy to techniques that decrease the disease pathogenesis is reflected in the current treatment procedures for psoriasis and atopic dermatitis. The surface area impacted by psoriasis greatly influences the treatment the doctor chooses. Topical corticosteroids, calcipotriene, or a combination of the two are usually part of the first regimen when plaque involvement is limited to less than 10% of the BSA [51-52]. Conventional narrowband UVB phototherapy, disease-modifying medications like methotrexate and cyclosporine, the retinoid acitretin, or an expanding list of biologics that target particular cytokine pathways may be used as first-line therapy for moderate-to-severe disease, where the substantial dermatologic burden justifies a more comprehensive systemic intervention [77].

The upper limit of therapeutic response has been steadily increased by biologics that target TNF-α (etanercept, adalimumab), IL-12/23 (ustekinumab), IL-17 (ixekizumab), and IL-23 (guselkumab). As a result, a growing %age of patients have achieved PASI 90 and PASI 100, which indicate 90% and 100% reductions in the Psoriasis Area and Severity Index, respectively [83].

The primary focus of atopic dermatitis treatment is the regular administration of emollients to maintain and repair the epidermal barrier. Topical corticosteroids are still the first-line treatment for flares, but calcineurin inhibitors, such as tacrolimus and pimecrolimus, are a better option for delicate areas like the face and neck [51-52]. In mild to severe situations, topical agents like Crisaborole provide an additional treatment option. Short courses of systemic medicines, usually cyclosporine, or phototherapy may be necessary for more severe illness. Dupilumab, a monoclonal antibody that targets the IL-4 and IL-13 pathways, has revolutionised the treatment landscape. The medication is available for patients six months of age and older (Table 6). Abrocitinib and upadacitinib, two recently approved oral Janus kinase inhibitors, can quickly reduce pruritus but need close safety monitoring [78].

There is still a significant gap between the strict recommendations of established professional standards and their use in everyday practice, even with the strength of contemporary treatment alternatives. Undertreatment patterns, lengthy diagnostic wait times, and a lack of biologic medicines disproportionately affect patients in low- and middle-income nations. It is common for comorbid illnesses to go unnoticed; for instance, psoriatic arthritis might go years without a diagnosis. At the same time, individuals with one or both inflammatory illnesses seldom receive mental health therapies as part of their care continuum.

Table 6: Treatment Modalities and Patient-Centred Strategies

Treatment Type	Psoriasis	Atopic Dermatitis
Topical Therapies	Corticosteroids, vitamin D analogues, coal tar,	Corticosteroids, calcineurin inhibitors, moisturisers
Systemic Therapies	Methotrexate, cyclosporine, retinoids,	Systemic immunosuppressants in severe cases
Biologics & Targeted Therapies	Anti-TNF, IL-17, IL-23 inhibitors,	Emerging biologics targeting specific pathways,
Patient Education & Psychosocial Support	Education on disease course, lifestyle, and comorbidity management,	Emphasis on skin care routines, triggers, and support systems,

5.7 Toward Patient-Centred, Personalised Care

Frameworks that specifically acknowledge and take into account the biological, psychological, and environmental factors that affect each patient are essential for the advancement of psoriasis and atopic dermatitis treatment. Algorithms that work for everyone are

no longer acceptable. The severity of the condition, the range of comorbidities, lifestyle choices, plans for reproduction, and the patient's stated values and preferences should all be considered while making therapeutic decisions. For example, a patient with obesity and metabolic syndrome may be directed toward an IL-23 inhibitor, partly because of its apparent cardiovascular protective profile. In contrast, a lady of reproductive age with severe psoriasis may refuse methotrexate because of teratogenic concerns. Care for atopic dermatitis must go beyond treating epidermal lesions and entail caregiver participation, structured psychological support networks, and systematic educational initiatives focused on trigger avoidance.

Collaborative decision-making in this paradigm takes on a procedural urgency and goes beyond principle. By definition, patients collaborate with clinicians to create the treatment plan, forcing the latter to systematically accomplish highly personalised, patient-defined outcomes, whether it be the unrelenting search for symptom-free, clear skin, the reduction of treatment-related iatrogenic load, or the restoration of restful sleep at night. Digital health infrastructures, advanced telemedical networks, and large patient registries like the Global Psoriasis Atlas are examples of complementary advancements that operate together to support ongoing clinical monitoring and provide quick, fair access to subspecialised care.

5.8 Evaluation of Approaches

Table 7 critically appraises key recent studies focused on evaluating these approaches, highlighting their strengths and limitations. While existing tools like PASI and SCORAD offer quantitative and patient-centred means to gauge disease activity, numerous knowledge gaps remain—particularly in integrating patient-reported outcomes, understanding comorbidities, optimising long-term treatment safety, and implementing personalised, multifactorial care in varied health systems. Moreover, the evolving landscape of immunomodulatory therapies calls for further investigation into sustained efficacy and holistic patient well-being.

By summarising these pivotal works, this evaluation aims to inform clinicians, researchers, and policymakers about current best practices while illuminating areas requiring intensified research and innovation. Addressing these gaps will be essential to advancing patient-centred care and improving the overall disease burden management for those affected by psoriasis and atopic dermatitis.

Table 7: Critical Evaluation of Current Approaches in Psoriasis and Atopic Dermatitis Management: Bridging Evidence and Knowledge Gaps

Study	Study Focus / Approach	Evaluation of Approach	Knowledge Gaps Highlighted
[55]	Development and validation of PASI scoring for psoriasis severity	Robust, widely used quantitative tool for lesion severity; standard in biologic drug studies	Limited incorporation of patient-reported outcomes, like pruritus or psychosocial impact
[56]	Use of PASI 75 as treatment efficacy threshold	Provides objective measurable endpoints to evaluate systemic and biologic therapies	Does not fully address individualised treatment goals or quality-of-life aspects
[58]	Combines patient-reported symptoms and observer assessment	Integrates subjective symptoms (itch, sleep loss) with objective signs, improving patient-centredness	Complexity hinders widespread routine use; it needs adaptation for diverse populations
[65]	Observational studies of nail changes indicating disease activity	Nail findings support diagnostic accuracy and disease monitoring	Lacks longitudinal data on the impact of nail symptoms on quality of life
[66]	Epidemiological studies linking AD onset to asthma, rhinitis, and allergies	Clarifies progression of atopic conditions, highlights the need for early intervention	Molecular mechanisms driving the transition remain unclear.
[67]	Cross-sectional and cohort analyses in pediatric populations	Demonstrates an increased risk of comorbid atopic disorders	Predictive biomarkers and prevention strategies are insufficiently developed

Study	Study Focus / Approach	Evaluation of Approach	Knowledge Gaps Highlighted
[78]	Clinical trials evaluating the safety and efficacy of JAK inhibitors	Offers immediate pruritus relief, an alternative to established biologics	Long-term safety data and real-world effectiveness are incompletely characterised
[79]	Protocols for reducing Staphylococcus aureus colonisation	Supports adjunctive therapies (e.g., bleach baths) to reduce flares	Optimal frequency and long-term benefits remain to be established
[80]	Conceptual approach integrating shared decision-making and multifaceted care	Emphasises personalised care, improves adherence and outcomes	Implementation barriers and scalability in diverse healthcare settings

6. Discussion

This comprehensive comparative study delineates the distinguishing clinical, immunopathologic, and therapeutic dimensions of psoriasis and atopic dermatitis, two of the most common chronic inflammatory skin diseases globally. Our findings affirm the profound burden both diseases impose on patients' quality of life, healthcare systems, and public health infrastructure, while underscoring critical differences in their phenotypic manifestations, immunological pathways, comorbidity profiles, and response to treatment modalities. The integration of patient-centred tools such as SCORAD and PASI indices advances precision in disease severity quantification and guides individualised therapeutic interventions. Below, we elaborate on the key insights emerging from our analysis, situate them within current scholarly evidence, and explore their clinical significance.

Firstly, the differential clinical presentation of psoriasis and atopic dermatitis is notably distinct yet occasionally overlapping, complicating diagnosis and management strategies. Psoriasis typically manifests as sharply demarcated, erythematous plaques with characteristic silvery scales predominantly on extensor surfaces, including the elbows, knees, scalp, and sacral region, with frequent nail involvement and confirmatory features such as the Koebner phenomenon and Auspitz's sign. Conversely, atopic dermatitis displays poorly defined eczematous patches characterised by intense pruritus, erythema, lichenification, excoriations, and xerosis, with age-dependent lesion distribution patterns—from exudative, crusted facial involvement in infants to lichenified flexural lesions in older children and adults.

Our data corroborate established epidemiological and clinical frameworks reported by other recent studies. For instance, Silverberg et al. [84] affirmed the age-specific morphological evolution of atopic dermatitis and its intense pruritic burden, emphasising symptom-driven management. Meanwhile, Boehncke and Schön [85] articulated the classical presentation and systemic implications of plaque psoriasis, highlighting the crucial relevance of precise phenotyping for treatment selection. The clinical overlap of erythema and scaling necessitates the application of validated scoring systems. The SCORAD index, by encompassing both subjective symptoms (pruritus and sleep loss) and objective lesion characteristics, captures the multifaceted morbidity of atopic dermatitis far more effectively than purely clinical assessments, aligning with our findings on its utility in chronic inflammatory dermatoses management. The PASI index's quantification of erythema, induration, and scaling serves as a gold standard for psoriasis severity, facilitating objective monitoring of therapeutic response, particularly in systemic and biologic treatment trials.

Secondly, the pathobiological divergence between the IL-23/Th17-dominated psoriasis and the filaggrin deficiency-driven Th2-skewed atopic dermatitis underpins distinct therapeutic responses and comorbidity spectra. Psoriasis's immunopathogenesis, with hyperactivation of IL-23 and IL-17 pathways leading to keratinocyte hyperproliferation, is strongly associated with systemic comorbidities, including psoriatic arthritis, increased cardiovascular risk, and metabolic syndrome. Our findings concur with those of Reich et al. [86], who emphasised the necessity of holistic management addressing not only cutaneous symptoms but also systemic inflammation to mitigate long-term morbidity.. In contrast, atopic dermatitis originates primarily from impaired epidermal barrier function, notably filaggrin insufficiency, and Th2-biased immune responses leading to pruritic, lichenified lesions and serving as a harbinger for the atopic march encompassing asthma and other allergic diseases. This immunologic distinctiveness guides the use of targeted biologics such as IL-4 receptor antagonists (e.g., dupilumab) in atopic dermatitis, compared to IL-17 and IL-23 inhibitors deployed in psoriasis. Our results reinforce the therapeutic paradigm shift documented by Simpson et al. [87], who highlighted improved patient outcomes with newer biologics addressing specific cytokine pathways.

Thirdly, patient-centred care emerges as pivotal in optimising treatment adherence and outcomes, tailoring interventions to disease severity, comorbidities, and individual preferences. The dynamic interplay between patient priorities—whether skin clearance, reduction of pruritus and sleep disruption, or minimisation of side effects—and the complexity of therapeutic regimens demands shared decision-making frameworks. This approach is supported by recent qualitative studies, such as those by Patel et al. [88], demonstrating improved satisfaction and psychological well-being when therapeutic goals align with patients' lived experiences and concerns. Moreover, comorbidity burden necessitates multidisciplinary cooperation; for example, dermatologists must coordinate with rheumatologists to manage psoriatic arthritis and with cardiologists for cardiovascular risk reduction in psoriasis patients.

Fourthly, addressing the psychosocial burden inherent to both diseases is critical. The persistent itch, visible skin changes, sleep disturbances, and associated anxiety and depression substantially degrade health-related quality of life. These sequelae perpetuate social stigma and economic costs through lost productivity and healthcare utilisation, as documented in population-based studies by Gupta et al. [89] and Lee et al. [90]. The inclusion of symptom-reduction, especially pruritus and sleep disturbance, in scoring systems like SCORAD underscores the importance of comprehensive outcome measures beyond mere skin lesion clearance. Our findings thus advocate integrated psychosocial support and caregiver assistance programs, aligning with recommendations from global consensus panels on chronic inflammatory skin diseases [91].

Fifthly, the challenge of early and precise diagnosis underlines the need for public awareness and clinician education, given the overlapping features that can mask the true disease identity—leading to suboptimal treatment. Enhanced differential diagnosis strategies employing clinical, immunohistochemical, and molecular biomarkers should be more widely disseminated in general dermatology and primary care settings. Our review of current literature echoes the call by Kim and Lee [92] for improved diagnostic algorithms incorporating patient-reported outcomes and objective measures to reduce misdiagnosis and delays. Additionally, equitable access to emerging therapies remains an unresolved issue, particularly in under-resourced health systems—a concern highlighted by our focus on real-world treatment considerations and access barriers.

7. Conclusion

Despite being categorised as chronic inflammatory dermatoses, psoriasis and atopic dermatitis differ significantly in their immunopathogenic causes, clinical manifestations, and systemic effects. While atopic dermatitis is based on a dysregulated epidermal barrier and is characterised by a preponderance of Th2-mediated inflammation that arises from both intrinsic and extrinsic stimuli, psoriasis is based on a self-sustaining Th17-driven autoimmune response. Physical infirmity, psychological morbidity, and increasing economic load are all polyhedral burdens imposed by each illness that are by no means limited to the epidermal plane. This complexity compels the implementation of integrated, lifelong management paradigms.

Biologic medicines have significantly improved clinical results and enabled targeted intervention along specific pathobiological cascades, revolutionising the treatment of chronic inflammatory disorders. However, the integration of new treatments into standard clinical practice frequently falls behind the rate of advancement, putting patients at risk for incorrect diagnoses, insufficient treatment rigour, and a failure to address comorbidities adequately.

To close these gaps, practising clinicians need to support patient-centred, tailored therapy models that are supported by continuous multidisciplinary cooperation and solid evidence. To ensure that all patients receive prompt, effective, and compassionate care, future research should place a strong emphasis on the early introduction of therapy, the identification and validation of prognostic biomarkers, and systematic measures to expand worldwide access to care.

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Conflict of interest

No conflict of interest

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