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Gut Microbiome and Kidney Disease: An Opinion

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Opinion

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Opinion

Microbiota is a complex group of microorganisms that colonizes host body and can promote both healthy and disease. Evidence suggests that maybe all organs of the human body pursue a specific microbiota, however, it is not a consensus when applied to central nervous system, for example. Female genital tract is a great system to understand host-microbiota interactions. During the secretory phase of the endometrial glands, several bacteria such as *Doderlein Bacillus* proliferate and digest glycogen in the reproductive system, which restricts the growth of other organisms, such as *Candida Albicans* and *Gardnerella spp*. However, the decline of hormonal stimuli during menopause, reduces or stop endometrial glands secretions, promoting decline of *Doderlein Bacillus* population, affecting vaginal acid-base balance, resource availability and letting a *free road* to *Candida Albicans* and *Gardnerella spp* recurrent infections.

Unfortunately, the microbiota does not exist just for our convenience. In the particularity of microbiota perspective, humans are a haunted house. Various antibodies and antimicrobial molecules produced by the host promote control of the quality, number, and distribution of microorganisms. For example, microbiome regulates the production of IgE both in gut mucosa, as well as in meninges. This survival pressure on the microbiota leads to the selection of bacteria with some antigens that are unrecognizable to the human immune system by an ancient mechanism called biological mimicry. In the same way that a butterfly resembling an owl's eye provides some survival advantage, peptide or protein mimicry in bacteria promotes some immune escape property. Increasing evidence suggests that bacterial mimicry promotes the development of autoimmunity in both humans and experimental models, through the breakdown of self and self-like immune tolerance.

Intestinal microbiota can affect different tissues through several different pathways. However, the loss of enterocytes adhesion leads antigen translocation to lamina propria and villus blood vessels, enabling the antigens to interact with different other tissues, activating robust immune response such as T_H17.

Intestinal microbiota is related to kidney disease in different aspects, promoting health or disease directly or indirectly. The abundance of actinobacteria is associated with the progression of IgA nephropathy, which also suggests it as a biomarker. In fact, the microbiota regulates the production of IgA. *Germ Free mice* have a smaller IgA repertoire in the intestine and distant tissues, recovered after intestinal recolonization. However, microbiota is not the root of all evil. The most important variable of microbiota affecting the immune system is their quality. Probiotics ameliorates IgA nephropathy reducing NLRP3 signaling. There are also data supporting that microbial metabolites, such as short-chain fatty acids, protects against proteinuria. Interestingly, *Lactobacillus* are linked to increase T_{reg} response and reduced T_H17 response, during experimental lupus.

In fact, microbiota and kidney diseases are a growing body of knowledge, with great works that guides our understanding. However, most of our knowledge in this area is acquired in experimental models and a long way to go towards a complete understanding of how microorganisms can be managed to improve kidney health during chronic non-infectious diseases.

Maybe our future perspective to manipulate microbiota, in way to prevent systemic inflammation, should be through vaccine development. Antibiotics seems, to me, as a huge irresponsible mistake, with can drives humanity to a major

resistance problem. Pre and probiotics, like vaccines, seems to be a good option to equilibrates bacterial populations, however, an immune education, in my view, is the state of art to control gut microorganisms.

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Not applicable

Consent for Publication

Not applicable

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