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Control of lupus nephritis by changes of gut microbiota

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Background: Characterized by persistent inflammation, systemic lupus erythematosus is a complex autoimmune disorder with no known cure. Immunosuppressants put patients to a higher risk of infections. New knowledge of disease modulators, such as symbiotic bacteria, can enable fine-tuning of parts of the immune system, rather than suppressing it altogether.

Results: Dysbiosis of gut microbiota promotes autoimmune disorders that damage extraintestinal organs. Here we report the role of gut microbiota in the pathogenesis of renal dysfunction in lupus. In a classical model of lupus nephritis, MRL/lpr, we found marked depletion of Lactobacillales in the gut microbiota. Increasing Lactobacillales in the gut improved renal function of these mice. Further studies revealed that MRL/lpr mice possessed a "leaky" gut, which was reversed by increased Lactobacillus colonization. Lactobacillus treatment decreased IL-6 and increased IL-10 in the gut, thereby providing an anti-inflammatory environment. It also increased IL-10 and decreased IgG2a in the circulation, the latter a major immune deposit in the kidney of MRL/lpr mice. Inside the kidney, Treg-Th17 balance was skewed towards Treg with Lactobacillus treatment. These beneficial effects were present in female and castrated male mice, but not unaltered males, suggesting that gut microbiota controls lupus nephritis in a sex hormone-dependent manner.

Conclusions: This work demonstrates essential mechanisms on how changes of the gut microbiota regulate lupus-associated immune responses. Our findings of the mechanisms of microbiota in the gut-kidney axis point to promising ways for new treatments of lupus.

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