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Psoriasis, gut and microbiome

DOI: 10.1111/bjd.18467

Linked Article: Hidalgo-Cantabrana et al. *Br J Dermatol* 2019; **181**:1287–1295.

‘All disease begins in the gut’, the phrase attributed to Hippocrates, may sound like an overstatement but empirical evidence has linked many chronic metabolic and immune disorders to gut malfunction. It is increasingly recognized that the microbiome can change or switch on a wide range of diseases including cancer, cardiometabolic diseases, allergies, skin diseases and obesity.¹ This is not surprising because recent advances in microbiome research have revealed that microbial cells outnumber human cells in our body by a ratio of around 10 : 1.

In this issue of the *BJD*, Hidalgo-Cantabrana et al. report on lower bacterial diversity and different relative abundance of bacterial taxa in patients with psoriasis.² They further suggest that gut microbiota dysbiosis could be one of the triggers in the manifestation of psoriasis in genetically predisposed individuals. The microbiome of patients with psoriasis has been explored and reported in earlier studies. The advantage of this study is the inclusion of a healthy cohort from the same geographical area to minimize the environmental impact. While the presence of gut dysbiosis appears to be common in most studies of psoriasis, the relative abundance/decrease of different genera varies, most likely due to sequencing methodologies and computational analysis.

Essentially, the 16S rRNA gene sequencing of gut microbiome represents only the first step of a scientific journey to reveal what kind of bacteria are in our gut; it provides a solid foundation for further scientific exploration and knowledge building. However, the clinical significance of organisms identified solely by 16S rDNA gene sequencing is unclear. In addition to viable cells, dormant and dead bacteria, as well as extracellular DNA, are targeted. The next essential step is to map the presence and quantity of live bacteria. This analysis may not necessarily yield the same results as 16S rRNA gene sequencing. Another limiting factor in this study is enrolling a patient cohort with established disease and a long clinical history (average of 25 years), making it difficult to establish any meaningful cause–effect relationship.

The ultimate goal of any clinical research is the translation of clinical discoveries into new diagnostics, treatment and preventive approaches. The gut microbiome is an attractive

therapeutic target because, theoretically, there is good amenability of the gut microbiome. Therapies could be based on environmental changes, diets, drugs or probiotic supplements. Evidence suggests that changing a diet dramatically leads to relatively quick gut microbiome changes, in which around 30–40% of the bacterial strains vary in their abundance.³ The big vision is to modulate or even prevent diseases via the microbiome alone or with other life factors. In this way, microbiome research is moving from an isolated offside position to become a beacon of hope with a lot of potential and possibilities.

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Conflicts of interest: none to declare.

CD8⁺ mycosis fungoides: a wolf in sheep's clothing?

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Linked Article: Doerschner et al. *Br J Dermatol* 2019; **181**:1296–1302.

In this issue of the *BJD*, Doerschner and colleagues report on a case of mycosis fungoides (MF) with involvement of the central nervous system (CNS). Complete and ongoing remission of CNS lesions for 17 months was achieved with systemic chemotherapy and autologous stem-cell transplantation (SCT). Interferon (IFN) alfa-2a was used successfully to treat the subsequent relapse confined to the skin.¹

Initially, the patient was timely diagnosed with advanced-stage MF of 5 months' duration restricted to the skin. Of note, the Prospective Cutaneous Lymphoma International Prognostic Index (PROCLIP) study has demonstrated a median diagnostic delay of 36 months in early-stage MF between first symptoms and initial diagnosis.² However, in the face of currently lacking biomarkers for predictable disease progression it is debatable whether patients will benefit from early diagnosis. Moreover, accurate patient education on their prognosis is challenging with 5-year survival rates ranging from 88% to 30% dependent