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Opinion Article

Microbiome and Metabolite Changes in Chronic Granulomatous Disease

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DESCRIPTION

Chronic Granulomatous Disease (CGD) is a rare, inherited immunodeficiency disorder characterized by the inability of phagocytes to produce reactive oxygen species, essential for killing certain bacteria and fungi. This defect leads to recurrent infections and granuloma formation. Recent research has shifted focus towards understanding the intestinal microbiome and metabolome in CGD patients, recognizing the gut's critical role in immune function and overall health. This article explores the unique microbiome and metabolome profiles in CGD patients and their implications for disease management and therapy. The intestinal microbiome consists of trillions of microorganisms, including bacteria, viruses, fungi, and protozoa, which play an important role in maintaining gut homeostasis and modulating immune responses. In CGD patients, studies have shown significant alterations in the composition and diversity of the gut microbiome compared to healthy individuals.

Microbial diversity and composition

CGD patients exhibit reduced microbial diversity, a hallmark of dysbiosis. Specifically, there is a notable decrease in beneficial bacteria such as *Bifidobacterium* and *Lactobacillus* species. These bacteria are vital for maintaining intestinal barrier integrity and modulating immune responses. Conversely, an increase in pathogenic bacteria, such as *Escherichia coli* and *Clostridium* species, has been observed. This shift towards a pathogenic microbiome can exacerbate the inflammatory state and contribute to the frequent gastrointestinal complications seen in CGD patients.

Functional implications

The altered microbiome in CGD patients impacts various metabolic pathways. For instance, reduced levels of Short-Chain Fatty Acids (SCFAs), particularly butyrate, have been reported. SCFAs are important for maintaining gut health, regulating inflammation, and supporting the immune system. The deficiency of these metabolites can compromise intestinal barrier function and promote chronic inflammation, exacerbating the symptoms of CGD. The metabolome represents the complete set of small-molecule metabolites found within a biological sample. In CGD patients, the metabolomic profile of the gut exhibits distinct alterations that reflect the underlying microbial dysbiosis and immune dysfunction. Metabolomic studies have identified disruptions in several key metabolic pathways in CGD patients.

These include amino acid metabolism, lipid metabolism, and bile acid metabolism. For example, abnormal levels of tryptophan and its metabolites have been observed, suggesting a dysregulation of the kynurenine pathway. This pathway is important for immune regulation and maintaining gut homeostasis. Additionally, alterations in bile acid metabolism can impact the gut microbiome composition and contribute to intestinal inflammation. Elevated levels of pro-inflammatory metabolites, such as arachidonic acid and its derivatives, have been detected in CGD patients. These metabolites are associated with increased production of inflammatory cytokines, contributing to the chronic inflammatory state characteristic of CGD. Furthermore, the presence of oxidative stress markers indicates an imbalance between pro-oxidant and antioxidant mechanisms, further highlighting the role of metabolic disturbances in disease pathogenesis

Clinical implications

Understanding the unique microbiome and metabolome signatures in CGD patients opens new avenues for targeted therapies and personalized medicine. Probiotic and prebiotic interventions aimed at restoring microbial balance and enhancing SCFA production may offer therapeutic benefits. Additionally, dietary modifications and supplementation with specific metabolites could help mitigate the metabolic disturbances observed in CGD.

CONCLUSION

The study of the intestinal microbiome and metabolome in Chronic Granulomatous Disease (CGD) underscores their profound impact on disease pathogenesis and clinical outcomes. CGD patients exhibit significant dysbiosis characterized by

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reduced microbial diversity and alterations in beneficial *versus* pathogenic bacteria. These changes contribute to chronic inflammation and gastrointestinal complications, exacerbating the immune deficiencies inherent in CGD.