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Mini Review

The Role of Blood Microbiome and Microbial Product Translocation in Disease Pathogenesis

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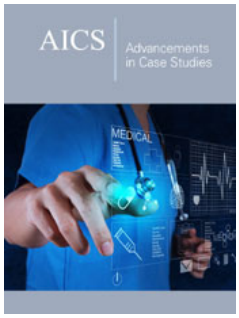

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Abstract

Circulation has been considered in a sterile condition in individuals without live microbial infections. However, recent publications from our group and others highlight detection of blood microbiome or microbial molecules in healthy individuals and individuals with various diseases, as well as their potential roles in disease pathogenesis. In this review, we introduce the published studies and research advancement in the field of blood microbial and microbial translocation in disease immunopathogenesis.

Keywords: Blood microbiome; Microbial product translocation; Disease pathogenesis

Introduction

Circulation has been considered in a sterile condition in individuals without live microbial infections. However, recent publications highlight detection of blood microbiome or microbial molecules in healthy individuals and individuals with various diseases without a live infection or sepsis. Due to the low blood levels of microbial biomass and background contaminations from experimental procedures, blood microbiome analysis has been met with technical challenges. Nonetheless, blood microbiome provides important information to study the interactions of microbiome and hosts, which is potentially critical for investigating their effects on host immune perturbations in various disease conditions.

Blood and tissue microbiome have been studied in some fields. In a recent study, blood and tissue microbiome has been assessed and cancer-specific microbial sequences have been identified in different types of cancer in humans [1]. In another study with seven different type of cancer, evidence was found that cancer-specific bacteria were detected within both immune cells and cancer tissues [2].

We have extensively investigated both quantity and quality of blood microbial translocation and their roles in HIV pathogenesis. In HIV, the magnitude of blood microbial translocation correlates with T cell activation, B cell activation and apoptosis, autoantibody production, persistent immune activation and inflammation, immune recovery after antiretroviral therapy, and organ complications [3-9]. Notably, not only the total amount of microbial translocation, but also the specific bacterium or its molecule translocation plays a role in HIV disease pathogenesis. In a recent study, we found that translocation of Staphylococcus aureus plays a role in autoantibody production in HIV, and provide evidence of systemic exposure of Staphylococcus aureus drove germinal central autoreactive B cell activation and autoantibody production in mice [8].

In autoimmune diseases, studies from Silverman's group and Kriegel's group reveal that translocation of gut pathobionts drive autoimmune diseases [10-13]. Our recent

studies in systemic lupus erythematosus (SLE) disease show that plasma microbial dysbiosis in the first-degree relatives of lupus patients compared to unrelated healthy controls, and plasma levels of microbial product translocation correlated with levels of autoantibodies in lupus patients and first-degree relatives of patients [14,15].

Studies in humans with chronic inflammatory diseases show that blood microbiome was detected and suggest that blood microbiome may play a role in the production of inflammation [16-18]. Moreover, blood and adipose tissue microbiome has been identified and suggest its role in tissue inflammation in obesity and type 2 diabetes; gut microbial translocation to the pancreatic lymph nodes triggered NOD2 singling pathway and contributed to type 1 diabetes [19,20]. Blood microbiome and microbial translocation have been studied in central nervous system diseases as well. An oral bacterium, Porphyromonas gingivalis, has detected to translocate to the brain and plays a role in Alzheimer's disease [21]. Moreover, bacterial LPS has shown to play a role in neurodegenerative diseases [22-24].

Conclusion

In summary, solid evidence has shown the detection of blood microbiome and microbial product translocation in humans without live infection. Further studies need to explore the mechanisms and causality using animals as well as in humans with treatment against specific microbes.

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