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(REVIEW ARTICLE)

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# *Candida Auris*: Review on worldwide spread, seems prone to antifungal and disinfectant therapies

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## Abstract

In recent years, *C. auris* has become a leading source of invasive fungal diseases, with outbreaks occurring across all continents. This fungus infection poses a severe hazard to healthcare, with outbreaks primarily affecting older patients with comorbidities and high fatality rates. Controlling epidemics has been challenging because to poor detection, fast spread, and resistance to environmental cleaning methods. *C. auris* is currently the major cause, or one of the leading causes of invasive fungal infections in many healthcare facilities, primarily due to its potential may exhibit or acquire resistance to many types of antifungal medications and due to their Ability to persevere in healthcare circumstances. Timely diagnosis by swift and reliable identification. techniques and dedication in infection control procedures can assist to limit the spread of *C. auris*, and prevent and manage epidemics

**Keywords:** *Candida Auris*; Antimicrobial Resistance; Environmental Conditions; Treatments; Hospital Outbreaks

## 1. Introduction

Infections caused by fungi are being growing in prominence as a global crisis to human health. Fungal infections affect approximately 1.7 billion individuals globally, with the majority of cases being superficial infections of the skin and mucosa [1]. Candida species are the most common cause of nosocomial fungal infections and the fourth greatest cause of hospital-acquired illnesses [2]. Annually, there are around 400,000 bloodstream infections caused by Candida species worldwide, with death rates surpassing 40%. Candida albicans is one of the most often seen Candida species, but non-albicans species such Candida tropicalis, Candida parapsilosis, and Candida glabrata have become more common in recent decades due to confined antifungal medication choices [3,4]. *Candida Auris*, an unfamiliar species of the Candida/Clavispora category, was first identified in a female patient's ear discharge in Japan in 2009 [5]. Considering the fact that the initial isolate of *Candida Auris* was identified more than a decade ago, there is still a worldwide screening strategy insufficient, as well as in underdeveloped countries, and there is an urgent need for effective therapy against this multidrug-resistant bacteria [8.7]. Indeed, *C. auris* has been detected in over 35 countries, with the majority of cases attributed to healthcare-associated person-to-person transmission [8].

*C. auris* has a high rate of transmission due to its capacity to colonize skin and other body locations and survive for weeks on abiotic surfaces and equipment [9]. The main concerns of *C. auris* infection include the high prevalence of antifungal treatment resistance [10], the ease of transmission among healthcare staff and patients in hospitals, and misidentification. The difficulty in correctly identifying *C. auris* strains, which are often misidentified as other pathogens, as well as inadequate diagnostic tools in many countries, leads to misidentification and incorrect therapy, resulting in multidrug resistance [11-14].

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*C. auris* has been linked to several types of malignant microbial infections. The most of documented data on patient infections and outcomes is derived from India, although there have also been reports from small numbers of patients in South Korea, Venezuela, South Africa, the United Kingdom, the United States, Colombia, and Canada. Invasive *C. auris* infection has been strongly linked to candidemia, including incidents related with CVC usage, as well as pericarditis, respiratory tract and urinary tract infections. *C. auris* infection is typically found in severely sick patients having surgical operations in intensive care units. Patients with significant underlying medical disorders, such as hematological malignancies or immunosuppressive diseases, are typically eligible for treatment. A study described a case of *C. auris* infection from a donor after lung transplant. Yeast was found in bronchoalveolar lavage samples before and after implantation, but was originally misinterpreted by biochemical and molecular tests.[15]. As in Table 1.

**Table 1** *Candida Auris* Infection cases by disease type reported in the literature

| Sr. No | Location of Isolation | Number of cases |
|---|---|---|
| 1 | Candidema | 291 |
| 2 | Central Venous Catheter Tip | 2 |
| 3 | CNS | 1 |
| 4 | ENT | 21 |
| 5 | Respiratory Tract | 18 |
| 6 | Urogenital System | 17 |
| 7 | Abdominal | 13 |
| 8 | Surgical Wounds on Skin and Soft Tissues | 12 |
| 9 | Bone | 2 |

## 2. Climate changes

The changing climate has a significant influence on malignant fungal infections. Pathogenic fungus, unlike humans, adapt fast and become more virulent as temperatures rise. Changes in fungal disease epidemiology have led to the creation of novel diseases, such as *Candida Auris*, which can tolerate high temperatures and adapt to human bodies. [16-18] Most fungal organisms cannot survive at human temperatures (36.5-37.5˚C and up to 40˚C during fever), preventing them from colonizing and causing diseases. Unlike similar Candida species, *C. auris* may thrive at high temperatures (>40˚C) [19-21]. An investigation analyzing the temperature resistance of *C. auris* to other Candida species indicated that climate change, notably global warming, may have influenced the pathogen's capacity to thrive at high temperatures [22]. *C. auris*, unlike other Candida species, can withstand higher salt concentrations (>10% NaCl, wt/vol) [19-21]. *C. auris* responds to high salt concentrations by forming pseudo hyphae-like morphologies, which may be adaptive under stressful conditions [19-21]. Thermotolerance and osmotolerance are properties that may contribute to *C. auris*' endurance and existence on biotic and abiotic surfaces for long periods of time [23-25].

Fungal pathogens can adapt to one environmental threat, making them more resilient to future challenges. This observation applies to a variety of human-induced factors that contribute to climate change. Fungal melanin protects against extreme temperature and pH stress, heavy metals, and radioactive isotopes, allowing some fungal taxa to thrive in metal-polluted areas, acidic environments, and radioactive wastelands following nuclear disasters.[26]. The presence of agricultural and industrial contaminants in rivers, lakes, and tap water significantly increases the chance of developing fungal illnesses. Contamination in water bodies and supply systems has been linked to increased proliferation and variety of fungus, including pathogenic species. Polluting substances like nitrates and iron can provide a favorable pH and nutritional environment for fungal development.[27] Contamination in freshwater can lead to microbial infections and threaten health care systems, even in developed countries.[28-30]

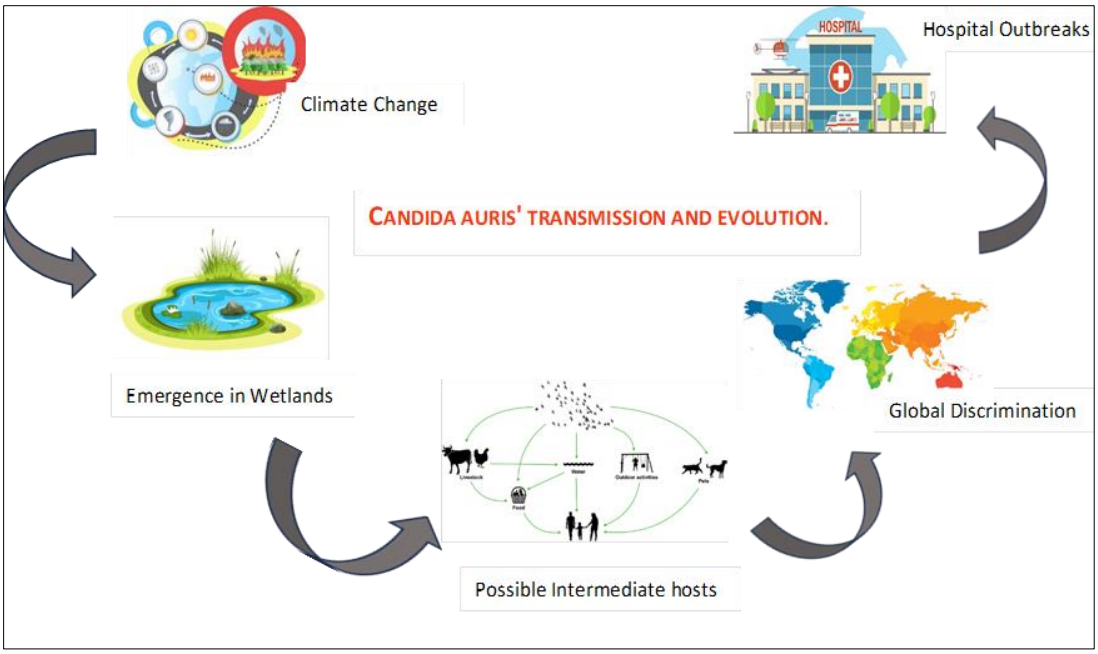


**Figure 1** Climate change effect in the emergence of *Candida Auris*

Rapid urbanization impacts both environmental change and microbiological ecosystems in different ways. Increased urban population density, along with industrial and biological variables like diminished vegetation cover, leads to elevated air and soil temperatures, as well as streams warming.[31]. Heat islands in urban areas set evolutionary pressure on microorganisms, resulting in increased fungal stress adaptability compared to rural isolates from neighboring locations.[32] Climate change impacts both fungal adaptability and host vulnerability to pathogenic fungus. UV radiation exposure has been related to negative effects on human immunity, including T-cell polarization, increased production of inhibitory cytokines, and altered complement activation.[33].

## 3. Biology

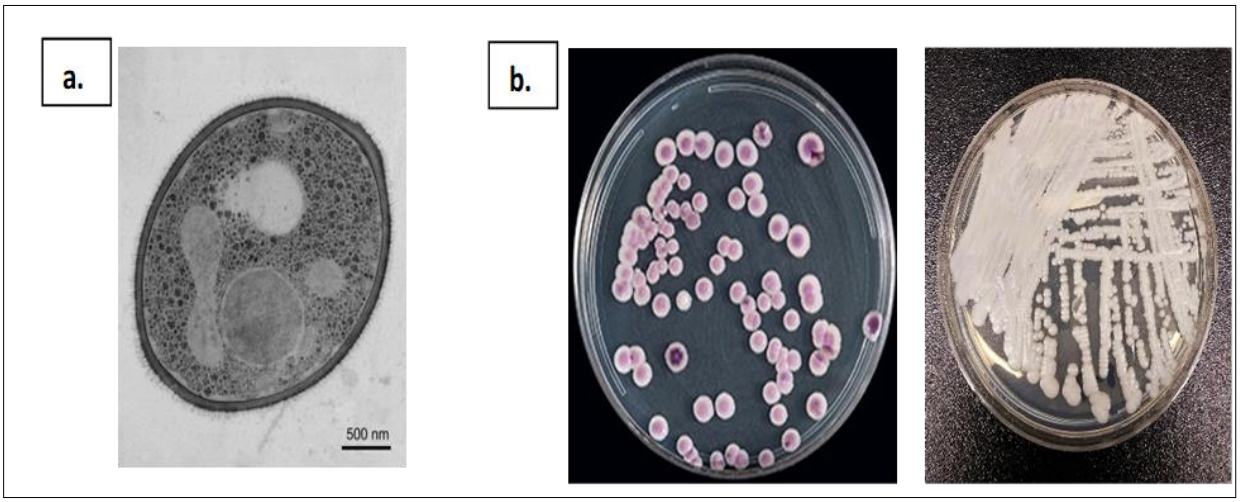


**Figure 2** a: *C. auris* Cell under Microscope. b: Developed colonies of the fungi in Chromogenic Agar Candida medium

- **Cell Biology:** *C. auris* develops pink to beige colonies on chromogenic agar Candida medium and grows well at 42°C, but growth is inconsistent at higher temperatures and does not occur in the presence of 0.01% cycloheximide. It produces oval or elongated yeast cells that might appear alone, in pairs, or in clusters. Importantly, no hyphal or pseudohyphal variants have been identified. [34]
- **Morphological Transitions:** *C. auris*, like other pathological Candida species, displays a variety of morphological phenotypes[35-38], while the regulatory mechanisms and significance of each morphology in *C. auris* remain unclear. Many *C. auris* isolates occur as single-celled yeast. However, certain natural *C. auris* isolates can produce huge clusters of pseudohyphal-like cells, with mother and daughter cells still connected [39-40]. In the Galleria mellonella infection model, aggregating cells are less virulent than non-aggregating cells, while being more resistant to antifungal drugs [41]. Pseudohyphal-like clusters in *C. auris* may indicate problem in cell division. Recent research found that genotoxic stressors induced DNA damage and disrupted replication forks in *C. auris*, leading to pseudohyphal development [42].
- Microorganisms often exploit morphological plasticity to adapt quickly to environmental changes [43,44]. Under specific environmental circumstances, both bacterial and fungal species can undergo morphological changes. Pathogenic Candida species, including C. albicans and C. tropicalis, can undergo a variety of morphological changes [45,46]. They can flip between cell types on their own or in response to environmental signals. C. albicans and C. tropicalis, for example, have well-studied morphological changes such as the yeast-hyphal transition and the white-opaque flip. Morphological flexibility in these species is crucial for disease and mating [45,46].
- Phenotypic shift occurs within colonies. Growing *C. auris* colonies on CHROMagar allows for morphological changes between pink, white, and dark purple phenotypes [47].
- **Filamentation:** Pathogenic Candida species rely on filamentous (hyphal or pseudohyphal) cell development to invade host tissues. The shift from yeast to filamentous growth forms of C. albicans has been extensively studied. Initially, it was thought that *C. auris* could only create pseudohyphae and not real hyphae. Recent research suggests that *C. auris* isolates can generate genuine hyphae under certain conditions.
- **Development of biofilms:** Biofilms are organized microbial communities that grow on both abiotic and biotic surfaces and are embedded in an extracellular matrix [48]. *C. auris* has been shown to create biofilms on surfaces, however these biofilms are weaker than those produced by C. albicans [36]. *C. auris* biofilm cells, like C. albicans biofilm cells, are more resistant to antifungal drugs than free-floating planktonic cells. *C. auris* biofilms have a role in the virulence, antifungal resistance, and survival of *C. auris* in the environment and host, albeit their relevance is unclear compared to other Candida species. Future research should focus on developing treatment techniques that target *C. auris* biofilms in both patients and the environment.

## 4. Rise and widespread within years

### 4.1. The development of *Candida Auris* as major pathogen

Whereas *C. auris* was initially identified in 2009, retroactive investigations of culture collections have revealed other *C. auris* isolates that were previously mistaken as C. haemulonii, including a bloodstream strain collected in 1996 [51,52]. *C. auris* is phylogenetically similar to other members of the C. haemulonii complex [53,54]. *C. auris* is extremely clonal, although whole genome sequencing analysis have discovered five different clades separated by thousands of single-nucleotide polymorphisms [54-56]. There are five geographically distinct clades: South Asian (Clade I), East Asian (Clade II), African (Clade III), South American (Clade IV), and Iranian (Clade V). Clade I, III, and IV isolates usually produce invasive infections and outbreaks, but Clade II isolates have a preference for the ear, which is uncommon among other isolates [58]. The Clade V isolate was originally discovered in a case of otomycoses [57,59].

*C. auris* strains differ in their resistance to fluconazole and susceptibility to other antibiotics, including echinocandins and amphotericin B. Many isolates are multidrug-resistant [40,71,73,89,95]. Several *C. auris* clades developed on various continents almost concurrently.

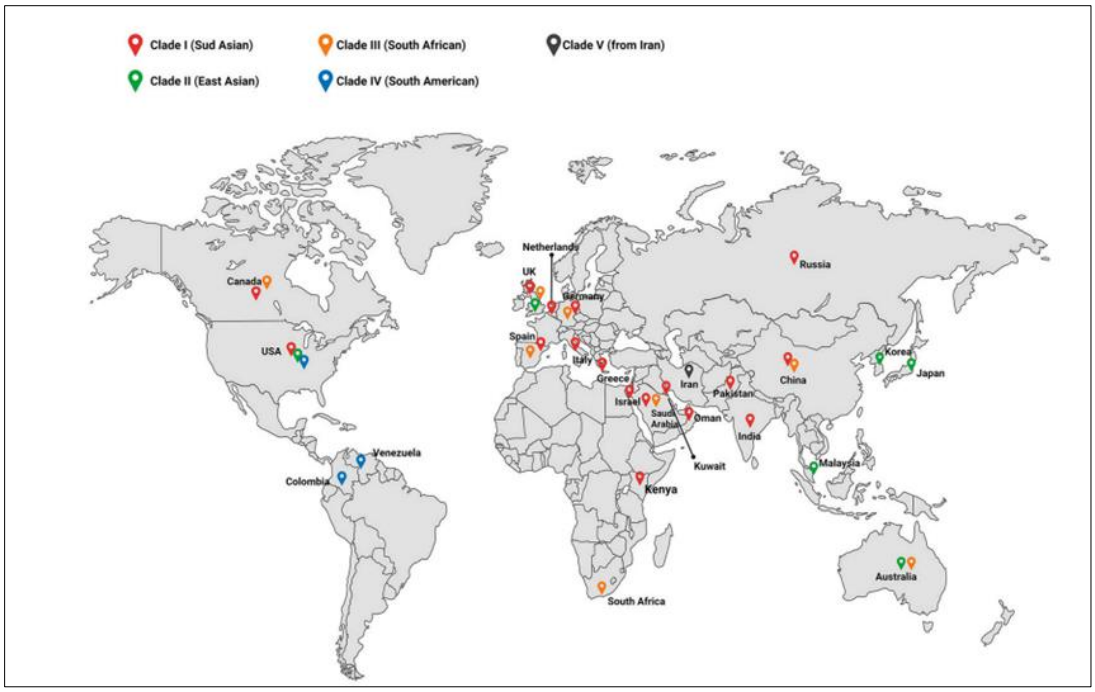


**Figure 3** A map showing the key clades of *C. auris*'s global distribution

### 4.2. *C. auris* alongside Covid-19

Research suggests that individuals with severe COVID-19 infection in the early stages of the pandemic are more likely to develop bacterial and fungal illnesses [65-66]. The COVID-19 pandemic may have led to increased spread of *C. auris*. This might be due to strain on the global healthcare system and inadequate infection prevention and control measures. The pandemic may have made it difficult to discover new instances [56]. During the peak of the pandemic, outbreaks of *C. auris* among extremely sick COVID-19 patients were only recorded in certain countries, including Italy and the USA [68]. During the pandemic, an epidemiological investigation in the UAE indicated an increase in COVID-19 and *C. auris* co-infection in hospitals with overburdened intensive care units [70]. Patients with major comorbidities received broad-spectrum antibiotics and immunosuppressive treatments. Additionally, they used urine and intravenous catheters [71]. An epidemic of *C. auris* was discovered in an ICU with COVID-19 patients in India. The mortality rate was high. Infection onset occurred 3-8 weeks after hospitalization. The majority of patients had central venous and urine catheters, required mechanical breathing, and had underlying chronic illnesses such diabetes and hypertension [72]. During the pandemic, Spain and Italy saw a rise in *C. auris* and multidrug-resistant organism infections, including nosocomial outbreaks in COVID-19 patients admitted to critical care units [72-74].

## 5. Common commercial antifungals and *candida auris* treatment failure:

*Candida Auris*, a multidrug-resistant species, causes substantial morbidity and death in immunocompromised persons globally [74]. *C. auris* has several critical properties, including easy dissemination in the environment, rapid transit among hospitalized patients [75,76], and resistance to standard disinfectants in healthcare settings [77]. *C. auris* is a potentially aggressive and worldwide developing disease, posing issues for infection control due to limited microbiological diagnostic tools and significant levels of antifungal resistance [78-81].

There are few antifungal medicines available for treating *C. auris* infections. In clinics, three types of antifungals (azoles, polyenes, and echinocandins) are often used to treat infected individuals [82]. Antifungal drugs significantly affect the development of *C. auris* in both planktonic and sessile states. *C. auris* can develop biofilms on medical surfaces in immunocompromised patients, as well as bio-surfaces [83,84]. The type and phenotypic characteristics of the isolates are significantly related [85]. Biofilms can lead to antifungal resistance and chronic infections in this species [86]. Antifungal resistance in *C. auris* is largely attributed to its biofilm characteristic [87].

### 5.1. Mechanisms of molecular resistance

The majority of clinical *C. auris* isolates are resistant to key antifungal classes (azoles, polyenes, or echinocandins), including multi-drug resistance (MDR) to more than two classes [13]. Research has focused on the molecular processes

of resistance development, leading to limited treatment options [13,89]. Accurate detection and identification of the *C. auris* pathogen is crucial for successful therapy [90,91]. Setting MICs for antifungals against *C. auris* strains has been requested, as increased MICs are a serious issue [92]. Monitoring antifungal resistance in various locations is crucial, as is implementing effective treatment recommendations [93].

### 5.2. Azole Resistance Mechanisms

Azole resistance in *C. auris* is acquired through various mechanisms, including overexpression of efflux pumps such as ATP-binding cassette (ABC) and major facilitator superfamily (MFS) transporters, which are known to cause deliberate multidrug resistance [94,95]. The *C. auris* genome includes three Mrr1 homologs and two Tac1 homologs. Mutations in the zinc cluster transcription factors Mrr1 and Tac1 lead to *C. auris*' intrinsic flu resistance [96].

### 5.3. Echinocandin Resistance Mechanisms

Echinocandins are the primary treatment for *C. auris* infections and seldom acquire resistance [13,97]. Resistance to echinocandins is often linked to mutations in the FKS1 gene, which encodes the enzyme (1,3)-β-D-glucan synthase. These mutations reduce the enzyme's affinity for the drug [98]. Sequencing 38 *C. auris* strains revealed an S639F amino acid alteration linked to pan-echinocandin resistance [99]. This mutation aligns with C. albicans FKS1's HS1 region [100].

### 5.4. Flucytosine resistance mechanisms (5-fluorocytosine)

Flucytosine, a nucleoside analog, prevents nucleic acid synthesis. Because this antifungal chemical is less often used than other medications, the mechanism of resistance is less understood, and less research have been conducted to determine *C. auris* resistance to this treatment. Mutations in the FUR1 gene have been linked to flucytosine resistance in non-*Candida Auris* Candida [101], whereas mutations in the FCY2 and FCY1 genes may also cause resistance to 5-fluorocytosine [102]. In resistant *C. auris*, a previously unknown missense mutation in the FUR1 gene resulted in an F211I amino acid substitution. Further research is needed to determine the underlying processes for the resistance to flucytosine tested in *Candida Auris* strain.[103].

### 5.5. Polyene Resistance Mechanisms

The processes behind polyene resistance in *C. auris* are poorly known. Whole-genome sequencing of resistant isolates revealed four unique non-synonymous mutations that are likely linked to AmB resistance. Overexpression of mutant ERG genes causes a drop in ergosterol concentration in the cellular membrane [13, 97] [98]. Mutations were found in genes related to C. albicans' FLO8 transcription factor and a membrane transporter [99].

**Table 2** Antifungal drugs are often employed to treat *C. auris* infections, and resistance mechanisms have been documented

| Antifungal Drugs Used | Mechanism of Action | Resistance Mechanism |
|---|---|---|
| Azoles | Inhibit the activity of lanosterol 14-α-demethylase enzyme; prevent converting lanosterol to ergosterol, leading to damaging integrity of cell membrane. | Overexpression of ATP-binding cassette (ABC) and major facilitator superfamily (MFS) transporters. ERG11 point mutation: Y132F and K143R. Mutation in zinc cluster transcription factors Mrr1 and Tac1. |
| Polyenes | Bind ergosterol molecules in the cytoplasmic membrane; disturb the permeability of cell membrane by formation of pores, causing oxidative damage. | Induction of genes associated with sterol biosynthetic process including ERG1, ERG2, ERG6, and ERG13. SNPs in different genomic loci related to increased resistance. |
| Echinocandins | Inhibit β-(1,3)-D-glucan synthase enzyme, leading to defective cell wall formation. | Hot-spot mutation in FKS1 gene associated with S639Y, S639P, and S639Y regions and FKS2. |
| Flucytosine | Inhibit the nucleic acid synthesis (DNA and RNA) of fungi. | Mutation of FUR1 gene, specifically missense mutation of FUR1 causing F211I amino acid substituted in the FUR1 gene in one flucytosine-resistant isolate. Mutations in the FCY2, FCY1 genes |

## 6. Novel treatments and combination therapies

To prevent *C. auris* infections, novel therapies, medicines, and technologies are being evaluated urgently. One of the potential ways appears to be synergistic interactions between chemicals and antifungals.

Researchers are exploring novel chemicals with anti-fungal properties to combat *C. auris* growth, both in vitro and in vivo. APX001A, a new inhibitor of the fungal protein Gwt1 (glycosylphosphatidylinositol-anchored wall transfer protein 1), has been tested against *C. auris*. In vitro and in an immunocompromised mouse model with disseminated infection, APX001A demonstrated effective antifungal efficacy with lower MIC50 and MIC90 than anidulafungin [100]. New antimicrobial peptides may be more effective than chemical antimicrobials in combating multidrug-resistant species due to their decreased risk of causing resistance [102].

Probiotic yeasts have been shown in clinical studies to effectively inhibit the growth of Candida spp., including C. albicans, Candida tropicalis, C. glabrata, Candida parapsilosis, Candida krusei, and *C. auris*. S. cerevisiae var. boulardii is the only commercially available probiotic yeast that may lower pathogen pathogenicity by blocking Candida spp. adherence and morphological transition, making it a prospective treatment alternative. [103] Evidence suggests that administering micafungin and AmB together may have therapeutic utility against *C. auris* and may be a good alternative to flucytosine [104,105], which can be hazardous to some patient populations with bone marrow disorders or to expectant mothers [106,107,108]. Currently, screening focuses on compounds with inhibitory effects, such as sulfamethoxazole, which exhibits strong synergistic interactions with Vcz and itraconazole in vitro [109]. Lopinavir inhibits HIV protease, but when combined with itraconazole, it has a synergistic impact against *C. auris* [110]. This drug sensitized C. albicans, C. tropicalis, C. krusei, and C. parapsilosis to azoles, most likely via altering efflux pump activity [111]. A previous study found synergistic interactions between suloctidil (a sulfur-containing aminoalcohol vasodilator) and Vcz, as well as ebselen (a synthetic organoselenium molecule) and anidulafungin [112]. Aprepitant, a medication used to alleviate nausea and vomiting, can also improve the antifungal activity of azoles against C. aureus by chemosensitizing [113]. The authors found that aprepitant/itraconazole interfered with metal ion homeostasis, resulting in reduced detoxifying capabilities of reactive oxygen species. [114]

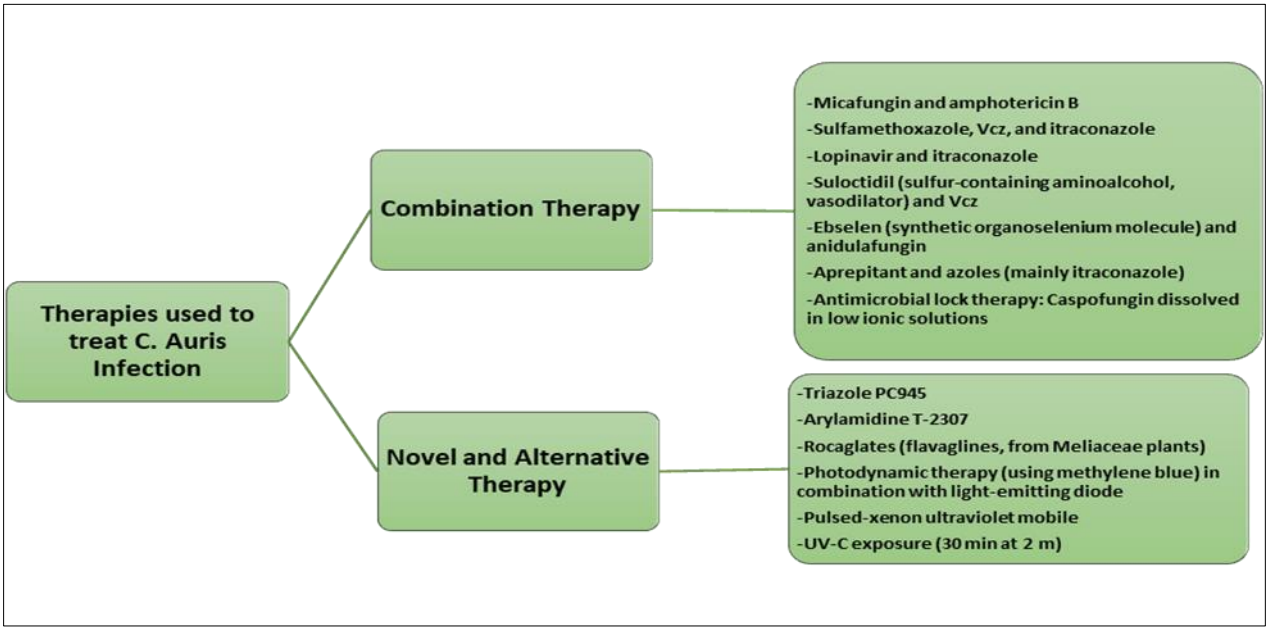


**Figure 4** Novel Alternative Therapies

Candida biofilms on indwelling catheters are difficult to treat. Antimicrobial lock treatment is a non-invasive method for removing persistent cells from a catheter.[115] Sumiyoshi and colleagues picked this technique. Caspofungin in low ionic solutions significantly reduced candidal biofilms, including resistant *C. auris*, in the catheter-lock treatment model [116].

New antifungal agents have also been investigated. PC945, a triazole, outperformed posaconazole, Vcz, and Flu as an inhibitor of *C. auris* isolates. T-2307, an arylamidine, effectively inhibits fungal mitochondrial membrane potential and has been tested against Candida species in vitro and in vivo, as well as *C. auris* [108]. In relation to novel and alternative

treatments , it was discovered that *C. auris* was surprisingly susceptible to translation inhibition by a class of compounds called rocaglates, which are naturally occurring products found in Meliaceae plants. These compounds activated a program that leads to cell death, and as a result, they demonstrated fungicidal activity against this yeast [109].

Another option is to irradiate photoactive dyes with a light source of the proper wavelength, resulting in the generation of reactive oxygen species, which is known as photodynamic treatment. Combining methylene blue with a light-emitting diode decreased the survivability of *C. auris*, both planktonic and biofilm. Finally, a pulsed-xenon UV transportable device successfully decreased the colony forming unit survival of *C. auris* .UV-C radiation was explored as a potential method of room disinfection. The greatest results for *C. auris* death were achieved after 30 minutes of UV-C exposure at 2 metres. *C. auris* may survive on surfaces for up to two weeks [114].

## 7. Conclusion

### 7.1. Questions and perspectives on the future

*C. auris* represents a new public global health hazard. *C. auris* is closely related to other pathogenic Candida species, but has distinct characteristics in biology, genetics, epidemiology, antifungal resistance, virulence, host adaptability, and transmission. At the moment, therapy options that are new or unconventional include photodynamic therapy, innovative triazoles (like PC945), and natural compounds (such rocaglates). Antimicrobial stewardship initiatives can improve patient outcomes, minimize antibiotic side effects, and decrease antimicrobial resistance. New diagnostic technologies have improved patient care and infection control, minimizing the risk of *C. auris* spread. To effectively prevent and treat *C. auris*, a collaborative effort among clinicians, laboratories, and healthcare institutes is necessary.

The emergence and biology of *C. auris* remain largely unknown, despite the fact that it has recently attracted a great deal of scientific research. What are the original habitats for *C. auris*? How did genetically diverse isolates arise globally simultaneously? How did multidrug resistance develop in *C. auris*? What allows *C. auris* to survive in clinical settings for extended periods of time? Is *C. auris* capable of sexual or parasexual reproduction, and if so, how did it become a pathogen? More study is needed to address these concerns.

We need to investigate the biology and genetics of antifungal resistance and pathogenicity in *C. auris*. Developing speedy and effective detection methods for *C. auris* can aid in identifying infections and distinguishing it from other Candida species. We will create disinfection techniques to effectively remove *C. auris* from surfaces, preventing future outbreaks. To tackle infections caused by *C. auris* and other fungal diseases, new and safe antifungals and treatment regimens with various pharmacological targets are needed.

## Compliance with ethical standards

*Disclosure of conflict of interest*

No conflict of interest to be disclosed.

## References

[1] Brown GD, Denning DW, Gow NA, Levitz SM, Netea MG, White TC. Hidden killers: human fungal infections. Sci Transl Med. 2012; 4(165):165rv13. https://doi.org/10.1126/scitranslmed.3004404 PMID: 23253612.

[2] Wisplinghoff H, Bischoff T, Tallent SM, Seifert H, Wenzel RP, Edmond MB. Nosocomial bloodstream infections in US hospitals: analysis of 24,179 cases from a prospective nationwide surveillance study. Clin Infect Dis. 2004; 39(3):309–317. Epub 2004 Aug 13. https://doi.org/10.1086/421946 PMID: 15306996.

[3] Pfaller MA. Epidemiology of candidiasis. J Hosp Infect. 1995; 30:329–338. https://doi.org/10.1016/ 0195-6701(95)90036-5 PMID: 7560969.

[4] Pfaller MA, Diekema DJ. Epidemiology of invasive candidiasis: a persistent public health problem. Clin Microbiol Rev. 2007; 20(1):133–163. https://doi.org/10.1128/CMR.00029-06 PMID: 17223626

[5] Satoh K, Makimura K, Hasumi Y, Nishiyama Y, Uchida K, Yamaguchi H. *Candida Auris* sp. nov., a novel ascomycetous yeast isolated from the external ear canal of an inpatient in a Japanese hospital. Microbiol Immunol. 2009; 53(1):41–44. https://doi.org/10.1111/j.1348-0421.2008.00083.x PMID: 19161556.

[6] Alvarado, M.; Bartolomé Álvarez, J.; Lockhart, S.R.; Valentín, E.; Ruiz-Gaitán, A.C.; Eraso, E.; de Groot, P.W.J. Identification of *Candida Auris* and related species by multiplex PCR based on unique GPI protein-encoding genes. Mycoses 2021, 64, 194–202.

[7] Abastabar, M.; Haghani, I.; Ahangarkani, F.; Rezai, M.S.; Taghizadeh Armaki, M.; Roodgari, S.; Kiakojuri, K.; Al-Hatmi, A.M.S.;Meis, J.F.; Badali, H. *Candida Auris* otomycosis in Iran and review of recent literature. Mycoses 2019, 62, 101–105.

[8] Saris, K.; Meis, J.F.; Voss, A. *Candida Auris*. Curr. Opin. Infect. Dis. 2018, 31, 334–340.

[9] Welsh, R.M.; Bentz, M.L.; Shams, A.; Houston, H.; Lyons, A.; Rose, L.J.; Litvintseva, A.P. Survival, persistence, and isolation of theemerging multidrug-resistant pathogenic yeast *Candida Auris* on a plastic health care surface. J. Clin. Microbiol. 2017, 55, 2996–3005.

[10] Hernando-Ortiz, A.; Mateo, E.; Perez-Rodriguez, A.; de Groot, P.J.W.; Quindós, G.; Erasoa, E. Virulence of *Candida Auris* from different clinical origins in Caenorhabditis elegans and Galleria mellonella host models. Virulence 2021, 12, 1063–1075.

[11] Forsberg, K.; Woodworth, K.; Walters, M.; Berkow, E.L.; Jackson, B.; Chiller, T.; Vallabhaneni, S. *Candida Auris*: The recent emergence of a multidrug-resistant fungal pathogen. Med. Mycol. 2019, 57, 1–12.

[12] Kordalewska, M.; Perlin, D.S. Identification of Drug Resistant *Candida Auris*. Front. Microbiol. 2019, 10, 1918.

[13] Mirabet, V.; Salvador, C.; Valentín, A.; Escobedo-Lucea, C.; Navarro, L.; Gimeno, C.; Pemán, J. Risk assessment of arterial allograft contamination from tissue donors colonized by *Candida Auris*. J. Hosp. Infect. 2021, 112, 49–53.

[14] Chakrabarti, A.; Sood, P. On the emergence, spread and resistance of *Candida Auris*: Host, pathogen and environmental tipping points. J. Med. Microbiol. 2021, 70, 001318.

[15] Jeffery-Smith A, Taori SK, Schelenz S, Jeffery K, Johnson EM, Borman A, *Candida Auris* Incident Management Team, Manuel R, Brown CS. *Candida Auris*: a review of the literature. Clinical microbiology reviews. 2018 Jan;31(1):10-128.

[16] Nnadi NE, Carter DA. Climate change and the emergence of fungal pathogens. PLoS Pathog 2021; 17: e1009503.

[17] Garcia-Bustos V, Cabañero-Navalon MD, Ruiz-Gaitán AC, et al. Climate change, animals, and *Candida Auris*: insights into the ecological niche of a new species from a One Health approach. Clin Microbiol Infect 2023; 29: 858–62.

[18] Casadevall A, Kontoyiannis DP, Robert V. Environmental *Candida Auris* and the global warming emergence hypothesis. mBio 2021; 12: e00360-21.

[19] Wang X, Bing J, Zheng Q, Zhang F, Liu J, Yue H, et al. The first isolate of *Candida Auris* in China: clinical and biological aspects. Emerg Microbes Infect. 2018; 7(1):93. https://doi.org/10.1038/s41426-018- 0095-0 PMID: 29777096.

[20] Tian S, Rong C, Nian H, Li F, Chu Y, Cheng S, et al. First cases and risk factors of super yeast *Candida Auris* infection or colonization from Shenyang, China. Emerg Microbes Infect. 2018; 7(1):128. Epub 2018 Jul 12. https://doi.org/10.1038/s41426-018-0131-0 PMID: 29992959.

[21] Welsh RM, Bentz ML, Shams A, Houston H, Lyons A, Rose LJ, et al. Survival, persistence, and isolation of the emerging multidrug-resistant pathogenic yeast *Candida Auris* on a plastic health care surface. J Clin Microbiol. 2017; 55(10):2996–3005. Epub 2017 Jul 28. https://doi.org/10.1128/JCM.00921-17 PMID: 28747370.

[22] Casadevall A, Kontoyiannis DP, Robert V. On the emergence of *Candida Auris*: climate change, azoles, swamps, and birds. mBio. 2019; 10(4). https://doi.org/10.1128/mBio.01397-19 PMID: 31337723

[23] Biswal M, Rudramurthy SM, Jain N, Shamanth AS, Sharma D, Jain K, et al. Controlling a possible outbreak of *Candida Auris* infection: lessons learnt from multiple interventions. J Hosp Infect. 2017; 97 (4):363–370. https://doi.org/10.1016/j.jhin.2017.09.009 PMID: 28939316.

[24] Sekyere JO. *Candida Auris*: a systematic review and meta-analysis of current updates on an emerging multidrug-resistant pathogen. Microbiologyopen. 2018; 7(4):e00578. Epub 2018 Jan 19. https://doi.org/

[25] Kean R, Sherry L, Townsend E, McKloud E, Short B, Akinbobola A, et al. Surface disinfection challenges for *Candida Auris*: an in-vitro study. J Hosp Infect. 2018; 98(4):433–436. https://doi.org/10.1016/ j.jhin.2017.11.015 PMID: 29203448.

[26] Robert VA, Casadevall A. Vertebrate endothermy restricts most fungi as potential pathogens. J Infect Dis 2009; 200: 1623–26.

[27] Cordero RJ, Casadevall A. Functions of fungal melanin beyond virulence. Fungal Biol Rev 2017; 31: 99–112.

[28] Ortiz-Vera MP, Olchanheski LR, da Silva EG, et al. Influence of water quality on diversity and composition of fungal communities in a tropical river. Sci Rep 2018; 8: 14799.

[29] Caggiano G, Diella G, Triggiano F, et al. Occurrence of fungi in the potable water of hospitals: a public health threat. Pathogens 2020; 9: 783

[30] Jenks JD, Aneke CI, Al-Obaidi MM, et al. Race and ethnicity: risk factors for fungal infections? PLoS Pathog 2023; 19: e1011025.

[31] Jenks JD, Prattes J, Wurster S, et al. Social determinants of health as drivers of fungal disease. EClinicalMedicine 2023; 66: 102325.

[32] Chapman S, Watson JEM, Salazar A, Thatcher M, McAlpine CA. The impact of urbanization and climate change on urban temperatures: a systematic review. Landsc Eco 2017; 32: 1921–35.

[33] McLean MA, Angilletta MJ, Williams KS. If you can't stand the heat, stay out of the city: thermal reaction norms of chitinolytic fungi in an urban heat island. J Therm Biol 2005; 30: 384–91.

[34] Norval M, Halliday GM. The consequences of UV-induced immunosuppression for human health. Photochem Photobiol 2011; 87: 965–77

[35] Oh BJ, Shin JH, Kim MN, Sung H, Lee K, Joo MY, et al. Biofilm formation and genotyping of Candida haemulonii, Candida pseudohaemulonii, and a proposed new species (*Candida Auris*) isolates from Korea. Med Mycol. 2011; 49(1):98–102. https://doi.org/10.3109/13693786.2010.493563 PMID: 20560864.

[36] Bentz ML, Sexton DJ, Welsh RM, Litvintseva AP. Phenotypic switching in newly emerged multidrugresistant pathogen *Candida Auris*. Med Mycol. 2018. Epub 2018 Oct 18. https://doi.org/10.1093/mmy/ myy100 PMID: 30329075.

[37] Wang X, Bing J, Zheng Q, Zhang F, Liu J, Yue H, et al. The first isolate of *Candida Auris* in China: clinical and biological aspects. Emerg Microbes Infect. 2018; 7(1):93. https://doi.org/10.1038/s41426-018- 0095-0 PMID: 29777096.

[38] Yue H, Bing J, Zheng Q, Zhang Y, Hu T, Du H, et al. Filamentation in *Candida Auris*, an emerging fungal pathogen of humans: passage through the mammalian body induces a heritable phenotypic switch. Emerg Microbes Infect. 2018; 7(1):188. Epub 2018 Nov 30. https://doi.org/10.1038/s41426-018-0187-x PMID: 30482894.

[39] Borman AM, Szekely A, Johnson EM. Comparative pathogenicity of United Kingdom isolates of the emerging pathogen *Candida Auris* and other key pathogenic Candida species. mSphere. 2016; 1(4). https://doi.org/10.1128/mSphere.00189-16 PMID: 27547827.

[40] Singh R, Kaur M, Chakrabarti A, Shankarnarayan SA, Rudramurthy SM. Biofilm formation by *Candida Auris* isolated from colonising sites and candidemia cases. Mycoses. 2019; 62(8):706–709. Epub 2019 May 28. https://doi.org/10.1111/myc.12947 PMID: 31132181.

[41] Bravo Ruiz G, Ross ZK, Gow NAR, Lorenz A. Pseudohyphal growth of the emerging pathogen *Candida Auris* is triggered by genotoxic stress through the S phase checkpoint. mSphere. 2020; 5(2). Epub 2020 Mar 13. https://doi.org/10.1128/mSphere.00151-20 PMID: 32161147.

[42] Justice SS, Hunstad DA, Cegelski L, Hultgren SJ. Morphological plasticity as a bacterial survival strategy. Nat Rev Microbiol. 2008; 6(2):162–168. https://doi.org/10.1038/nrmicro1820 PMID: 18157153.

[43] Jain N, Hasan F, Fries BC. Phenotypic switching in fungi. Curr Fungal Infect Rep. 2008; 2(3):180–188. https://doi.org/10.1007/s12281-008-0026-y PMID: 19768140

[44] Huang G. Regulation of phenotypic transitions in the fungal pathogen Candida albicans. Virulence. 2012; 3(3):251–261. https://doi.org/10.4161/viru.20010 PMID: 22546903.

[45] Whiteway M, Bachewich C. Morphogenesis in Candida albicans. Annu Rev Microbiol. 2007; 61:529– 553. Epub 2007 May 18. https://doi.org/10.1146/annurev.micro.61.080706.093341 PMID: 17506678.

[46] Biswas S, Van Dijck P, Datta A. Environmental sensing and signal transduction pathways regulating morphopathogenic determinants of Candida albicans. Microbiol Mol Biol Rev. 2007; 71(2):348–376. Epub 2007 Jun 8. https://doi.org/10.1128/MMBR.00009-06 PMID: 17554048

[47] Nobile CJ, Johnson AD. Candida albicans biofilms and human disease. Annu Rev Microbiol. 2015; 69:71–92. https://doi.org/10.1146/annurev-micro-091014-104330 PMID: 26488273.

[48] Hall-Stoodley L, Costerton JW, Stoodley P. Bacterial biofilms: from the natural environment to infectious diseases. Nat Rev Microbiol. 2004; 2(2):95–108. Epub 2004 Mar 26. https://doi.org/10.1038/ nrmicro821 PMID: 15040259

[49] Kim, M.N.; Shin, J.H.; Sung, H.; Lee, K.; Kim, E.C.; Ryoo, N.; Jung, S.I.; Park, K.H.; Kee, S.J.; Kim, S.J.; et al. Candida haemulonii and closely related species at 5 university hospitals in Korea: Identification, antifungal susceptibility and clinical features. Clin. Infect. Dis. 2009, 48, e57–e61.

[50] Lee, W.G.; Shin, J.H.; Uh, Y.; Kang, M.G.; Kim, S.H.; Park, K.H.; Jang, H.-C. First three reported cases of nosocomial fungemia caused by *Candida Auris*. J. Clin. Microbiol. 2011, 49, 3139–3142.

[51] Kathuria, S.; Singh, P.K.; Sharma, C.; Prakash, A.; Masih, A.; Kumar, A.; Meis, J.F.; Chowdhary, A. Multidrug-resistant *Candida Auris* misidentified as Candida haemulonii: Characterization by matrix-assisted laser desorption ionization-time of flight mass spectrometry and DNA sequencing and its antifungal susceptibility profile variability by Vitek 2, CLSI broth microdilution, and Etest method. J. Clin. Microbiol. 2015, 53, 1823–1830.

[52] Cendejas-Bueno, E.; Kolecka, A.; Alastruey-Izquierdo, A.; Theelen, B.; Groenewald, M.; Kostrzewa, M.; Cuenca-Estrella, M.; Gomez-Lopez, A.; Boekhout, T. Reclassification of the Candida haemulonii complex as Candida haemulonii (C. haemulonii group I), C. duobushaemulonii sp. Nov. (C. haemulonii group II), and C. haemulonii var. vulnera var. nov.: Three multiresistant human pathogenic yeasts. J. Clin. Microbiol. 2012, 50, 3641–3651.

[53] Lockhart, S.R.; Etienne, K.A.; Vallabhaneni, S.; Farooqi, J.; Chowdhary, A.; Govender, N.P.; Colombo, A.; Calvo, B.; Cuomo, C.A.; Desjardins, C.A.; et al. Simultaneous emergence of multidrug-resistant *Candida Auris* on 3 continents confirmed by whole-genome sequencing and epidemiological analyses. Clin. Infect. Dis. 2017, 64, 134–140.

[54] Sharma, C.; Kumar, N.; Pandey, R.; Meis, J.F.; Chowdhary, A. Whole genome sequencing of emerging multidrug-resistant *Candida Auris* isolates in India demonstrates low genetic variation. New Microbes New Infect. 2016, 13, 77–82.

[55] Chow, N.A.; de Groot, T.; Badali, H.; Abastabar, M.; Chiller, T.M.; Meis, J.F. Potential fifth clade of *Candida Auris*, Iran, 2018. Emerg. Infect. Dis. 2019, 25, 1780–1781.

[56] Kwon, Y.J.; Shin, J.H.; Byun, S.A.; Choi, M.J.; Won, E.J.; Lee, D.; Lee, S.Y.; Chun, S.; Lee, J.H.; Choi, H.J.; et al. *Candida Auris* clinical isolates from South Korea: Identification, antifungal susceptibility, and genotyping. J. Clin. Microbiol. 2019, 57, e01624-18.

[57] Abastabar, M.; Haghani, I.; Ahangarkani, F.; Rezai, M.S.; Taghizadeh Armaki, M.; Roodgari, S.; Kiakojuri, K.; Al-Hatmi, A.M.S.; Meis, J.F.; Badali, H. *Candida Auris* otomycosis in Iran and review of recent literature. Mycoses 2019, 62, 101–105.

[58] Chowdhary, A.; Anil Kumar, V.; Sharma, C.; Prakash, A.; Agarwal, K.; Babu, R.; Dinesh, K.R.; Karim, S.; Singh, S.K.; Hagen, F.; et al. Multidrug-resistant endemic clonal strain of *Candida Auris*, India. Eur. J. Clin. Microbiol. Infect. Dis. 2014, 33, 919–926.

[59] Chowdhary, A.; Prakash, A.; Sharma, C.; Kordalewska, M.; Kumar, A.; Sarma, S.; Tarai, B.; Singh, A.; Upadhyaya, G.; Upadhyay, S.; et al. A multicentre study of antifungal susceptibility patterns among 350 *Candida Auris* isolates (2009–17) in India: Role of ERG11 and FKS1 genes in azole and echinocandin resistance. J. Antimicrob. Chemother. 2018, 73, 891–899.

[60] Chaabane, F.; Graf, A.; Jequier, L.; Coste, A.T. Review on antifungal resistance mechanisms in the emerging pathogen *Candida Auris*. Front. Microbiol. 2019, 10, 2786.

[61] Lockhart, S.R.; Etienne, K.A.; Vallabhaneni, S.; Farooqi, J.; Chowdhary, A.; Govender, N.P.; Colombo, A.; Calvo, B.; Cuomo, C.A.; Desjardins, C.A.; et al. Simultaneous emergence of multidrug-resistant *Candida Auris* on 3 continents confirmed by whole-genome sequencing and epidemiological analyses. Clin. Infect. Dis. 2017, 64, 134–140.

[62] Kean, R.; Sherry, L.; Townsend, E.; McKloud, E.; Short, B.; Akinbobola, A.; Mackay, W.G.; Williams, C.; Jones, B.L.; Ramage, G. Surface disinfection challenges for *Candida Auris*: An in-vitro study. J. Hosp. Infect. 2018, 98, 433–436.

[63] Chowdhary, A.; Sharma, A. The lurking scourge of multidrug resistant *Candida Auris* in times of COVID-19 pandemic. J. Glob. Antimicrob. Resist. 2020, 22, 175–176.

[64] Tsai, C.S.; Lee, S.S.; Chen, W.C.; Tseng, C.H.; Lee, N.Y.; Chen, P.L.; Li, M.C.; Syue, L.S.; Lo, C.L.; Ko, W.C.; et al. COVID-19- associated candidiasis and the emerging concern of *Candida Auris* infections. J. Microbiol. Immunol. Infect. 2023, 56, 672–679.

[65] Sticchi, C.; Raso, R.; Ferrara, L.; Vecchi, E.; Ferrero, L.; Filippi, D.; Finotto, G.; Frassinelli, E.; Silvestre, C.; Zozzoli, S.; et al. Increasing Number of Cases Due to *Candida Auris* in North Italy, July 2019–December 2022. J. Clin. Med. 2023, 12, 1912.

[66] Thomsen, J.; Abdulrazzaq, N.M.; Oulhaj, A.; Nyasulu, P.S.; Alatoom, A.; Denning, D.W.; Al Dhaheri, F.; Consortium, U.A.S.; Menezes, G.A.; Moubareck, C.A.; et al. Emergence of highly resistant *Candida Auris* in the United Arab Emirates: A retrospective analysis of evolving national trends. Front. Public Health 2023, 11, 1244358.

[67] Senok, A.; Alfaresi, M.; Khansaheb, H.; Nassar, R.; Hachim, M.; Al Suwaidi, H.; Almansoori, M.; Alqaydi, F.; Afaneh, Z.; Mohamed, A.; et al. Coinfections in Patients Hospitalized with COVID-19: A Descriptive Study from the United Arab Emirates. Infect. Drug Resist. 2021, 14, 2289–2296.

[68] Bagheri Lankarani, K.; Akbari, M.; Tabrizi, R.; Vali, M.; Sekhavati, E.; Heydari, S.T.; Khodadadi, H.; Ahmadizar, F. *Candida Auris*: Outbreak fungal pathogen in COVID-19 pandemic: A systematic review and meta-analysis. Iran. J. Microbiol. 2022, 14, 276–284.

[69] Magnasco, L.; Mikulska, M.; Giacobbe, D.R.; Taramasso, L.; Vena, A.; Dentone, C.; Dettori, S.; Tutino, S.; Labate, L.; Di Pilato, V.; et al. Spread of Carbapenem-Resistant Gram-Negatives and *Candida Auris* during the COVID-19 Pandemic in Critically Ill Patients: One Step Back in Antimicrobial Stewardship? Microorganisms 2021, 9, 95.

[70] Mulet Bayona, J.V.; Tormo Palop, N.; Salvador Garcia, C.; Fuster Escriva, B.; Chanza Avino, M.; Ortega Garcia, P.; Gimeno Cardona, C. Impact of the SARS-CoV-2 Pandemic in Candidaemia, Invasive Aspergillosis and Antifungal Consumption in a Tertiary Hospital. J. Fungi 2021, 7, 440.

[71] Meis, J.F.; Chowdhary, A. *Candida Auris*: A global fungal public health threat. Lancet Infect. Dis. 2018, 18, 1298–1299.

[72] Chow, N.A.; Gade, L.; Tsay, S.V.; Forsberg, K.; Greenko, J.A.; Southwick, K.L.; Barrett, P.M.; Kerins, J.L.; Lockhart, S.R.; Chiller, T.M.; et al. Multiple introductions and subsequent transmission of multidrug-resistant *Candida Auris* in the USA: A molecular epidemiological survey. Lancet Infect. Dis. 2018, 18, 1377–1384.

[73] Chowdhary, A.; Sharma, C.; Meis, J.F. *Candida Auris*: A rapidly emerging cause of hospital-acquired multidrug-resistant fungal infections globally. PLoS Pathog. 2017, 13, e1006290.

[74] Durante, A.J.; Maloney, M.H.; Leung, V.H.; Razeq, J.H.; Banach, D.B. Challenges in identifying *Candida Auris* in hospital clinical laboratories: A need for hospital and public health laboratory collaboration in rapid identification of an emerging pathogen. Infect. Control Hosp. Epidemiol. 2018, 39, 1015–1016.

[75] Lockhart, S.R.; Jackson, B.R.; Vallabhaneni, S.; Ostrosky-Zeichner, L.; Pappas, P.G.; Chiller, T. Thinking beyond the common Candida species: Need for species-level identification of Candida due to the emergence of multidrug-resistant *Candida Auris*. J. Clin. Microbiol. 2017, 55, 3324–3327.

[76] Mulet Bayona, J.V.; Salvador García, C.; Tormo Palop, N.; Gimeno Cardona, C. Evaluation of a novel chromogenic medium for Candida spp. identification and comparison with CHROMagarTM Candida for the detection of *Candida Auris* in surveillance samples. Diagn. Microbiol. Infect. Dis. 2020, 98, 115168.

[77] Arendrup, M.C.; Prakash, A.; Meletiadis, J.; Sharma, C.; Chowdhary, A. Comparison of EUCAST and CLSI reference microdilution mics of eight antifungal compounds for *Candida Auris* and associated tentative epidemiological cutoff values. Antimicrob. Agents Chemother. 2017, 61, e00485-17.

[78] Pristov, K.E.; Ghannoum, M.A. Resistance of Candida to azoles and echinocandins worldwide. Clin. Microbiol. Infect. 2019, 25, 792–798.

[79] Jeffery-Smith, A.; Taori, S.K.; Schelenz, S.; Jeffery, K.; Johnson, E.M.; Borman, A.; Manuel, R.; Browna, C.S. *Candida Auris*: A review of the literature. Clin. Microbiol. Rev. 2018, 31.

[80] Horton, M.V.; Nett, J.E. *Candida Auris* Infection and Biofilm Formation: Going beyond the Surface. Curr. Clin. Microbiol. Rep. 2020, 7, 51–56.

[81] Singh, R.; Kaur, M.; Chakrabarti, A.; Shankarnarayan, S.A.; Rudramurthy, S.M. Biofilm formation by *Candida Auris* isolated from colonising sites and candidemia cases. Mycoses 2019, 62, 706–709.

[82] Dominguez, E.G.; Zarnowski, R.; Choy, H.L.; Zhao, M.; Sanchez, H.; Nett, J.E.; Andes, D.R. Conserved Role for Biofilm Matrix Polysaccharides in *Candida Auris* Drug Resistance. mSphere 2019, 4, e00680-18.

[83] Romera, D.; Aguilera-Correa, J.J.; Gadea, I.; Viñuela-Sandoval, L.; García-Rodríguez, J.; Esteban, J. *Candida Auris*: A comparison between planktonic and biofilm susceptibility to antifungal drugs. J. Med. Microbiol. 2019, 68, 1353–1358.

[84] Hou, X.; Lee, A.; Jiménez-Ortigosa, C.; Kordalewska, M.; Perlin, D.S.; Zhao, Y. Rapid detection of ERG11-associated azole resistance and FKS-associated echinocandin resistance in *Candida Auris*. Antimicrob. Agents Chemother. 2019, 63, 1–7.

[85] Spivak, E.S.; Hanson, K.E. *Candida Auris*: An Emerging Fungal Pathogen. J. Clin. Microbiol. 2018, 56, 1–10.

[86] Lockhart, S.R. *Candida Auris* and multidrug resistance: Defining the new normal. Fungal Genet. Biol. 2019, 131, 103243.

[87] Aljindan, R.; Aleraky, D.M.; Mahmoud, N.; Abdalhamid, B.; Almustafa, M.; Abdulazeez, S.; Francis Borgio, J. Drug resistanceassociated mutations in erg11 of multidrug-resistant *Candida Auris* in a tertiary care hospital of eastern Saudi Arabia. J. Fungi 2021, 7, 1–9.

[88] Sharma, C.; Kumar, N.; Pandey, R.; Meis, J.F.; Chowdhary, A. Whole genome sequencing of emerging multidrug resistant *Candida Auris* isolates in India demonstrates low genetic variation. New Microbes New Infect. 2016, 13, 77–82.

[89] Chatterjee, S.; Alampalli, S.V.; Nageshan, R.K.; Chettiar, S.T.; Joshi, S.; Tatu, U.S. Draft genome of a commonly misdiagnosed multidrug resistant pathogen *Candida Auris*. BMC Genom. 2015, 16, 1–16.

[90] Mayr, E.-M.; Ramírez-Zavala, B.; Krüger, I.; Morschhäuser, J. A Zinc Cluster Transcription Factor Contributes to the Intrinsic Fluconazole Resistance of *Candida Auris*. mSphere 2020, 5, e00279-20.

[91] Chaabane, F.; Graf, A.; Jequier, L.; Coste, A.T. Review on Antifungal Resistance Mechanisms in the Emerging Pathogen *Candida Auris*. Front. Microbiol. 2019, 10, 2788.

[92] Frías-De-león, M.G.; Hernández-Castro, R.; Vite-Garín, T.; Arenas, R.; Bonifaz, A.; Castañón-Olivares, L.; Acosta-Altamirano, G.; Martínez-Herrera, E. Antifungal resistance in *Candida Auris*: Molecular determinants. Antibiotics 2020, 9, 568.

[93] Escandón, P.; Chow, N.A.; Caceres, D.H.; Gade, L.; Berkow, E.L.; Armstrong, P.; Rivera, S.; Misas, E.; Duarte, C.; Moulton-Meissner, H.; et al. Molecular epidemiology of *Candida Auris* in Colombia Reveals a Highly Related, Countrywide Colonization with Regional Patterns in Amphotericin B Resistance. Clin. Infect. Dis. 2019, 68, 15–21.

[94] Kordalewska, M.; Lee, A.; Park, S.; Berrio, I.; Chowdhary, A.; Zhao, Y.; Perlin, D.S. Understanding echinocandin resistance in the emerging pathogen *Candida Auris*. Antimicrob. Agents Chemother. 2018, 62, e00238-18.

[95] Chowdhary, A.; Prakash, A.; Sharma, C.; Kordalewska, M.; Kumar, A.; Sarma, S.; Tarai, B.; Singh, A.; Upadhyaya, G.; Upadhyay, S.; et al. A multicentre study of antifungal susceptibility patterns among 350 *Candida Auris* isolates (2009–17) in India: Role of the ERG11 and FKS1 genes in azole and echinocandin resistance. J. Antimicrob. Chemother. 2018, 73, 891–899.

[96] Rhodes, J.; Abdolrasouli, A.; Farrer, R.A.; Cuomo, C.A.; Aanensen, D.M.; Armstrong-James, D.; Fisher, M.C.; Schelenz, S. Genomic epidemiology of the UK outbreak of the emerging human fungal pathogen *Candida Auris* article. Emerg. Microbes Infect. 2018, 7, 1–12

[97] Vandeputte, P.; Ferrari, S.; Coste, A.T. Antifungal resistance and new strategies to control fungal infections. Int. J. Microbiol. 2012, 68, 15–21.

[98] Hager, C.L.; Larkin, E.L.; Long, L.; Abidi, F.Z.; Shaw, K.J.; Ghannoum, M.A. In vitro and in vivo evaluation of the antifungal activity of APX001A/APX001 against *Candida Auris*. Antimicrob. Agents Chemother. 2018, 62.

[99] Colombo, A.L.; Dal Mas, C.; Rossato, L.; Shimizu, T.; Oliveira, E.B.; Da Silva Junior, P.I.; Meis, J.F.; Hayashi, M.A.F. Effects of the natural peptide crotamine from a south american rattlesnake on *Candida Auris*, an emergent multidrug antifungal resistant human pathogen. Biomolecules 2019, 9, 205.

[100] Kunyeit, L.; KA, A.-A.; Rao, R.P. Application of Probiotic Yeasts on Candida Species Associated Infection. J. Fungi 2020, 6, 189.

[101] Jaggavarapu, S.; Burd, E.M.; Weiss, D.S. Micafungin and amphotericin B synergy against *Candida Auris*. The Lancet Microbe 2020, 1, e314–e315.

[102] Zhu, Y.C.; Barat, S.A.; Borroto-Esoda, K.; Angulo, D.; Chaturvedi, S.; Chaturvedi, V. Pan-resistant *Candida Auris* isolates from the outbreak in New York are susceptible to ibrexafungerp (a glucan synthase inhibitor). Int. J. Antimicrob. Agents 2020, 55, 105922.

[103] Fujii, S.; Yabe, K.; Kariwano-Kimura, Y.; Furukawa, M.; Itoh, K.; Matsuura, M.; Horimoto, M. Developmental toxicity of flucytosine following administration to pregnant rats at a specific time point of organogenesis. Congenit. Anom. 2019, 59, 39–42.

[104] Vermes, A.; Guchelaar, H.J.; Dankert, J. Flucytosine: A review of its pharmacology, clinical indications, pharmacokinetics, toxicity and drug interactions. J. Antimicrob. Chemother. 2000, 46, 171–179.

[105] Eldesouky, H.E.; Li, X.; Abutaleb, N.S.; Mohammad, H.; Seleem, M.N. Synergistic interactions of sulfamethoxazole and azole antifungal drugs against emerging multidrug-resistant *Candida Auris*. Int. J. Antimicrob. Agents 2018, 52, 754–761.

[106] Eldesouky, H.E.; Salama, E.A.; Lanman, N.A.; Hazbun, T.R.; Seleem, M.N. Potent synergistic interactions between lopinavir and azole antifungal drugs against emerging multidrug-resistant *Candida Auris*. Antimicrob. Agents Chemother. 2021, 65, 1–5.

[107] De Oliveira, H.C.; Monteiro, M.C.; Rossi, S.A.; Pemán, J.; Ruiz-Gaitán, A.; Mendes-Giannini, M.J.S.; Mellado, E.; Zaragoza, O. Identification of Off-Patent Compounds That Present Antifungal Activity against the Emerging Fungal Pathogen *Candida Auris*. Front. Cell. Infect. Microbiol. 2019, 9, 1–10.

[108] Eldesouky, H.E.; Lanman, N.A.; Hazbun, T.R.; Seleem, M.N. Aprepitant, an antiemetic agent, interferes with metal ion homeostasis of *Candida Auris* and displays potent synergistic interactions with azole drugs. Virulence 2020, 11, 1466–1481.

[109] Walraven, C.J.C.J.; Lee, S.A.S.A. Antifungal lock therapy. Antimicrob. Agents Chemother. 2013, 57, 1–8.

[110] Sumiyoshi, M.; Miyazaki, T.; Makau, J.N.; Mizuta, S.; Tanaka, Y.; Ishikawa, T.; Makimura, K.; Hirayama, T.; Takazono, T.; Saijo, T.; et al. Novel and potent antimicrobial effects of caspofungin on drug-resistant Candida and bacteria. Sci. Rep. 2020, 10, 1–12.

[111] Rudramurthy, S.M.; Colley, T.; Abdolrasouli, A.; Ashman, J.; Dhaliwal, M.; Kaur, H.; Armstrong-James, D.; Strong, P.; Rapeport, G.; Schelenz, S.; et al. In vitro antifungal activity of a novel topical triazole PC945 against emerging yeast *Candida Auris*. J. Antimicrob. Chemother. 2019, 74, 2943–2949.

[112] Wiederhold, N.P.; Najvar, L.K.; Jaramillo, R.; Olivo, M.; Patterson, H.; Connell, A.; Fukuda, Y.; Mitsuyama, J.; Catano, G.; Patterson, T.F. The novel arylamidine T-2307 demonstrates in vitro and in vivo Activity against *Candida Auris*. Antimicrob. Agents Chemother. 2020, 64.

[113] Iyer, K.R.; Whitesell, L.; Porco, J.A.; Henkel, T.; Brown, L.E.; Robbins, N.; Cowen, L.E. Translation inhibition by rocaglates activates a species-specific cell death program in the emerging fungal pathogen *Candida Auris*. MBio 2020, 11, 1–17.

[114] Tan, J.; Liu, Z.; Sun, Y.; Yang, L.; Gao, L. Inhibitory Effects of Photodynamic Inactivation on Planktonic Cells and Biofilms of *Candida Auris*. Mycopathologia 2019, 184, 525–531.