

Review

Balneotherapy as Adjunctive Care in Atopic Dermatitis: Effects on Skin Barrier, Microbiome, and Pruritus

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Abstract

Atopic dermatitis (AD) is a chronic inflammatory skin disease in which type 2 immune activation, epidermal barrier dysfunction, microbial dysbiosis, and neurosensory pathways interact to sustain disease activity and symptom burden. Despite major advances with biologics and Janus kinase inhibitors, residual xerosis, pruritus, sleep disturbance, and barrier fragility may persist in otherwise controlled patients. This narrative review evaluates balneotherapy as adjunctive supportive care in AD, focusing on skin barrier function, microbiome-related effects, pruritus, and clinical integration. A narrative literature review was conducted using PubMed/MEDLINE, Ovid, and Scopus from database inception to 31 December 2025. Search terms addressed AD, type 2 inflammation, skin barrier dysfunction, transepidermal water loss, pruritus, microbiome dysbiosis, thermal spring water, spa therapy, seawater therapy, Dead Sea climatotherapy, hydrotherapy, and balneophototherapy. Clinical, mechanistic, translational, and methodological studies were considered. Available evidence suggests that water-based interventions may improve selected patient-centred outcomes, particularly xerosis-related discomfort, pruritus, sleep impairment, quality of life, and global severity scores. Thermal spring water programmes, Dead Sea climatotherapy, seawater-based approaches, and balneophototherapy differ substantially in exposure characteristics, co-interventions, and outcome reporting. Objective endpoints such as transepidermal water loss, skin hydration, tape stripping biomarkers, and microbiome profiling may help clarify biological plausibility, although current evidence is limited by heterogeneity, short follow-up, and difficulty isolating water-specific effects. Balneotherapy should be positioned as an adjunctive, supportive intervention rather than an alternative to evidence-based anti-inflammatory therapy in AD, particularly for selected patients with persistent barrier- and symptom-dominant residual burden.

Keywords: atopic dermatitis; balneotherapy; skin barrier; transepidermal water loss; pruritus; skin microbiome



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1. Introduction

Atopic dermatitis (AD) is a chronic relapsing inflammatory dermatosis increasingly conceptualised as a systemic, type 2-skewed disease in which epithelial barrier dysfunction and immune activation interact bidirectionally with microbial and neurosensory pathways. Over the last decade, targeted systemic therapies, most prominently IL-4/IL-13 pathway blockade and oral Janus kinase inhibitors (JAKi), have transformed outcomes for patients with moderate-to-severe AD, enabling rapid control of visible eczema and meaningful improvements in patient-reported outcomes. Yet, inflammatory control does not uniformly translate into complete resolution of the lived burden of disease. Persistent xerosis, barrier fragility, pruritus and sleep disturbance, and a tendency to flare in response to irritants or microbial triggers remain common drivers of impaired quality of life and ongoing rescue topical use, even in patients otherwise responding well to systemic therapy.

This residual burden is clinically coherent when AD is viewed as a coupled barrier-microbiome-itch disorder rather than a purely lesion-based inflammatory phenotype. Longitudinal paediatric data show that disease flares track with dynamic shifts in the skin microbiome, supporting the concept of AD as a volatility-prone ecosystem rather than a static state [1]. Reviews of the AD microbiome further support the clinical relevance of dysbiosis, often characterised by reduced diversity and *Staphylococcus aureus* predominance, as a contributor to disease instability and flare biology [2].

Within contemporary AD care, this creates a rationale for adjunctive, evidence-aware non-pharmacological strategies aimed at reinforcing barrier resilience and improving symptom control alongside topical anti-inflammatory therapy and systemic agents. Balneotherapy, delivered through thermal spring water programmes, seawater/Dead Sea regimens, or combined approaches such as balneophototherapy, has a long history in inflammatory dermatoses but is variably standardised and often discussed without linkage to modern outcome measurement. A clinically useful synthesis should therefore address which outcome domains are most consistently affected, which patient profiles may plausibly benefit, and how effects can be evaluated using feasible contemporary tools such as TEWL assessment and tape stripping-based biomarkers. A practical comparator for “water-based adjuncts” is the broader bathing literature in AD, which clarifies how bathing regimens can be positioned and evaluated in routine care [3].

2. Evidence Acquisition

This narrative review was conceived to inform clinically practical, barrier- and symptom-oriented integration of balneotherapy as adjunctive care in type 2 AD within contemporary management pathways that include biologics and Janus kinase inhibitors. To strengthen reporting quality and internal consistency, the manuscript was developed in alignment with core principles captured by the Scale for the Assessment of Narrative Review Articles (SANRA), focusing on justification of the topic, transparency of literature identification, scientific reasoning, and balanced presentation of evidence.

A literature search was conducted in PubMed/MEDLINE, Ovid, and Scopus from database inception to 31 December 2025. Searches combined free-text keywords and Medical Subject Headings (MeSH) addressing the target condition and relevant biological domains, including AD/atopic eczema, type 2 inflammation, skin barrier, transepidermal water loss, filaggrin, natural moisturising factor, ceramides, pruritus, and neuroimmune pathways. Intervention terms covered balneotherapy, thermal spring water, spa therapy, hydrotherapy, seawater, Dead Sea climatotherapy, and balneophototherapy, and were paired with outcome- and implementation-oriented terms including quality of life, itch, sleep, patient-reported outcomes, microbiome, *S. aureus*, dysbiosis, tape stripping, and non-invasive skin assessment.

Eligible records included clinical trials, observational studies, systematic and scoping reviews, narrative reviews with clear clinical or mechanistic relevance, translational and mechanistic studies, and methodological papers addressing outcome assessment in AD, balneotherapy, hydrotherapy, climatotherapy, seawater-based interventions, balneophototherapy, skin barrier function, pruritus, and microbiome-related endpoints. Priority was given to studies directly involving patients with AD and water-based interventions. When direct AD-specific evidence was limited, studies addressing related bathing-based interventions, skin barrier assessment, microbial colonisation, pruritus, or outcome measurement were considered if they provided clinically relevant context or mechanistic plausibility. English-language articles were prioritized; non-English records were considered when an English abstract was available and the study was directly relevant to balneotherapy or water-based supportive care in AD.

Exclusion criteria included articles without relevance to AD or water-based dermatological interventions, non-dermatological balneotherapy studies, purely opinion-based articles without identifiable supporting evidence, duplicate publications, studies lacking clinically interpretable outcomes, and reports in which the intervention or outcome domains could not be clearly extracted. Searches were iteratively refined to capture mechanistic and translational studies supporting biological plausibility, clinical trials and observational cohorts evaluating water-based interventions in AD, systematic and scoping reviews across dermatology and thermal medicine, and methodological papers relevant to objective barrier and microbiome assessment.

Additional records were identified through manual screening of reference lists from included articles and relevant reviews, and by targeted retrieval of key documents underpinning measurement standardisation and clinical framing. Specifically, we retrieved methodological references for in vivo barrier measurements and tape-stripping workflows, and contemporary AD systemic-therapy guidance to ensure clinically coherent positioning of adjunctive balneotherapy. Given the heterogeneity of the evidence base, including variability in intervention definitions, treatment duration and co-interventions, outcome measures, patient populations, and follow-up, formal systematic screening, quantitative pooling, and risk-of-bias assessment were not undertaken. Evidence was selected and interpreted according to clinical relevance, directness of evidence, methodological transparency, and contribution to the barrier–microbiome–pruritus framework, with recognition of limitations inherent to small trials, non-randomised designs, site-specific interventions, and studies in which multifactorial exposures such as climate, ultraviolet radiation, and structured care components could not be disentangled from water-based effects.

Because terminology and scope vary across publications, we did not assume strict equivalence among different water-based interventions. Rather, we adopted a pragmatic taxonomy distinguishing thermal spring water programmes, seawater-based programmes and Dead Sea climatotherapy, and balneophototherapy, mapping reported outcomes to shared clinical domains rather than to a uniform mechanistic construct. Translational and mechanistic observations, including changes in barrier biomarkers or microbial composition, were incorporated only when they plausibly supported observed clinical effects and were interpreted as supportive, hypothesis-generating evidence rather than definitive proof of causality. Overall, the synthesis was structured around practical clinical questions relevant to current dermatology practice, including which residual disease domains persist despite modern systemic therapy, which patient phenotypes may benefit from adjunctive balneotherapy, how such interventions can be integrated with biologics and JAK inhibitors, which objective and patient-reported outcomes are feasible in routine and research settings, and what safety considerations should guide patient selection.

3. Evidence Synthesis

3.1. Pathophysiological Framework: Why Barrier, Microbiome, and Pruritus Matter

3.1.1. Skin Barrier Dysfunction in AD

Barrier dysfunction in AD is multifactorial and measurable, involving impaired corneocyte cohesion, altered lipid lamellae, reduced filaggrin-derived natural moisturising factor (NMF), and shifts in skin surface pH, which collectively increase TEWL and lower the threshold for irritation and inflammation. Guidance from EEMCO provides core methodological standards for TEWL and in vivo hydration measurements, underscoring the need for strict environmental and procedural control when TEWL is used as an endpoint [4,5]. In AD populations, instrumental hydration/TEWL measures correlate variably with severity and quality-of-life metrics, supporting their use as complementary, rather than substitutive, outcomes [6]. Importantly, type 2 cytokines do not function solely as inflammatory mediators; targeted therapy can be accompanied by measurable improvements in barrier function and/or hydration in adults with AD in observational and mechanistic studies [7–10]. At the same time, discordant instrumental patterns have been reported (e.g., TEWL improvement without parallel hydration gain), reinforcing that barrier biology is multidimensional and may not normalise uniformly [11].

3.1.2. Skin Microbiome Dysbiosis

Beyond flare-linked microbiome volatility [1], paediatric AD is characterised by strain-level dynamics of *S. aureus*/*S. epidermidis* and disease-relevant microbial diversity patterns [12]. Early-life colonisation with commensal staphylococci has also been associated with lower later AD risk, supporting a clinically meaningful developmental window for microbiome–barrier interactions [13]. Contemporary reviews describe how *S. aureus* contributes to AD persistence via barrier disruption and immune amplification, and why dysbiosis should be interpreted with attention to absolute burden and longitudinal stability rather than relative abundance alone [2,14–16].

3.1.3. Neuroimmune Axis and Pruritus

Pruritus is the dominant symptom for many patients with AD and often the principal determinant of sleep quality, psychosocial functioning, and perceived disease control. Scratching provides transient relief but mechanically disrupts the stratum corneum, increases TEWL, and promotes microfissuring, facilitating antigen penetration and microbial perturbation and thereby amplifying both inflammation and dysbiosis [3,6]. Clinically, this makes itch reduction more than symptomatic palliation: it is a plausible lever for interrupting the itch–scratch–barrier damage cycle and improving disease stability. Adjunctive interventions should therefore be evaluated not only by lesion scores but also by itch and sleep outcomes, ideally paired with barrier or microbiome measures that support biological plausibility.

3.2. Biological Plausibility of Balneotherapy in AD

3.2.1. Effects on the Skin Barrier

A primary rationale for balneotherapy in AD is the potential to improve stratum corneum hydration and skin comfort, thereby reducing xerosis-driven symptom burden and supporting barrier function. Reviews of thermal water use in chronic dermatoses and centre-based hydrotherapy programmes support hydration- and comfort-related benefits as plausible clinical drivers, while highlighting the importance of protocol details and co-interventions [17,18]. A systematic review of balneotherapy across dermatological diseases further underscores heterogeneity in water composition, programme structure, and endpoints, which complicates attribution of durable “barrier repair” beyond transient hydration [19].

3.2.2. Effects on the Skin Microbiome

Balneotherapy may plausibly influence microbiome dynamics through indirect mechanisms, including changes in skin hydration, pH, scratching behaviour, and barrier integrity. However, direct evidence that balneotherapy durably reduces *S. aureus* burden or normalises dysbiosis in AD remains limited. A useful comparator is the bleach-bath literature, where split-body and placebo-controlled trials have evaluated barrier outcomes and *S. aureus* colonisation [20–22]. Microbiome-focused studies and meta-analyses of bleach baths further show that water-based adjunctive regimens may improve selected clinical or barrier-related outcomes, although microbial dysbiosis may not fully normalise even when symptoms improve [23–26].

3.2.3. Effects on Pruritus and Symptom Burden

The most immediately plausible and clinically relevant impact of balneotherapy is on symptom burden, particularly itch, dryness-related discomfort, and sleep. In practice, spa-based care may also improve outcomes through structured routines that reduce behavioural and environmental triggers and reinforce adherence to skincare; these programme effects are clinically meaningful and should be acknowledged explicitly to avoid overstating water-specific mechanisms [17–19].

3.3. Taxonomy of Water-Based Interventions in AD

A major barrier to interpreting the balneotherapy literature in AD is that “water-based therapy” is often used as an umbrella label for exposures that are not interchangeable. For clinical and research clarity, modalities should be separated by core exposure, including water composition and delivery, treatment intensity and duration, embedded co-interventions, and health resort or treatment context [17–19]. In practical terms, meaningful reporting should include mineral profile, water temperature range, immersion duration and frequency, concomitant ultraviolet exposure when applicable, and post-bath skincare protocol. Table 1 summarizes reported characteristics from specific health resort or treatment contexts rather than implying that protocols or mechanisms are interchangeable across centres.

Table 1. Reported Characteristics, Site-Specific Protocols, Concomitant Interventions, and Endpoints of Water-Based Interventions in AD.

Intervention	Reported Setting/Health Resort Context	Key Characteristics	Typical Protocol	Mechanism/Co-interventions	Main Outcomes
Thermal Spring Water	Comano Spa, Italy [27,28]	Site-specific thermal spring water; mineral composition should not be generalized across centres	Daily immersion, commonly 15–20 min at 34–37 °C for 2–3 weeks in reported spa protocols	May improve hydration and skin comfort; typically combined with emollient protocols	Improvement in SCORAD and EASI scores, reduction in itch VAS, improvement in quality of life, reduced need for topical corticosteroids in reported thermal spa programmes [27,28]
Dead Sea Climatotherapy	Dead Sea region [29–32]	Hypertonic mineral-rich water within a specific climatic environment	Bathing combined with controlled solar ultraviolet exposure; duration usually 2–4 weeks	Potential keratolytic and anti-inflammatory effects related to mineral-rich water, climate, and controlled ultraviolet exposure	Improvement in SCORAD and quality of life, prolonged remission; difficulty in isolating the effect of water from ultraviolet exposure [29–32]

Table 1. Cont.

Intervention	Reported Setting/Health Resort Context	Key Characteristics	Typical Protocol	Mechanism/Co-interventions	Main Outcomes
Balneophototherapy	Synchronous outpatient balneophototherapy settings using salt-water or brine solution plus NB-UVB [33,34]	High-concentration salt solution, natural or artificial, combined with ultraviolet exposure	Synchronous immersion with narrowband UVB exposure according to defined phototherapy protocols	Combined salt-water immersion and NB-UVB exposure; effects on UV delivery and cumulative dose are protocol-dependent	Greater reduction in SCORAD and EASI compared with narrowband UVB alone in reported balneophototherapy protocols [33,34]
Seawater/Thalassotherapy	Site-specific seawater or thalassotherapy settings reported in the literature [35]	Sodium chloride-rich seawater or seawater-derived solutions with variable trace elements depending on site and protocol	Variable immersion protocols, typically at 30–35 °C	Potential keratolytic and comfort-related effects; effects on bacterial load remain protocol-dependent	Improvement in disease severity and quality of life; significant heterogeneity across studies [35]

Balneotherapy (thermal spring water immersion) refers to bathing in mineral-rich thermal waters delivered as structured spa programmes, commonly as daily or near-daily sessions over a defined course. Paediatric data from Comano describe clinical improvement with centre-based balneotherapy [27], and response-associated characteristics have been explored in subsequent work [28]. Seawater-based programmes and Dead Sea climatotherapy represent distinct exposures in which bathing is embedded in an environmental context that may include unique climate and, in many protocols, planned ultraviolet (UV) exposure [29–32]. Balneophototherapy (salt-water bathing combined with UV exposure) is best considered a phototherapy-adjacent modality; both synchronous outpatient approaches and randomised comparisons against narrowband UVB have been reported in AD [33,34]. Finally, water-based adjuncts used in routine practice, such as controlled bathing recommendations and dilute sodium hypochlorite baths, provide a comparator framework for standardised protocols and measurable microbial/barrier endpoints [3,20–26].

3.4. Clinical Evidence in AD

Overall, the clinical literature suggests that water-based interventions can improve AD symptom burden and quality of life, but the strength of inference depends on how tightly the exposure is defined and how much of the observed effect may plausibly be derived from the surrounding programme. Most structured spa or climatotherapy courses are time-limited, commonly spanning 1–3 weeks, and follow-up is frequently short or inconsistently reported, which limits conclusions about durability and flare prevention beyond the treatment window [29–32,35]. Against this background, the most consistent and clinically defensible benefits of balneotherapy in AD concern patient-centred outcomes, including pruritus, sleep impairment, xerosis-related discomfort, skin comfort, and quality of life [17–19,27–35]. These domains are particularly relevant in patients with residual burden despite otherwise appropriate anti-inflammatory treatment [7–11]. Conversely, claims of durable disease modification, sustained barrier repair, or microbiome normalization remain insufficiently supported unless accompanied by objective longitudinal data, such as TEWL, hydration, pH, tape-stripping biomarkers, quantitative *S. aureus* assessment, or microbiome profiling [1,4–6,12,36–39].

3.4.1. Dead Sea Climatotherapy and Seawater-Based Programmes

Dead Sea climatotherapy studies in AD describe improvements in clinical outcomes and quality of life during treatment courses [29–32]. Paediatric UV climatotherapy has also been reported, supporting feasibility in selected children under structured protocols [31]. Long-term follow-up comparisons against topical steroid regimens have been explored, but durability and causal attribution remain constrained by the multicomponent nature of the exposure [32]. A recent systematic review focusing on seawater therapy highlights methodological heterogeneity across non-pharmacological comparators and outcome reporting in this space [35].

3.4.2. Thermal Spring Water Programmes (Site-Specific Evidence)

Thermal spring water programmes delivered in dermatology-oriented spa centres may be more readily translatable because they often embed explicit skincare protocols and dermatology supervision. The Comano paediatric experience reports clinical improvement with balneotherapy [27], and subsequent work suggests that response may not be uniform across patients, supporting phenotype-aware positioning [28].

3.4.3. Balneophototherapy

Balneophototherapy has been evaluated as a strategy combining bathing with UV-based anti-inflammatory treatment. Early outpatient synchronous approaches using Dead Sea salt solution plus NB-UVB reported safety and efficacy signals [33], and a prospective randomised trial comparing synchronous balneophototherapy with NB-UVB monotherapy provides higher-level evidence while also highlighting protocol-dependence and feasibility constraints [34].

3.5. Objective Outcome Measures for Evaluating Balneotherapy

A major limitation of the existing balneotherapy literature in AD is the reliance on global severity scores and short-term symptom reporting without systematic integration of objective endpoints that can support biological plausibility and distinguish transient hydration effects from measurable changes in barrier-related outcomes. For balneotherapy, the most informative endpoint families are non-invasive barrier assessment, tape stripping biomarkers, and microbiome profiling, ideally captured longitudinally beyond the immediate treatment course.

3.5.1. Non-Invasive Barrier Assessment

EEMCO guidance provides methodological standards for TEWL and in vivo hydration measurements, including environmental control, acclimatisation, site selection, and washout windows for topical products [4,5]. In AD cohorts, correlations among TEWL, hydration, severity indices and QoL measures support their use as complementary endpoints, particularly when interventions aim to reduce “residual burden” beyond lesion extent [6].

3.5.2. Tape Stripping and Epidermal Biomarkers

Tape stripping enables minimally invasive capture of immune and barrier abnormalities in paediatric AD [36] and has been applied for monitoring inflammatory mediators during topical therapy [37]. Proteomic profiling on tape strips can identify biomarkers responsive to targeted therapy and support feasibility of non-invasive translational endpoints [38]. Concordance and complementarity between tape strip biomarkers and biopsy-derived measures have also been evaluated, reinforcing tape stripping as a practical bridge between clinic and mechanism [39].

3.5.3. Microbiome Assessment

Microbiome outcomes should prioritise quantitative *S. aureus* burden, diversity, and longitudinal stability rather than single time-point relative abundance snapshots. The flare-linked temporal volatility of the paediatric AD microbiome provides a benchmark for longitudinal study design [1], and strain-level diversity work illustrates why species-level data may be insufficient [12]. Microbiome-focused studies in the bleach-bath field show that measurable ecological shifts can occur, but also that dysbiosis may not “normalise” even when itch or barrier measures improve—an instructive caution for interpreting microbiome changes in balneotherapy [23,26].

3.6. Clinical Positioning in Contemporary AD Management

Balneotherapy is best framed as adjunctive supportive care aimed at improving domains that often remain clinically relevant despite optimised pharmacologic treatment (xerosis, barrier fragility, itch, sleep impairment, and day-to-day volatility) rather than as a substitute for evidence-based anti-inflammatory therapy. In selected settings, thermal spring water programmes have described clinically meaningful improvements in paediatric AD [27], and response-associated characteristics have been explored [28], supporting a phenotype-aware approach rather than a uniform recommendation.

3.6.1. Which Patients Are Most Likely to Benefit?

The highest-yield candidates are those whose limiting factor appears barrier- and symptom-dominant rather than uncontrolled inflammatory activity. Patients with moderate-to-severe AD who are otherwise controlled on systemic therapy may still present residual barrier impairment or dysbiosis-associated volatility; observational and mechanistic studies documenting barrier and hydration changes during dupilumab therapy further support the clinical relevance of “barrier endpoints” even in the targeted-therapy era [7–11].

3.6.2. Mechanistic Complementarity: Systemic Immunomodulation and Physicochemical Barrier Support

The integration of balneotherapy with targeted systemic agents may be conceptualized as a clinically coherent model of complementary care (Figure 1). While biologics and JAK inhibitors achieve suppression of type 2 inflammatory and pruritogenic pathways through upstream cytokine or signalling blockade, they may not immediately resolve all downstream biophysical consequences of chronic eczema, including xerosis, crusting, scaling, superficial barrier instability, and dysbiosis-associated disease volatility. In this coupled framework, balneotherapy may complement systemic efficacy through physicochemical and behavioural mechanisms, including stratum corneum hydration, softening and removal of crusts or scales, reduction of xerosis-related discomfort, and reinforcement of structured post-bath emollient use. These effects may indirectly support the cutaneous microenvironment by reducing scratching and limiting mechanical barrier disruption, both of which are closely linked to microbial imbalance and flare propensity in AD [3,14,15]. However, direct evidence that balneotherapy specifically reduces *S. aureus* biofilm, durably lowers microbial burden, or normalizes the AD microbiome remains limited. This complementary approach may help bridge the temporal gap between rapid pharmacological itch and inflammation control and the slower recovery of functional barrier resilience, thereby addressing the multidimensional nature of residual burden in selected patients.

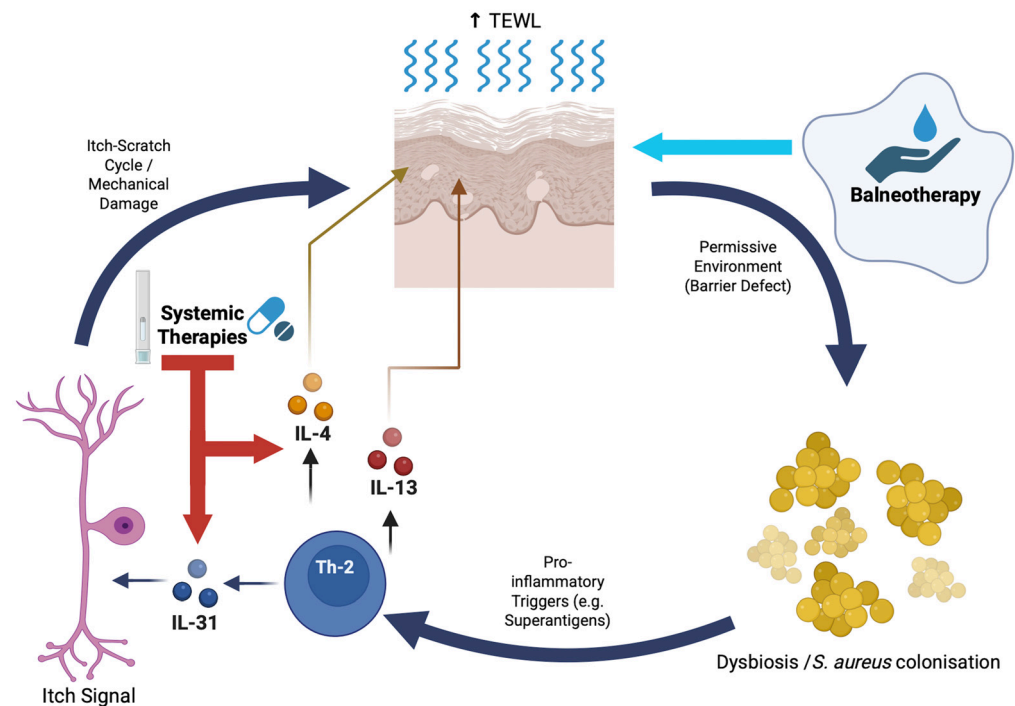


Figure 1. Hypothesized mechanistic complementarity of systemic therapy and balneotherapy. The diagram summarizes a proposed model in which systemic therapies provide upstream control of type 2 inflammation and pruritogenic signalling, while balneotherapy and structured water-based care may address downstream physical and symptomatic burden. By hydrating the stratum corneum, softening crusts or scales, reducing xerosis-related discomfort, and reinforcing post-bath emollient use, balneotherapy may support barrier stability and the cutaneous microenvironment.

3.6.3. Integration with Biologics and JAK Inhibitors

Integration should prioritise treatment continuity. Balneotherapy may be considered after stabilisation on systemic therapy to consolidate symptom control and reinforce long-term skincare behaviour, with realistic targets focused on itch/sleep, xerosis-related discomfort, and reduced need for rescue topical anti-inflammatory courses.

3.6.4. Practical Management During Balneotherapy

Implementation success depends on standardising operational elements that drive tolerability and durability (temperature/exposure duration, avoidance of harsh cleansers, immediate post-bath emollient application, and a clear flare plan). The broader bathing literature in AD provides a practical framework for integrating water-based interventions with routine topical management [3].

3.7. Safety Considerations and Contraindications

Balneotherapy in AD should be approached with a risk-aware framework appropriate to a barrier-compromised disease. In programmes where UV exposure is embedded (Dead Sea climatotherapy or balneophototherapy), attention to UV dose protocols and patient selection is essential, as highlighted in paediatric climatotherapy and balneophototherapy studies [31–34]. In addition, the bleach-bath literature underscores that water-based adjuncts can be safe when protocols are standardised, but tolerability and irritation risks must be managed pragmatically [20–26].

3.8. Research Gaps and Future Directions

The field now needs studies that are both mechanistically anchored and clinically implementable. Systematic reviews across thermal water/hydrotherapy and balneotherapy

emphasise that incomplete protocol reporting and heterogeneous endpoints are major obstacles to reproducibility [17–19]. Future research should prioritise standardised reporting of water composition, temperature, exposure duration/frequency, and embedded co-interventions, alongside pragmatic add-on designs and follow-up beyond programme completion. Objective endpoints should include TEWL/hydration/pH (standardised per EEMCO guidance) [4,5], tape stripping biomarkers [36–39], and microbiome outcomes anchored to absolute *S. aureus* burden and longitudinal stability [1,12].

4. Conclusions

Balneotherapy can be positioned as adjunctive supportive care for selected patients with AD, with the most consistent clinical benefits reported in symptom burden, particularly xerosis-related discomfort, pruritus, sleep impairment, and quality of life. Evidence from thermal spring water programmes [27,28] and Dead Sea climatotherapy/balneophototherapy studies [29–34] supports potential benefit in selected settings, but limited long-term follow-up and variable protocol reporting constrain mechanistic attribution and durability claims. Balneotherapy should be judged primarily by patient-centred outcomes and barrier-related measurements. Future research should prioritise reproducible protocol reporting, pragmatic add-on designs, and integration of objective barrier and microbiome endpoints to clarify which regimens and which patient phenotypes derive the greatest incremental benefit [4,5,36–39].

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Abbreviations

The following abbreviations are used in this manuscript:

AD	Atopic Dermatitis
JAKi	Janus Kinase inhibitors
IL	Interleukin
TEWL	Transepidermal Water Loss
NMF	Natural Moisturising Factor
QoL	Quality of Life
VAS	Visual Analogue Scale
SCORAD	Scoring Atopic Dermatitis
EASI	Eczema Area and Severity Index
CDLQI	Children’s Dermatology Life Quality Index

NB-UVB	Narrowband Ultraviolet B
UV	Ultraviolet

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