The Microbiome and Immunity

Aziz Koleilat*

Associate Professor, Beirut Arab University, Senior pediatric consultant, Makassed University General Hospital, Lebanon

*Corresponding Author: Dr. Aziz Koleilat, Associate Professor, Beirut Arab University, Senior pediatric consultant, "Makassed University General Hospital" (MUGH), IRB Member; MUGH, Vice General Secretary, "Pan Arab Society, Pediatric Gastroenterology, Hepatology & Nutrition" (PASPGHAN), Lebanon.

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Understanding the concept of the microbiome and recognizing the important cellular players in gut tolerance and the immunomodulatory effects of microbiota on the mucosal immune system will lead to the core of the origin of many diseases.

Microbiota is "the ecological community of commensal, symbiotic and pathogenic microorganisms that literally share our body space" [1]. The human microbiome is all of our microbes’ genes. It can be regarded as a counterpart to the human genome (all of our genes). The genes in our microbiome are more than the genes in our genome [2].

The gastrointestinal tract is a mucosal surface that is continuously exposed to foreign microbes and antigens. Nevertheless, it is protected by a large variety of immunologically active structures and cells. Lamina propria dendritic cells are essential in determining if the response to a certain antigen will be inflammatory or anti-inflammatory in nature. The intestinal microbiome has a major effect on mucosal immune regulation [3].

A breakdown or disruption in the immune tolerance of the host will lead to unwanted inflammatory immune responses against innocuous antigens. This will result in allergic, inflammatory or infectious diseases. Preservation of immune tolerance plays an important role in protecting against allergic and autoimmune diseases, where microbiota plays the role [4].

The immunomodulatory function of commensal species serves as an appealing approach for developing new therapeutics for the treatment of allergic diseases [4]. The microbiome provides many nutritional benefits to the host such as by synthesizing vitamins and short chain-fatty acids. It is also necessary for the development and function of the intestinal immune system [3]. It was considered that healthy microbiota exists when there is balance between symbionts, commensal organisms, and pathobionts (dormant bad bacteria). Changes in this balance might cause dysbiosis, which was associated with several pathologies, such as inflammatory bowel diseases, atopy and infection [4].

Bibliography


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