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Microbiota and Rheumatoid Arthritis: Suggestions and Outlook Editorial Letter

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Background

Rheumatoid arthritis (RA) is a chronic autoimmune disorder where genetic and environmental factors contribute to the pathogenesis of disease.

RA leads to functional disability; it was found that 30% of patients are unable to work after 3 years of disease. The gut contains about 29% of all micro-organisms that live in and on the human body. Their function is to help in digestion, nutrition, energy production and vitamin synthesis. Gut microbiota are an environmental factor that regulate the immune system thus providing health benefits to their host.

Among the commensal community, mucosa associated commensal species such as segmented filamentous bacteria (SFB) are a minority regulating strongly the host immune response. Autoimmune diseases, especially RA, are involved in such modulation by gut microbiota. SFB drive autoimmune arthritis in the K/BxN mouse model of arthritis by triggering signals that instruct gut T helper 17 (Th17) cells to differentiate and help in autoantibody production.

T follicular helper (Tfh) cells contribute in the production of high-titer antibodies (Abs) through helping B cells to produce high affinity Abs and long-lived plasma cells by inducing somatic hypermutation and class switching.

The frequency of Tfh cells significantly increased in RA peripheral blood and this was accompanied by increased level of anti-citrullinated protein autoantibodies (ACPAs) indicating the possible involvement of Tfh cells in development of RA.

In a K/BxN RA mouse model, SFB interfered with Tfh differentiation in Peyer's Patches and supported antigen specific Tfh dissemination at systemic sites which could also support the contribution of both SFB and Tfh in RA pathogenesis.

The effects of long-term antibiotics on the microbiota are not yet understood and the emergence of resistance due to genetic alteration in microbiota, still to be investigated.

In conclusion, the crucial role of the microbiota in health and disease is well firmly established. Studies must be undertaken to better understand human microbiome in terms of composition, metabolic pathways and its role in RA. There is evidence that targeted modulation of the microbiota may improve clinical outcome in RA suggesting that personalized treatment strategies based on patient microbiome profiles may increase drug efficacy, safety, and prognosis. This might benefit in establishing a highly individualized management for each RA patient.