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Article

Skin Dysbiosis in Atopic Dermatitis and Psoriasis

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Abstract: The skin is a complex ecosystem containing bacteria, fungi and viruses that play a role in maintaining homeostasis, protecting against pathogens and immune training. A disruption of this balance that is, skin dysbiosis is increasingly recognised as a significant factor in inflammatory skin diseases, including atopic dermatitis (AD) and psoriasis. This review includes 20 sources from the studies and reviews published between 2017 and 2026, focusing on the skin microbiome, mycobiome, the skin–gut axis, and microbiome-targeted interventions for AD and psoriasis.

Keywords: Skin Dysbiosis, Atopic Dermatitis, Psoriasis

Introduction

The skin is a complex ecosystem containing bacteria, fungi and viruses, which play a role in maintaining homeostasis, protecting against pathogens and immune education. A disruption of this balance that is, skin dysbiosis is increasingly recognised as a significant factor in inflammatory skin diseases, including atopic dermatitis (AD) and psoriasis [1].

Whilst the clinical manifestations of AD and psoriasis overlap to some extent, their immunological axes and microbial profiles differ. AD is primarily associated with barrier dysfunction and a Th2-type inflammation, whereas psoriasis is more commonly associated with the Th17 axis and keratinocyte hyperproliferation [2].

The aim of this study is to present an IMRAD review of the differences and similarities in cutaneous dysbiosis in these two dermatoses, with a focus on microbiota composition, pathogenetic mechanisms and therapeutic prospects [3].

Materials and Method

The review includes 20 sources selected from the studies and reviews published between 2017 and 2026, focusing on the skin microbiome, the mycobiome, the skin–gut axis, and microbiome-

targeted interventions for atopic dermatitis and psoriasis. Priority was given to systematic reviews, large-scale comparative studies and papers containing data on mechanisms or therapies.

The analysis was structured around three areas:

1. Characteristics of the skin's microbial composition in atopic dermatitis and psoriasis;
2. Pathogenetic mechanisms linking dysbiosis to inflammation;
3. Evidence on therapeutic modulation of the microbiome.

Results

In atopic dermatitis, the most consistent finding is a reduction in bacterial diversity, particularly in the affected areas, and the dominance *of Staphylococcus aureus*. *S. aureus* is detected on the affected skin in more than 90 per cent of patients with AD, and its overabundance correlates with disease severity and flare-ups. At the same time, the abundance of protective commensals, including *Cutibacterium* and coagulase-negative staphylococci, decreases, thereby weakening resistance to colonization [4],[5].

Psoriasis presents a different picture: the skin microbiome differs in composition from that of healthy skin, with higher diversity and heterogeneity, as well as reduced community stability, being more commonly reported. In psoriatic lesions and on unaffected skin of patients, a relative enrichment *of S. aureus* and a deficiency of *S. epidermidis* and *Propionibacterium/Cutibacterium acnes* were detected [6]. A large-scale comparative study showed that atopic dermatitis is characterised by the dominance of a single species, whereas in psoriasis, coexisting microbial communities with weaker links to the host transcriptome predominate [7],[8].

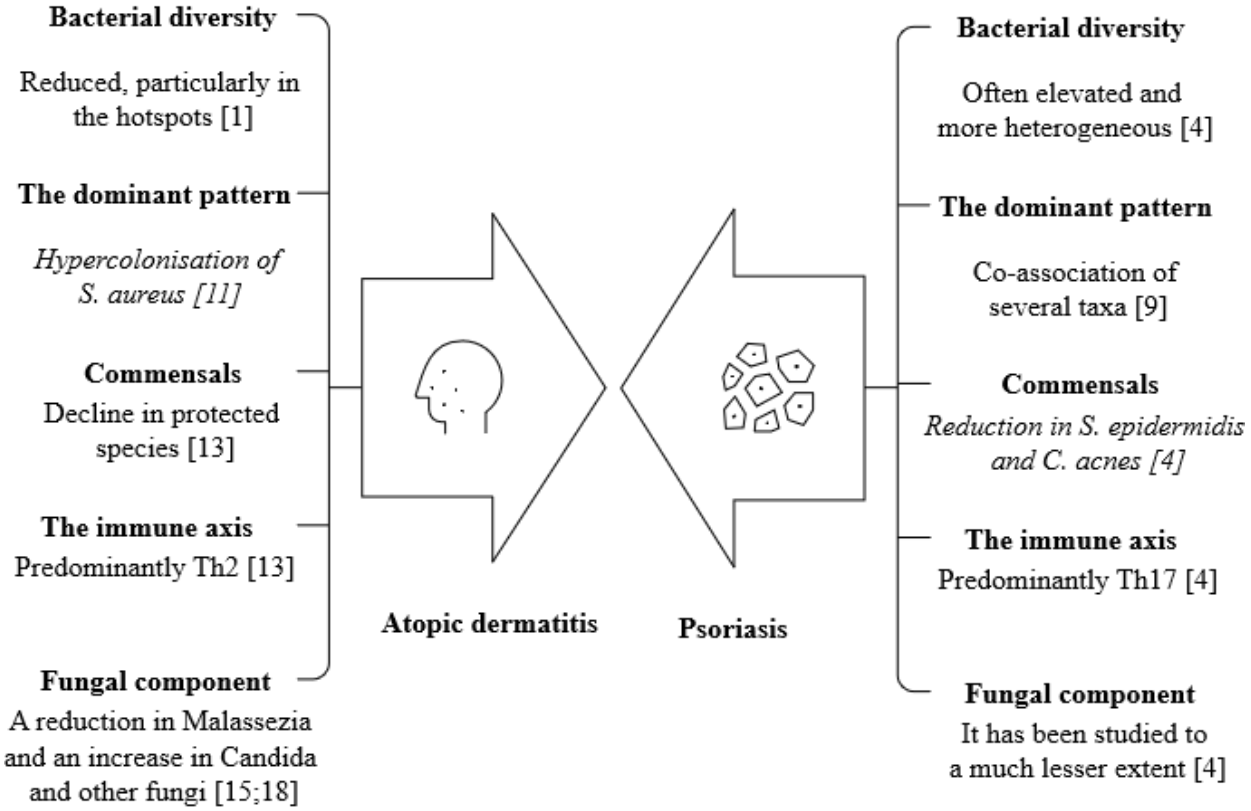

Table 1. Comparison of microbial characteristics in atopic dermatitis and psoriasis

In AD, the virulence *of S. aureus* is significant not only in quantitative but also in qualitative terms. The Agr quorum-sensing system induces the expression of toxins, including δ-toxin, which exacerbates type 2 inflammation and barrier damage [9]. The secretion of toxins and proteases by *S.*

aureus further exacerbates the impairment of the epidermal barrier and disrupts the already altered immune response. It has been shown separately that early-life colonisation by *S. aureus* strains with a functional Agr system is associated with a higher risk of subsequent development of AD [10],[11].

Experimental data on psoriasis point to the possible immunological significance of these microbial shifts. In a mouse model, neonatal colonisation *by S. aureus* induced marked Th17 polarisation, whereas *S. epidermidis* did not produce such an effect. This supports the hypothesis that the loss of immunoregulatory commensals may facilitate pathogen-mediated maintenance of inflammation via the Th17 axis [12],[13].

The fungal component of AD has been less well studied than the bacterial component, but a systematic review and a longitudinal study confirm that severe AD is associated with a depletion *of Malassezia* and an increase in *non-Malassezia* fungi, including *Candida*. A positive correlation between *Candida* and *Staphylococcus* suggests possible intermicrobial interactions that exacerbate inflammation [14],[15].

Discussion

A comparison of the data shows that dysbiosis in atopic dermatitis and psoriasis differs qualitatively. Atopic dermatitis is characterised by a low-diversity microbiome dominated by *S. aureus*, whereas psoriasis is more commonly associated with a more complex and unstable reorganisation of the microbial community [16]. This is consistent with the differences between Th2 inflammation in atopic dermatitis and Th17 inflammation in psoriasis. However, the question of causality remains open. Several reviews explicitly state that it is as yet unclear whether dysbiosis is the root cause of the disease or a secondary consequence of inflammation and barrier dysfunction. Nevertheless, animal data and clinical-microbiological observations make a causal contribution of dysbiosis in AD more likely than in psoriasis [17]. The skin–gut axis is of particular interest. Reviews and systematic data indicate that gut dysbiosis is also associated with AD and psoriasis, whilst in psoriasis a reduction in Actinobacteria and an increase in Firmicutes have been described, correlating with inflammatory markers. In AD, a reduction in beneficial gut bacteria, including *Bifidobacterium* and *Lactobacillus*, has been reported, which is associated with flare-ups and disease severity [18].

Therapeutic prospects

Microbiome-based approaches for AD appear to be the most advanced, but clinical data remain mixed. Therapies using commensal staphylococci have shown a reduction in *S. aureus* colonisation and an improvement in clinical indices in early trials. Commensals such as *S. hominis*, *S. lugdunensis* and *S. epidermidis* are capable of enhancing innate antimicrobial defence against *S. aureus* [19].

- Topical bacteriotherapy reduced *S. aureus* colonisation and improved the EASI in small RCTs.
- Indirect methods, including UVB and anti-inflammatory therapy, may also alter the microbiome and reduce the pathogen load.
- New platforms include prebiotics, probiotics, postbiotics, phage therapy and engineered live biotherapeutics.

For psoriasis, microbiome-targeted interventions have been studied to a lesser extent. There is evidence of the potential impact of probiotics and other methods of modulating the microbiota, but the evidence base is as yet less robust, and clinical effects are difficult to distinguish from the immunomodulatory effects of concomitant therapy. In a clinical context, personalised treatment decisions should be made in consultation with a registered doctor; the information provided here reflects the research literature and does not constitute individual medical advice [20].

Conclusion

Skin dysbiosis in atopic dermatitis and psoriasis is indeed an important part of the pathogenesis, but manifests differently: in atopic dermatitis, a loss of diversity and the expansion *of S. aureus* predominate, whilst in psoriasis, there is a more heterogeneous and less stable community

restructuring with a probable link to the Th17 axis. The question of which comes first – inflammation or dysbiosis – remains open; however, the microbiome has already become a promising therapeutic target for both conditions.

REFERENCES

[1] R. D. Bjerre, J. Bandier, L. Skov, L. Engstrand, and J. D. Johansen, "The role of the skin microbiome in atopic dermatitis: A systematic review," *Br. J. Dermatol.*, vol. 177, no. 5, pp. 1272–1278, 2017, doi: 10.1111/bjd.15390.

[2] L. Bonzano, F. Borgia, R. Casella, A. Miniello, E. Nettis, and S. Gangemi, "Microbiota and IL-33/31 axis linkage: Implications and therapeutic perspectives in atopic dermatitis and psoriasis," *Biomolecules*, vol. 13, no. 7, Art. no. 1100, 2023, doi: 10.3390/biom13071100.

[3] S. Carmona-Cruz, L. Orozco-Covarrubias, and M. Sáez-de-Ocariz, "The human skin microbiome in selected cutaneous diseases," *Front. Cell. Infect. Microbiol.*, vol. 12, Art. no. 834135, 2022, doi: 10.3389/fcimb.2022.834135.

[4] H.-W. Chang, D. Yan, R. K. Singh, J. Liu, X. Lu, D. Uçmak, K. M. Lee, L. Afifi, D. Fadrosh, J. M. Leech, K. S. Vasquez, M. Lowe, M. D. Rosenblum, T. Scharschmidt, S. Lynch, and W. Liao, "Alteration of the cutaneous microbiome in psoriasis and potential role in Th17 polarization," *Microbiome*, vol. 6, no. 1, Art. no. 154, 2018, doi: 10.1186/s40168-018-0533-1.

[5] Y. Chen, C. Peng, L. Zhu, J. Wang, Q. Cao, X. Chen, and J. Li, "Atopic dermatitis and psoriasis: Similarities and differences in metabolism and microbiome," *Clin. Rev. Allergy Immunol.*, vol. 66, no. 3, pp. 294–315, 2024, doi: 10.1007/s12016-024-08995-3.

[6] B. De Pessemier, L. Grine, M. Debaere, A. Maes, B. Paetzold, and C. Callewaert, "Gut–skin axis: Current knowledge of the interrelationship between microbial dysbiosis and skin conditions," *Microorganisms*, vol. 9, no. 2, Art. no. 353, 2021, doi: 10.3390/microorganisms9020353.

[7] A.-L. Demessant-Flavigny, S. Connétable, D. Kerob, M. Moreau, L. Aguilar, and A. Wollenberg, "Skin microbiome dysbiosis and the role of *Staphylococcus aureus* in atopic dermatitis in adults and children: A narrative review," *J. Eur. Acad. Dermatol. Venereol.*, vol. 37, suppl. 5, pp. 3–17, 2023, doi: 10.1111/jdv.19125.

[8] R. Fölster-Holst, "The role of the skin microbiome in atopic dermatitis—Correlations and consequences," *J. Dtsch. Dermatol. Ges.*, vol. 20, no. 5, pp. 571–577, 2022, doi: 10.1111/ddg.14709.

[9] N. Fyhrquist, G. Muirhead, S. Prast-Nielsen, M. Jeanmougin, P. Olah, T. Skoog, G. Jules-Clement, M. Feld, M. Barrientos-Somarribas, H. Sinkko, E. H. van den Bogaard, P. L. J. M. Zeeuwen, G. Rikken, J. Schalkwijk, H. Niehues, W. Däubener, S. K. Eller, H. Alexander, D. Pennino, S. Suomela, I. Tessas, E. Lybeck, A. M. Baran, H. Darban, R. S. Gangwar, U. Gerstel, K. Jahn, P. Karisola, L. Yan, B. Hansmann, S. Katayama, S. Meller, M. Bylesjö, P. Hupé, F. Levi-Schaffer, D. Greco, A. Ranki, J. M. Schröder, J. Barker, J. Kere, S. Tsoka, A. Lauerma, V. Soumelis, F. O. Nestle, B. Homey, B. Andersson, and H. Alenius, "Microbe-host interplay in atopic dermatitis and psoriasis," *Nat. Commun.*, vol. 10, no. 1, Art. no. 4703, 2019, doi: 10.1038/s41467-019-12253-y.

[10] C. Hülpüsch, R. Rohayem, M. Reiger, and C. Traidl-Hoffmann, "Exploring the skin microbiome in atopic dermatitis pathogenesis and disease modification," *J. Allergy Clin. Immunol.*, vol. 154, no. 1, pp. 31–41, 2024, doi: 10.1016/j.jaci.2024.04.029.

[11] L. F. Koh, R.-Y. Ong, and J. E. Common, "Skin microbiome of atopic dermatitis," *Allergol. Int.*, vol. 71, no. 1, pp. 31–39, 2022, doi: 10.1016/j.alit.2021.11.001.

[12] M. Mazur, H. Tomczak, M. Lodyga, R. Czajkowski, R. Żaba, and Z. Adamski, "The microbiome of the human skin and its variability in psoriasis and atopic dermatitis," *Adv. Dermatol. Allergol.*, vol. 38, no. 2, pp. 205–209, 2021, doi: 10.5114/ada.2021.106197.

[13] H. Okamoto and Y. Nakamura, "Dysbiosis in the pathogenesis of atopic dermatitis," *J. Dermatol.*, vol. 53, no. 3, pp. 388–398, 2026, doi: 10.1111/1346-8138.70191.

[14] H. Okamoto, S. Li, and Y. Nakamura, "The role of skin dysbiosis and quorum sensing in atopic dermatitis," *JID Innov.*, vol. 5, no. 4, Art. no. 100377, 2025, doi: 10.1016/j.xjidi.2025.100377. The publisher/PubMed records confirm the three authors, volume 5, issue 4, article 100377, and DOI.

[15] T. Rong, R. Li, and R. Wang, "Dysbiosis of skin mycobiome in atopic dermatitis," *Mycoses*, vol. 65, no. 3, pp. 285–293, 2022, doi: 10.1111/myc.13402.

[16] A. Rušanac, Z. Škibola, M. Matijašić, H. Č. Paljetak, and M. Perić, "Microbiome-based products: Therapeutic potential for inflammatory skin diseases," *Int. J. Mol. Sci.*, vol. 26, no. 14, Art. no. 6745, 2025, doi: 10.3390/ijms26146745. The publisher record confirms the five-author list, volume 26, issue 14, article 6745, and publication date of July 14, 2025.

[17] M. F. Sanchez-Lopez, P. A. Barrero-Caicedo, H. M. Olmos-Carval, A. F. Torres-Medina, and J. P. Alzate-Granados, "Relationship between skin and gut microbiota dysbiosis and inflammatory skin diseases in adult patients: A systematic review," *The Microbe*, vol. 7, Art. no. 100342, 2025, doi: 10.1016/j.microb.2025.100342.

[18] B. Schmid, A. Künstner, A. Fähnrich, E. Bersuch, P. Schmid-Grendelmeier, H. Busch, M. Glatz, and P. P. Bosshard, "Dysbiosis of skin microbiota with increased fungal diversity is associated with severity of disease in atopic dermatitis," *J. Eur. Acad. Dermatol. Venereol.*, vol. 36, no. 10, pp. 1811–1819, 2022, doi: 10.1111/jdv.18347.

[19] N. E. van Hout, G. Nevot, P. A. M. Jansen, A. A. Mannan, P. L. J. M. Zeeuwen, D. J. Hijnen, R. J. Tanaka, M. Güell, and E. H. van den Bogaard, "From prebiotics to engineered microbes: Microbe-inspired treatments for atopic dermatitis," *Br. J. Dermatol.*, vol. 194, no. 3, pp. 420–428, 2026, doi: 10.1093/bjd/ljaf451.

[20] X.-E. Zhang, P. Zheng, S.-Z. Ye, X. Ma, E. Liu, Y.-B. Pang, Q.-Y. He, Y.-X. Zhang, W.-Q. Li, J.-H. Zeng, and J. Guo, "Microbiome: Role in inflammatory skin diseases," *J. Inflamm. Res.*, vol. 17, pp. 1057–1082, 2024, doi: 10.2147/JIR.S441100.