

## PROBIOTICS IN ATOPIC DERMATITIS: DOES GUT MICROBIOME MODULATION ENHANCE SKIN CONDITION?

Kamila Łukańko<sup>1\*</sup> , Aleksandra Blachucik<sup>1</sup> , Wiktoria Marciniak<sup>1</sup> ,  
Karolina Biegańska<sup>1</sup> , Joanna Nowicka<sup>2</sup> 

<sup>1</sup> Students' Scientific Society of Microbiologists, association at the Department of Microbiology,  
Faculty of Medicine, Wrocław Medical University

<sup>2</sup>Department of Microbiology, Faculty of Medicine, Wrocław Medical University

Submitted on 13 March 2026, accepted on 09 September 2026

**Abstract:** Atopic dermatitis (AD) is a chronic condition driven by epidermal barrier disruption and multiphasic immune dysregulation involving type 2, Th1, Th17, and Th22 pathways. Beyond local cutaneous pathology, systemic interactions mediated by the bidirectional gut-skin axis play an integral role, in which intestinal dysbiosis, depletion of short-chain fatty acid-producing taxa, and compromised epithelial integrity exacerbate cutaneous symptoms. This review evaluates the therapeutic potential and mechanistic pathways of targeted probiotic administration to restore microenvironmental equilibrium and alleviate cutaneous pathology. The underlying biological mechanisms-encompassing epithelial junction reinforcement, short-chain fatty acid biosynthesis, and cytokine rebalancing-are examined alongside current clinical evidence. Therapeutic interventions utilizing selected bacterial strains have been shown to significantly reduce SCORAD indices, indicating that probiotic supplementation represents a viable, safe adjunctive strategy for managing disease severity in both pediatric and adult populations

1. Introduction. 2. Pathophysiology of atopic dermatitis. 3. Probiotics. 4. Probiotics in atopic dermatitis. 5. Limitations. 6. Summary.

**Keywords:** gut dysbiosis, eczema, butyrate, epidermal barrier.

### 1. Introduction

Atopic Dermatitis (AD) is one of the most common chronic inflammatory diseases, which typically manifests with eczema, dry skin, and pruritus (Lugović-Mihić et al. 2023). It has a multifactorial etiology involving genetic and environmental factors, immune dysregulation, epidermal barrier dysfunction, and intestinal dysbiosis (Díez-Madueño et al. 2024). Although the condition can affect individuals of any age, it is predominantly found in the pediatric population, with a prevalence of up to 20% in children and remaining below 10% in adults. The clinical presentation of AD is age-dependent, but its chronic inflammatory nature, associated with the overreaction of Th2 and Th22 lymphocytes, remains a consistent trait. While immune cell dysfunction and epidermal barrier disruption remain fundamental pathological fea-

tures of AD, in recent years scientists have paid more attention to bidirectional communication between the intestine and the skin. As a result, they established that this connection, known as the gut-skin axis, also plays an important role in the pathogenesis of atopic dermatitis (Simonetti et al. 2021; Díez-Madueño et al. 2024; Blicharz et al. 2025). The intestinal microbiome, with functionally vital taxa such as *Lactobacillus* and *Bifidobacterium*, plays a crucial role in immune system maturation and the maintenance of systemic homeostasis, so any disruption to this microbial balance can cause serious consequences for the host. In AD patients, particularly adults, gut dysbiosis is consistently observed and closely related to disease persistence (Díez-Madueño et al. 2024). Beyond mere correlation, a Mendelian randomization-based study found plausible evidence (Xue et al. 2023). It suggests a relationship between the occurrence of atopic dermatitis and gut

\* Corresponding authors: Kamila Łukańko, Students' Scientific Society of Microbiologists, association at the Department of Microbiology, Faculty of Medicine, Wrocław Medical University, Wrocław, Poland; e-mail: [lukankokamila@gmail.com](mailto:lukankokamila@gmail.com)

© 2026 Kamila Łukańko et al.

This is an open access article licensed under the Creative Commons Attribution-NonCommercial-NoDerivs License (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

#### Cite as:

Probiotics in Atopic Dermatitis: Does Gut Microbiome Modulation Enhance Skin Condition? Łukańko K. et al., ADV MICROBIOL-NY, 2026, 65, 2, 154-160, <https://doi.org/10.2478/am-2026-0013>

microbiota composition, supporting the hypothesis that microbial alterations are significant factors in disease development.

Microbial metabolites and the structural integrity of the intestinal wall primarily drive the interaction between skin condition and gut health. One recent study found that AD severity is strongly associated with circulating gut-derived metabolites and biomarkers linked to increased intestinal permeability (Blicharz et al. 2025). A compromised gut barrier promotes the entry of pro-inflammatory mediators and metabolic products into the bloodstream, contributing, inter alia, to the exacerbation of cutaneous inflammation. In light of these findings, modulating the intestinal microbiota via probiotic supplementation represents a promising adjunctive therapy, especially with specific strains that have shown efficacy in reducing AD symptoms among children and adolescents (Andrade et al. 2022). Furthermore, the systemic translocation of microbial products and gut-derived inflammatory mediators triggers oxidative stress (OS) by overwhelming the host's antioxidant defenses. Available data suggest that the exacerbation of skin lesions in AD is triggered by an intestine-derived OS that creates a feedback loop sustaining chronic inflammation (Simonetti et al. 2021).

Although significant associations link gut dysbiosis, barrier dysfunction, and oxidative stress to AD pathogenesis, the mechanisms by which these factors converge to drive skin inflammation are not yet fully understood. Therefore, this study aims to analyze the bidirectional connection within the gut-skin axis and the influence of intestinal microbiota on inflammatory reactions, especially in AD. It also evaluates the role of probiotic interventions in rebalancing the gut ecosystem and improving skin condition, which may reduce atopic symptoms.

## 2. Pathophysiology of atopic dermatitis

Dysfunction of the epidermal barrier is the main cause of atopic dermatitis (Van Den Bogaard et al. 2023). These abnormalities result from genetic factors, including mutations in the filaggrin (FLG) gene, a protein essential for the structural integrity of the epidermis. In a healthy body, filaggrin is broken down into free amino acids, which form part of the natural moisturizing factor (NMF). Filaggrin deficiency leads to decreased NMF levels, elevated skin pH, and the activation of endogenous proteases, resulting in weak-

ened intercellular junctions and excessive transepidermal water loss (TEWL). A damaged barrier loses its protective function, allowing environmental allergens, microorganisms, and irritants to penetrate deep into the tissue and trigger an immune response, even in skin without visible lesions (Hansen-Sackey and Hartono 2025).

Antigen penetration through the permeable epidermis stimulates keratinocytes to secrete alarmins, including TSLP, IL-25, and IL-33, which activate an inflammatory cascade dominated by a type 2 response (Hansen-Sackey and Hartono 2025). Th2 lymphocytes produce cytokines that are important for the development of atopic dermatitis, such as IL-4 and IL-13. These compounds not only drive inflammation and pruritus but also operate in a vicious cycle by inhibiting the expression of barrier proteins, including filaggrin and loricrin, which exacerbate epidermal damage and increase susceptibility to external factors. While the Th2/Th22 immune axis primarily drives acute lesions, the immunopathology of atopic dermatitis exhibits marked complexity and chronicity-dependent plasticity. In the chronic phase of the disease, there is a pronounced shift toward a mixed inflammatory infiltrate characterized by activated Th1 cells producing IFN- $\gamma$  and regulated by upstream cytokines such as IL-12.

Furthermore, the Th17 pathway contributes significantly to chronic tissue remodeling, epidermal hyperproliferation, and antimicrobial defense through the secretion of IL-17 and IL-22 (Brunner et al. 2017). Among the major factors affecting disease course is skin dysbiosis, characterized by a drastic reduction in microbiome diversity and the dominance of *Staphylococcus aureus*. This bacterium uses a quorum-sensing mechanism to monitor population density and colonizes the skin in most patients with exacerbations of atopic dermatitis (Okamoto et al. 2025). After reaching sufficient population density, *S. aureus* activates toxin and protease production. These proteases directly damage keratinocytes and promote mast cell degranulation, exacerbating inflammation. This phenomenon is intensified by a deficiency of commensal strains, such as *Staphylococcus epidermidis*, which protect healthy skin by producing natural antimicrobial peptides (Kim and Kim 2019).

The pathophysiology of atopic dermatitis is closely linked not only to the skin microbiome but also to the gut microbiota, which is described by the concept of the “gut-skin axis”. Studies show that patients

with AD exhibit profound gut dysbiosis, characterized by reduced overall microbial diversity and compositional shifts extending beyond the depletion of classical commensals such as *Bifidobacterium* spp. and *Lactobacillus* spp. In particular, patients often show a marked loss of key short-chain fatty acid (SCFA)-producing obligate anaerobes, most notably *Faecalibacterium prausnitzii* and members of the *Ruminococcaceae* and *Lachnospiraceae* families. Concurrently, an expansion of opportunistic and pro-inflammatory taxa, including *Enterobacteriaceae* and certain *Clostridium* spp. (such as *Clostridium perfringens*) and *Clostridium difficile*, has been observed. This dysbiotic state leads to a deficient production of immunomodulatory SCFAs, especially butyrate, which is essential for intestinal epithelial barrier integrity and regulatory T cell differentiation. Consequently, impaired barrier function (“leaky gut”) facilitates the translocation of bacterial endotoxins and systemic pro-inflammatory metabolites into the circulation, driving systemic inflammation that exacerbates cutaneous pathology (Haidar et al. 2025). Furthermore, early colonization of the gut by normal bacterial flora is important for immune system development. Given that microbial perturbations directly compromise systemic and cutaneous immune homeostasis, microbiota-targeted therapeutic strategies, including the administration of specific probiotics, prebiotics, and postbiotics, represent a promising adjunct approach in the management of atopic dermatitis (Kim and Kim 2019).

### 3. Probiotics

Probiotics are defined as live microorganisms that, when administered in adequate amounts, confer health benefits to the host (Hill et al. 2014). Their beneficial effects are mediated through a variety of biological mechanisms, primarily within the gastrointestinal tract, but with systemic consequences involving immune, metabolic, and neuroendocrine pathways. Importantly, probiotic mechanisms are strain-specific and should not be automatically extrapolated to other strains within the same species or genus. It also depends on host factors, microbial composition, and environmental conditions (Plaza-Diaz et al. 2019). Microorganisms most commonly used as probiotics and documented to have health benefits belong to several bacterial genera, including *Lactobacillus*, *Bifidobacterium*, *Enterococcus*, *Streptococcus*, *Pediococcus*, *Leuconostoc*, *Bacillus*, and the yeast genus *Saccharo-*

*myces*. In addition, a selected strain of *Escherichia coli* Nissle 1917 has shown positive effects. Strains belonging to *Bifidobacterium* spp. and *Lactobacillus* spp. are especially popular (Fijan 2014).

One fundamental mechanism of probiotics is the competitive exclusion of pathogenic microorganisms. Certain probiotic strains compete with pathogens for adhesion sites on the intestinal epithelium and for available nutrients within the gut lumen (Plaza-Diaz et al. 2019; Latif et al. 2024). By occupying epithelial binding receptors, these strains may reduce pathogen attachment and colonization. Additionally, some probiotic strains can produce antimicrobial substances, such as organic acids, hydrogen peroxide, and bacteriocins, which inhibit the growth of pathogenic species (Latif et al. 2024). Furthermore, certain strains of *Bifidobacterium* and *Lactobacillus* can produce compounds that reduce intestinal pH, which also inhibit the development of enteric pathogens by creating an unfavorable environment and promoting beneficial bacteria (Markowiak and Śliżewska 2017).

Another mechanism involves modulating gut microbiota composition. Probiotic supplementation may help restore microbial balance after disturbances such as antibiotic treatment, infection, or dietary changes (Markowiak and Śliżewska 2017; Plaza-Diaz et al. 2019). Certain probiotic strains can influence microbial composition and its protective functions by promoting the growth of beneficial commensal bacteria and suppressing opportunistic or pathogenic species (Latif et al. 2024).

Probiotic supplementation may also directly or indirectly influence the production of SCFAs, including acetate, butyrate, and propionate, which are primarily generated through microbial fermentation of dietary fibers and carbohydrates (Markowiak and Śliżewska 2017; Plaza-Diaz et al. 2019). These metabolites serve as energy sources for enterocytes and, through their beneficial influence on adipose tissue and liver, play a pivotal role in immune regulation, energy homeostasis, and metabolism. They activate G-protein-coupled receptors, which regulate immune responses, glucose metabolism, and lipid homeostasis. Butyrate is especially interesting because it exhibits anti-inflammatory properties by inhibiting histone deacetylases, a mechanism that alters gene expression and influences functions such as cell proliferation, apoptosis, and differentiation. SCFAs can regulate cytokine expression in T cells and the generation of regulatory T cells (Treg) through their effect on histone deacetylase inhibition (Koh et al. 2016; Markowiak and Śliżewska 2017).

|                                               |                                                                                                                                                                                                                                                                                 |
|-----------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Probiotics and other pathogens                | <ul style="list-style-type: none"><li>- competitive exclusions of pathogenic bacteria through taking over nutrients and place for adherence to gut mucosa</li><li>- production of anti-microbial substances (SCFA, organic acids, hydrogen peroxide and bacteriocins)</li></ul> |
| Improvements in epithelial barrier functions  | <ul style="list-style-type: none"><li>- stimulating gut mucosa to produce more mucin proteins</li><li>- stimulating expression of tight junction proteins which prevents translocation of bacteria into the blood</li></ul>                                                     |
| Regulation of immune system in the human body | <ul style="list-style-type: none"><li>- modulating responses of dendritic cells, macrophages and T lymphocytes</li><li>- increasing production of anti-inflammatory cytokines by interacting with epithelial cells</li></ul>                                                    |
| Production of neurotransmitters               | <ul style="list-style-type: none"><li>- influencing levels of serotonin, dopamine and GABA transmitters in the gut-brain axis</li></ul>                                                                                                                                         |

**Tab.1** Probiotics – mechanisms of action, authors’ own elaboration based on (Latif et al. 2024)

Another mechanism describes a significant immunomodulatory effect exerted by certain probiotic strains through interactions with both the innate and adaptive immune systems, primarily at the level of the intestinal mucosa. They engage pattern recognition receptors, such as Toll-like receptors expressed on intestinal epithelial cells, dendritic cells, and macrophages. These interactions modulate cytokine production, characterized by increased anti-inflammatory cytokines like interleukin-10 and transforming growth factor- $\beta$ , which can control the expression of pro-inflammatory mediators (e.g., tumor necrosis factor- $\alpha$ , interleukin-6). For example, *Lactobacillus casei* CRL 431 has been shown to stimulate IL-10 release, while *Limosilactobacillus reuteri* 100-23 has been associated with increased IL-10 and TGF- $\beta$  production. (Koh et al. 2016; Bron et al. 2017; Mazziotta et al. 2023).

Furthermore, selected probiotic strains influence antigen presentation and may contribute to regulatory T cell differentiation, resulting in immune tolerance and the maintenance of intestinal immune homeostasis. Therefore, the immune system can become tolerant to beneficial strains (Plaza-Diaz et al. 2019; Mazziotta et al. 2023). Through these immunomodulatory effects, beneficial strains help limit excessive inflammatory responses (particularly relevant in inflammatory and autoimmune conditions) while preserving effective immune defense against pathogenic microorganisms.

It may also be relevant to atopic dermatitis, in which dysregulated immune responses result in chronic inflammation.

Probiotics play a significant role in maintaining and strengthening the intestinal epithelial barrier. Selected probiotic strains, especially *L. rhamnosus* ATCC 7469 and *Lactiplantibacillus plantarum* WCFS1, enhance the expression of occludin, claudins, E-cadherins, and zonula occludens-1 proteins, which take part in improving tight-junction integrity, thereby reducing intestinal permeability (Bron et al. 2017; Plaza-Diaz et al. 2019). Certain probiotic strains from *Lactobacillus* spp. can also stimulate mucus production by goblet cells through increased mucin gene expression, providing an additional physical barrier against microbial translocation. By strengthening epithelial integrity, some strains help prevent the passage of pathogens, toxins, and pro-inflammatory molecules into the systemic circulation (Bron et al. 2017; Latif et al. 2024). Because the gut-skin axis may link intestinal barrier integrity with systemic immune regulation, understanding its underlying mechanisms may improve the treatment of atopic dermatitis.

Emerging evidence suggests that probiotics can influence the bidirectional communication between the gut and the central nervous system, known as the gut-brain axis. This interaction occurs through neural, immune, and metabolic pathways. Certain

probiotic strains can modulate the production of neurotransmitters and neuromodulators, such as serotonin and gamma-aminobutyric acid (GABA). They may influence dopamine levels, which may affect mood, behavior, gut motility, and stress-related pathways. In the literature, e.g., *L. rhamnosus* JB-1 is described as possibly impactful for increasing levels of glutamate, glutamine, N-acetyl aspartate, and GABA (Cryan et al. 2019; Latif et al. 2024). Stress and psychological factors are known to impact atopic dermatitis activity and symptom severity directly; thus, these beneficial effects should be considered important.

#### 4. Probiotics in atopic dermatitis

Atopic and allergic diseases are characterized by a predominance of Th2 immune responses, including elevated levels of IL-4, IL-5, and IL-13, which promote IgE synthesis. Probiotics have been shown to reduce the severity of atopic dermatitis by inhibiting the Th2-mediated response and improving the Th1/Th2 ratio (Jang et al. 2012). By inhibiting the Th2 cell response, cytokines such as IL-4, IL-5, and IL-13 are no longer released. Probiotics also stimulate the secretion of IL-10 and transforming growth factor- $\beta$  (TGF- $\beta$ ), cytokines essential for suppressing excessive immune activation. Probiotics can alleviate inflammation by reducing proinflammatory cytokines, IL-4, IL-6, tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), INF- $\gamma$ , and high-sensitivity C-reactive protein (hsCRP), and by increasing the expression of IL-10 and Treg-related cytokines at mesenteric lymph nodes (Kim et al. 2013).

Dysbiosis, characterized by reduced abundance of beneficial taxa (*Lactobacillus* spp., *Bifidobacterium* spp.) and increased pathogenic species, has been associated with Th2-skewed inflammation, impaired regulatory T cell function, and elevated immunoglobulin E in AD patients (Kader et al. 2021). Probiotics modulate the intestinal microbiome and immune status by improving the intestinal barrier; these effects reduce allergic phenomena and AD severity (Lin et al. 2015).

Adults with atopic dermatitis and obesity represent a clinically relevant population, as obesity is associated with an increased prevalence of AD and may intensify cutaneous inflammation through immune, metabolic, and microbiome-related mechanisms (Zhang & Silverberg, 2015). In this context, gut dysbiosis may disrupt the gut-skin axis, suggesting that restoring intestinal microbial homeostasis could influence systemic inflammation and AD severity (Shang & Zhao, 2024). Beyond conventional probiotic supplementation, fe-

cal microbiota transplantation (FMT) has emerged as a potential microbiome-based therapeutic approach, with exploratory clinical results in adults with moderate-to-severe AD suggesting improvements in disease severity and modulation of the gut microbiota. However, further studies are needed to prove its efficacy and safety (Liu et al. 2024).

Prakoeswa CRS et al. (2020) showed that *Lactiplantibacillus plantarum* IS-10506 may help reduce clinical symptoms in adults with atopic dermatitis, as shown by decreases in the SCORAD index, IL-4, and IL-17 levels, IgE concentrations, along with an increase in Foxp3+ levels (Prakoeswa et al. 2020). An imbalance in cellular immunity, particularly between Th1 and Th2 cells and between Th17 and Foxp3+ Treg cells, underlies the development of AD, and a key finding of this study is that probiotics beneficially modulate both Th2 and Th1 adaptive immune responses.

Yamamoto K et al. (2016) demonstrated that *Lactobacillus acidophilus* L-92 improved clinical outcomes, including SCORAD and EASI reduction, sustained remission, and immunomodulatory effects (Yamamoto et al. 2016). The intake of L-92 reduced subjective symptoms in adult AD patients. Furthermore, sustained ingestion of L-92 was suggested to suppress scratching behavior, maintain remission of skin symptoms, and significantly increase transforming growth factor- $\beta$  and IL-12 levels at the end of the treatment period. This study suggested that L-92 suppresses type-2-helper-T-cell-dominant inflammation by activating regulatory T cells and type 1 helper T cells.

In newborns, the composition of *Bifidobacterium* species in the intestinal microbiota has been shown to influence the risk of developing allergic diseases (Akay et al. 2014). A study comparing fecal levels of different *Bifidobacterium* species in allergic and non-allergic children found significantly higher concentrations of *Bifidobacterium longum* in healthy individuals. These findings suggest a protective role for *B. longum* in preventing bronchial asthma and allergic dermatitis.

Consistent with these findings, Cukrowska et al. (2021) demonstrated that in children up to two years old with atopic dermatitis, the administration of a probiotic preparation containing *Lactobacillus rhamnosus* LOCK 0900 and LOCK 0908, together with *Lactobacillus casei* LOCK 0918, was safe and produced favorable clinical outcomes (Cukrowska et al. 2021). Dietary supplementation for three months significantly decreased atopic dermatitis severity, as evaluated using the SCORAD index.

## 5. Limitations

A limitation of this study is its exclusive focus on assessing the therapeutic potential of probiotics in AD, whilst disregarding the impact of other alternative or complementary treatment methods or conventional therapy. The analysis covers various age groups, which also makes it difficult to draw definitive conclusions. Future studies should consider the therapeutic process as a whole, along with factors such as the patient's age, comorbidities, and medications.

## 6. Summary

In conclusion, the gut-skin axis is a fundamental component of atopic dermatitis. Gut dysbiosis and increased intestinal permeability drive systemic pro-inflammatory cascades that aggravate cutaneous barrier dysfunction. Based on the literature analyzed in this review, probiotic supplementation is a biologically plausible and safe adjunctive therapeutic strategy that can modulate systemic immunity and mitigate disease severity. The clinical efficacy of probiotics is strictly strain-specific and varies substantially by target age demographic. In pediatric populations, particularly during early immune development, interventions aimed at establishing bifidobacterial dominance (notably *Bifidobacterium longum*) and multi-strain preparations, such as the consortium of *L. rhamnosus* LOCK 0900, *L. rhamnosus* LOCK 0908, and *L. casei* LOCK 0918, have demonstrated clinical efficacy in significantly decreasing SCORAD indices and mitigating allergic sensitization (Cukrowska et al. 2021). In adult cohorts, where chronic inflammation features complex Th1/Th17/Th22 profiles and distinct metabolic comorbidities, specific strains such as *L. plantarum* IS-10506 and *L. acidophilus* L-92 have shown superior benefits in inducing clinical remission, reducing pruritus, and restoring Treg-driven homeostasis alongside TGF- $\beta$  and IL-12 upregulation (Prakoeswa et al. 2020; Yamamoto et al. 2016). Despite these promising findings, clinical translation is currently hindered by study heterogeneity, variable intervention durations, and unstandardized dosing regimens. These age-dependent variations and strain-specific outcomes underscore the need for further rigorous, large-scale randomized controlled trials. Future research should prioritize identifying optimal strain consortia, the optimal intervention window across distinct age groups, and how host-specific metabolic factors influence therapeutic responsiveness.



ORCID

Kamila Łukańko: <https://orcid.org/0009-0004-1737-2094>

Aleksandra Blachucik: <https://orcid.org/0009-0002-6768-0680>

Wiktoria Marciniak: <https://orcid.org/0009-0005-1666-173X>

Karolina Biegańska: <https://orcid.org/0009-0005-6888-9170>

Joanna Nowicka: <https://orcid.org/0000-0002-4001-4189>

## Conflict of interest

The authors do not report any financial or personal connections with other persons or organizations, which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

## References

- Akay HK, Tokman HB, Hatipoglu N, Hatipoglu H, Siraneci R, Demirci M, Borsa BA, Yuksel P, Karakullukcu A, Kangaba AA, Sirekbasan S, Aka S, Torun MM, Kocazeybek BS. The relationship between bifidobacteria and allergic asthma and/or allergic dermatitis: a prospective study of 0–3 years-old children in Turkey. *Anaerobe*, 2014 Aug; 28: 98–103. <https://doi.org/10.1016/j.anaerobe.2014.05.006>
- Andrade PDSMA, Silva JME, Carregaro V, Sacramento LA, Roberti LR, Aragon DC, Carmona F, Roxo-Junior P. Efficacy of Probiotics in Children and Adolescents With Atopic Dermatitis: A Randomized, Double-Blind, Placebo-Controlled Study. *Front Nutr*, 2022 Jan; 8: 833666. <https://doi.org/10.3389/fnut.2021.833666>
- Blicharz L, Samborowska E, Zagożdżon R, Bukowska-Ośko I, Czuwara J, Zych M, Roszczyk A, Perlejewski K, Makowska K, Nowaczyk J, Zaremba M, Kazimierska K, Waśkiel-Burnat A, Dadlez M, Samochocki Z, Olszewska M, Radkowski M, Rudnicka L. Severity of atopic dermatitis is associated with gut-derived metabolites and leaky gut-related biomarkers. *Sci Rep*, 2025 Jul; 15(1): 26146. <https://doi.org/10.1038/s41598-025-09520-y>
- Bron PA, Kleerebezem M, Brummer R-J, Cani PD, Mercenier A, MacDonald TT, Garcia-Ródenas CL, Wells JM. Can probiotics modulate human disease by impacting intestinal barrier function? *Br J Nutr*, 2017 Jan; 117(1): 93–107. <https://doi.org/10.1017/S0007114516004037>
- Brunner PM, Guttman-Yassky E, Leung DY. The immunology of atopic dermatitis and its reversibility with broad-spectrum and targeted therapies. *J Allergy Clin Immunol*, 2017 Apr; 139(4 Suppl): S65–S76. <https://doi.org/10.1016/j.jaci.2017.01.011>
- Cryan JE, O'Riordan KJ, Cowan CSM, Sandhu KV, Bastiaanssen TFS, Boehme M, Codagnone MG, Cussotto S, Fulling C, Golubeva AV, et al. The Microbiota-Gut-Brain Axis. *Physiological Reviews*, 2019 Oct; 99(4): 1877–2013. <https://doi.org/10.1152/physrev.00018.2018>
- Cukrowska B, Ceregra A, Maciorkowska E, Surowska B, Zegadło-Mylik MA, Konopka E, Trojanowska I, Zakrzewska M, Biera JB, Zakrzewski M, et al. The Effectiveness of Probiotic *Lactobacillus rhamnosus* and *Lactobacillus casei* Strains in Children with Atopic Dermatitis and Cow's Milk Protein Allergy: A Multi-center, Randomized, Double Blind, Placebo Controlled Study. *Nutrients*, 2021 Apr; 13(4): 1169. <https://doi.org/10.3390/nu13041169>
- Díez-Madueño K, de la Cueva Dobao P, Torres-Rojas I, Fernández-Gosende M, Hidalgo-Cantabrana C, Coto-Segura P. Gut Dysbiosis and Adult Atopic Dermatitis: A Systematic Review. *J Clin Med*, 2024 Dec; 14(1): 19. <https://doi.org/10.3390/jcm14010019>

- Fijan S.** Microorganisms with Claimed Probiotic Properties: An Overview of Recent Literature. *Int J Environ Res Public Health*, 2014 May; 11(5): 4745–4767. <https://doi.org/10.3390/ijerph110504745>
- Haidar L, Bănărescu CE, Uța C, Zimbru E-L, Zimbru R-I, Tîrziu A, Pătrașcu R, Șerb A-F, Georgescu M, Nistor D, et al.** Beyond the Skin: Exploring the Gut–Skin Axis in Chronic Spontaneous Urticaria and Other Inflammatory Skin Diseases. *Biomedicines*, 2025 Aug; 13(8): 2014. <https://doi.org/10.3390/biomedicines13082014>
- Hansen-Sackey EB, Hartono S.** Atopic Dermatitis: Pathophysiology and Emerging Treatments. *Allergies*, 2025 Nov; 5(4): 40. <https://doi.org/10.3390/allergies5040040>
- Hill C, Guarner F, Reid G, Gibson GR, Merenstein DJ, Pot B, Morelli L, Canani RB, Flint HJ, Salminen S, et al.** The International Scientific Association for Probiotics and Prebiotics consensus statement on the scope and appropriate use of the term probiotic. *Nat Rev Gastroenterol Hepatol*, 2014 Aug; 11(8): 506–514. <https://doi.org/10.1038/nrgastro.2014.66>
- Jang SO, Kim HJ, Kim YJ, Kang MJ, Kwon JW, Seo JH, Kim HY, Kim BJ, Yu J, Hong SJ.** Asthma Prevention by *Lactobacillus Rhamnosus* in a Mouse Model is Associated With CD4+CD25+Foxp3+ T Cells. *Allergy Asthma Immunol Res*, 2012 May; 4(3): 150–156. <https://doi.org/10.4168/aaair.2012.4.3.150>
- Kader HA, Azeem M, Jwayed SA, Al-Shehhi A, Tabassum A, Ayoub MA, Hetta HF, Waheed Y, Iratni R, Al-Dhaheri A, et al.** Current Insights into Immunology and Novel Therapeutics of Atopic Dermatitis. *Cells*, 2021 Jun; 10(6): 1392. <https://doi.org/10.3390/cells10061392>
- Kim H-J, Kim HY, Lee S-Y, Seo J-H, Lee E, Hong S-J.** Clinical efficacy and mechanism of probiotics in allergic diseases. *Korean J Pediatr*, 2013 Sep; 56(9): 369–376. <https://doi.org/10.3345/kjp.2013.56.9.369>
- Kim JE, Kim HS.** Microbiome of the Skin and Gut in Atopic Dermatitis (AD): Understanding the Pathophysiology and Finding Novel Management Strategies. *J Clin Med*, 2019 Apr; 8(4): 444. <https://doi.org/10.3390/jcm8040444>
- Koh A, De Vadder F, Kovatcheva-Datchary P, Bäckhed F.** From Dietary Fiber to Host Physiology: Short-Chain Fatty Acids as Key Bacterial Metabolites. *Cell*, 2016 Jun; 165(6): 1332–1345. <https://doi.org/10.1016/j.cell.2016.05.041>
- Latif A, Shehzad A, Niazi S, Zahid A, Ashraf W, Iqbal MW, Rehman A, Riaz T, Aadil RM, Khan IM, et al.** Corrigendum: Probiotics: mechanism of action, health benefits and their application in food industries. *Front Microbiol*, 2024 Feb; 15: 1378225. <https://doi.org/10.3389/fmicb.2024.1378225>
- Lin R-J, Qiu L-H, Guan R-Z, Hu S-J, Liu Y-Y, Wang G-J.** Protective effect of probiotics in the treatment of infantile eczema. *Exp Ther Med*, 2015 May; 9(5): 1593–1596. <https://doi.org/10.3892/etm.2015.2299>
- Lugović-Mihic L, Meštrović-Štefekov J, Potočnjak I, Cindrić T, Ilić I, Lovrić I, Skalicki L, Bešlić I, Pondeljnak N.** Atopic Dermatitis: Disease Features, Therapeutic Options, and a Multidisciplinary Approach. *Life*, 2023 Jun; 13(6): 1419. <https://doi.org/10.3390/life13061419>
- Markowiak P, Śliżewska K.** Effects of Probiotics, Prebiotics, and Synbiotics on Human Health. *Nutrients*, 2017 Sep; 9(9): 1021. <https://doi.org/10.3390/nu9091021>
- Mazziotta C, Tognon M, Martini F, Torreggiani E, Rotondo JC.** Probiotics Mechanism of Action on Immune Cells and Beneficial Effects on Human Health. *Cells*, 2023 Jan; 12(1): 184. <https://doi.org/10.3390/cells12010184>
- Okamoto H, Li S, Nakamura Y.** The Role of Skin Dysbiosis and Quorum Sensing in Atopic Dermatitis. *JID Innovations*, 2025 Jul; 5(4): 100377. <https://doi.org/10.1016/j.xjidi.2025.100377>
- Plaza-Diaz J, Ruiz-Ojeda FJ, Gil-Campos M, Gil A.** Mechanisms of Action of Probiotics. *Adv Nutr*, 2019 Jan; 10(Suppl 1): S49–S66. <https://doi.org/10.1093/advances/nmy063>
- Prakoeswa CRS, Bonita L, Karim A, Herwanto N, Umborowati MA, Setyaningrum T, Hidayati AN, Surono IS.** Beneficial effect of *Lactobacillus plantarum* IS-10506 supplementation in adults with atopic dermatitis: a randomized controlled trial. *J Dermatol Treat*, 2022 May; 33(3): 1491–1498. <https://doi.org/10.1080/09546634.2020.1836310>
- Shang D, Zhao S.** Molecular mechanisms of obesity predisposes to atopic dermatitis. *Front Immunol*, 2024 Nov; 15: 1473105. <https://doi.org/10.3389/fimmu.2024.1473105>. Erratum in: *Front Immunol*. 2026 Apr 20;17:1850518. <https://doi.org/10.3389/fimmu.2026.1850518>
- Simonetti O, Bacchetti T, Ferretti G, Molinelli E, Rizzetto G, Bellachioma L, Offidani A.** Oxidative Stress and Alterations of Para-oxonases in Atopic Dermatitis. *Antioxidants*, 2021 Apr; 10(5): 697. <https://doi.org/10.3390/antiox10050697>
- van den Bogaard EH, Elias PM, Goleva E, Berdyshev E, Smits JPH, Danby SG, Cork MJ, Leung DYM.** Targeting Skin Barrier Function in Atopic Dermatitis. *J Allergy Clin Immunol Pract*, 2023 May; 11(5): 1335–1346. <https://doi.org/10.1016/j.jaip.2023.02.005>
- Xue Y, Zhang L, Chen Y, Wang H, Xie J.** Gut microbiota and atopic dermatitis: a two-sample Mendelian randomization study. *Front Med*, 2023 Jun; 10: 1174331. <https://doi.org/10.3389/fmed.2023.1174331>
- Yamamoto K, Yokoyama K, Matsukawa T, Kato S, Kato S, Yamada K, Hirota T.** Efficacy of prolonged ingestion of *Lactobacillus acidophilus* L-92 in adult patients with atopic dermatitis. *J Dairy Sci*, 2016 Jul; 99(7): 5039–5046. <https://doi.org/10.3168/jds.2015-10605>
- Zhang A, Silverberg JI.** Association of atopic dermatitis with being overweight and obese: a systematic review and metaanalysis. *J Am Acad Dermatol*, 2015 Apr; 72(4): 606–616.e4. <https://doi.org/10.1016/j.jaad.2014.12.013>