Gut Microbiota and its Role in Immune Response to Helminth Infections

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The microbiota is an ecological community of commensal, symbiotic, and pathogenic microorganisms. The gut microbiota has evolved as a new important player in the pathophysiology of many intestinal diseases. Recently, special interest has been arisen on gut microbiota-host interaction because extensive evidence has shown that intestinal microbiota composition has an important role in human health and disease such as infectious diseases. Dysbiosis of the microbiota has been related to a plethora of infections and still increasing number of diseases. Studies have mostly focused on analyzing the associations between disease and an aberrant microbiota composition. Generally, the Th2 lymphocytes response is accepted as the responsible mechanism for most striking clinical features in helminth infection. The Th2 response results in the production of IL-4 (helps B cells switch from an IgM response to IgG, IgA, and, more importantly, IgE), IL-5 (an eosinophilopoietic cytokine), and IL-13 (associated with the hyper-reactivity of the asthmatic lung).

One of the most physiological functions that is affected by the composition of the microbiota is immune responses, but it has not been clearly demonstrated if these immune effects can occur in the absence of gut bacteria. Therefore, it is possible that certain elements of immune regulation by helminths is caused by alterations to the bacterial communities of the host.

In an experimental study on virus-helminth co-infection, helminths impaired antiviral immunity, but the immune regulation was still present in germ-free mice, indicating that regulation can occur independent of the microbiota. One possibility is that helminths (or the immune response to helminths) are the driving force favoring specific bacterial communities that are more immune regulatory. Future investigations would show that how these interactions may impact the regulation of the immune system.

Ethical Approval
Not applicable.

Conflict of Interest Disclosures
The authors declare that they have no conflict of interests.

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