doi:10.36849/JDD.M0922

Dysbiosis, (Barrier) Dysfunction, and Dermatoses: A Chicken-and-Egg Dilemma

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Acne vulgaris (AV) and rosacea are two of the most common dermatoses diagnosed and managed by dermatologists.[1,2] Despite this and our improved understanding of the unique pathogenesis of each, there has been little focus on general skin care and how it may affect physiologic functioning of the epidermis until recently. Under optimal homeostatic conditions, the skin generally provides a physical, chemical, and immune layer that deftly balances moisture, temperature, and biochemical substrates.[3] These factors create biomes specific to different areas of the body including the skin, teeth, and internal/external mucosa, where various microorganisms (including bacteria, viruses, and fungi) can thrive.[3] Given that bacteria alone outnumber human cells more than 10 to 1 and have over 100 times more genes than humans, the microbiome, and the factors that support it, cannot be ignored.[4] While alterations in epidermal barrier function and the disruption of the cutaneous microbiome(CM) have been studied in a few dermatoses, most notably atopic dermatitis, these may not be the first to come to mind when treating AV and/or rosacea.

One of the most prominent pillars of AV pathogenesis is the follicular colonization with *Cutibacterium acnes*. Although typically a commensal organism, virulent strains of *C. acnes*—phylotype IA1 in particular—have been associated with AV and worsening acne severity.[5] With the onset of puberty, there is also a notable change in epidermal barrier function as measured by increased transepidermal water loss (TEWL).[6] TEWL is further compounded in patients with AV, which may be up to 50% greater depending on acne severity.[7]

Rosacea pathogenesis is thought to be a complex, interconnected web of dysregulated innate and adaptive immunity combined with hyperreactive neurovasculature. In practice, many of the secondary criteria for rosacea (eg, burning, stinging, edema, dryness)[8] are also symptoms of epidermal barrier deficiency. Multiple studies have demonstrated that the addition of physiologic moisturizers and/or cleansers to standard-of-care regimens may augment treatment outcomes for mild-moderate disease.[9-13] Unlike AV in which one organism has been implicated, rosacea has been associated with multiple organisms including bacteria (eg, *Streptococcus epidermidis, Bacillus oleronius*, and *Helicobacter pylori*), *Demodex* species, a decreased abundance of *C. acnes*, and even the general overgrowth of bacteria in the small intestine (SIBO).[14] Although a singular perpetrator has not been conclusively identified, multiple studies have found dysbiosis within the rosacea cutaneous (and enteric) microbiome.

Both AV and rosacea have varying degrees of epidermal barrier dysfunction, which may be further altered by topical and even systemic therapies. Similarly, AV and rosacea have unique cutaneous microbiomes and microbiome derangement that can be both directly and indirectly affected by common treatment options, including the indiscriminate use of broad-spectrum antibiotics. With derangements at baseline that may be further exacerbated by current therapeutic paradigms, discussion of proper skin care is close to becoming a cornerstone of care. Every acne and rosacea encounter should endeavor to discuss key factors in moisturizers and cleansers: physiologic to slightly acidic pH; scent-/fragrance-free; emollients to soften the skin; humectants, such as glycerin and hyaluronic acid, to attract water; occlusives like petrolatum to seal them in, and proper lipid composition to potentially supplement those deficient in disease states.

A final interesting concept to consider: the chicken and the egg. A healthy microbiome is dependent on a functioning epidermis with adequate moisture and nutrients for survival and balance. In turn, a healthy microbiome can quell virulent organisms and provide a suitable pH to optimize biochemical and immunological epidermal processes. In AV and rosacea, there are data to suggest that both dysbiosis and epidermal barrier dysfunction have roles to play. But does one precede (or even supersede) the other? And, more importantly, if dermatologists have the potential to repair and even restore both, how much might it matter in clinical practice? While research continues to elucidate the role of both dysbiosis and barrier dysfunction in acne and rosacea, it behooves us to employ quality skin care to restore and repair so the chicken and egg concept is irrelevant. No visit for either disease is complete until quality skin care has been discussed.

REFERENCES

1. Landis ET, Davis SA, Taheri A, Feldman SR. Top dermatologic diagnoses by age. *Dermatol Online J.* 2014;20(4). doi:10.5070/d3204022368.
2. Peck GM, Roberson FA, Feldman SR. Why do patients in the united states seek care from dermatologists? *Dermatol Ther.* 2022;12(4):1065-1072. doi:10.1007/s13555-022-00706-0.
3. Baldwin HE, Bhatia ND, Friedman A, Eng RM, Seite S. The role of cutaneous microbiota harmony in maintaining a functional skin barrier. *J Drugs Dermatol.* 2017;16(1):12-18.
4. NIH HMP Working Group, Peterson J, Garges S, et al. The NIH Human Microbiome Project. *Genome Res.* 2009;19(12):2317-2323. doi: 10.1101/gr.096651.109.
5. Cunliffe WJ, Gould DJ. Prevalence of facial AV in late adolescence and in adults. *Br Med J.* 1979;1(6171):1109-1110. doi:10.1136/bmj.1.6171.1109
6. Pappas, A., Dunn, K., Cula, G.O., et al. Barrier and microbiome changes in the facial skin of children as they approach puberty. Presented at: 26th European Academy of Dermatology and Venerology (EADV) Congress: P006513; September 17, 2017; Geneva Switzerland.
7. Yamamoto A, Takenouchi K, Ito M. Impaired water barrier function in AV. *Arch Dermatol Res.* 1995;287(2):214-218. doi:10.1007/BF01262335
8. Gallo RL, Granstein RD, Kang S, et al. Standard classification and pathophysiology of rosacea: The 2017 update by the National Rosacea Society Expert Committee. *J Am Acad Dermatol.* 2018;78(1):148-155. doi:10.1016/j.jaad.2017.08.037.
9. Pelle MT, Crawford GH, James WD. Rosacea: II. Therapy. *J Am Acad Dermatol.* 2004;51(4):499-514. doi:10.1016/j.jaad.2004.03.033
10. Draelos ZD. Cosmetics in acne and rosacea. *Semin Cutan Med Surg.* 2001;20(3):209-214. doi:10.1053/sder.2001.27556
11. Del Rosso JQ. The use of moisturizers as an integral component of topical therapy for rosacea: clinical results based on the assessment of skin characteristics study. *Cutis.* 2009;84(2):72-76.
12. Subramanyan K. Role of mild cleansing in the management of patient skin. *Dermatol Ther.* 2004;17 (Suppl 1):26-34. doi:10.1111/j.1396-0296.2004.04s1003.x
13. Draelos ZD, Green BA, Edison BL. An evaluation of a polyhydroxy acid skin care regimen in combination with azelaic acid 15% gel in rosacea patients. *J Cosmet Dermatol.* 2006;5(1):23-29. doi:10.1111/j.1473-2165.2006.00219.x
14. Holmes AD. Potential role of microorganisms in the pathogenesis of rosacea. *J Am Acad Dermatol.* 2013;69(6):1025-1032. doi:10.1016/j.jaad.2013.08.006

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