

# Respiratory Microbiome Dysbiosis in COVID-19 and its Impact on Disease Severity

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## Introduction

The respiratory tract harbors a complex and dynamic microbial ecosystem that plays a crucial role in maintaining mucosal immunity and protecting against respiratory pathogens. In health, this microbial community exists in a state of balanced homeostasis, contributing to the regulation of inflammation and the integrity of epithelial barriers. However, infections such as SARS-CoV-2, the virus responsible for COVID-19, have the potential to disturb this equilibrium. Emerging evidence suggests that respiratory microbiome dysbiosis—characterized by a loss of microbial diversity, overgrowth of opportunistic pathogens, and depletion of commensals—may significantly influence the host immune response, viral pathogenesis, and clinical outcomes in COVID-19 patients. Understanding the nature and consequences of respiratory microbiome alterations during SARS-CoV-2 infection is critical to identifying potential biomarkers of disease severity and developing adjunctive microbiome-targeted therapies [1].

## Description

This study investigates the composition and functional changes of the respiratory microbiome in patients with varying severities of COVID-19, ranging from mild symptoms to critical illness. Nasopharyngeal and Bronchoalveolar Lavage (BAL) samples from hospitalized patients were analyzed using 16S rRNA gene sequencing and metatranscriptomic profiling to characterize bacterial taxa, community structure, and microbial gene expression [2]. The data were then correlated with clinical parameters, including viral load, inflammatory cytokine levels, and markers of respiratory dysfunction. Compared to healthy controls and patients with mild COVID-19, individuals with severe or critical illness displayed marked reductions in microbial diversity and a shift toward dominance by potentially pathogenic bacteria such as *Pseudomonas*, *Staphylococcus*, and *Acinetobacter*. Conversely, beneficial commensals such as *Corynebacterium* and *Doligranulum*, which are known to support mucosal immunity, were significantly depleted [3].

Functional analysis of the microbial communities revealed altered expression of genes involved in immune modulation, biofilm formation, and antimicrobial resistance. These microbial shifts were associated with heightened local and systemic inflammation, including elevated IL-6, IL-8, and interferon-gamma levels. Moreover, patients with profound dysbiosis showed increased epithelial injury and poorer oxygenation, indicating a potential link between microbial imbalance and respiratory compromise. Notably, dysbiosis persisted even after viral clearance in some patients, suggesting a lasting impact on respiratory microbial ecology and potential implications for long-term recovery or susceptibility to secondary infections [4,5].

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## Conclusion

In conclusion, this study provides compelling evidence that respiratory microbiome dysbiosis is closely associated with COVID-19 severity and contributes to disease pathogenesis through modulation of host immune responses and epithelial barrier function. The findings highlight the respiratory microbiome as both a marker and a modulator of disease progression, offering new avenues for risk stratification and therapeutic intervention. Restoring microbial balance through targeted probiotics, microbiome-based diagnostics, or preemptive antimicrobial stewardship may represent a valuable complement to current antiviral and immunomodulatory treatments. As the pandemic continues to evolve, integrating microbiome research into the broader understanding of COVID-19 could yield novel insights into host-pathogen interactions and improve outcomes in respiratory viral infections.

## Acknowledgment

None.

## Conflict of Interest

None.

## References

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