The epithelial barrier hypothesis has been just published.
This article introduces the ‘epithelial barrier hypothesis’, which proposes that the increase in epithelial barrier-damaging agents linked to industrialization, urbanization and modern life underlies the rise in allergic, autoimmune and other chronic conditions. Furthermore, it discusses how the immune responses to dysbiotic microbiota that cross the damaged barrier may be involved in the development of these diseases.
There has been a steep increase in allergic and autoimmune diseases, reaching epidemic proportions and now affecting more than one billion people worldwide. These diseases are more common in industrialized countries, and their prevalence continues to rise in developing countries in parallel to urbanization and industrialization. Intact skin and mucosal barriers are crucial for the maintenance of tissue homeostasis as they protect host tissues from infections, environmental toxins, pollutants and allergens. A defective epithelial barrier has been demonstrated in allergic and autoimmune conditions such as asthma, atopic dermatitis, allergic rhinitis, chronic rhinosinusitis, eosinophilic esophagitis, coeliac disease and inflammatory bowel disease. In addition, leakiness of the gut epithelium is also implicated in systemic autoimmune and metabolic conditions such as diabetes, obesity, multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus, ankylosing spondylitis and autoimmune hepatitis. Finally, distant inflammatory responses due to a ‘leaky gut’ and microbiome changes are suspected in Alzheimer disease, Parkinson disease, chronic depression and autism spectrum disorders.

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