

# The Respiratory Micro biome Composition of COVID-19 Patients

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## Editorial

COVID-19, a novel coronavirus disease classified as a pandemic by the World Health Organization, has so far resulted in over 150 million reported cases and 3 million deaths worldwide. Infection with its causative agent, the novel coronavirus SARS-CoV-2, results in a wide range of clinical manifestations: it is estimated that approximately 80% of infected individuals are asymptomatic or have only mild respiratory and/or gastrointestinal symptoms, while the remaining 20% develop acute respiratory distress syndrome, requiring hospitalisation and oxygen support, and 25% of cases require critical care. Despite a concerted global research effort, many questions about the full spectrum of disease severity remain unanswered. Independent studies from different countries, however, agree that age and gender, as well as type 2 diabetes and obesity, are major risk factors for disease severity and patient death. Other potential risk factors for critical condition and death include the patient's viral load at the time of admission to the hospital and the specific immune response to infection, with the manifestation of an abnormal immune response in critical patients characterised by dysregulated levels of pro-inflammatory cytokines and chemokines, which some studies have linked to organ failure.

Despite its close relationship with the immune system and known associations with host health, little is known about the respiratory microbiota's role in modulating COVID-19 disease severity or its potential as a prognostic marker. Previous research into other pulmonary disorders has found that lung microbiota members may aggravate symptoms and contribute to their severity, possibly through direct crosstalk with the immune system and/or through bacteremia and secondary infections. The first studies of the COVID-19 respiratory microbiome revealed elevated levels of opportunistic pathogenic bacteria. Reports on bacterial diversity, on the other hand, are contradictory. While some studies show a low microbial diversity in COVID-19 patients who recover, others show an increase in the COVID-19 associated microbiota diversity. These contradictory findings could be attributed to differences in sampling location (upper or lower respiratory tract), patient severity, disease stage, treatment, or other confounders. While these preliminary findings suggest that the lung microbiome may be exacerbating or mitigating COVID-19 progression, the precise mechanisms remain unknown. As a result, there is an urgent need for studies that identify and address confounders in order to distinguish true signals from noise.

Researchers used nasopharyngeal swabs and bronchoalveolar lavage (BAL) samples to look for possible links between COVID-19 severity and evolution and upper and lower respiratory tract microbiota. In conjunction with viral load determination and nCounter immune profiling, they longitudinally

profiled the nasopharyngeal microbiome of 58 COVID-19 patients during intensive care unit (ICU) treatment and after discharge to a traditional hospital ward following clinical improvement. They profiled microbial reads in cross-sectional single-cell RNA-seq data from bronchoalveolar lavage (BAL) samples of 22 COVID-19 patients and 13 pneumonitis controls with negative COVID-19 qRT-PCR obtained from the same hospital for the lower respiratory tract. They were able to:

- (1) Identify potential confounders of COVID-19 microbiome associations
- (2) Investigate how microbial diversity evolves throughout hospitalisation
- (3) Investigate microbe-host cell interactions, and
- (4) Establish a link between the respiratory microbiome and SARS-CoV-2 viral load, as well as COVID-19 disease severity, by combining these data.

Overall, our findings point to the existence of links between the microbiota and specific immune cells in the context of COVID-19 disease. These interactions may be influenced by mechanical ventilation and the clinical practises associated with it, and thus may influence COVID-19 disease progression and resolution [1-5].

## Conflict of Interest

None.

## References

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