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The Rise of *Candida auris*: A Review of a Globally Emerging Multidrug-Resistant Pathogen

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Abstract

The emerging multidrug-resistant fungal pathogen *Candida auris* poses a serious global public health threat. Since its identification in 2009, *C. auris* has rapidly spread worldwide and can cause difficult-to-treat nosocomial outbreaks and invasive infections with high mortality. *C. auris* exhibits frequent resistance to azoles, echinocandins, and polyenes, creating challenges for its clinical management. Its ability to persistently colonise patients and contaminate hospital environments also facilitates easy transmission. This review summarises current knowledge on *C. auris* global epidemiology, mechanisms of antifungal resistance, limitations of current treatments, and emerging novel therapies under investigation. Ongoing research priorities include improving diagnostics, elucidating pathogenesis, developing new therapies, and optimising infection control. Mitigating the public health risks of this formidable fungal pathogen remains an urgent goal requiring focused efforts across multiple domains.

Keywords: *Candida auris,* Multidrug resistance, Antifungal resistance, Fungal pathogenesis, Global epidemiology, Nosocomial outbreaks

Introduction

Candida auris has emerged as a serious global public health threat over the past decade (Prayag et al., 2022). Since its initial identification 10 years ago, *C. auris* has been reported in over 30 countries across 6 continents (Borman & Johnson, 2020). This pathogenic yeast is capable of causing large hospital outbreaks, particularly in high-dependency units, due to its ability to persistently colonise patients and contaminate hospital environments (Borman & Johnson, 2020). Whole-genome sequencing has revealed at least 4

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distinct clonal lineages of *C. auris*, each associated with specific geographical regions: clade I (Southern Asia), clade II (East Asia), clade III (South Africa), and clade IV (South America). A potential fifth clade from Iran has been proposed, though not yet linked to nosocomial outbreaks (Borman & Johnson, 2020). In India, *C. auris* has emerged as the predominant *Candida* species among hospitalised patients with candidemia (Prayag et al., 2022) (Dixit et al., 2022). A study in a tertiary care hospital in Western India found *C. auris* to be the most frequent cause of candidemia, with universal fluconazole resistance (Prayag et al., 2022). Furthermore, a study in a North Indian tertiary care hospital reported *C. auris* as a leading nosocomial fungal pathogen in COVID-19 patients, underscoring its public health threat (Dixit et al., 2022). Together, these findings highlight the growing candidemia burden posed by *C. auris* in Indian healthcare settings.

Biological Makeup and Clinical Importance of Candida auris

Candida auris is a multidrug-resistant pathogenic yeast that has rapidly emerged as a global public health threat due to its unique cellular characteristics and challenging clinical profile (Borman & Johnson, 2020; Burrack et al., 2022).

Cellular Morphology and Genomic Characteristics

At the cellular level, *C. auris* is an oval budding yeast distinguished by a robust cell wall composed predominantly of beta-glucans, chitin, and mannoproteins. This complex cell wall not only provides structural integrity but also contributes to inherent resistance by limiting the penetration of antifungal agents (Chakrabarti & Sood, 2021; de Jong et al., 2022). Genomic studies have revealed significant genetic diversity among *C. auris* isolates, with distinct clonal lineages emerging across different geographic regions. Such diversity includes variations in key resistance genes and regulatory elements that underpin the pathogen's adaptive capacity (Burrack et al., 2022; Kordalewska & Perlin, 2022).

Metabolic Flexibility and Stress Response

Candida auris exhibits remarkable metabolic plasticity, which allows it to thrive under diverse and often hostile environmental conditions. Its ability to withstand high salinity, desiccation, and exposure to disinfectants commonly used in healthcare settings is a direct consequence of adaptive stress response pathways and altered metabolic regulation (Chakrabarti & Sood, 2021). Moreover, rapid changes in gene expression in response to environmental cues enable *C. auris* to efficiently form biofilms—a structured community of cells embedded in an extracellular matrix that enhances survival under antifungal pressure and environmental stress (Černáková et al., 2021; Du et al., 2020).

Virulence and Mechanisms of Pathogenicity

The pathogenicity of *C. auris* is multifactorial. One of its most clinically significant features is its capacity for biofilm formation, which confers a protective niche against host immune responses and antifungal agents, thereby facilitating persistent infections (Chowdhary et al., 2018; Rybak et al., 2022). In addition to biofilms, *C. auris* secretes a range of hydrolytic enzymes that contribute to tissue invasion and immune modulation. This ability to evade host defences is further compounded by its rapid adaptation to antifungal pressure, which is often mediated by mutations in target enzymes and the upregulation of efflux pumps (Du et al., 2020; Rybak et al., 2022).

Clinical Impact and Epidemiological Considerations

Clinically, *C. auris* is associated with severe invasive infections, most notably candidemia and deep-seated organ infections, which carry high morbidity and mortality rates (Prayag et al., 2022; Dixit et al., 2022). The organism's propensity to cause nosocomial outbreaks is especially problematic in intensive care units and other high-dependency settings where patients are often immunocompromised. Delayed or inaccurate identification—stemming from its phenotypic similarity to other *Candida* species—can lead to inappropriate treatment regimens and further exacerbate patient outcomes (Borman & Johnson, 2020; Kordalewska & Perlin, 2022). Moreover, the environmental resilience of *C. auris*, including its ability to persist on medical equipment and surfaces, plays a critical role in its rapid inter- and intra-hospital dissemination (Chakrabarti & Sood, 2021; Forsberg et al., 2019).

Factors contributing to the global spread of *Candida auris*

Several key factors have enabled the rapid global dissemination of the multidrug-resistant fungal pathogen *Candida auris*. Firstly, *C. auris* exhibits resilience to disinfectants, desiccation, and high salinity, facilitating environmental survival and transmission (Chakrabarti & Sood, 2021). The pathogen also readily colonises patients and contaminates fomites (Chakrabarti & Sood, 2021). Secondly, difficulties identifying *C. auris* via traditional laboratory methods often delay diagnosis and targeted antifungal therapy (Watkins et al., 2022). Thirdly, multidrug resistance hinders treatment options (Chakrabarti & Sood, 2021; Du et al., 2020; Kilburn et al., 2022). Fourthly, specific adaptations like skin persistence factors promote Colonization and infection (Watkins et al., 2022). Additionally, *C. auris* can persist on surfaces for prolonged periods, enabling nosocomial spread (Chakrabarti & Sood, 2021). The emergence of *C. auris* also coincided with deteriorating host, pathogen, and environmental conditions in the regions where it first emerged, likely reaching a tipping point for its evolution

and dissemination (Chakrabarti & Sood, 2021). Global travel and trade subsequently facilitated intercontinental transmission (Du et al., 2020), while inadequate infection prevention and control in healthcare settings enabled local outbreaks (Hinrichs et al., 2022).

The resilience of *C. auris*, difficulties in laboratory identification, multidrug resistance, evolving virulence factors, environmental persistence, specific selective pressures, and global connectedness have all contributed to the rapid dissemination of this fungal pathogen worldwide. Understanding these factors is key to controlling its spread.

Identification of *Candida auris*

Several methods can identify the emerging nosocomial pathogen *Candida auris*, which is often misidentified by conventional approaches (de Jong et al., 2022; Kordalewska & Perlin, 2022). Whole genome sequencing is considered the gold standard for genotyping *C. auris* but is expensive and time-consuming. More simple, rapid and inexpensive alternatives include ITS region sequencing and microsatellite typing for identification and genotyping of *C. auris* (de Jong et al., 2022). MALDI-TOF mass spectrometry has largely replaced phenotypic methods for yeast identification and profiling in routine clinical, diagnostic, and research microbiology laboratories and can reliably identify *C. auris* from cultures of clinical or environmental surveillance samples (Abdolrasouli & Fraser, 2022). Chromogenic media like CHROMagar *Candida* Plus allow presumptive *C. auris* screening based on colony characteristics showing white colonies with blue-green halos that are more evident after 72 hours of incubation at 35°C than after 48 hours, though distinguishing closely related species such as *Candida haemulonii*, *Candida pseudohaemulonii*, and *Candida duobushaemulonii* still requires additional testing (Tamura et al., 2022). Molecular approaches like the ClaID, which is a colony PCR-based clade identification system, rapidly identify *C. auris* clades using clade-specific DNA sequences (Narayanan et al., 2022). Spectroscopic techniques like Raman spectroscopy and imaging employ analysis of representative Raman spectra from different *C. auris* samples by means of a customised machine-learning algorithm linked to a Raman database in order to decode structural differences at the molecular scale. Raman analyses of metabolites reveal clear differences in cell walls and membrane structure and thus provide a rapid on-site/real-time taxonomic identification and metabolic profiling of different *C. auris* clades/subclades (Pezzotti et al., 2022). Identification and characterisation of the ABC transporter repertoire in *Candida* species are of high relevance because overexpression of these in different *Candida* species results in antifungal resistance. These could be used for building bioinformatic pipelines that can be used to identify different *Candida* species based on the heterogeneous expression of ABC transporter proteins (Banerjee et al., 2022).

A range of advanced molecular, spectroscopic, chromogenic, and bioinformatic methods, therefore, could facilitate accurate identification and characterisation of the emerging multidrug-resistant yeast *C. auris* in healthcare and research settings. Integration of these technologies can overcome the limitations of conventional identification approaches.

Candida auris: resistance to common antifungal drugs

Candida auris is an emerging multidrug-resistant fungal pathogen that poses significant challenges for patient treatment and hospital disinfection (Chaabane et al., 2019). *C. auris* exhibits frequent resistance to the azole antifungals as well as amphotericin B, with isolates resistant to all major antifungal classes reported (Rybak et al., 2022). Recent CDC breakpoints indicate approximately 90% *C. auris* isolates found in the USA are fluconazole-resistant, 30% amphotericin B-resistant, and under 5% echinocandin-resistant (Chaabane et al., 2019). Resistance trends in New York and New Jersey isolates from 2016-2020 reveal nearly universal fluconazole resistance (99.8%), 50% amphotericin B resistance, and increasing echinocandin resistance (from 0 to 4%) and pan-resistance (from 0 to <1%) in New York but not for New Jersey, highlighting regional differences (Kilburn et al., 2022). A molecular diagnostic platform was evaluated on a panel of clinical skin swabs, enabling rapid identification of FKS1 and ERG11 mutations conferring echinocandin and azole resistance, respectively, which indicates that rapid molecular detection of resistance mutations directly from patient samples is critical for guiding treatment and infection control (Kordalewska & Perlin, 2022). In vitro evolution of 17 new clinical isolates of *C. auris* from clades I and IV determined how quickly resistance mutations arise, the stability of resistance in the absence of a drug, and the impact of genetic background on evolutionary trajectories (Burrack et al., 2022). In vitro studies demonstrate *C. auris* can quickly evolve antifungal resistance via genomic and phenotypic changes, including karyotype alterations, aneuploidy, acquisition of point mutations, and increases in MIC values within the populations (Burrack et al., 2022). Fluconazole resistance mutations arise readily, persist stably without fitness costs, and confer high-level resistance (Burrack et al., 2022). Most South African *C. auris* isolates also exhibited primarily azole resistance, with resistance mutations even in phenotypically susceptible strains (Maphanga et al., 2021). Strategies to overcome resistance include efflux pump inhibitors like farnesol combined with azoles, which enhance azole activity against resistant isolates by modulating efflux pump gene expression (Dekkerová et al., 2022). Thus, the rapid emergence of multidrug resistance in *C. auris* poses serious challenges for treatment and infection control worldwide. Therefore, ongoing surveillance coupled with strategies to overcome resistance will be critical.

Molecular resistance mechanisms of *Candida auris*

Candida auris exhibits intrinsic and acquired resistance to multiple antifungal classes, posing significant treatment challenges (Chaabane et al., 2019; Jangir et al., 2023; Rybak et al., 2022). Several molecular mechanisms likely underlie this multidrug resistance (Figure 1).

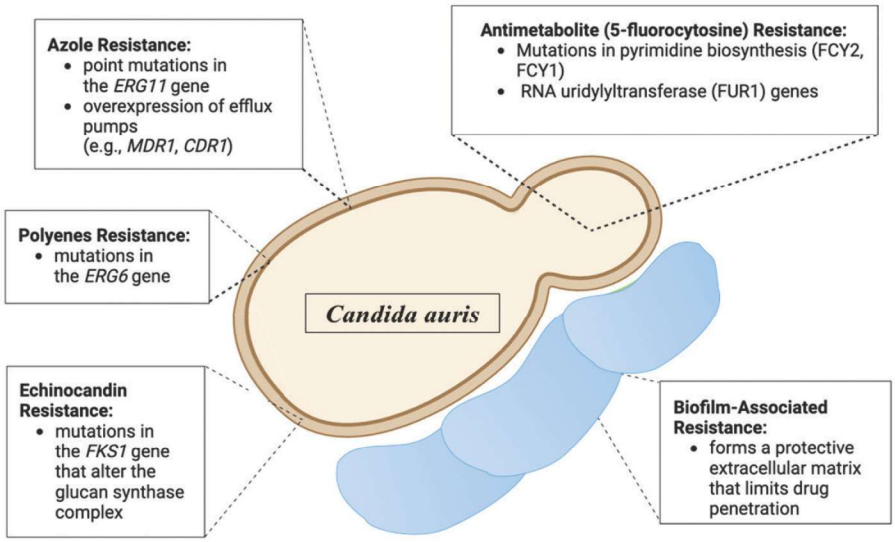


Figure 1: Molecular mechanisms of antifungal drug-resistant in *Candida auris*

Genetic diversity across *C. auris* isolates facilitates rapid development of antifungal resistance in vitro and in vivo (Burrack et al., 2022). Differential gene expression between resistant and susceptible isolates, including upregulation of efflux pumps and ergosterol biosynthesis genes such as SNQ2, CDR4, ARB1, MDR1, MRR1, and ERG genes (Zhou et al., 2021). Multi-omics approach comparing drug-resistant and drug-susceptible *C. auris* strains showed major differences in carbon utilisation, downstream lipid and protein content, suggesting a multi-factorial mechanism of drug resistance (Zamith-Miranda et al., 2019). Mutations in resistance genes result in phenotypically susceptible isolates (Table 1) (Dekkerová et al., 2022). Overexpression of efflux pumps reduces intracellular azole levels, and the efflux pump inhibitor farnesol can restore fluconazole susceptibility (Du et al., 2020). Fluconazole resistance is mostly explained by mutations in the ERG11 gene, as well as a higher number of gene copies and overexpression of efflux pumps, while mutations in FKS1 lead to caspofungin resistance by reducing drug binding (Frías-De-León et al., 2020). Amphotericin B resistance arises from mutations in ERG6 causing reduced ergosterol content, which was identified as a novel resistance mechanism (Rybak et al., 2022). Mutations in pyrimidine biosynthesis (FCY2, FCY1) and RNA uridylyltransferase (FUR1) genes confer 5-fluorocytosine resistance (Frías-De-León et al., 2020) (Table 1).

Gene	Mutation Type / Alteration	Effect on Drug Resistance	Experimental Evidence
ERG11	Point mutations; gene amplification	Reduces binding affinity of azoles, leading to decreased drug efficacy	Frías-De-León et al. (2020); Burrack et al. (2022)
FCY2, FCY1, FUR1	Mutations	5-fluorocytosine resistance	Frías-De-León et al. (2020)
FKS1	Point mutations	Alters glucan synthase structure, diminishing echinocandin binding	Kordalewska & Perlin (2022); Chaabane et al. (2019)
ERG6	Mutations	Disrupts ergosterol biosynthesis, thereby lowering polyene binding	Rybak et al. (2022)
SNQ2, CDR4, ARB1, MDR1, MRR1	Overexpression (often via regulatory mutations)	Enhances drug efflux, reducing intracellular concentrations of antifungal agents	Du et al. (2020); Chakrabarti & Sood (2021); (Zhou et al., 2021)

Table 1: key genetic manipulation/alterations and their impact on antifungal drug resistance in *Candida auris*

C. auris resistance thus involves multiple complex molecular mechanisms, including target site mutations, efflux pump overexpression, and metabolic changes like ergosterol depletion. Further elucidation of these pathways will inform strategies to overcome antifungal resistance in this emerging pathogen. Ongoing research is still needed to fully characterise the multidrug resistance of *C. auris* (Chaabane et al., 2019).

Candida auris: Current challenges in developing effective treatments

Candida auris is resistant to multiple classes of antifungal drugs, including azoles, echinocandins, and polyenes (Forsberg et al., 2019; M. Jain et al., 2022). This makes it difficult to find effective treatment options, as the few available drugs may be ineffective against *C. auris* infections (Černáková et al., 2021). *C. auris* can be misidentified as other yeasts by commonly available identification methods (Forsberg et al., 2019). This can lead to delayed or incorrect diagnosis, resulting in inappropriate treatment and potential spread of the infection (Černáková et al., 2021). *C. auris* has the ability to

form biofilms, which are naturally more resistant to drugs. Biofilms provide a protective environment for the fungus, making it harder for antifungal agents to penetrate and eradicate the infection (Černáková et al., 2021). *C. auris* can persist and colonise patients for extended periods, potentially indefinitely, and can persist in the healthcare environment (Forsberg et al., 2019). This ability to colonise patients and persist in the environment contributes to the spread of *C. auris* in healthcare settings (Forsberg et al., 2019). The limited number of available antifungal drugs effective against *C. auris* further complicates treatment (Černáková et al., 2021). The emergence of strains with multiple drug resistance is a cause for concern (Dany G. Kramer, 2022). Developing new antifungal agents with novel mechanisms of action is crucial to combating *C. auris* infections (M. Jain et al., 2022). *C. auris* has the potential to cause nosocomial outbreaks, especially in healthcare settings such as nursing homes and intensive care units (Chowdhary et al., 2018). Controlling the spread of *C. auris* requires robust infection prevention and control measures, including surveillance, communication, and environmental cleaning (Chowdhary et al., 2018).

Novel treatments for Candida auris

The emerging multidrug-resistant phenomenon in *Candida auris* resulted in nosocomial invasive infections and outbreaks in more than 47 countries worldwide and thus necessitated the development of novel therapeutic approaches, as current antifungals are often inadequate (Kamli et al., 2022; Xin et al., 2023). Several promising alternatives to supplement or replace conventional antifungals have recently emerged. Intravenous immunoglobulin (IVIG) has long been approved as a standard treatment for patients with immunodeficiency disorders who are also susceptible to fungal infection. Studies have shown that human IVIG can provide protection against *Candida auris* and *Candida albicans* disseminated infections in mouse models (Xin et al., 2023). Ibrexafungerp is a first-in-class triterpenoid antifungal agent that inhibits fungal cell wall glucan synthesis and demonstrates broad in vitro activity against *Candida* species, including *C. auris* (Ghannoum et al., 2020). Defensin-like peptide D-lp1 is a plant defensin peptide that exhibits fungicidal effects against *C. auris* by disrupting biofilms and virulence (Kamli et al., 2022). Rezafungin is a novel echinocandin with enhanced stability and pharmacokinetics that achieves high plasma drug exposure and allows for once-weekly dose administration. It has shown promising results in the treatment of disseminated *C. auris* infection using a mouse model of disseminated candidiasis (Hager et al., 2018). Two novel transdermal agents, 1% terbinafine and 1% clotrimazole in a proprietary Advanced Penetration Technology formulation (APT), have been shown to significantly reduce fungal burden compared to control groups in a murine skin colonisation model of *C. auris* (Ghannoum et al., 2020). While further research is needed, these emerging non-traditional antifungal therapies

show promise against *C. auris* and could supplement current suboptimal treatment options. Evaluation in clinical trials will determine their ultimate utility in combatting this challenging multidrug-resistant pathogen.

Combination therapy approaches against *Candida auris*

The multidrug resistance of *Candida auris* has compelled investigations into combination antifungal therapy. While clinical evidence remains limited, initial findings suggest potential synergies. In one of the cases, reports indicate combining echinocandins and amphotericin B may help overcome breakthrough infections with reduced echinocandin susceptibility (Al-Obaid et al., 2022). In vitro studies demonstrate synergistic interactions between Isavuconazole and Echinocandins against *C. auris*, with combination therapy superior to monotherapy (Caballero et al., 2021, 2023). In another study, photodynamic therapy, which involves using visible light in combination with photosensitising compounds, showed efficacy against *Candida auris* biofilms. Blue light alone inhibited and disrupted the biofilms, and the addition of photosensitising compounds improved its antibiofilm potential. Red light and green light also showed inhibitory effects when combined with photosensitising compounds (Bapat & Nobile, 2021). A clinical case report described the successful treatment of invasive *C. auris* infection using a three-drug regimen of micafungin, amphotericin B, and posaconazole (Shah et al., 2022).

While promising, clinical evidence for combination therapy against *C. auris* remains scarce, and optimal combinations are unclear. Additional in vivo and clinical studies are needed to validate synergies suggested by in vitro data. Until more robust evidence exists, combination antifungal therapy should be considered experimental and in consultation with infectious disease experts when facing refractory *C. auris* infections. Ongoing research on synergistic combinations is crucial for overcoming the treatment challenges posed by this multidrug-resistant yeast.

Candida auris: Potential future directions for research

Elucidating specific molecular mechanisms underlying *C. auris* virulence and skin colonisation could reveal novel therapeutic targets, as current understanding remains limited (Victor Garcia-Bustos, 2021; Watkins et al., 2022). Improving the accuracy and efficiency of *C. auris* diagnostic methods is essential, as conventional approaches lack sensitivity. Leveraging molecular techniques like PCR and MALDI-TOF MS could enable rapid confirmation (Dany G. Kramer, 2022). Given the paucity of effective treatments, discovering innovative antifungal agents with novel mechanisms is imperative (Victor Garcia-Bustos, 2021). Approaches such as antimicrobial peptides, combinational therapy, immunotherapies, nanoparticles, and drug repurposing merit exploration to overcome drug resistance (Bandara

& Samaranayake, 2022). Resilient infections like *C. auris* pose a risk of nosocomial transmission and potential high morbidity and mortality to patients with impaired immune systems (Chowdhary et al., 2018), thus optimising protocols for surveillance, communication, disinfection, and clinical management could limit future outbreaks (Giacobbe et al., 2021). Finally, elucidating *C. auris* pathogenesis and drug resistance mechanisms via genomic and transcriptomic approaches could provide vital insights. Comparative analyses of resistant isolates may inform the development of targeted therapies. (K. Jain et al., 2022; Watkins et al., 2022).

Candida auris: Conclusion and future outlook

The emerging fungal pathogen *Candida auris* represents a considerable public health threat, given its propensity for multidrug resistance and nosocomial transmission. Effective treatment poses challenges due to intrinsic resistance, biofilm formation, environmental persistence, and nosocomial transmission (Černáková et al., 2021; Chowdhary et al., 2018; Forsberg et al., 2019). Ongoing research aims to elucidate resistance mechanisms, develop diagnostic methods, and discover new therapies. Comparative genomics reveals the upregulation of drug efflux, ergosterol biosynthesis, and MAPK signalling genes in azole-resistant isolates (M. Jain et al., 2022). Improved speciation by MALDI-TOF MS and PCR overcomes the limitations of conventional methods (Ravichandran et al., 2022). New antifungal targets are being explored, including eugenol derivatives with anti-*C. auris* activity in preliminary tests (Alam et al., 2023). However, significant challenges remain in translating these findings into clinical treatments. Continued surveillance and outbreak readiness are critical as *C. auris* spreads globally (Meyer et al., 2021; Moore et al., 2022).

Exploring the molecular mechanisms conferring antifungal resistance in *C. auris* stands out as a research priority, as does characterising the epidemiology and virulence factors driving its global dissemination and pathogenicity. The development of rapid and accurate diagnostics platforms and novel disinfection protocols are urgently needed to enable early identification of *C. auris* and interrupt its spread in healthcare settings. Most crucially, the design of new antifungal agents and therapeutic strategies represents an urgent priority against this multidrug-resistant fungus. Ongoing research across diverse domains, including *C. auris* biology, diagnostics, drug development, and infection control is essential to fully understand and mitigate the public health challenges posed by this formidable fungal pathogen. In summary, advancing our understanding of *C. auris* pathogenesis while concurrently improving diagnostics, therapies, and infection control measures represents a key near-term goal to limit *C. auris*. Focused efforts bridging these multiple areas are critical to counter the public health threats posed by the emergence of *C. auris*.

Author's contribution

Vishal Bhoir: conducted literature review, drafted the manuscript and editing; Dr Bela Nabar reviewed and approved the final version.

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Conflict of Interest

The authors declare no conflict of interest.

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