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Fluconazole Resistance in Candida auris: Environmental and Clinical Insights.

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Introduction

Candida auris is an emerging multidrug-resistant fungal pathogen that has rapidly become a global health concern. First identified in 2009 in Japan, *C. auris* has since been reported in over 50 countries across six continents. Its ability to cause invasive infections, persist in healthcare environments, and resist multiple antifungal agents particularly fluconazole has made it a formidable challenge for clinicians and infection control specialists [1, 2].

Fluconazole, a triazole antifungal, has long been a first-line treatment for candidiasis. However, *C. auris* exhibits high levels of intrinsic and acquired resistance to fluconazole, complicating treatment and contributing to high mortality rates in infected patients. Understanding the environmental and clinical factors driving this resistance is essential for developing effective containment and therapeutic strategies [3, 4].

Fluconazole targets the fungal enzyme lanosterol 14α-demethylase, encoded by the ERG11 gene, which is essential for ergosterol biosynthesis. Mutations in ERG11, such as Y132F, have been identified in fluconazole-resistant *C. auris* isolates and are known to reduce drug binding affinity [5, 6].

Additionally, overexpression of efflux pump genes like CDR1, CDR2, and MDR1 contributes to active drug expulsion from fungal cells, further diminishing fluconazole efficacy [7, 8].

Recent studies have also revealed adaptive aneuploidy—chromosomal changes that enhance resistance under drug pressure—as a mechanism of fluconazole resistance. These genetic adaptations allow *C. auris* to rapidly evolve resistance during treatment, even in the absence of prior exposure.Clinically, fluconazole resistance in *C. auris* presents significant challenges. Infections are often misdiagnosed due to phenotypic similarities with other *Candida* species, delaying appropriate treatment. In intensive care units (ICUs), *C. auris* has caused outbreaks of bloodstream infections with mortality rates exceeding 30% [9, 10].

Conclusion

A study from a tertiary care trauma center in Delhi, India, found that while 45% of *C. auris* isolates exhibited low minimum inhibitory concentrations (MICs) for fluconazole, the majority were resistant, necessitating alternative therapies such as echinocandins or amphotericin B. However, resistance to these agents is also emerging, underscoring the urgency of developing new antifungals and diagnostic tools.One of the most alarming features of *C. auris* is its ability to persist in healthcare environments. It can survive on surfaces for weeks and resist standard disinfectants, facilitating nosocomial transmission. Environmental stress resistance—including tolerance to high temperatures, pH extremes, and oxidative stress—enhances its survival in hospital settings.A study comparing *C. auris* to other *Candida* species found that it was relatively thermotolerant and resistant to combinatorial stress conditions, such as high pH and heat, which are common in hospital laundering protocols. This resilience contributes to its rapid spread and difficulty in eradication.

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