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Exploring the Role of Microbial Dysbiosis in Chronic Inflammatory Diseases: A Pathological Perspective

Yuki Nakamura[1]*, Akiko Suzuki[2] and Kenji Sato[2]

[1]Department of Pathology, University of Tokyo, Bunkyo, Tokyo 113-8654, Japan
[2]Department of Immunology, Osaka University, Suita, Osaka 565-0871, Japan

Introduction

Microbial dysbiosis, an imbalance in the composition of the gut microbiota, has garnered increasing attention for its potential role in the pathogenesis of Chronic Inflammatory Diseases (CIDs). The human gastrointestinal tract harbors a diverse and dynamic microbial community that plays a crucial role in maintaining immune homeostasis, metabolism, and overall health. However, disruptions to this balance—whether due to diet, antibiotic use, or other environmental factors—can lead to dysbiosis, which in turn has been implicated in the development of various CIDs, including Inflammatory Bowel Disease (IBD), Rheumatoid Arthritis (RA), and even neuroinflammatory conditions like Multiple Sclerosis (MS). The growing body of research underscores the significance of the gut microbiota in disease processes, suggesting that microbial dysbiosis is not merely a consequence of chronic inflammation but may also be a driving factor [1].

Recent studies have identified specific alterations in microbial composition associated with these diseases, highlighting the potential for targeted therapies that restore microbial balance. For example, in IBD, there is often a decrease in beneficial commensals such as *Faecalibacterium prausnitzii* and an increase in pro-inflammatory bacteria like *Escherichia coli*. Similarly, RA has been linked to the overrepresentation of *Prevotella copri* and a corresponding decrease in microbial diversity. These findings not only advance our understanding of the etiological role of gut microbiota in CIDs but also open new avenues for therapeutic interventions, such as probiotics, prebiotics, and Fecal Microbiota Transplantation (FMT) [2].

Description

Chronic inflammatory diseases such as Inflammatory Bowel disease, Rheumatoid arthritis, and Multiple sclerosis are characterized by persistent inflammation and tissue damage. While genetic predisposition and environmental factors are well-established contributors to these conditions, recent research has increasingly pointed to microbial dysbiosis as a critical factor in their pathogenesis. Dysbiosis is characterized by a reduction in microbial diversity, an imbalance between beneficial and harmful bacteria, and alterations in microbial metabolic functions. This imbalance can lead to immune dysregulation, increased gut permeability, and systemic inflammation, which are hallmarks of many chronic inflammatory diseases [3].

In IBD, for instance, studies have shown that patients exhibit a significant reduction in anti-inflammatory bacteria, such as *Faecalibacterium prausnitzii* and *Akkermansia muciniphila*, alongside an increase in pathogenic bacteria like *Escherichia coli* and *Clostridium difficile*. These microbial shifts contribute to the breakdown of the intestinal barrier, allowing bacterial products such as Lipopolysaccharides (LPS) to enter the bloodstream, triggering systemic inflammation. In Rheumatoid arthritis, similar patterns of dysbiosis have been observed, with a notable increase in *Prevotella copri*, which is associated with disease severity. The presence of this bacterium has been linked to the activation of Th17 cells, a subset of pro-inflammatory T cells, further exacerbating the inflammatory response [4].

Moreover, the role of microbial dysbiosis in neuroinflammatory diseases like MS is also gaining recognition. Research has shown that MS patients often have altered gut microbiota, with a decrease in *Bacteroides* and an increase in *Akkermansia*, which is associated with disease activity and severity. These alterations can influence the gut-brain axis, a bidirectional communication pathway between the gut microbiota and the central nervous system, leading to neuroinflammation and demyelination. The cumulative evidence across various Chronic Inflammatory Diseases highlights the central role of microbial dysbiosis in their pathogenesis and underscores the potential for microbiota-targeted therapies [5].

Conclusion

The exploration of microbial dysbiosis in the context of chronic inflammatory diseases has provided invaluable insights into the complex interplay between the gut microbiota and the immune system. It is becoming increasingly clear that microbial imbalances are not merely a consequence of chronic inflammation but are actively involved in the initiation and progression of these diseases. The consistent association of dysbiosis with conditions such as Inflammatory Bowel disease, Rheumatoid arthritis, and Multiple sclerosis underscores the importance of maintaining a balanced microbiome for overall health. Future research should focus on identifying the precise microbial signatures associated with different chronic inflammatory diseases and elucidating the mechanisms through which these microbes influence disease processes. Such studies will be critical in developing effective microbiota-based therapies, which could revolutionize the treatment of CIDs. Probiotics, prebiotics, and FMT are already showing promise in restoring microbial balance and alleviating disease symptoms, but further clinical trials are needed to validate their efficacy and safety.

In conclusion, microbial dysbiosis represents a significant and modifiable risk factor for chronic inflammatory diseases. As our understanding of the gut microbiota continues to expand, so too does the potential for novel therapeutic strategies that target this crucial aspect of human health. Addressing dysbiosis may not only help in managing existing conditions but also in preventing the onset of chronic inflammation, thereby improving the quality of life for millions of individuals worldwide.

Acknowledgement

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Conflict of Interest

None.

***Address for Correspondence:** Yuki Nakamura, Department of Pathology, University of Tokyo, Bunkyo, Tokyo 113-8654, Japan, E-mail: y.nakamura@u-tokyo.ac.jp*

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