SCIENCE LETTERS

ISSN: 1675-7785
eISSN: 2682-8626

Available online at
http://myjms.mohe.gov.my/index.php/sl

Science Letters 20(2) 2026, 265-284

Science Letters

The roles of postbiotics in modulating the gut-skin axis in atopic dermatitis: A scoping review

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ARTICLE INFO

Article history:
Received 06 April 2026
Revised 05 May 2026
Accepted 06 May 2026
Published 30 June 2026

Keywords:
Atopic dermatitis
Postbiotics
Gut-skin axis
Immune modulation
Skin barrier function
Scoping review

DOI: 10.24191/scl.v20i2.11601

ABSTRACT

Atopic dermatitis (AD) is a persistent inflammatory condition, where immune dysregulation, skin barrier disruption, and alterations in gut microbiota converge along the gut-skin axis. This scoping review maps the existing evidence on postbiotics' role in modulating the gut-skin axis in AD. A total of 23 peer-reviewed articles were included in the review, comprising 5 human clinical studies, 3 animal studies, 3 *in vitro* studies, and 12 systematic reviews or position papers. The most frequently investigated postbiotic components were short-chain fatty acids (n = 5), heat-killed or inactivated bacterial preparations (n = 6), and microbial cell components or metabolites (n = 4). Most studies focused on immune modulation (n = 14), followed by barrier function (n = 10), and microbiota-related outcomes (n = 6). Of the human clinical studies, 60% (n = 3) evaluated oral postbiotic interventions, while 40% (n = 2) assessed topical formulations. The majority of animal and *in vitro* studies reported improvements in skin barrier integrity, including enhanced keratinocyte differentiation and reduced transepidermal water loss (TEWL) (n = 6 studies). A subset of studies (n = 6) demonstrated reductions in pro-inflammatory cytokines such as TNF-α and IL-6, and modulation of T-cell balance. Microbiota-related outcomes were less frequently reported, with three studies suggesting postbiotics' potential influence on microbial composition and short-chain fatty acid production. Despite these promising findings, substantial heterogeneity was observed across studies with respect to postbiotic type, study design, and outcome measures, leading to limited standardization. This review underscores the need for further high-

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https://doi.org/10.24191/scl.v20i2.11601

quality, well-powered human clinical trials using standardized postbiotic interventions to explore their therapeutic potential in AD. Future research should also address gaps in the understanding of the effects of specific postbiotics on both immune and barrier functions.

1.0 INTRODUCTION

Atopic dermatitis is characterized by chronic inflammation that is central to disease progression, impaired skin barrier function, immune dysregulation, and recurrent disease flares. Alterations in the intestinal microbiota, referred to as dysbiosis, have been increasingly implicated in the development and exacerbation of inflammatory skin diseases, including atopic dermatitis, through their influence on systemic and cutaneous immune-response patterns (Trompette *et al.*, 2022; Xiao *et al.*, 2023). This interaction is conceptualised within the gut–skin axis, which describes the bidirectional interactions between the gut microbiota, the immune system, and skin homeostasis (Kim *et al.*, 2024). Accordingly, modulation of the gut microbiota and its bioactive derivatives have been proposed as a strategy to prevent or attenuate inflammation and improve clinical outcomes in atopic dermatitis.

Interest in microbiota-directed interventions, particularly postbiotics, as potential therapeutic agents has increased substantially. Postbiotics are defined as non-viable microbial cells, their components, or metabolites that confer health benefits to the host (Lima & Paulino, 2024). Compared with probiotics, postbiotics may offer advantages in terms of safety, stability, and standardization (Yoshitake *et al.*, 2022). Postbiotic preparations may include heat-killed or inactivated bacteria, microbial cell wall components, and microbial metabolites such as short-chain fatty acids (SCFAs) (Aguilar-Toalá *et al.*, 2018; Lima & Paulino, 2024). These preparations are available in different formulations and can be administered orally or topically, depending on the intended therapeutic target.

The precise mechanisms by which postbiotics reduce inflammation and enhance skin barrier function remain incompletely understood. However, evidence suggests that postbiotics may modulate immune responses by regulating cytokine production, influencing T-cell differentiation, and attenuating IgE-mediated pathways, while also supporting epithelial and keratinocyte function (Chen *et al.*, 2022; Ogawa *et al.*, 2016). SCFAs of microbial origin have been shown to induce keratinocyte differentiation, enhance epidermal barrier integrity, and exert anti-inflammatory effects through host metabolic and signalling pathways (Żółkiewicz *et al.*, 2020). In addition, non-viable bacterial components may directly interact with pattern recognition receptors on immune and epithelial cells, thereby modulating inflammatory signalling cascades without the risks associated with live microbial colonization (Salminen *et al.*, 2021).

Interventions targeting the gut microbiota and its derivatives have been investigated across a range of inflammatory and immune-mediated conditions, including allergic, metabolic, gastrointestinal, and dermatological diseases. Meta-analyses and systematic reviews focusing predominantly on probiotics and synbiotics in atopic dermatitis have reported inconsistent effects on clinical severity scores and inflammatory biomarkers, with outcomes influenced by strain specificity, dosage, treatment duration, and population characteristics (Liscano *et al.*, 2025; Park *et al.*, 2021; Xu *et al.*, 2022). Notably, much of this evidence is derived from adult populations, with comparatively limited data in pediatric cohorts or studies involving non-viable microbial preparations.

Responses to microbiota-targeted interventions in infants and children may differ from those observed in adults due to developmental variations in gut microbiota composition, immune maturation, and skin barrier development. The early-life gut microbiota is less diverse, frequently dominated by bifidobacteria, and remains highly dynamic during the first years of life (Kim, 2024). Moreover, immune and inflammatory pathways continue to mature throughout childhood, suggesting that early-life interventions may yield distinct immunomodulatory and long-term health effects (Paz & Lio, 2024).

Despite growing interest in postbiotics, the literature remains fragmented, with substantial heterogeneity in definitions, components, experimental models, and outcome measures (Eraghieh Farahani *et al.*, 2025; Sulaiman *et al.*, 2025). A wide range of postbiotic preparations has been evaluated

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in clinical, animal, and in vitro studies, often assessing diverse immunological, microbiota-related, or barrier-function endpoints, which complicate cross-study comparisons (Amobonye *et al.*, 2025; Sulaiman *et al.*, 2025). Furthermore, it remains unclear which specific postbiotic components, routes of administration, and study designs are most appropriate for modulating the gut–skin axis in atopic dermatitis (Asefa *et al.*, 2025).

Given this heterogeneity and the exploratory nature of the field, a scoping review methodology was selected rather than a systematic review or meta-analysis. This approach aims to map the existing evidence on the effects of postbiotics on the gut–skin axis in atopic dermatitis (Arksey & O'malley, 2005; Peters *et al.*, 2024). Specifically, this scoping review seeks to identify the types of postbiotic components investigated, the experimental models and study designs employed, the reported immunological and barrier-related outcomes, and the key knowledge gaps to inform future research directions (Lee *et al.*, 2025). The research questions guiding this scoping review are: (1) What components of postbiotics have been investigated in relation to the gut–skin axis in atopic dermatitis?; (2) What experimental models and study designs have been used to investigate postbiotic-based modulation of the gut–skin axis in atopic dermatitis?; (3) What immunological outcomes have been reported following postbiotic interventions in atopic dermatitis? and (4) What effects of postbiotics on gut microbiota composition and skin or intestinal barrier function have been reported in atopic dermatitis?

2.0 MATERIALS AND METHODS

2.1 Search strategy

A comprehensive literature search was conducted to identify peer-reviewed studies using multiple electronic databases and publishing platforms, including PubMed (MEDLINE), ScienceDirect (Elsevier), MDPI, Frontiers, and ResearchGate. All retrieved records were imported into reference management software, and titles and abstracts were screened for eligibility. Articles meeting the inclusion criteria were subsequently reviewed in full text. In addition, the reference lists of included articles and relevant review papers were manually screened to identify further eligible studies.

The search strategy was developed to capture studies investigating postbiotics or postbiotic-related components in the context of the gut–skin axis and atopic dermatitis. Search terms were adapted to the specific requirements of each database and publishing platform. The detailed search strategy for PubMed (MEDLINE) is presented in Table 1, and comparable combinations of keywords were applied across the remaining databases.

Table 1. Search strategy for PubMed (MEDLINE)

Search	Search term(s) and combinations
1	(postbiotic* OR “heat-killed bacteria” OR “inactivated bacteria” OR paraprobiotic* OR “microbial metabolite*” OR “short-chain fatty acid*” OR SCFA*): ti, ab, kw
2	(“atopic dermatitis” OR eczema OR “allergic skin disease*”): ti, ab, kw
3	(“gut–skin axis” OR microbiota OR microbiome OR inflammation OR cytokine* OR immune OR barrier OR keratinocyte*): ti, ab, kw
4	(randomised controlled trial OR RCT OR clinical trial OR animal study OR in vitro): ti, ab, kw
5	(review OR protocol OR editorial): ti, ab, kw
6	1 AND 2 AND 3 AND 4 NOT 5

*Wildcard representing one or more characters in the search

2.2 Eligibility criteria

The eligibility criteria for inclusion in the review were based on the population, intervention, comparator, outcomes, and study design (PICOS) framework. The types of studies included were studies with: human subjects of any age with a diagnosis of atopic dermatitis, as well as animal and *in vitro* models

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investigating mechanisms associated with atopic dermatitis and the gut–skin axis; postbiotic interventions, such as non-viable or heat-killed microorganisms, microbial cell components, or microbial metabolites (e.g., short-chain fatty acids), administered via oral or topical routes; unexposed groups, placebo, vehicle control, routine treatment, or no postbiotic intervention; immunological outcomes (e.g., inflammatory or anti-inflammatory biomarkers), microbiota-related outcomes, and skin or intestinal barrier-function outcomes; randomised controlled trials, non-randomised interventional studies, animal studies, in vitro experimental studies, and review articles. Meanwhile, conference abstracts, commentaries, case reports, and articles published in languages other than English were excluded.

2.3 Data extraction and analysis

A data extraction form was developed and standardised to guide the data collection process. Data extraction was performed by the reviewers. The extracted data was entered into a Microsoft Excel spreadsheet and subsequently organized for synthesis. The information collected was the details of publication (first author, year of publication, journal, and study design), characteristics of the study (study population or experimental model, sample size, age group, health status, and geographical location), characteristics of the intervention **(**type of postbiotic, formulation, route of administration (oral or topical), comparator, and duration of the intervention) and outcomes of interest (immunological outcomes, microbiota-related outcomes, and skin or intestinal barrier-function outcomes relevant to atopic dermatitis).

Given that the included studies investigated postbiotic interventions across diverse populations, study designs, and outcome measures, the extracted data were grouped and synthesized descriptively according to study design, postbiotic type, and outcome category. A meta-analysis was not conducted due to substantial heterogeneity among studies, including variations in postbiotic components, intervention duration, outcome measures, and study populations. Therefore, findings were summarized using a narrative and descriptive approach to identifying overarching trends and research gaps within the existing literature.

3.0 RESULTS

In this scoping review, 23 articles were included after screening the records identified through the literature search. Figure 1 illustrates the study selection process. The included studies were conducted across diverse geographical regions, including Asia, Europe, and North America (Pattapulavar *et al.*, 2025; Trompette *et al.*, 2022).

3.1 Study designs and experimental models

Out of the 23 included studies, 5 were human clinical studies, 3 were animal studies, 3 were in vitro experimental studies, and 12 were narrative reviews, systematic reviews, or position papers (Pattapulavar *et al.*, 2025; Trompette *et al.*, 2022). The main types of human studies were randomised, placebo-controlled trials and pilot clinical studies investigating oral or topical postbiotic interventions. Animal models of atopic dermatitis (including murine models) were primarily employed to study immunological and barrier-related mechanisms, whereas in vitro models (including keratinocyte-based models of inflammation or barrier-function models) were used to investigate cellular inflammatory and barrier responses (Fujii, 2020; Kim *et al.*, 2019).

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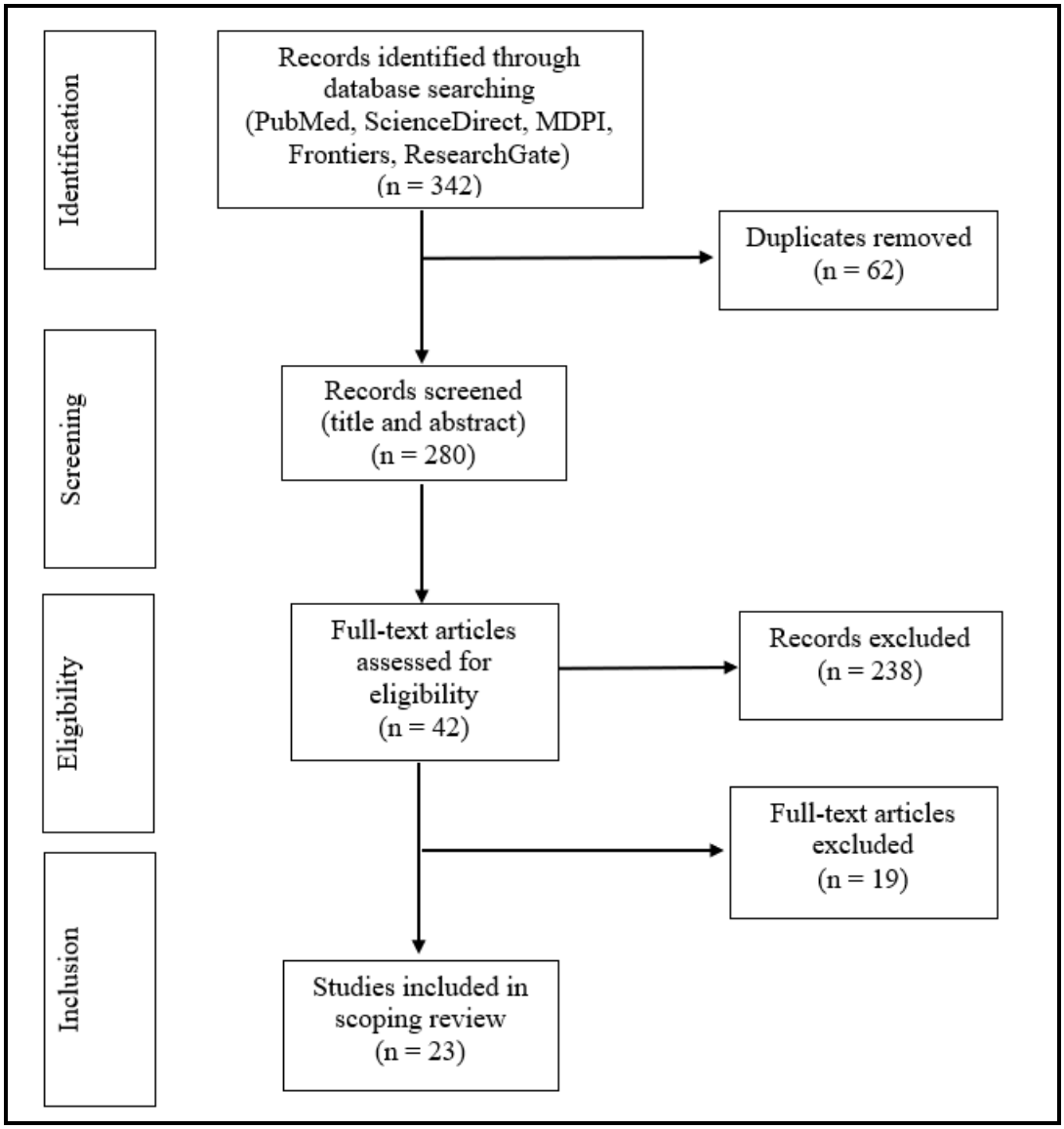


Figure 1. Flow diagram of the study selection process for the scoping review on the role of postbiotics in modulating the gut–skin axis in atopic dermatitis.

3.1 Study designs and experimental models

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and barrier-related mechanisms, whereas in vitro models (including keratinocyte-based models of inflammation or barrier-function models) were used to investigate cellular inflammatory and barrier responses (Fujii, 2020; Kim *et al.*, 2019).

3.2 Postbiotic components and interventions.

The number of postbiotic components examined varied considerably across the included studies. Five studies assessed short-chain fatty acids (SCFAs) as microbial metabolites or functional derivatives of microbial activity (Park *et al.*, 2021; Prajapati *et al.*, 2025; Trompette *et al.*, 2022; Vázquez-Frias *et al.*, 2025; Xiao *et al.*, 2023). Six studies investigated heat-killed or inactivated bacterial preparations, including strains of *Lactiplantibacillus plantarum*, *Lacticaseibacillus paracasei*, *Lactobacillus brevis*, and mixed bacterial formulations (Chen *et al.*, 2022; Lee *et al.*, 2025; Ogawa *et al.*, 2016; Piqué *et al.*, 2019; Yoshitake *et al.*, 2022). Four studies examined microbial cell components or secreted metabolites, primarily in experimental and mechanistic settings (Aguilar-Toalá *et al.*, 2018; Hashemi *et al.*, 2025; Pattapulavar *et al.*, 2025; Żółkiewicz *et al.*, 2020). One randomised, double-blind clinical trial evaluated a topical postbiotic formulation in patients with mild-to-moderate atopic dermatitis (Kim *et al.*, 2024).

The most common form of postbiotic intervention was oral administration, and the duration of intervention ranged from short-term experimental exposure to several weeks in clinical trials.

3.3 Immunological outcomes

Immunological outcomes were reported in 14 out of the 23 included studies (Chen *et al.*, 2022; Hashemi *et al.*, 2025; Lima & Paulino, 2024; Pattapulavar *et al.*, 2025; Paz & Lio, 2024; Piqué *et al.*, 2019; Prajapati *et al.*, 2025; Vázquez-Frias *et al.*, 2025; Xiao *et al.*, 2023; Xu *et al.*, 2022; Żółkiewicz *et al.*, 2020). Of these, eight studies reported reductions in pro-inflammatory markers, including inflammatory cytokines and IgE-mediated pathways (Chen *et al.*, 2022; Lee *et al.*, 2025; Lima & Paulino, 2024; Pattapulavar *et al.*, 2025; Prajapati *et al.*, 2025; Vázquez-Frias *et al.*, 2025; Żółkiewicz *et al.*, 2020). Five studies demonstrated modulation of immune cell balance, particularly involving T-cell subsets and immunoregulatory pathways (Chen *et al.*, 2022; Hashemi *et al.*, 2025; Paz & Lio, 2024; Piqué *et al.*, 2019; Xu *et al.*, 2022). One study reported mixed immunological findings, with certain cytokines reduced while others remained unchanged (Ogawa *et al.*, 2016).

Immunological endpoints were predominantly assessed in animal and in vitro models, whereas only three clinical studies reported immune-related outcomes as secondary endpoints.

3.4 Microbiota-related outcomes.

Microbiota-related outcomes were examined in six articles (Micu *et al.*, 2025; Park *et al.*, 2021; Prajapati *et al.*, 2025; Trompette *et al.*, 2022; Vázquez-Frias *et al.*, 2025; Xiao *et al.*, 2023). Three studies investigated the composition of the gut or skin microbiota, including alterations in microbial diversity or relative abundance, while three studies analysed microbial metabolic activity, particularly pathways related to short-chain fatty acid (SCFA) production. Collectively, these findings suggest a potential modulatory effect of postbiotics on microbiota composition and function in relation to skin inflammation. However, microbiota-related outcomes were largely exploratory and inconsistently reported across studies.

3.5 Barrier-function outcomes

Barrier-function outcomes were reported in 10 studies (Hashemi *et al.*, 2025; Kim *et al.*, 2024; Lee *et al.*, 2025; Ogawa *et al.*, 2016; Park *et al.*, 2021; Prajapati *et al.*, 2025; Trompette *et al.*, 2022; Vázquez-Frias *et al.*, 2025; Xu *et al.*, 2022; Yoshitake *et al.*, 2022). Six studies demonstrated improvements in skin barrier integrity, including enhanced keratinocyte differentiation and reduced transepidermal water loss (Kim *et al.*, 2024; Lee *et al.*, 2025; Trompette *et al.*, 2022; Vázquez-Frias *et al.*, 2025; Xu *et al.*, 2022;

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Yoshitake *et al.*, 2022). Four studies documented protective effects against inflammatory or oxidative damage in keratinocytes or animal models of atopic dermatitis (Hashemi *et al.*, 2025; Ogawa *et al.*, 2016; Park *et al.*, 2021; Prajapati *et al.*, 2025). Barrier-related outcomes were observed across human, animal, and in vitro studies.

3.6 Evidence gaps

Although the number of postbiotic studies in humans has increased, only five human clinical trials were identified. Moreover, substantial variability was observed in the definitions of postbiotics, specific ingredients, routes of administration, and outcome measures. This heterogeneity precluded quantitative synthesis, and the existing evidence base does not support meta-analysis. Collectively, these findings underscore the need for further well-designed human studies investigating standardised postbiotic interventions with clinically relevant immunological, microbiota-based, and barrier-function outcomes (Lima & Paulino, 2024; Pattapulavar *et al.*, 2025; Paz & Lio, 2024; Salminen *et al.*, 2021; Vázquez-Frias *et al.*, 2025).

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Table 2. Studies evaluating the effect of postbiotics on the gut–skin axis in atopic dermatitis

NO	Author, Year	Study Design and Population	Intervention(s)	Biomarker(s)	Significant Intervention Effects (Anti-Inflammatory)	Overall Effect (Pro-Inflammatory)
1	Xu et al., 2022	*In vitro* experimental study; UVB-induced keratinocyte inflammation model	Heat-killed *Lacticaseibacillus paracasei*	Oxidative Damage: MDA, PC, ROS Antioxidant: GSH, SOD, CAT, GPx, GST, GR, Nrf2, Sirt1, PGC-1α Skin Photoaging: COL1A1, MMP-1, MMP-2, MMP-9, SA-β-gal → Melanogenesis: TYR, TYRP-1, MITF, p-PKA, p-CREB	Anti-inflammatory: ↓ Oxidative stress (MDA, PC, ROS), ↑ Antioxidant enzymes (SOD, CAT, GPx) Barrier Function: ↑ COL1A1, ↓ MMP-1, MMP-2, MMP-9 Anti-melanogenesis: ↓ Melanin, ↓ Tyrosinase, Suppressed TYRP-1 & TYR via PKA/CREB/MITF	Anti-inflammatory (PL): ↓ Oxidative damage & photoaging via antioxidants, modulated Nrf2/Keap1 Barrier-enhancing (PL): ↑ Collagen, ↓ MMPs Anti-melanogenic (PL): ↓ Melanin & Tyrosinase activity, inhibited melanogenesis

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2	Trompette et al., 2022	Experimental study; murine and keratinocyte models	Postbiotic metabolites (short-chain fatty acids)	Keratinocyte Markers: IVL, Lor, FLG Inflammatory Markers: IL-33, IL-4Rα, S100A8, CXCL1	Anti-inflammatory: ↓ IL-33, IL-4Rα, S100A8, CXCL1; ↓ TH2 cytokines (IL-4, IL-5) Barrier Enhancement: ↑ IVL, Lor, FLG; ↑ Stratum corneum thickness, lipid synthesis (ceramides, cholesterol)	Anti-inflammatory: ↓ Inflammation, ↓ IgE production Barrier-enhancing: ↑ Skin barrier, ↓ Allergen penetration, ↑ Hydration

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3	Chen et al., 2022	Experimental animal study; house dust mite–induced atopic dermatitis mouse model	Heat-killed probiotic mixture	Immune T Cell Markers: IL-4+CD4+ T cells (Th2), IFN-γ+CD4+ T cells (Th1), CD4+CD25+Foxp3+ T cells (Tregs) IgE Markers: Total IgE, HDM-specific IgE	Immune Modulation: ↓ IL-4+CD4+ T (Th2), ↓ IFN-γ+CD4+ T (Th1) in spleen; no change in Tregs (CD4+CD25+Foxp3+) IgE Regulation: ↓ Total IgE and HDM-specific IgE in serum, alleviated skin lesions, and inflammation (↓ epidermal thickening)	Anti-inflammatory: Heat-killed MP01 & MP02 modulated immune balance, ↓ IgE, and reduced inflammation in AD mice
4	Lee et al., 2025	*In vitro* experimental study; keratinocyte-based AD model	Heat-killed *Lactiplantibacillus plantarum* postbiotics	Inflammatory Cytokines: IL-6, IL-8, TSLP, TARC Skin Barrier Genes: INV, FLG, LOR Antioxidants: DPPH, ABTS, SOD-like activity	Anti-inflammatory: ↓ IL-6, IL-8, TSLP, TARC after postbiotic treatment Suppressed inflammation in TNF-α/IFN-γ-stimulated HaCaT cells	Skin Barrier Restoration: ↑ INV, FLG, LOR expression Anti-inflammatory Action: Postbiotics ↓ Inflammatory cytokines, enhancing skin health
5	Xiao et al., 2023	Narrative review; inflammatory skin diseases	Postbiotic metabolites (SCFAs)	Keratinocyte markers: FLG, KRT1 Inflammatory markers: IL-6, ICAM-1, IL-17, TNF-α	Anti-inflammatory: ↓ IL-6, ICAM-1 in HaCaT cells; ↓ TNF-α, IL-6, IL-8 in skin inflammation Barrier-enhancing: ↑ FLG, KRT1 expression; enhanced skin barrier through mitochondrial metabolism and keratinocyte differentiation	Anti-inflammatory (SCFAs): Butyrate, propionate ↓ pro-inflammatory cytokines and inhibited mast cell activation Barrier-enhancing (SCFAs): Improved skin barrier by promoting keratinocyte differentiation and lipid synthesis

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6	Aguilar-Toalá et al., 2018	Narrative review; functional foods and human health	Postbiotics (non-viable microbial cells, components, and metabolites)	Skin Barrier Markers: FLG, LOR, IVL Inflammatory Markers: sIL-2R, IL-13, CCL17 (TARC), CCL22 (MDC) Allergic Marker: IgE	Anti-inflammatory: ↓ sIL-2R, IL-13, CCL17, CCL22 (significant decrease vs placebo) Barrier Enhancement: ↑ FLG, LOR, IVL Clinical Improvement: ↓ TEWL, reduced pruritus, ↑ skin moisture, improved EASI & IGA scores	No Significant Improvement: IgE unchanged Placebo Trend (Pro-inflammatory): ↑ sIL-2R, IL-13, CCL17, CCL22
7	Żółkiewicz et al., 2020	Narrative review; probiotics, prebiotics, and postbiotics	Postbiotics (heat-killed bacteria and microbial metabolites)	Skin Barrier Markers: FLG, LOR, IVL Inflammatory Markers: sIL-2R, IL-13, CCL17 (TARC), CCL22 (MDC) Allergic Marker: IgE	Anti-inflammatory: ↓ sIL-2R, IL-13, CCL17, CCL22 (significant vs placebo) Barrier Enhancement: ↑ FLG, LOR, IVL Clinical Improvement: ↓ TEWL, reduced pruritus, ↑ skin moisture, improved EASI & IGA scores	No Significant Improvement: IgE unchanged Placebo Trend (Pro-inflammatory): ↑ Inflammatory markers (sIL-2R, IL-13, CCL17, CCL22)
8	Kim, S. R., 2024	Narrative review; inflammatory airway disease	Microbiota-derived metabolites and immune pathways	Inflammatory Markers: sST2, MPO, IL-8, S100A9 Immune Markers: IL-33, ST2 receptor	Neutrophilic Inflammation: Elevated sST2 in uncontrolled asthma (UA) linked to ↑ MPO, IL-8, S100A9. Increased ST2 expression on neutrophils. Cytokine Regulation: IL-33 ↑ MPO and ERK/MAPK activation in neutrophils, enhancing airway inflammation.	Anti-inflammatory: sST2 as a diagnostic biomarker for UA, indicating neutrophilic inflammation. Barrier-enhancing: Targeting the IL-33/ST2 pathway could reduce neutrophilic inflammation, potentially improving asthma control.
9	Hashemi et al., 2025	Narrative review; dermatological and wound-healing contexts	Postbiotics (non-viable microbes and microbial metabolites)	Skin Healing Markers: Collagen, elastin, MMP-1, MMP-9 Inflammation Markers: IL-8, TNF-α, IL-10, TGF-β Barrier Integrity: TEWL, hydration, skin pH	Anti-inflammatory: ↓ IL-8, TNF-α, ROS, NO Wound Healing: Stimulated collagen synthesis, angiogenesis, tissue regeneration Antimicrobial: ↓ Pathogen growth; supported skin microbiome balance	Anti-inflammatory: ↓ Inflammation in wound areas Barrier-enhancing: ↑ Skin barrier, wound closure, tissue regeneration Pro-healing: ↑ Collagen production, ↓ Scar tissue

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10	O'Neill et al., 2016	Narrative review; gut–skin axis in health and disease	Microbiota-derived metabolites and immune pathways	Inflammatory Markers: IL-17, IL-10, TNF-α, TGF-β Gut Markers: SCFAs (butyrate, acetate, propionate) Skin Markers: TEWL, FLG, collagen deposition	Anti-inflammatory: ↑ IL-10, TGF-β; ↓ TNF-α, IL-17; modulated immune response via Tregs Barrier Function: ↑ Filaggrin expression, ↓ TEWL, ↑ Collagen deposition, improved skin hydration Microbiome Modulation: Probiotics improved skin thickness, sebocyte production, and gut microbiota (Lactobacillus reuteri) improved skin phenotype and immune response	Anti-inflammatory: ↓ Inflammation in gut and skin Barrier-enhancing: ↑ Skin hydration, elasticity, and barrier function Immune Modulation: Tregs and SCFAs modulated immune response, improving skin health
11	Pattapulavar et al., 2025	Narrative review; next-generation therapeutics	Probiotic-derived postbiotics	Immunomodulatory Markers: IL-6, TNF-α, IL-8, IL-10, IL-1β Antioxidant Markers: ROS, DPPH radical scavenging activity Cancer-related Markers: Bax, caspase-3, caspase-9 (apoptosis), cell viability	Anti-inflammatory: ↓ IL-6, TNF-α, IL-8, IL-1β; ↑ IL-10 for anti-inflammatory response Antioxidant: Strong free radical scavenging (DPPH, H2O2); ↑ Antioxidant capacity, reduced oxidative stress Anti-cancer: Induced apoptosis in cancer cells (HT-29, A549); cytotoxic effects in pancreatic, lung, and colon cancer; ↓ Proliferation in colon cancer (HCT-116)	Anti-inflammatory: Postbiotics ↓ Inflammatory cytokines, promoted balanced immune response Antioxidant: ↓ Oxidative stress, improved redox balance Anti-cancer: Induced apoptosis, inhibited cancer cell proliferation, especially in colorectal cancer models

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12	Piqué et al., 2019	Narrative review; human and experimental studies	Heat-killed (tyndallized) probiotics	Gut Health Markers: IL-8, TNF-α, sIgA, ZO-1 (tight junctions) Immune Response Markers: IL-12, TLR2 activation, IL-10	Anti-inflammatory: ↓ TNF-α, IL-8, sIgA; ↑ IL-10 (immune tolerance) Barrier Function: ↑ ZO-1 expression; improved intestinal barrier integrity, protection against pathogen adhesion Pathogen Protection: Competed with enteropathogens, produced bacteriocins, EPS to inhibit pathogen growth	Anti-inflammatory: ↓ Systemic and intestinal inflammation via immune modulation Barrier-enhancing: ↑ Gut barrier function, epithelial integrity Probiotics Protection: Heat-killed probiotics protected against intestinal infections and dysbiosis
13	Micu et al., 2025	Narrative review; clinical and microbiome studies in atopic dermatitis	Probiotics and postbiotic-related mechanisms along the gut–skin axis	Skin and Immune Markers: IL-4, IL-13, TNF-α, IL-6, IL-10, TGF-β Microbiome Markers: Staphylococcus aureus, Lactobacillus spp., Bifidobacterium spp. Barrier Function Markers: TEWL, filaggrin	Anti-inflammatory: ↓ TNF-α, IL-6, IL-4 (type 2 inflammation); ↑ IL-10, TGF-β (anti-inflammatory) Barrier Function: ↑ Filaggrin expression; ↓ TEWL in skin lesions; modulated Staphylococcus aureus burden Microbiome Modulation: ↑ Lactobacillus, Bifidobacterium; ↓ Proteobacteria in gut	Anti-inflammatory: Probiotics ↓ skin inflammation, modulated immune response Barrier-enhancing: Improved skin barrier, ↑ Hydration, ↓ Microbial translocation Gut-skin Axis: Probiotics influenced both gut and skin microbiomes, supporting systemic immune regulation

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14	Prajapati et al., 2025	Narrative review; skin health and microbiome	Postbiotics and microbiome-derived metabolites	Skin Markers: Filaggrin, TEWL, skin hydration, IL-17A, IL-4 Microbiome Markers: Staphylococcus epidermidis, Cutibacterium acnes, Corynebacterium spp. Inflammatory Markers: IL-10, TNF-α, IL-6, TLR2	Anti-inflammatory: ↓ IL-6, TNF-α, IL-4, IL-17A; ↑ IL-10, reducing inflammation Barrier Function: ↑ Filaggrin, ↓ TEWL, improved skin hydration; strengthened skin barrier by modulating microbiome (↑ Staphylococcus epidermidis, Cutibacterium acnes) Microbiome Modulation: ↑ Beneficial bacteria (Staphylococcus epidermidis); ↓ Pathogens (Staphylococcus aureus); modulated immune response via T-cell interactions	Anti-inflammatory: Postbiotics ↓ IL-4, IL-6 Barrier-enhancing: Improved skin hydration, integrity, and barrier function Microbiome-modulating: Supported growth of beneficial bacteria, reduced pathogenic dominance
15	Zerihun et al., 2025	Narrative review; chronic disease and therapeutic perspectives	Postbiotics (microbial metabolites and inactivated microbes)	Skin Barrier Markers: Occludin, claudin, MUC2 Inflammatory Markers: IL-10, IL-1β, IL-6, TNF-α, COX-2, TLR4 Allergic/Other Markers: NF-κB	Anti-inflammatory: ↓ IL-6, TNF-α, IL-4, IL-17A; ↑ IL-10 Barrier Enhancement: ↑ Filaggrin, ↑ Skin hydration, ↓ TEWL Clinical Improvement: ↓ TEWL, ↑ Skin hydration	No Significant Improvement: Not reported Pro-inflammatory Trend: Not reported
16	Lima & Paulino, 2024	Systematic review of randomised controlled trials; atopic dermatitis populations	Oral postbiotic preparations	Clinical Parameters: SCORAD Immunological Markers: TARC, IgE, eosinophil count Gut Microbiome: specific microbial taxa	Adults: ↓ SCORAD, ↓ inflammatory markers, ↓ IgE; benefit with heat-killed Lactobacillus postbiotics Pediatrics: mixed results; 3/6 studies showed no significant improvement; higher doses linked to better outcomes	Anti-inflammatory: ↓ TARC, ↓ IgE, associated with less skin inflammation Gut Microbiome: microbial shifts reported in some trials, suggesting a role in AD management

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17	Liscano et al., 2025	Systematic review and meta-analysis; patients with atopic dermatitis and psoriasis	Microbial interventions, including postbiotic-related approaches	Skin Markers: SCORAD, PASI Inflammatory Markers: IL-4, TNF-α, IL-10, IL-17, IgE Barrier Function: TEWL, skin hydration, filaggrin levels	AD: ↓ SCORAD, ↓ IL-4, IgE, eosinophils; improved skin hydration and ↓ TEWL Psoriasis: mixed effects on PASI; some improvement, some no significant change; improved DLQI; modulation of Th17-related cytokines	Anti-inflammatory: ↓ TNF-α, IL-4, IL-17, IgE in AD Barrier-enhancing: improved skin hydration and barrier function in AD Psoriasis: trend toward improvement, but PASI reduction inconsistent
18	Kim et al., 2024	Randomised, double-blind, vehicle-controlled trial; adolescents and adults with mild-to-moderate atopic dermatitis	Topical *Streptococcus*-derived postbiotic emollient	Skin Barrier Markers: FLG, LOR, IVL Inflammatory Markers: sIL-2R, IL-13, CCL17 (TARC), CCL22 (MDC), IgE	Anti-inflammatory: ↓ sIL-2R, IL-13, CCL17, CCL22 Barrier Enhancement: ↑ FLG, LOR, IVL Clinical Improvement: ↓ TEWL, reduced pruritus, ↑ skin moisture	No Significant Improvement: IgE unchanged Placebo Trend (Pro-inflammatory): ↑ sIL-2R, IL-13, CCL17, CCL22
19	Yoshitake et al., 2022	Randomised, placebo-controlled, double-blind study; healthy adults	Heat-killed *Lactiplantibacillus plantarum* L-137	Skin Moisture Markers: Water content of the stratum corneum (SC) Skin Barrier Markers: TEWL	Moisture Enhancement: ↑ SC water content at forearm and face → Barrier Improvement: ↓ TEWL at face; improved skin moisture and barrier function in older participants and those with dry skin	Barrier-enhancing: HK L-137 ↑ skin hydration and ↓ TEWL, especially in older participants or those with dry skin
20	Ogawa et al., 2016	Randomised, double-blind, placebo-controlled trial; adults with dry skin	Heat-killed *Lactobacillus brevis* SBC8803	Skin Hydration Markers: TEWL, corneal hydration (skin conductance)	Skin Hydration Improvement: ↓ TEWL at neck (8-week) and lower eye region (4-week) in 50 mg/day group; ↑ corneal hydration at neck (12-week) in 25 mg/day group	Barrier-enhancing: improved skin hydration, especially in individuals with low habitual intake of lactic fermentation products, particularly at neck and eye regions

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21	Salminen et al., 2021	Expert consensus statement (ISAPP)	Postbiotics (definition and scope)	Immune Markers: IL-8, TNF-α, IL-10, TLR4, TLR2 Gut Barrier Markers: tight junction proteins (ZO-1, occludin), SCFAs	Immune Modulation: ↓ TNF-α, IL-8; ↑ IL-10; modulated TLR2/TLR4 signaling Barrier Function: ↑ ZO-1, occludin; ↑ SCFAs (acetate, propionate, butyrate); improved epithelial barrier function	Anti-inflammatory: ↓ pro-inflammatory cytokines and modulated immune responses via TLR pathways Barrier-enhancing: strengthened epithelial barrier and reduced gut permeability
22	Park et al., 2021	Pilot observational study; healthy adult participants	Endogenous postbiotic metabolites (short-chain fatty acids)	Skin Barrier Markers: TEWL, hydration levels, skin pH Microbiome Markers: Cutibacterium, Staphylococcus, Lactobacillus, Anaerobacillus SCFA Markers: propionate, acetate, butyrate	Microbial Modulation: ↑ Cutibacterium and propionate in moist skin; ↓ Staphylococcus vs dry skin Skin Hydration & Barrier: ↑ hydration, ↓ TEWL in moist skin, indicating stronger barrier	Anti-inflammatory: higher propionate linked to better skin hydration and barrier function Barrier-enhancing: ↑ Cutibacterium and hydration associated with ↓ TEWL and better barrier integrity
23	Vázquez-Frias et al., 2025	Position paper; paediatric clinical practice	Postbiotics in pediatric populations	Gut Health Markers: α-/β-defensins, secretory IgA, gut microbiota composition Inflammatory Markers: IL-8, TNF-α, IL-10, IL-1β Gut Maturation Markers: Bifidobacterium, Lactobacillus, Streptococcus thermophilus	Anti-inflammatory: ↓ IL-8, TNF-α, IL-1β, ROS; ↑ IL-10, secretory IgA Gut Maturation: ↑ Bifidobacterium, Lactobacillus; enhanced gut barrier and immune maturation Gut Protection: ↓ pathogen colonization and infection risk	Anti-inflammatory: reduced inflammation, improved immune response Gut-health Supporting: supported recovery from acute diarrhea Barrier-enhancing: promoted gut health and gastrointestinal protection in pediatric populations

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4.0 DISCUSSION

The studies included in this scoping review were conducted across highly diverse populations, experimental models, and geographical settings, encompassing human clinical research, animal models, in vitro experiments, and narrative or systematic reviews. Study populations comprised both healthy individuals and patients with atopic dermatitis, while experimental investigations utilised allergen-induced animal models and keratinocyte-based inflammatory systems. A wide range of postbiotic preparations was evaluated, including short-chain fatty acids, heat-killed or inactivated bacterial strains, microbial metabolites, and topical postbiotic formulations. This substantial heterogeneity in study populations, postbiotic components, outcome measures, and study designs reflects the fragmented nature of the current evidence base (Tjiu & Lu, 2025; Vázquez-Frias *et al.*, 2025; Zhou *et al.*, 2024).

Overall, the evidence regarding the impact of postbiotics on immune and inflammatory pathways in atopic dermatitis remains inconclusive (Liscano *et al.*, 2025). Although several studies reported reductions in pro-inflammatory cytokines or modulation of immune cell balance, these effects were often pathway-specific and not consistently observed across all assessed biomarkers (Al-Qahtani *et al.*, 2024). As a result, generalisation across studies and definitive conclusions regarding anti-inflammatory efficacy are not currently possible. This inconsistency precludes quantitative synthesis and supports the appropriateness of a scoping review methodology rather than a systematic review or meta-analysis (Prajapati *et al.*, 2025; Trompette *et al.*, 2022).

Evidence from human clinical studies remains limited. Only a small number of randomised controlled trials have directly evaluated postbiotic interventions in patients with atopic dermatitis, with most focusing primarily on barrier-related outcomes rather than comprehensive immunological profiling (Kim *et al.*, 2024). Improvements in transepidermal water loss and barrier integrity were more consistently reported than reductions in a wide range of inflammatory biomarkers. Collectively, these findings suggest that postbiotics may exert more pronounced effects on epithelial and barrier function than on systemic immunomodulation (Kim *et al.*, 2024; Lee *et al.*, 2025; Yoshitake *et al.*, 2022).

Conversely, animal and in vitro research demonstrated more consistent immunomodulatory and barrier-restoring effects. Investigations in murine models of atopic dermatitis showed decreases in IgE production, improved T-cell balance, and attenuation of inflammatory reactions following administration of heat-killed bacterial preparations or postbiotic metabolites (Chen *et al.*, 2022; Hashemi *et al.*, 2025). Similarly, keratinocyte-based experiments demonstrated reduced inflammatory signalling, improved oxidative stress responses, and restoration of barrier-related gene expression in response to postbiotic treatment (Lee *et al.*, 2025; Xu *et al.*, 2022). Although these findings provide valuable mechanistic insights, their translational relevance to human disease remains uncertain, given differences in immune system complexity, microbiota composition, and disease chronicity (O'Neill *et al.*, 2016).

A notable observation across the included studies is the frequent lack of correlation between biomarker changes and clinical or functional outcomes. Some studies reported improvements in skin barrier function or clinical symptoms with minimal or inconsistent alterations in inflammatory biomarkers (Wahhab *et al.*, 2024). This suggests that commonly measured cytokines and systemic markers may not fully capture the mechanisms through which postbiotics influence disease activity in atopic dermatitis. Greater emphasis may therefore be warranted on localised effects on keratinocyte metabolism, epidermal integrity, and tissue-level immune regulation (Park *et al.*, 2021; Trompette *et al.*, 2022; Żółkiewicz *et al.*, 2020).

Interpretation of the literature is further complicated by the heterogeneity of postbiotic preparations. Postbiotics encompass a broad spectrum of biologically active substances, and their effects appear highly dependent on formulation, strain origin, and mode of administration. Consensus statements and review articles have highlighted the absence of standardised definitions, reporting frameworks, and outcome measures, limiting reproducibility and comparability across studies (Lima & Paulino, 2024; Salminen *et al.*, 2021; Żółkiewicz *et al.*, 2020). Such non-standardisation remains a significant barrier to translating experimental findings into clinical practice.

Overall, the existing evidence suggests that postbiotics have potential as modulators of the gut–skin axis in atopic dermatitis, despite current limitations. Compared with live probiotics, postbiotics offer advantages in safety, stability, and regulatory control, which may be particularly important for individuals with compromised skin barriers or immune dysfunction (Paz & Lio, 2024). Experimental and preliminary clinical data indicate that postbiotics may modulate immune responses, microbiota-related pathways, and barrier function without the risks associated with live microbial administration (Aguilar-Toalá *et al.*, 2018; Paz & Lio, 2024; Vázquez-Frias *et al.*, 2025).

Nevertheless, several important research gaps were identified in this scoping review. There is a need for well-designed human studies with adequately powered sample sizes, standardised postbiotic interventions, and harmonised outcome measures that integrate clinical severity assessments, barrier-function parameters, and mechanistic biomarkers (Pattapulavar *et al.*, 2025). Future research should determine whether observed effects

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are specific to individual postbiotic components or attributable to broader postbiotic classes, and whether modulation of the gut–skin axis can be translated into sustained clinical benefit in atopic dermatitis (Trompette *et al.*, 2022; Żółkiewicz *et al.*, 2020).

5.0 CONCLUSION

This scoping review provides a comprehensive overview of the existing evidence regarding the role of postbiotics in the regulation of the gut–skin axis in atopic dermatitis. The findings suggest that postbiotics have the potential to modulate immune responses, microbiota-related pathways, and skin barrier function, particularly through localised and pathway-specific mechanisms. Preliminary experimental and early clinical data indicate that postbiotics may enhance barrier integrity and regulate inflammatory signalling without the risks associated with live microbial administration (Kim *et al.*, 2024; Trompette *et al.*, 2022; Xu *et al.*, 2022).

However, the overall evidence base remains heterogeneous and fragmented. Although positive effects have been reported, particularly in experimental models and selected clinical trials, consistent improvements across a broad range of inflammatory biomarkers or robust clinical endpoints have not been demonstrated. Variability in postbiotic preparations, study methodologies, and outcome measures limits the ability to draw definitive conclusions regarding clinical benefit in atopic dermatitis (Paz & Lio, 2024; Vázquez-Frias *et al.*, 2025).

Future research should prioritise well-designed human studies evaluating standardised postbiotic interventions with harmonised outcome measures and integrated assessment of clinical severity, immunological parameters, microbiota-related indices, and barrier-function outcomes. Such investigations are necessary to clarify the therapeutic potential of postbiotics and to define their role within the broader framework of gut–skin axis modulation in atopic dermatitis.

Acknowledgements/Funding

The authors would like to acknowledge the Faculty of Applied Sciences, Universiti Teknologi MARA, Shah Alam, Selangor, Malaysia, for academic support in the preparation of this manuscript. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of interest statement

The authors declare that this research was conducted in the absence of any commercial, financial, or personal relationships that could be construed as a potential conflict of interest.

Authors' contributions

NH carried out the literature search, data extraction, analysis, and manuscript drafting. MFI conceptualised the research idea, supervised the review process, provided intellectual input, and revised the manuscript. Mohd IMY, SML and KR contributed to manuscript review, intellectual input, and final approval of the article.

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