SKIN

SHORT COMMUNICATION

Beyond Acne Vulgaris: Role of Cutibacterium Acnes in Atopic Dermatitis and Psoriasis

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Dear Editor,

Cutibacterium acnes is a commensal bacterium and important component of the normal skin microbiome[1]. *C. acnes* has been linked to the development of acne vulgaris (AV) attributable to its effects on sebum production and promotion of inflammatory cascades.[1] Recent advances have shown that AV results from disruptions in the abundance of *C. acnes* phylotypes on the skin, leading to significant changes in homeostasis usually maintained by normal skin microbiota.[2] Many studies have reviewed its role in AV, but limited work has been done to describe how *C. acnes* may impact other common dermatologic conditions. Thus, the goal of this review was to discuss the role of *C. acnes* in both atopic dermatitis (AD) and psoriasis.

A comprehensive search was performed using PubMed, Web of Science, and Embase using the terms "Cutibacterium acnes" or "Propionibacterium acnes" AND "Atopic dermatitis" or "Eczema" or "Psoriasis" according to PRISMA guidelines. Three researchers performed full text review and data extraction (MG,AF,NK), with any discrepancies settled by a fourth (HM). All English experimental studies were included.

Thirteen studies met inclusion criteria (**Table 1**). Lesional skin in patients with AD was shown to have higher concentrations of *Staphylococcus aureus* and lower concentrations of *C. acnes* when compared to healthy individuals (n=9 studies). Additionally, AD lesions were shown to host significantly different *C. acnes* phylotypes than healthy skin (n=1 studies). Furthermore, *C. acnes* colonization was less in adult AD lesions compared to healthy control adults, but no difference was seen in children (n=1). Lesional skin in patients with psoriasis was shown to have higher concentrations of *Cornybacterium sp.* and lower concentrations of *C. acnes* than non-lesional skin of psoriasis patients and skin in healthy controls (n=2).

Overall, the relative concentration of *C. acnes* in both AD and psoriatic lesions was shown to be decreased when compared to healthy skin. Although the exact mechanism remains unknown, a relative decrease in *C. acnes* concentration likely allows for the overgrowth of *S. aureus* and *Cornybacterium sp.*. One hypothesis is that *C. acnes* inhibits growth of other bacterial species through propionic acid, its fermentation product.[3]

Table 1. Bacteria in Disease Affected Skin vs. Comparators of Included Studies.

Study Number	Sample Size (Patients)	Comparator [lesional (L), non-lesional (NL), healthy control (C)]	Bacterial Concentration in Disease Affected Skin vs. Comparator[a]
1[b]	AD: n=1 Control: n=2	L vs. C	• Decreased: *C. acnes:* Vaccination with *P. acnes* successfully prevented clinical manifestations in the skin of AD mice)
2	AD: n=4 Control: n=10	L vs. C	• Different strains of *C. acnes*: 13/28 strains from AD patients were ST6 strain; only 1/22 from healthy patients were classified as ST6 strain
3	AD: n=128 Control: n=68	L vs. C	• Decreased: *C. acnes*
4	AD: n=7 Control: n=7	L vs. NL	• Increased: *S. aureus* • Decreased: *C. acnes*
5	AD: n=34 Control: n=54	L vs. C	• Increase: *S. aureus* • Decreased: *C. acnes*
6	AD: n=10 Control: n=5	L vs. C	• Increased: phages PHL041/PHL092, *S. aureus* • Decreased: *C. acnes*
7	AD: n=39 Control: n=15	L vs. C	• Increased: *S. aureus, S. epidermis* and *S. capitis* in dermotype B • Decreased: *C. acnes, Dermacoccus*, and *Methylobacterium*
8	AD: n=17 Control: n=9	L vs. C	• Increased: *S. aureus* • Decreased: *C. acnes*
9	Control: n=9 AD: n=20	L vs. NL and C	• The most lantibiotic synthesis genes were matched to *C. acnes* on healthy individuals, followed by nonlesional AD skin, followed by lesional AD skin
10	Psoriasis: n=119 AD: n=82 Control: n=115	L vs. C	• Increase*: S. aureus* (AD), *Cornyebacterium* (psoriasis) • Decreased: *C. acnes* (AD and psoriasis)
11	Psoriasis: n=26 Control: n=28	L and NL vs. C	• Increased: *S. aureus* • Decreased: *S. epidermis and C. acnes*
12	Psoriasis: n=114 Control: n=114	L vs. NL	• Decreased: *C. acnes* (vs. non-lesional)
13	Control: n=16 Psoriasis: n=16	L vs. NL and C	• Increased: *Cornyebacterium* • Decreased: *C. acnes* (vs. non-lesional and control)

[b]Animal study: mice.

Additionally, in mouse models, vaccination against *C. acnes* induced Th-1 type cytokines that improved AD symptoms, suggesting *C. acnes* plays an important role in immune-mediated maintenance of the skin microbiome.[4]

Much work remains to be done on the implications of *C. acnes* and its relationship with other commensal skin flora, but future therapies may benefit from altering the dermal microbiome. Probiotics have been studied for their potential treatment of AV and AD, with bacteria such as *Staphylococcus epidermidis* creating succinic acid by-products which slows the growth of other bacterial species.[5] Future research should investigate this relationship further. Additionally, the treatment of AV with oral antibiotics is common practice, yet little work investigating the effects of antibiotics on AV patients with concurrent AD or psoriasis exists.

Limitations of this study include small sample size and heterogeneity of methodology across studies. Furthermore, the role of additional bacterial and fungal species were not evaluated in this review.

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