The Evolution of Biologic Therapies for Psoriasis and Atopic Dermatitis: A Systematic Review of Randomized Controlled Trial

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Abstract

Background:
Psoriasis and atopic dermatitis are chronic, immune-mediated inflammatory skin diseases that cause a substantial, lifelong burden on patients globally. Over the past two decades, the therapeutic paradigm has shifted dramatically from broad immunosuppression to highly targeted biologic therapies designed to interrupt key cytokine pathways driving clinical disease activity.

Methods:
To comprehensively evaluate this transition, we conducted a systematic review of randomized controlled trials assessing the efficacy and safety of biologic treatments for moderate-to-severe psoriasis and atopic dermatitis. We systematically searched major electronic databases for peer-reviewed published trials, extracting data on targeted mechanisms, clinical efficacy outcomes, and safety profiles.

Result:
The evolution in psoriasis treatment progressed sequentially from early tumor necrosis factor inhibitors to highly specific interleukin (IL)-12/23, IL-17, and selective IL-23 blockades. These newer agents consistently demonstrate unprecedented rates of complete skin clearance and durable responses. Similarly, the atopic dermatitis landscape has been revolutionized by biologics targeting type 2 inflammation, particularly the IL-4 and IL-13 pathways. These interventions provide profound relief from intractable pruritus and persistent lesions in patients refractory to conventional therapies, while largely avoiding the off-target toxicities of older systemic agents.

Conclusions:
The advent and evolution of biologic therapies represent a watershed moment in dermatology. By enabling precision immunology, these targeted treatments have fundamentally transformed disease management, offering patients realistic expectations for sustained remission and significantly enhanced quality of life.

Introduction

Psoriasis and atopic dermatitis (AD) are chronic, immune-mediated inflammatory skin diseases that cause substantial, lifelong morbidity through persistent lesions, pruritus, sleep disruption, and impaired quality of life, with ripple effects on mental health, work productivity, and healthcare utilization.[1] AD is similarly common and increasingly recognized as a major public health problem; recent global estimates suggest roughly 204 million people are affected (prevalence 2.6%), including 101 million

adults and 103 million children.[2] Beyond the skin, systemic inflammation and chronic disease burden are clinically relevant because they intersect with comorbidities and long-term risk.[3,4]

Over the past two decades, the therapeutic paradigm for moderate-to-severe psoriasis and AD has shifted from broad immunosuppression toward targeted biologic therapies designed to interrupt key cytokine pathways driving disease activity. In psoriasis, successive generations of biologics (e.g., TNF inhibition followed by IL-12/23, IL-17, and IL-23 pathway targeting) have raised expectations for durable skin clearance and comorbidity-aware care, while in AD, biologics targeting type 2 inflammation (notably IL-4/IL-13 and related pathways) have expanded options for patients whose disease remains uncontrolled on topical therapy.[5] Given the fast pace of approvals, expanding indications, and accumulating long-term safety and real-world effectiveness data, clinicians need an integrated synthesis of how biologic strategies have evolved and what that evolution means for treatment selection across both diseases. Therefore, this systematic review aims to summarize and critically evaluate the evolution of biologic therapies for psoriasis and atopic dermatitis, focusing on changes in targeted mechanisms, clinical efficacy outcomes, and safety evidence over time.

Material And Methods

Study design and reporting framework

This systematic review was designed and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline.[6] An a priori plan was developed to prespecify the review question, eligibility criteria, outcomes of interest, screening procedures, data extraction items, and risk-of-bias assessment approach. Only randomized controlled trials (RCTs) were eligible for inclusion.

Review question and PICO framework

The review question was structured using the PICO framework. The population comprised children and adults diagnosed with psoriasis and/or atopic dermatitis. The intervention was biologic therapy used for either condition, including agents targeting TNF and interleukin pathways relevant to disease mechanisms (for example IL-12/23, IL-17, IL-23 for psoriasis and IL-4/IL-13–related pathways for atopic dermatitis). Comparators included placebo, standard of care, non-biologic systemic therapy, phototherapy, or another biologic agent, as evaluated within randomized controlled trial designs. Outcomes included clinically relevant efficacy measures and safety outcomes. For psoriasis, efficacy outcomes included PASI response thresholds and physician global assessments where reported; for atopic dermatitis, efficacy outcomes included EASI response thresholds, investigator global assessments, and pruritus measures such as numerical rating scales. Safety outcomes included overall adverse events, serious adverse events, serious infections, discontinuations due to adverse events, and other key safety signals as reported.

Information sources and search strategy

A comprehensive search of PubMed, EMBASE, and Scopus was performed from database inception through 15 February 2026. The search strategy combined controlled vocabulary with free-text keywords. In PubMed, Medical Subject Headings (MeSH) were used alongside title/abstract keywords; in EMBASE, the strategy was adapted using Emtree terms; and in Scopus, keyword-based searching across titles, abstracts, and indexed terms was applied. Search concepts included the disease terms for psoriasis and atopic dermatitis/eczema, the intervention terms for biologics and monoclonal antibodies, and biologic targets and agent names to ensure sensitivity. Reference lists of included articles and relevant review papers were hand-searched to identify additional eligible studies, and all retrieved citations were deduplicated prior to screening.

Study selection process

All authors conducted study selection independently from title and abstract screening through full-text eligibility assessment and continued independently

through the risk-of-bias stage. At each step, disagreements regarding inclusion or exclusion were resolved through discussion until consensus was reached. The selection process was documented using a PRISMA flow diagram, including reasons for exclusion at the full-text stage. Studies were included only if they were randomized controlled trials evaluating biologic therapies for psoriasis and/or atopic dermatitis.

Data extraction and data items

Data were extracted using a predefined extraction framework and performed independently by all authors. Extracted items included publication details, study design and setting, participant characteristics and disease severity, prior and concomitant therapies, biologic agent and target/class, dosing and duration, comparator details when applicable, follow-up time, and prespecified efficacy and safety outcomes. Where outcomes were reported in multiple formats, data were captured in the form most comparable across studies, and any discrepancies in extracted values were resolved by discussion.

Risk of bias assessment

Risk of bias for included randomized controlled trials was assessed using the Cochrane Risk of Bias tool, version 2 (RoB 2). Trials were evaluated across the RoB 2 domains: bias arising from the randomization process, bias due to deviations from intended interventions, bias due to missing outcome data, bias in measurement of the outcome, and bias in selection of the reported result. Each domain and the overall risk of bias were judged as low risk, some concerns, or high risk. All authors performed assessments independently, and discrepancies were resolved through discussion to reach consensus.

Synthesis of results

A qualitative, narrative synthesis was conducted without meta-analysis. Results were synthesized by disease, biologic class or target, and chronology to describe the evolution of biologic therapies, with emphasis on clinically meaningful efficacy outcomes and safety profiles across trials and real-world evidence.

Result

A total of 469 records were identified through database searching (PubMed, n = 167; EMBASE, n = 199; Scopus, n = 103), and 35 duplicates were removed prior to screening, leaving 434 records for title and abstract screening; of these, 408 records were excluded and 26 reports were sought for retrieval, with none unavailable (n = 0), resulting in 26 full-text reports assessed for eligibility. Following full-text review, 12 reports were excluded, most commonly because they evaluated small molecules/JAK inhibitors, topical agents only, phototherapy only, or conventional systemic drugs only (n = 12), reported outcomes that were not relevant or not extractable (n = 3), or were animal/in vitro studies (n = 2), and 9 studies were ultimately included in the review (Figure 1).[7–15]

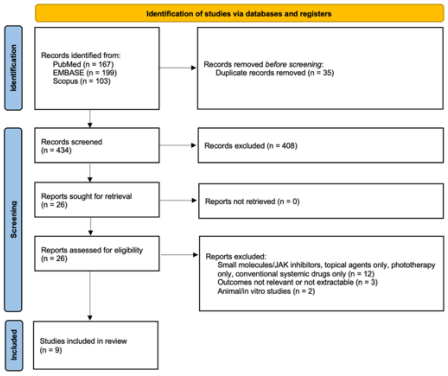

Figure 1. PRISMA flow diagram of the study selection process.

Study characteristics

The included evidence base comprised randomized controlled trials and real-world observational studies evaluating biologic therapies across psoriasis and atopic dermatitis, with follow-up ranging from short-term induction periods (commonly 12–16 weeks) to longer maintenance and extension phases. Across psoriasis studies, outcomes were most frequently reported as PASI response thresholds (PASI 75/90/100) and physician global assessments, allowing comparison of the

clinical “ceiling” achieved by different biologic classes over time. Across atopic dermatitis studies, EASI response thresholds, IGA, itch severity (often numerical rating scales), and quality-of-life measures were commonly reported, reflecting the clinical priority of both skin clearance and itch/sleep improvement.

Psoriasis efficacy trends across biologic classes

Across the evolution of biologics for moderate-to-severe plaque psoriasis, later pathway-targeted agents consistently demonstrated higher short-term skin clearance rates than earlier biologic strategies, particularly when PASI 90 and PASI 100 were used as contemporary treatment targets. In head-to-head evidence, the IL-17 era marked a notable step-change in higher-level clearance; for example, secukinumab achieved PASI 90 in 79.0% at week 16 compared with 57.6% for ustekinumab in the CLEAR trial. Subsequent IL-23–targeted therapy further consolidated high response rates; in VOYAGE 1, guselkumab achieved PASI 90 in 73.3% at week 16 and outperformed adalimumab (49.7%). Across IL-23 comparisons, risankizumab demonstrated week-16 PASI 90 response rates in the range of approximately 73.1%–80.2% versus 41.9%–46.0% for ustekinumab (reported across analyses of the UltIMMa program), consistent with the modern expectation of near-clear or clear skin for many patients. More recently, dual IL-17A/IL-17F blockade also showed strong induction efficacy in comparative trials; bimekizumab achieved PASI 90 in 85.5% at week 16 compared with 74.3% for secukinumab in a head-to-head study. Collectively, these findings support a clinically meaningful trend toward faster and deeper clearance, which is directly relevant to reducing symptom burden, improving quality of life, and enabling treat-to-target approaches in routine practice.

Atopic dermatitis efficacy trends across biologic classes

For atopic dermatitis, the introduction of targeted biologics expanded therapeutic options beyond topical therapy and broad immunosuppression, with consistent improvements in clinician-reported severity and patient-centered outcomes such as itch. Across phase 3 monotherapy evidence for dupilumab, pooled week-16 results have shown EASI-75 rates around 47.7%–50.2% for dupilumab regimens versus 13.3% for placebo, reflecting robust short-term disease control in moderate-to-severe populations. IL-13–targeted biologics also demonstrated statistically significant efficacy versus placebo in pivotal programs; in ECZTRA 1 and ECZTRA 2, tralokinumab achieved EASI-75 at week 16 in 25.0% and 33.2%, respectively, compared with 12.7% and 11.4% with placebo. More recently, lebrikizumab phase 3 trials reported higher induction responses, with EASI-75 at week 16 occurring in 58.8% in one trial compared with 16.2% with placebo. These results align with the clinical goal of improving visible disease while also addressing itch and sleep disruption, which are often the dominant drivers of patient distress and functional impairment.

Safety outcomes

Across both diseases, safety reporting commonly included overall adverse events, serious adverse events, serious infections, and discontinuations due to adverse events. In psoriasis, comparative trials generally supported acceptable short-term safety profiles across biologic classes, with ongoing emphasis on infection monitoring and long-term pharmacovigilance as exposure durations increase. In atopic dermatitis, dupilumab maintenance data have supported sustained benefit without new major safety signals in longer-term use, and controlled studies consistently

tracked class-relevant events such as conjunctivitis alongside general infection outcomes. Overall, the clinically relevant interpretation across the evolution of biologics is that increasing efficacy has been accompanied by increasingly mature safety datasets, though the strength of inference for rare harms remains dependent on long-term extensions and real-world registries.

Risk of bias assessment

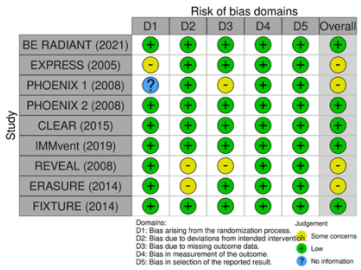

Figure 2. Cochrane RoB version 2 for risk of bias analysis.

Across the included randomized controlled trials, the overall risk of bias was predominantly low, with 5 studies rated low risk and 4 rated as having some concerns (Figure 2). The “some concerns” ratings were driven mainly by issues in the randomization process (D1) and, in a subset of trials, deviations from intended interventions (D2) and/or missing outcome data (D3), while outcome measurement (D4) and selective reporting (D5) were consistently low risk (Figure 2). Overall, these assessments suggest the evidence base is generally methodologically robust, though findings from trials with “some concerns” should be interpreted with modest caution.

Discussion

The evolution of biologic therapies represents a watershed moment in the management of chronic, immune-mediated skin diseases. As demonstrated by the evidence synthesized in this systematic review, the therapeutic landscape for psoriasis and AD has fundamentally shifted from broad, non-specific immunosuppressants to highly targeted, mechanistically driven interventions.[16,17] By isolating and neutralizing key pathogenic cytokines, successive generations of biologics have not only raised the bar for clinical efficacy but also significantly reduced the off-target toxicities traditionally associated with older systemic therapies.[17] This review confirms that the treatment paradigm has transitioned from mere disease mitigation to the realistic pursuit of sustained clinical remission and profound improvements in patient quality of life.

In the context of psoriasis, our findings illustrate a distinct, step-wise trajectory of targeted therapy, progressing from early TNF inhibitors to more specific downstream IL blockades. The advent of IL-12/23, IL-17, and selective IL-23 inhibitors has redefined treatment expectations, with randomized controlled trials consistently demonstrating high proportions of patients achieving near-complete or complete skin clearance (PASI 90 and PASI 100).[18–20] Furthermore, targeting the IL-23/Th17 axis has shown remarkable durability of response and favorable, long-term safety profiles. This evolution reflects a much deeper understanding of psoriasis pathophysiology and highlights the clinical value of increasingly precise immunological targeting in addressing both cutaneous symptoms and associated systemic comorbidities.[19]

Parallel to the advancements in psoriasis, the biologic revolution in atopic dermatitis has profoundly altered its clinical management, primarily through the targeted modulation of type 2 inflammation. The introduction of agents targeting the IL-4 and IL-13 pathways has provided a highly efficacious systemic option for the large population of patients whose disease remains refractory to topical therapies. The trial data indicate that these targeted biologics are highly successful in resolving persistent cutaneous lesions and intractable pruritus thereby rescuing patients from severe sleep disruption and

psychosocial distress.[21] As the therapeutic armamentarium for AD continues to expand rapidly, clinicians are becoming better equipped to step away from broad immunosuppressants like cyclosporine or systemic corticosteroids.[22–24]

Despite the robust evidence supporting these biologic advancements, several limitations of this systematic review must be acknowledged. First, the inherent heterogeneity among the included RCTs complicates direct, cross-trial comparisons. Second, while RCTs provide the highest level of controlled efficacy data, their strictly defined inclusion criteria and relatively short-term primary observation periods may not fully capture rare or long-latency adverse events, underscoring the need to interpret these findings alongside real-world pharmacovigilance data. Additionally, the relative scarcity of head-to-head comparative trials, particularly among the newest agents, limits definitive conclusions regarding absolute superiority. Finally, the potential for publication bias, favoring trials with positive outcomes, cannot be entirely ruled out.

Conclusion

In conclusion, the systematic evolution of biologic therapies has dramatically transformed the prognostic horizons for both psoriasis and atopic dermatitis, successfully shifting the standard of care toward precision immunology. Successive generations of targeted biologics have yielded unprecedented levels of skin clearance, durable symptom relief, and improved safety profiles compared to traditional systemic agents. As our understanding of immune-mediated pathophysiology continues to deepen, future research should prioritize identifying reliable predictive biomarkers to guide personalized treatment selection, expanding head-to-head clinical trials to establish clear therapeutic hierarchies, and continuously monitoring long-term real-world safety. Ultimately, this ongoing biologic evolution empowers clinicians to deliver highly effective, comorbidity-aware care, profoundly improving the lifelong trajectory of individuals burdened by these debilitating skin diseases.

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