Gut microbiota influence the development and function of different host defense mechanisms of innate and adaptive immunity

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Microbiome and Gluten
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Key insights
Intestinal dysbiosis is a hallmark of several immune disorders, including celiac disease. Increased levels of pathobionts may activate the proinflammatory pathways that trigger a breakdown in gluten tolerance and promote disease onset. This, in turn, further drives the gut microbial community towards a state of dysbiosis, resulting in a vicious circle that disrupts the host-microbiota homeostatic balance. The genes that predispose an individual towards celiac disease modulate the infant’s gut colonization process, highlighting the importance of the gut microbiome in tipping the balance towards health or disease.

Current knowledge
The gut microbiome plays a key role in the host’s defense mechanisms against pathogens and is also involved in the maturation, maintenance and function of the mucosal immune system. The microbiome is a highly complex entity that exhibits many redundant functions and is capable of evolving in response to genetic and environmental factors. Thus, although a large body of literature exists on the features of a healthy microbiome, it is difficult to pinpoint the microbial signature of celiac disease. Further research is needed to understand how intestinal bacteria interact within the host environment to promote celiac disease.

Practical implications
Although gluten is the main environmental trigger, the timing of disease onset following gluten exposure varies greatly between individuals, suggesting that other factors are involved. The pathogenic mechanisms that contribute towards celiac disease may be triggered by viral or bacterial infections that could amplify the response to gluten in predisposed individuals. In addition, individuals with celiac disease have perturbations in the gut microbial signature, such as abnormal levels of Bacteroides spp., Bifidobacterium spp. and Staphylococcus spp. Not surprisingly, celiac disease patients also show alteration in the levels of metabolites (i.e. short-chain fatty acids) and molecules (α-defensins, TLR2, TLR4) involved in host-microbial interactions.

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