

indicating more severe disease. When used by dermatologists, the CDASI provides valid and reliable measures of cutaneous disease activity and damage that are sensitive to change over time, making it a suitable instrument for longitudinally monitoring cutaneous disease in individuals with DM.^{2–5}

In this issue of the *BJD*, Tiao *et al.*⁶ assessed the reliability of the CDASI when used by dermatologists, rheumatologists and neurologists, the latter two groups representing specialists who also routinely care for patients with DM and participate in DM clinical trials. When independently evaluating individuals with DM, no significant differences in mean reported CDASI activity scores were found among the three groups. CDASI damage scores were significantly higher when reported by rheumatologists, but no difference was found between dermatologists and neurologists. The inter-rater reliability of CDASI activity between dermatologists and rheumatologists was good, indicating that the two physician groups scored DM cutaneous activity similarly. However, this same reliability metric was less robust for neurologists. Inter-rater reliability of CDASI damage was excellent for all groups. The intrarater reliability for CDASI activity was excellent for all physicians, indicating reproducibility when used by an individual examiner, while intrarater reliability of damage scores was excellent only for dermatologists and rheumatologists. Despite greater variability, Physician's Global Assessment activity and damage scores correlated with CDASI and were similar among the three groups. The authors conclude that the CDASI is a reliable and valid outcome instrument for measuring cutaneous DM disease activity and damage when used by dermatologists and rheumatologists, with more limited utility for neurologists.

Until now, the reliability and validity of the CDASI has been demonstrated for dermatologists alone. The results from this study⁶ extend the use of this instrument to nondermatologists and represent a meaningful step towards ensuring the inclusion of skin disease outcomes with respect to treatment response in all future DM-centred investigations, whether led by dermatologists or not. This and other similar efforts⁷ provide a platform for the systematic evaluation of novel and repurposed therapies for rare autoimmune and inflammatory diseases that involve the skin. Expanding the use of the CDASI to nondermatologists will undoubtedly increase the number of rigorously designed studies that include a focus on skin outcomes and will thereby serve to enhance the data available to guide treatment of the frequently refractory cutaneous manifestations of DM.

Conflicts of interest

None declared.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

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Atopic dermatitis and gut microbiota

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Linked Article: Nowrouzian *et al.* *Br J Dermatol* 2017; **176**:439–445.

During the past decades, we have learnt that the interplay between the environmental microflora and the immune system is essential for educating the immune system and thus for our survival.^{1,2} This interplay takes place at the barriers of the body (skin, lung and gut). It is probable that this 'education' mostly occurs in early life. It has been described that a lower diversity of the gut flora or an imbalance in the composition of the gut microbiome are associated with development of allergy.³ In a similar way it has been shown that a greater diversity of skin commensals improves the function of the immune system, while a decreased diversity is associated with disease, e.g. flare-ups of atopic dermatitis (AD), when colonized with superantigen (SAg)-producing *Staphylococcus aureus*.⁴ Although much interest has focused on the connection between the gut microbiome and the development of allergy we still today do not understand the relationship.

Establishment of the microbiome of the gut, mucous membranes and skin starts immediately following birth or even

before,⁵ and the mode of birth, either vaginal or by caesarean section, affects the newborn's initial microbiota. The mother's vaginal and faecal flora are usually assumed to be the main source of bacteria; however, the origin of *S. aureus* may also be external, thus not only obtained from the parents but also from hospital staff members.⁶ *Staphylococcus aureus* produces a wide variety of virulence factors, including surface-associated adhesins such as MSCRAMMs (microbial surface components recognizing adhesive matrix molecules) and secreted SAGs, cytolytins and other exoenzymes. The SAGs comprise staphylococcal enterotoxins (SEs), toxic shock syndrome *S. aureus* toxin-1 and SE-like SAGs, and the enterotoxin gene cluster (*egc*) encodes the most prevalent SAGs among *S. aureus* strains. However, a difference in the prevalence of *egc* genes between nasal carriage and invasive (i.e. bacteraemic) isolates among adults has been reported⁷ that might indicate the significance of these SAGs in colonization. Nevertheless, the presence of the *egc* cluster is firmly linked to specific clonal complexes of *S. aureus* and these clonal lineages may harbour specific sets of genes that are required for successful colonization.

The multifactorial pathogenesis of AD is still somewhat of an enigma. There is a multigenetic basis, an impaired barrier function, a deviation of the innate immune response, as well as changes in the adaptive response, and there are several well-known environmental risk factors.^{8–10} Accumulated data indicate that AD is primarily due to the impaired diffusion barrier function associated with inflammation, increased penetration of irritants and allergens, and deviating innate response. Sensitization will thus be a secondary phenomenon. The complexity of AD pathogenesis and the possibility of clonal diversity among *S. aureus* could be one explanation of why the connection between gut flora and eczema development has not been fully established.

Staphylococcus aureus is often found on the skin in AD, mostly on inflamed skin,¹¹ and flares of AD are associated with *S. aureus* SAGs.⁴ In this issue of the BJD, Nowrouzian et al.¹² report on their observational study, in which they looked more closely at commensal *S. aureus* strains in the gut microbiome during the first 18-month postpartum period along with concomitantly registered development of AD and allergy signs. Authors of the current study have previously suggested that commensal *S. aureus* in the gut microflora carrying combinations of SAGs stimulate and educate the immune system and thus modify the development of AD.¹³ In the current study,¹² there was found to be a negative correlation between AD development and the occurrence of certain adhesins and SAGs, supporting the idea that the composition of the gut microbiome (perhaps specific *S. aureus* strains) in early childhood has an impact on AD and allergy development. The results open the door for more mechanistic study designs as there is a rapid development of techniques for identifying and classifying microbes.

Conflicts of interest

None declared.

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A criteria-validated sun-exposure questionnaire to evaluate skin cancer prevention campaigns

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Melanoma and nonmelanoma skin cancer remain significant health and economic cost burdens in many countries.^{1,2} However, skin cancer incidence is now decreasing in younger age