

The Role of Smoking in Changing Lung Microbes and Causing TB

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DESCRIPTION

Smoking is a well-established risk factor for Tuberculosis (TB), a serious infectious disease caused by *Mycobacterium tuberculosis* (*M. tb*). Beyond its direct effects on lung health and immune function, smoking-induced microbial dysbiosis—an imbalance in microbial communities—plays a significant role in the pathogenesis and progression of TB. Understanding how smoking alters microbial ecosystems can provide insights into TB susceptibility, disease progression, and treatment outcomes. Tuberculosis remains a major global health issue, particularly in regions with high rates of smoking. Smoking exacerbates the risk of developing TB and contributes to poorer outcomes in those with the disease. The mechanisms through which smoking impacts TB are multifaceted, involving direct effects on the respiratory system, modulation of immune responses, and alterations in microbial communities.

Effect of smoking on respiratory microbiota

The respiratory microbiota, comprising bacteria, viruses, and fungi, plays an important role in maintaining lung health and defending against infections. Smoking disrupts this delicate balance, leading to microbial dysbiosis that can influence TB susceptibility and progression. Smoking reduces microbial diversity in the respiratory tract, leading to an overgrowth of pathogenic bacteria and a decrease in beneficial species. This dysbiosis creates an environment that can facilitate the colonization and persistence of *M. tb*. For example, smoking-induced changes can promote the growth of bacteria such as *Streptococcus pneumoniae* and *Haemophilus influenzae*, which can interact with or enhance the pathogenicity of *M. tb*. Smoking-induced dysbiosis affects the immune system's ability to respond to infections. Smoking impairs immune function by causing chronic inflammation and reducing the effectiveness of immune cells that are important for controlling TB.

Mechanisms linking smoking-induced dysbiosis to TB

Altered host-microbe interactions: Smoking-induced dysbiosis changes the interactions between the host and microbial

communities in the lungs. An imbalanced microbiota can disrupt normal immune responses and increase susceptibility to TB by impairing the lung's defense mechanisms. For instance, the reduced abundance of beneficial microbes may decrease the production of antimicrobial peptides and other protective factors that help prevent TB infection.

Enhanced pathogen survival: The inflammatory environment created by smoking can enhance the survival and replication of *M. tb*. Chronic inflammation caused by smoking disrupts the lung's microbial balance and provides a more favorable environment for the persistence of *M. tb*. Additionally, smoking-induced damage to the respiratory epithelium can facilitate the entry and colonization of *M. tb*.

Effect on treatment outcomes: Smoking-induced dysbiosis can affect the effectiveness of TB treatment. The altered microbial environment may influence drug metabolism and immune responses, potentially reducing the efficacy of anti-TB medications. Smokers with TB may experience slower treatment responses and increased risk of drug-resistant strains due to the interplay between smoking, microbial dysbiosis, and treatment regimens.

Implications for tuberculosis management

Addressing smoking as part of TB prevention strategies is important. Smoking cessation can improve respiratory microbial balance, enhance immune function, and reduce the risk of TB infection and progression. Public health initiatives should emphasize the importance of quitting smoking to reduce TB risk and improve overall lung health. For individuals with TB who smoke, a comprehensive approach to treatment should include strategies to manage smoking-induced dysbiosis. This may involve smoking cessation programs, as well as interventions aimed at restoring a healthy microbial balance in the respiratory tract. Additionally, tailored TB treatment plans that consider the impact of smoking on drug metabolism and immune responses can improve outcomes. Studies exploring how smoking alters the respiratory microbiota and its impact on TB susceptibility and treatment can inform new strategies for managing and preventing the disease.

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CONCLUSION

Smoking-induced microbial dysbiosis has significant implications for tuberculosis, influencing disease susceptibility, progression, and treatment outcomes. By disrupting the respiratory microbiota and impairing immune function, smoking creates conditions that favour the persistence and severity of TB. Addressing smoking-related dysbiosis through prevention, treatment, and research is essential for improving

TB management and reducing the global burden of this infectious disease. Investigating the potential for microbiome-based therapies and interventions to mitigate the effects of smoking on TB can offer promising avenues for improving patient care. The altered respiratory microbiota further contributes to immune dysfunction, making it more difficult for the body to mount an effective response against *M. tb*. This impaired immunity increases susceptibility to TB infection and enhances disease progression.