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Staphylococcus aureus in atopic dermatitis: a diverse problem?

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Linked Article: Clausen *et al. Br J Dermatol* 2017; **177**: 1394–1400.

Skin colonization with *Staphylococcus aureus* and eczema/atopic dermatitis (AD) are inextricably linked. Through microbiome analysis the dynamic changes in *S. aureus* colonization occurring during a flare cycle have been elegantly shown.[1] However, the question remains: does *S. aureus* 'drive' or 'follow' flares of AD? The answer could well be both, starting with the defective skin barrier exhibited by patients with AD creating a more favourable environment for *S. aureus* growth, and then progressing to the potentiation of proallergic inflammation by established colonies.[2]

In this issue of the *BJD*, Clausen *et al.*[3] present evidence on the relationship between skin colonization by *S. aureus* and *FLG* gene mutations, which result in loss of functional filaggrin and predispose to a defective skin barrier. A cohort of 101 adult patients with AD was recruited, comprising 31 *FLG* mutation carriers. *Staphylococcus aureus* skin colonization was found in 77% of the carriers compared with only 44% of the noncarriers. This may suggest an increased risk of *S. aureus* colonization in carriers, or simply reflect the established association between both factors and AD severity.

The clonal diversity of *S. aureus* was also investigated. Far from being a homogeneous population, *S. aureus* exists as diverse clones with distinct pathogenic properties derived from the numerous clonal lineages.[4] It follows that certain clones may be more adept at triggering or exacerbating AD than others. More than 90% of all patients were colonized by only one clone across all test sites, although a total of 18 clones were detected in the cohort. The diversity of clones differed from that of healthy populations, suggesting disease specificity. There was no association between any of the clones and AD severity; however, the sample size was too small following stratification for robust analysis, and such associations have been reported previously.[5] Of the clones identified, CC1 was the most common, being found in 22% of the colonized patients, and markedly more prevalent in *FLG* mutation carriers than noncarriers.

During infection, *S. aureus* growth follows a clonal expansion pattern, whereby inhospitable conditions, including drug treatments, lead to selection of the clones best equipped to survive.[4] Certain clones, such as CC1, could therefore be better equipped to capitalize on the skin changes resulting from a *FLG* mutation. An increase in skin-surface pH is one such change already associated with *S. aureus* growth.[6] Perhaps by characterizing the specific *S. aureus* clones colonizing the skin of patients with AD, and by understanding how skin defects favour their growth, new strategies could be developed to restrict their growth and avert their contribution to proallergic inflammation.

Conflicts of interest

None declared.

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Real-world effect of biologics on quality of life in psoriasis

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A well-planned and well-run clinical registry is like a goldmine: just waiting for the surveyors to find another rich seam.

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