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Candida Albicans: Pathogenesis, Antifungal Resistance, And Emerging Challenges

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Abstract

Candida albicans is a versatile fungal species that normally lives harmlessly within the human microbiota but can become pathogenic under certain conditions, especially in immunocompromised or critically ill individuals. This review summarizes current insights into its morphological adaptability, virulence traits, biofilm development, host immune interactions, and mechanisms of antifungal resistance. The discussion also covers the clinical burden of candidiasis—from mucosal infections to invasive disease—and emerging strategies to address drug resistance. Recognized by the World Health Organization (WHO) as a critical fungal pathogen, C. albicans demands urgent attention toward improved diagnostics, therapeutics, and preventive measures.

Introduction

Candida albicans is a diploid yeast capable of multiple growth forms, commonly residing on human skin and mucosal surfaces such as the mouth, gut, and genitourinary tract. While it typically exists as a harmless commensal organism, disruptions in the host's immune defenses or microbiota can trigger its pathogenic transformation. The resulting infections range from mild mucosal candidiasis to severe bloodstream infections like candidemia.

The growing number of immunocompromised patients and the increased use of invasive medical devices have amplified the clinical importance of C. albicans. Designated by the WHO as a critical fungal pathogen, it represents a major healthcare challenge due to its diverse virulence factors and rising antifungal resistance (3,11,8).

Literature Review

Morphological Plasticity and Virulence Factors

C. albicans exhibits remarkable morphological flexibility, shifting between yeast, pseudohyphal, and hyphal forms depending on environmental stimuli. This morphological switching is a key factor in its pathogenicity—enhancing adhesion, tissue invasion, and immune evasion. Regulatory genes such as EFG1, CPH1, and HWP1 govern this transition, with the hyphal state being closely associated with tissue penetration and mature biofilm formation.

In addition, virulence-related enzymes like secreted aspartyl proteases (SAPs) and phospholipases, along with surface adhesins, support colonization and tissue damage (12,13,1).

Biofilm Formation

Biofilm formation represents one of the most critical survival strategies of C. albicans. These complex microbial communities, composed of yeast and filamentous cells within an extracellular matrix, enable persistent colonization on both host tissues and medical devices. Biofilms confer strong resistance to antifungal agents and immune responses. The cAMP-PKA and MAP kinase signaling pathways play vital roles in biofilm regulation. Recent advances have identified novel biofilm inhibitors, offering promising leads for antifungal therapy (12,6).

Host-Pathogen Interactions

The fungal cell wall of C. albicans is recognized by host pattern recognition receptors (PRRs) such as Dectin-1 and Toll-like receptors (TLRs), triggering immune responses essential for fungal clearance. However, C. albicans can modify its surface by masking β-glucans, especially in the hyphal phase, allowing it to escape immune detection.
The Th17 immune response is particularly critical for mucosal defense, and deficiencies in this pathway are strongly linked to recurrent fungal infections (14,15,16).

Antifungal Resistance

Resistance to antifungal agents, particularly azoles like fluconazole, has become an increasing concern worldwide. Mechanisms driving this resistance include overexpression of efflux pumps (CDR1, CDR2, MDR1), mutations or upregulation of ERG11, chromosomal abnormalities, and biofilm-mediated tolerance.
Global surveillance data highlight an upward trend in resistance rates, complicating treatment outcomes (17,18,19). New combination therapies—such as using amantadine alongside azoles—are being explored as potential ways to overcome these limitations (20).

Discussion and Conclusion

C. albicans continues to be a clinically important opportunistic pathogen due to its adaptability and multifaceted virulence mechanisms. Its ability to switch morphologies, form biofilms, and evade immune detection allows it to thrive in various host environments while resisting antifungal treatments.
The growing prevalence of antifungal resistance poses a serious clinical challenge, emphasizing the need for innovative therapies and improved diagnostic methods. Advances in genomics and molecular biology have shed light on the mechanisms behind its pathogenicity and drug resistance, guiding future drug and vaccine development.

To mitigate resistance and reduce infection rates, continuous surveillance and antifungal stewardship are essential. Future research should focus on targeted drug delivery, immunotherapeutic approaches, and microbiome-based interventions to restore host-fungal balance. A coordinated multidisciplinary effort is crucial to effectively manage C. albicans infections and lessen their global health impact.

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