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Fungal pathogens and cystic fibrosis: A hidden connection.

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Introduction

Cystic fibrosis (CF) is a genetic disorder characterized by the production of thick, sticky mucus that obstructs airways and predisposes patients to chronic respiratory infections. While bacterial pathogens such as *Pseudomonas aeruginosa* and *Staphylococcus aureus* have long dominated the clinical landscape of CF, fungal pathogens are increasingly recognized as significant contributors to disease progression. This hidden connection between fungal colonization and CF exacerbations is gaining attention, revealing complex interactions that challenge traditional paradigms of CF care [1].

For ABPA, corticosteroids and antifungals are used in combination. However, long-term steroid use can suppress immunity and promote fungal growth. Emerging therapies, including biologics targeting IgE and eosinophils, offer promise but require further validation. Prophylactic antifungal use is controversial due to resistance risks and uncertain benefits. Personalized treatment based on fungal load, immune status, and clinical presentation is recommended. CFTR modulators like lumacaftor/ivacaftor have transformed CF care by improving mucus clearance and reducing bacterial infections. Their impact on fungal colonization is under investigation. Preliminary data suggest a reduction in fungal burden, possibly due to improved airway hydration and mucociliary function. Historically, fungi were considered benign colonizers in CF airways. However, improved detection methods—including molecular diagnostics and culture techniques—have revealed a diverse fungal microbiota with potential pathogenic roles. Among the most frequently isolated fungi are *Aspergillus fumigatus*, *Candida albicans*, *Candida dubliniensis*, *Scedosporium apiospermum*, and *Exophiala dermatitidis*. Longitudinal studies have shown that fungal prevalence and diversity in CF patients have increased over the past two decades, likely due to prolonged antibiotic use, immunomodulatory therapies, and environmental exposure. These factors disrupt bacterial-fungal balance and create niches for fungal persistence [2].

However, long-term effects on fungal ecology and resistance patterns remain unknown. Monitoring fungal dynamics in patients receiving modulators is essential. Integrating fungal surveillance into routine CF care will enable early detection, targeted therapy, and improved patient outcomes. *A. fumigatus* is the most commonly detected mold in CF airways. It can exist in several clinical forms: Asymptomatic presence without inflammation. A hypersensitivity reaction causing wheezing, pulmonary infiltrates, and lung function decline. Rare but severe, especially in immunocompromised patients. Azole resistance in *A. fumigatus* is a growing concern, driven by environmental fungicide use and prolonged azole therapy. Resistant strains may retain virulence and evade treatment, complicating management strategies. *Candida albicans* and *C. dubliniensis* are frequently isolated from CF sputum. Once considered harmless, persistent colonization has now been linked to accelerated lung function decline [3].

A 16-year study found that patients colonized with *C. albicans* experienced a 2.6% annual decrease in predicted forced expiratory volume (ppFEV1), while *C. dubliniensis* was associated with a 7.6% decline. The pathogenic potential of Candida may stem from biofilm formation, immune modulation, and synergistic interactions with bacteria. However, causality remains difficult to establish due to overlapping microbial communities. Beyond the usual suspects, CF patients are increasingly

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colonized by rare fungi such as: *Scedosporium apiospermum* and *Lomentospora prolificans*: Associated with pulmonary exacerbations and resistant to multiple antifungals. *Exophiala dermatitidis*: A black yeast linked to airway inflammation. *Rasamsonia argillacea*: Emerging mold with unclear clinical significance. These fungi often appear in advanced disease stages and may contribute to refractory symptoms. Their detection requires specialized culture media and molecular tools, underscoring the need for standardized mycological protocols [4].

Recent studies suggest that fungal colonization may influence the frequency and severity of pulmonary exacerbations in CF. During periods of clinical instability, detection rates of *Aspergillus* increase significantly—by up to 25% using molecular biomarkers like galactomannan and lateral flow assays. However, distinguishing colonization from infection remains challenging, and the clinical relevance of fungal isolates must be interpreted in context. Some laboratories use agar slants with limited surface area, potentially missing mixed fungal populations common in CF airways. Molecular diagnostics offer improved sensitivity but may detect non-viable organisms, leading to overdiagnosis. Treatment of fungal infections in CF is nuanced. Azoles remain the mainstay for *Aspergillus*, but resistance necessitates susceptibility testing. Amphotericin B and echinocandins are alternatives, though toxicity and limited efficacy pose challenges [5].

Conclusion

Fungal pathogens in cystic fibrosis represent an underappreciated but clinically significant aspect of disease management. From common molds like *Aspergillus fumigatus* to emerging species, fungi contribute to airway inflammation, exacerbations, and lung function decline. As diagnostic tools and therapeutic options evolve, recognizing and addressing this hidden connection will be vital to optimizing care for CF patients.

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