RESEARCH PAPER

Candida auris: A Systematic Review of Epidemiology, Antifungal Resistance, and Advances in Laboratory Diagnostics

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ABSTRACT

Background:
C. auris is an emerging multidrug-resistant fungal pathogen associated with healthcare-acquired infections, persistent colonization, and outbreaks. Its resistance to multiple antifungal agents and frequent misidentification by conventional laboratory methods pose significant challenges to clinical management and infection prevention.

Objective:
To review the global epidemiology, antifungal resistance patterns, underlying resistance mechanisms, and current laboratory diagnostic approaches for *C. auris*.

Methods:
This systematic review was designed to synthesize relevant literature published between January 2020 and October 2026. The search strategy included PubMed/MEDLINE, Scopus, Web of Science, Embase, and supplementary sources. Relevant observational studies, outbreak investigations, surveillance reports, diagnostic accuracy studies, and systematic reviews were considered. Findings were organized into epidemiology and transmission, antifungal resistance, and laboratory diagnosis.

Results:
The available evidence highlights the global spread of *C. auris*, geographically distinct genetic clades, and healthcare-associated transmission, particularly among critically ill patients with prolonged hospitalization and invasive medical devices. Fluconazole resistance is frequently reported, while resistance to amphotericin B and echinocandins, including multidrug-resistant and pan-resistant isolates, further limits treatment options. Mutations involving *ERG11* and *FKS1* contribute to azole and echinocandin resistance, respectively. Conventional biochemical identification may lead to misidentification. MALDI-TOF mass spectrometry with updated databases, species-specific PCR, real-time PCR, and DNA sequencing provide more reliable identification. Antifungal susceptibility testing remains important for guiding treatment.

Conclusion:
Early and accurate identification, susceptibility testing, strengthened surveillance, and effective infection-control measures are essential to address the growing clinical challenge posed by *C. auris*. Improved diagnostic capacity and standardized resistance monitoring are particularly important in resource-limited healthcare settings.

Keywords: *C. auris*; epidemiology; antifungal resistance; MALDI-TOF MS; polymerase chain reaction; laboratory diagnosis; systematic review.

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INTRODUCTION

C. auris is an emerging multidrug-resistant fungal pathogen that has become a major concern in clinical microbiology and global public health. Unlike many other Candida species, it can persist on human skin and environmental surfaces, survive in healthcare settings, and spread between patients. Its ability to cause serious infections, combined with difficulties in identification and treatment, has established C. auris as an important emerging healthcare-associated pathogen [1, 2].

The clinical significance of *C. auris* extends beyond bloodstream infections to include invasive candidiasis and asymptomatic colonization. Infections are particularly concerning among critically ill patients, individuals with multiple comorbidities, and those requiring prolonged hospitalization or invasive medical devices. Persistent colonization and environmental contamination facilitate transmission, making early detection and effective infection prevention essential [1, 3].

Since its recognition, *C. auris* has been reported across multiple continents, with distinct genetic clades associated with different geographical regions. Healthcare-associated outbreaks have highlighted the importance of patient screening, environmental disinfection, and surveillance. Prolonged hospital stays, intensive care admission, and exposure to complex healthcare procedures may increase opportunities for acquisition and transmission [2, 3].

Antifungal resistance is a defining characteristic of *C. auris*. Many isolates exhibit resistance to fluconazole, while resistance to amphotericin B and echinocandins has also been documented. Some isolates demonstrate multidrug resistance, restricting therapeutic options and increasing the complexity of clinical management. Consequently, accurate identification and antifungal susceptibility testing are important for guiding treatment decisions [1, 3].

Conventional biochemical identification systems may misidentify *C. auris* as related yeast species, delaying appropriate infection control. MALDI-TOF mass spectrometry with updated reference databases, species-specific PCR, real-time PCR, and DNA sequencing offer more reliable identification. Whole-genome sequencing additionally supports outbreak investigation and genomic surveillance [2, 4, 5].

Despite these advances, differences in regional epidemiology, antifungal susceptibility, and diagnostic capacity remain important challenges. This systematic review therefore aims to critically synthesize evidence published between 2020 and 2026 on the epidemiology, antifungal resistance, and contemporary laboratory diagnostic approaches for *C. auris*, highlighting implications for clinical management, surveillance, and infection prevention [6].

MATERIALS AND METHODS

Study Design

This systematic review was designed to evaluate the epidemiology, antifungal resistance patterns, and current laboratory diagnostic approaches for *C. auris*. The review methodology was based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement to promote transparent reporting, reproducibility, and comprehensive documentation of the study selection process. The review protocol, eligibility criteria, search strategy, and data synthesis methods were defined before the formal screening of records.

Literature Search Strategy

A comprehensive literature search was planned across PubMed/MEDLINE, Scopus, Web of Science, and Embase, where accessible. Google Scholar was to be used as a supplementary source to identify additional relevant publications. The search covered studies published from 1 January 2020 to 19 October 2026.

Search terms were developed using combinations of keywords and Boolean operators, including "*C. auris*," "epidemiology," "geographical distribution," "healthcare-associated outbreak," "antifungal resistance," "multidrug resistance," "antifungal susceptibility testing," "laboratory diagnosis," "MALDI-TOF mass spectrometry," "polymerase chain reaction," and "whole-genome sequencing." The search strategy was adapted to the indexing system and syntax of each database. Reference lists of eligible articles and relevant reviews were also examined to identify additional studies.

Eligibility Criteria

Studies were considered eligible if they were published in English between 2020 and 2026 and provided relevant evidence concerning the epidemiology, clinical distribution, healthcare-associated transmission, antifungal resistance, susceptibility testing, or laboratory diagnosis of *C. auris*. Original research articles, observational studies, outbreak investigations, diagnostic accuracy studies, surveillance reports, and relevant systematic reviews were considered for inclusion.

Publications were excluded if they were unrelated to the review objectives, lacked sufficient methodological information, duplicated previously identified records, or consisted of editorials, opinion pieces, or conference abstracts without

adequate supporting data. Studies published before the specified period were excluded from the primary evidence synthesis, although older publications could be consulted for background information where necessary.

Study Selection Process

All retrieved records were to be compiled in a reference management system, and duplicate records were removed before screening. Titles and abstracts were assessed against the predefined eligibility criteria, followed by full-text evaluation of potentially relevant articles. Ideally, two independent reviewers would conduct screening, with disagreements resolved through discussion or consultation with a third reviewer. The reasons for excluding full-text articles were documented. The selection process was to be reported using the PRISMA 2020 flow diagram.

Data Extraction

Data were to be extracted using a standardized data extraction form. The information collected included the first author, publication year, country or geographical region, study design, study population, sample size, *C. auris* identification methods, antifungal susceptibility results, resistance patterns, diagnostic performance measures, and principal findings. Particular attention was given to regional differences in epidemiology, resistance to fluconazole, amphotericin B and echinocandins, and the performance of MALDI-TOF mass spectrometry, molecular assays, real-time PCR, and sequencing-based techniques.

Quality Assessment and Data Synthesis

The methodological quality of included primary studies was to be assessed using appropriate design-specific critical appraisal tools, such as relevant Joanna Briggs Institute checklists. Diagnostic accuracy studies were to be evaluated using the QUADAS-2 framework where applicable. Quality assessments would be interpreted in relation to study design and potential sources of bias.

Because the review addressed heterogeneous epidemiological, microbiological, and diagnostic outcomes, a narrative synthesis was planned. Findings were to be organized into three principal domains: epidemiology and transmission, antifungal resistance, and laboratory diagnosis. Quantitative meta-analysis would be considered only if sufficient clinical and methodological comparability existed across studies. The review findings would be interpreted in light of geographical variation, differences in laboratory methods, and limitations in the available evidence.

AIMS & OBJECTIVES

General Objective

- To systematically review the available evidence on the global epidemiology, antifungal resistance, and current laboratory diagnostic approaches for *C. auris*.

Specific Objectives

1. To summarize the global epidemiology and geographical distribution of *C. auris*, including its emergence, healthcare-associated transmission, and reported outbreaks.
2. To evaluate the prevalence and mechanisms of antifungal resistance in *C. auris*, with particular emphasis on resistance to azoles, amphotericin B, and echinocandins.
3. To compare the diagnostic performance, strengths, and limitations of current laboratory diagnostic approaches, including MALDI-TOF mass spectrometry, molecular assays, real-time PCR, and sequencing-based methods.
4. To identify existing gaps in epidemiological surveillance, antifungal susceptibility testing, and early laboratory detection, and to highlight priorities for improving clinical diagnosis and infection prevention.

Study Selection

Study selection was planned in accordance with the PRISMA 2020 guidelines. All records identified through the database searches and supplementary sources were to be compiled in a reference management system, and duplicate records were to be removed before screening. Titles and abstracts were to be screened against the predefined eligibility criteria to identify potentially relevant publications. Full texts of potentially eligible articles were then to be assessed for final inclusion.

Where feasible, two independent reviewers were to conduct the screening process. Disagreements regarding study eligibility were to be resolved through discussion and consensus, with a third reviewer consulted when necessary. Reasons for excluding studies at the full-text assessment stage were to be documented. The study identification, screening, eligibility assessment, and inclusion process was to be summarized using a PRISMA 2020 flow diagram.

Quality Assessment and Risk of Bias

The methodological quality and potential risk of bias of included studies were to be assessed using tools appropriate to their respective study designs. Diagnostic accuracy studies were to be evaluated using the Quality Assessment of Diagnostic Accuracy Studies-2 (QUADAS-2) tool, which assesses patient selection, the index test, the reference standard, and flow and timing.

For prevalence studies, an appropriate critical appraisal tool, such as the relevant Joanna Briggs Institute (JBI) checklist for prevalence studies, was to be used to evaluate sampling methods, participant selection, measurement procedures, and

the appropriateness of statistical analyses. Observational studies and outbreak investigations were to be assessed using suitable design-specific appraisal criteria.

Quality assessment findings were to be considered when interpreting the evidence on geographical distribution, antifungal resistance patterns, and laboratory diagnostic performance. Studies with methodological limitations were not necessarily to be excluded solely on the basis of their quality scores; instead, their limitations were to be considered in the overall interpretation of the findings.

EPIDEMIOLOGY OF *C. auris*

Global emergence and geographic distribution

C. auris (currently also referred to as Candidozyma auris in updated taxonomic literature) is an emerging, multidrug-resistant fungal pathogen that has become a major concern for global public health because of its ability to cause healthcare-associated infections, persist on hospital surfaces, spread between patients, and produce outbreaks in healthcare facilities. The organism was first described in 2009 after being isolated from the external auditory canal of a patient in Japan. Subsequent investigations identified invasive infections, including bloodstream infections, and established its importance as a nosocomial pathogen [7], [8].

Unlike many other clinically important Candida species, *C. auris* can persist on the skin of colonized patients and survive for prolonged periods on healthcare-associated surfaces and equipment. These characteristics facilitate transmission through direct patient contact and contaminated healthcare environments. Outbreaks have been reported in intensive care units (ICUs), neonatal and paediatric intensive care units, long-term care facilities, and other hospital settings. The organism's ability to cause persistent transmission, combined with frequent antifungal resistance and difficulties in routine laboratory identification, makes its control particularly challenging [7], [9].

The global distribution of *C. auris* has expanded substantially since its initial recognition. Epidemiological investigations have documented cases across all six World Health Organization (WHO) regions. However, the actual burden remains uncertain because surveillance capacity, laboratory identification methods, case definitions, and reporting practices differ considerably between countries [2], [3].

A systematic review and meta-analysis published in 2020 identified more than 4,733 reported cases across over 33 countries in the literature available up to October 2019. These figures represent the historical evidence included in that review, rather than the current global case count. Subsequent surveillance and genomic investigations have documented additional geographic spread and continuing transmission in healthcare settings [3], [6].

The emergence of *C. auris* is particularly concerning because its distribution is not explained solely by the international spread of a single strain. Whole-genome sequencing studies have demonstrated genetically distinct populations associated with different geographic regions, suggesting that several lineages emerged independently or became established separately before subsequent international dissemination [8], [6].

Geographic clades and their epidemiological significance

Whole-genome sequencing has traditionally identified four major geographic clades of *C. auris*. Additional genetically distinct lineages have subsequently been reported, including a fifth clade associated with Iran and a sixth reported from Singapore and Bangladesh. These findings demonstrate that the organism's genetic diversity is greater than initially recognized [8], [10], [11], [12].

Clade	Principal geographic association	Epidemiological significance
Clade I	South Asia, particularly India and Pakistan	Frequently associated with healthcare-associated outbreaks, invasive infections and multidrug resistance.
Clade II	East Asia, particularly Japan and South Korea	Initially associated predominantly with ear infections; invasive disease has also been documented.
Clade III	Africa, particularly South Africa	Associated with healthcare outbreaks, invasive infections and substantial antifungal resistance.
Clade IV	South America, particularly Venezuela and Colombia	Associated with bloodstream infections, hospital transmission and geographically variable antifungal susceptibility.
Clade V	Iran	A genetically distinct lineage identified in 2018.
Clade VI	Singapore and Bangladesh	A more recently recognized divergent lineage, demonstrating previously underappreciated genetic diversity.

Hospital and healthcare-associated outbreaks

Healthcare-associated transmission is a defining epidemiological feature of *C. auris*. Unlike infections that arise primarily from a patient's own endogenous microbiota, *C. auris* can spread through colonized patients, contaminated hands of healthcare personnel, shared medical equipment and environmental surfaces. Its persistence in the healthcare environment can facilitate repeated transmission even after the initial case has been recognized [7], [9].

Outbreaks have been documented in ICUs, neonatal intensive care units (NICUs), paediatric intensive care units (PICUs), long-term care facilities and other hospital wards. Patients may remain colonized without symptoms and act as reservoirs for transmission. Delayed recognition of the organism, inadequate screening and difficulty distinguishing colonization from infection can allow transmission to continue before appropriate infection-prevention measures are introduced [9], [2].

Several features contribute to outbreak propagation:

- Environmental persistence: The organism can persist on bed rails, mattresses, reusable equipment and other frequently touched surfaces.
- Patient colonization: Colonized patients may carry the organism on their skin or at other body sites without clinical evidence of infection.
- Frequent contact with healthcare equipment: Shared thermometers, blood pressure cuffs, monitors and other reusable equipment may contribute to transmission if cleaning and disinfection are inadequate.
- Prolonged hospital stays: Longer exposure to healthcare environments increases opportunities for acquisition.
- Delayed laboratory identification: Conventional laboratory methods may misidentify the organism, delaying isolation and targeted infection-control interventions.
- High-risk clinical environments: ICUs and neonatal units often involve invasive devices, frequent patient handling, antimicrobial exposure and patients with substantial underlying illness.

An outbreak investigation in an Eastern Indian tertiary-care hospital described diagnostic challenges and outbreak-control measures in paediatric and neonatal intensive care settings, highlighting the regional importance of early recognition and coordinated infection prevention [13]. In another Indian tertiary-care setting, investigators described transmission dynamics and containment strategies over a four-year period, emphasizing the need for sustained surveillance and infection-control interventions [14].

Effective outbreak control generally requires prompt laboratory confirmation, contact precautions, screening of epidemiologically linked patients when indicated, thorough environmental cleaning with products effective against *C. auris*, appropriate disinfection of shared equipment, and communication between clinical, microbiology and infection-control teams [9], [2].

Risk factors for *C. auris* colonization and infection

C. auris disproportionately affects patients with substantial healthcare exposure and underlying medical conditions. However, it is important to distinguish risk factors for acquiring colonization from risk factors for developing invasive infection. A patient may become colonized without developing disease, whereas invasive infection is more likely when host vulnerability and disruption of normal protective barriers are present [9], [15].

ICU admission and prolonged hospitalization

ICU admission is a frequently reported characteristic among patients with *C. auris* infection. Critically ill patients often require prolonged hospitalization, mechanical ventilation, repeated procedures, broad-spectrum antibiotics and invasive devices. These exposures increase contact with healthcare environments in which *C. auris* may circulate [9], [15].

In an international multicentre observational study involving 10 centres across five countries, recent ICU admission was reported in 63% of patients, while 96% had a history of antibiotic treatment. These figures describe the characteristics of the study population and should not be interpreted as population-level risk estimates or proof that each exposure independently causes infection [15].

Invasive medical devices

Invasive devices are important clinical considerations because they can breach natural protective barriers and provide a route for bloodstream or other invasive infections. Commonly reported devices and procedures include:

- Central venous catheters.
- Urinary catheters.
- Mechanical ventilation and associated airway equipment.
- Recent surgery and other invasive procedures.

In the international genomic epidemiological investigation by Lockhart et al., clinical information was available for 41 patients. A central venous catheter was documented in 73%, and a urinary catheter in 61%. The median time from hospital admission to diagnosis was 19 days, with an interquartile range of 9–36 days [8].

These observations demonstrate the frequent coexistence of invasive devices and healthcare

exposure in affected patients. Nevertheless, the percentages should be interpreted as descriptive findings among selected cases rather than as estimates of the independent effect of each device.

Underlying illness and immunocompromised states

Reported underlying conditions include diabetes mellitus, malignancy, chronic kidney disease, other severe chronic illnesses and conditions requiring immunosuppressive treatment. Critical illness and immunocompromise may increase susceptibility to invasive fungal disease, while repeated healthcare contact may increase opportunities for colonization [8], [15].

In the study by Lockhart et al., diabetes mellitus was reported in 41% of patients with available clinical information. In the international multicentre study, diabetes and renal failure were reported in 63% and 52% of patients, respectively [8], [15]. Differences between these estimates may reflect patient selection, clinical settings, sample size and geographic variation.

Broad-spectrum antibiotics and previous antifungal exposure

Broad-spectrum antibiotic exposure is commonly reported among patients with *C. auris* infection. Antibiotic use can disrupt the normal bacterial microbiota, potentially facilitating fungal colonization, while prolonged treatment often reflects severe underlying illness and extensive healthcare exposure. Previous antifungal treatment is also clinically relevant because *C. auris* frequently exhibits reduced susceptibility or resistance to commonly used antifungal agents [8], [15].

However, observational associations do not establish that antibiotic or antifungal exposure independently causes acquisition. Future epidemiological studies should account for duration of hospitalization, illness severity, device use, prior colonization and other confounding variables.

Colonization versus invasive infection

An essential component of *C. auris* epidemiology is the distinction between colonization and clinical infection. Colonization refers to the presence of the organism on the skin or at another body site without evidence that it is causing disease. In contrast, infection occurs when the organism invades tissues or produces a clinically significant infectious process.

Colonization is epidemiologically important because an asymptomatic carrier can serve as a reservoir for transmission within a healthcare facility. Patients with colonization may remain undetected unless appropriate screening and laboratory identification methods are used. Colonization does not necessarily progress to invasive disease, and a positive culture from a non-sterile site does not, by itself, establish invasive infection [9], [2], [3].

Invasive *C. auris* infections include candidemia and other forms of invasive candidiasis. Bloodstream infections are particularly concerning in critically ill patients, especially those with central venous catheters, severe underlying illness or impaired immune function. A positive blood culture generally requires prompt clinical assessment and appropriate treatment rather than being interpreted as simple colonization [8], [15].

For epidemiological studies, colonization and infection should therefore be recorded as separate outcomes. Screening results from skin or other non-sterile sites should not be combined indiscriminately with culture-confirmed invasive infections when calculating prevalence, incidence or mortality.

Mortality and clinical outcomes

C. auris infection is associated with substantial mortality, particularly among hospitalized patients with bloodstream infections and serious underlying illnesses. However, mortality estimates vary across studies because of differences in patient populations, infection sites, clinical severity, treatment availability and definitions of attributable versus all-cause mortality [8], [3], [15].

In the international genomic epidemiological study by Lockhart et al., 24 of 41 patients with available clinical information died during hospitalization, corresponding to a crude in-hospital mortality of 59%. In the international multicentre study by Lone et al., the reported 30-day all-cause mortality was 37% [8], [15].

These findings should not be interpreted as demonstrating that *C. auris* alone caused every recorded death. Patients affected by the organism often have severe comorbidities and critical illness, both of which contribute independently to poor outcomes. Establishing attributable mortality requires an appropriate comparison group and adjustment for confounding factors.

Factors that may influence outcomes include:

- Severity of the underlying illness.
- Presence of bloodstream or other invasive infection.
- Delayed diagnosis and initiation of effective antifungal treatment.
- Antifungal resistance and limited therapeutic options.
- Persistence of infected intravascular devices or other uncontrolled sources.
- Availability of intensive care and appropriate infection-management facilities.

A systematic review and meta-analysis published in 2020 documented the substantial reported disease burden and mortality associated with *C. auris*, while a subsequent WHO-priority-pathogen review highlighted major gaps in comparable incidence and outcome data [3], [6].

India and South Asia: epidemiological evidence

India and neighbouring South Asian countries are particularly important in the epidemiology of *C. auris* because the South Asian lineage, designated clade I, is associated with the region and has subsequently been identified in multiple countries. Early investigations in India documented healthcare-associated candidemia and demonstrated that *C. auris* could cause hospital outbreaks involving patients with substantial underlying illness [8], [10].

A landmark investigation by Chowdhary et al. reported a clonal outbreak of *C. auris* candidemia involving 12 patients in two hospitals in Delhi. The isolates had been collected during 2009–2011, although the report was published in 2013. The findings established the clinical importance of the organism in India and provided early evidence of healthcare-associated transmission [10].

Subsequent studies demonstrated that the burden of *C. auris* varies considerably across hospitals and patient populations. A multicentre Indian study of candidemia patients in 27 ICUs reported a prevalence of approximately 5.3%, while a separate single-centre study from 2016–2017 reported a substantially higher proportion of 39%. These figures should not be directly compared without considering differences in study design, patient selection, sampling and laboratory methods [3].

India has also reported outbreaks in paediatric and neonatal intensive care settings. The investigation published in 2024 from Eastern India highlighted diagnostic difficulties and the need for timely identification and outbreak-control measures [13]. Such findings are important because neonatal and paediatric patients may have prolonged hospital stays, invasive device exposure and substantial clinical vulnerability.

In Pakistan, *C. auris* was identified in hospital-associated infections and was included in the international genomic investigation that established the presence of geographically distinct lineages. The combined evidence from India and Pakistan demonstrates the importance of surveillance, molecular epidemiology and coordinated infection prevention across South Asia [8].

Despite increasing awareness, the epidemiological burden in the region remains incompletely characterized. Differences in laboratory capacity, access to molecular identification, routine screening practices and reporting systems may result in under-recognition. Consequently, hospital-based prevalence estimates should not be interpreted as national prevalence estimates, and the absence of published cases does not necessarily indicate the absence of transmission.

Study	Country /setting	Study period or publication year	Sample size	Principal epidemiological findings
Chowdhary et al. [10]	India; two hospitals in Delhi	Isolates from 2009–2011; published 2013	12 patients	Early report of a clonal outbreak of *C. auris* candidemia.
Lockhart et al. [8]	India, Pakistan, South Africa and Venezuela; hospital-associated cases	2012–2015; published 2017	54 isolates; clinical data for 41 patients	Demonstrated geographically distinct clades, frequent bloodstream infection, invasive-device exposure and high crude mortality.
Systematic review and meta-analysis [6]	International	Literature through 6 October 2019; published 2020	More than 4,733 reported cases	Documented international spread, variation in reported infection patterns and substantial mortality; counts reflect the literature available at that time.
Lone et al. [15]	Five countries; 10 participating centres	Published 2021	Multicentre observational study	Reported ICU exposure, comorbidities, device use and 37% 30-day all-cause mortality.
WHO-priority-pathogen systematic review [3]	International; studies across WHO regions	Published 2024	Multiple studies; heterogeneous denominators	Reported healthcare-associated outbreaks in all six WHO regions and substantial variation in reported prevalence.
Eastern India outbreak investigation [13]	India; PICU and NICU	Published 2024	See full article	Described diagnostic challenges and outbreak-control measures in paediatric and neonatal intensive care settings.
Gaikwad [14]	India; tertiary-care hospital	Four-year period; reported 2024	See full article	Examined transmission dynamics and containment strategies in a tertiary-care healthcare setting.

ANTIFUNGAL RESISTANCE IN *C. auris*

Antifungal resistance is one of the most important clinical and microbiological characteristics of *C. auris*. The organism frequently exhibits reduced susceptibility to multiple antifungal agents, complicating the management of invasive infections and increasing the importance of antifungal susceptibility testing (AFST), molecular resistance surveillance and appropriate therapeutic monitoring [8], [16].

Three major antifungal classes are particularly relevant: azoles, polyenes and echinocandins. Fluconazole resistance is highly prevalent in many studied populations, amphotericin B resistance occurs in a clinically important subset of isolates, and echinocandin resistance, although less common in many surveillance datasets, is especially concerning because echinocandins are recommended as initial treatment for most patients older than two months with clinical *C. auris* infection [8], [16], [17].

Resistance frequencies differ by geographic region, genetic clade, healthcare setting, testing method and surveillance period. Therefore, resistance estimates from one country or institution should not be generalized to all *C. auris* isolates.

Fluconazole resistance and susceptibility to other azoles

Fluconazole resistance

Fluconazole resistance is the most frequently reported antifungal resistance phenotype in *C. auris*. Many isolates demonstrate high minimum inhibitory concentrations (MICs), sometimes exceeding 256 µg/mL. This phenotype substantially limits the reliability of fluconazole as empirical or definitive treatment for invasive *C. auris* infections [8], [16].

In a major international genomic investigation, Lockhart et al. demonstrated substantial variation in antifungal susceptibility among isolates belonging to different geographic clades. Fluconazole resistance was particularly widespread among isolates from several major lineages [8]. More recent genomic surveillance in the United States has continued to demonstrate a high prevalence of fluconazole resistance, although the proportion varies between clades [12].

Fluconazole inhibits lanosterol 14α-demethylase, an enzyme involved in ergosterol biosynthesis. Resistance may result from alterations in the target enzyme, increased expression of resistance-associated genes, enhanced drug efflux and other mechanisms affecting the availability or activity of the drug within the fungal cell [16], [18].

The principal molecular mechanism associated with azole resistance in *C. auris* involves changes in ERG11, which encodes lanosterol 14α-demethylase. Specific amino acid substitutions can reduce the susceptibility of the target enzyme to azoles. The contribution of these substitutions varies according to the genetic background of the isolate and the specific antifungal agent [8], [18].

ERG11 mutations and azole resistance

The ERG11 substitutions Y132F and K143R are among the best-characterized resistance-associated changes in *C. auris*. Their distribution differs among clades, and they have been associated with elevated fluconazole MICs. Additional ERG11 substitutions and alterations in gene expression may contribute to the resistance phenotype [8], [18].

Other mechanisms include increased activity or expression of efflux pumps, particularly those associated with the ATP-binding cassette and major facilitator superfamily transporter systems. These mechanisms can reduce intracellular antifungal exposure and may contribute to resistance beyond that explained by ERG11 alterations alone [16], [18].

Importantly, detection of an ERG11 mutation should not automatically be interpreted as proof of resistance to every azole. Genotype–phenotype relationships are drug-specific and may depend on other molecular factors. Phenotypic AFST remains important when selecting treatment.

Susceptibility to other azoles

Voriconazole, posaconazole and itraconazole have shown variable in-vitro activity against *C. auris*. Fluconazole resistance does not invariably imply resistance to every second-generation triazole, although cross-resistance can occur. Consequently, susceptibility to another azole should not be assumed from its drug class or from the fluconazole result alone [16], [17].

The Centers for Disease Control and Prevention (CDC) does not provide established *C. auris*-specific tentative resistance breakpoints for voriconazole and other second-generation triazoles in its current guidance. Fluconazole susceptibility may provide contextual information, but treatment decisions involving alternative azoles require individualized interpretation of laboratory results and clinical circumstances [17].

Amphotericin B resistance

Amphotericin B is a polyene antifungal agent that acts principally through interactions with ergosterol in fungal membranes, disrupting membrane function. It has been used in the treatment of invasive *C. auris* infection, particularly when resistance to other antifungal classes is present or treatment response is inadequate [16], [19].

Amphotericin B susceptibility is variable across *C. auris* isolates. Elevated MICs and resistant isolates have been reported in multiple geographic settings, but the proportion varies substantially according to the study population, clade and interpretation criteria [8], [12].

Mechanisms of amphotericin B resistance

Potential mechanisms include alterations in ergosterol biosynthesis and changes in the

composition or organization of the fungal cell membrane. Changes affecting ergosterol abundance or membrane sterol composition may reduce the effectiveness of amphotericin B. However, the molecular basis of resistance is less completely characterized than the relationship between ERG11 alterations and azole resistance or between FKS1 alterations and echinocandin resistance [16], [19].

Amphotericin B resistance should therefore be described primarily as a phenotypic finding unless a specific molecular mechanism has been demonstrated experimentally.

Interpretation of amphotericin B MICs

The CDC currently uses a tentative amphotericin B MIC threshold of ≥2 µg/mL to identify isolates considered resistant for surveillance and clinical interpretation. This threshold is based on expert assessment and supporting pharmacokinetic/pharmacodynamic evidence, rather than a validated *C. auris*-specific clinical breakpoint established by CLSI [17].

An MIC of 2 µg/mL should consequently be interpreted using the applicable laboratory method and current guidance. A result at or above the threshold is concerning but does not independently determine the patient's clinical outcome.

Echinocandin resistance and treatment implications

Echinocandins, including micafungin, caspofungin and anidulafungin, inhibit β-1,3-D-glucan synthase, an enzyme essential for fungal cell-wall synthesis. They are important agents for treating invasive *C. auris* infections because resistance to fluconazole is widespread and amphotericin B resistance is also documented [16], [17], [20].

Although echinocandin resistance has historically been less frequent than fluconazole resistance, resistant isolates have been reported in several countries. More recent surveillance has demonstrated that echinocandin resistance and pan-resistance are emerging concerns [12], [21].

FKS1 mutations and echinocandin resistance

The FKS1 gene encodes a catalytic subunit of β-1,3-D-glucan synthase, the target of echinocandins. Amino acid substitutions in conserved hotspot regions of Fks1 can reduce the susceptibility of the enzyme to these drugs, resulting in elevated echinocandin MICs [20], [21].

Important resistance-associated substitutions include changes at serine 639, particularly S639F, S639P and S639Y. Other substitutions, including changes at F635 and D642, have also been identified in resistant isolates [12], [20].

The relationship between FKS1 mutations and echinocandin resistance is clinically important because resistance can emerge during treatment and may also be transmitted through the spread of resistant strains. In a recent US genomic investigation, 49 of 53 echinocandin-resistant isolates with available genomic data carried an FKS1 mutation, supporting the importance of molecular surveillance alongside phenotypic testing [12].

However, not every FKS1 sequence variation is associated with clinically meaningful resistance. Interpretation should consider the specific amino acid substitution, its location, the antifungal agent and the phenotypic susceptibility results.

Clinical implications

Echinocandins remain the recommended initial treatment for most patients older than two months with clinical *C. auris* infection, according to current CDC guidance. Nevertheless, treatment response should be monitored carefully, and susceptibility testing is recommended [22].

When echinocandin resistance is demonstrated, or when a patient fails to improve after approximately five days of echinocandin therapy, the CDC advises consideration of liposomal amphotericin B, taking into account susceptibility results and the clinical circumstances. Treatment options for echinocandin-resistant and pan-resistant infections remain limited, and evidence for alternative agents is still developing [22].

An important distinction is that colonization alone does not require antifungal treatment. A positive screening result from a non-invasive site should not automatically trigger antifungal therapy in the absence of clinical infection [22].

Multidrug-resistant and pan-resistant isolates

Multidrug resistance in *C. auris* refers to resistance affecting multiple antifungal classes. Pan-resistance generally refers to resistance to all three major classes used for candidiasis: azoles, polyenes and echinocandins. Definitions should be stated explicitly because some reports use broader terminology to describe resistance to additional agents [16], [23].

Multidrug-resistant isolates are well documented, and the prevalence of resistance differs between clades and geographical regions. The simultaneous presence of fluconazole resistance, elevated amphotericin B MICs and echinocandin resistance substantially reduces the number of routinely available treatment options [12], [23].

Ostrowsky et al. reported pan-resistant *C. auris* isolates in New York in 2019, demonstrating resistance to all three major antifungal classes and highlighting the potential for healthcare-associated transmission of isolates with severely restricted treatment options [23].

A subsequent report described isolates resistant to four antifungal classes, including flucytosine. This finding illustrates that the resistance phenotype may extend beyond the three principal classes routinely assessed in *C. auris* surveillance [24].

In the 2026 US genomic epidemiological investigation, resistance patterns varied by clade. Among isolates tested against individual drugs, 91% were fluconazole-resistant, 12% were

amphotericin B-resistant and 7% were echinocandin-resistant. These estimates came from a selected genomic surveillance dataset and should not be interpreted as universal prevalence figures [12].

Pan-resistance is especially concerning because routine treatment options may be exhausted. Management requires specialist consultation, susceptibility-guided decision-making, assessment of infection source control and consideration of investigational agents where appropriate. Evidence supporting these alternatives remains limited [22].

Antifungal susceptibility testing (AFST)

AFST is essential for characterizing the resistance profile of *C. auris*, informing treatment decisions and identifying resistant isolates that may require enhanced infection-control measures. Because susceptibility patterns vary geographically and over time, a historical resistance profile should not substitute for testing the patient's own isolate when testing is available [17], [12].

Other testing methods

- Commercial broth microdilution panels: Useful for standardized testing, provided the panel and interpretation are appropriate for the organism and drug.
- Gradient diffusion (Etest): Can provide MIC estimates, but method-dependent differences may occur. Results near an interpretive threshold require careful assessment.
- Molecular resistance testing: Detection of ERG11 or FKS1 alterations can help characterize resistance mechanisms but does not replace phenotypic testing in every circumstance.
- Whole-genome sequencing: Useful for investigating resistance-associated variants, genetic clades and transmission of resistant strains.

For clinical and surveillance studies, the reference method should be clearly stated, and MICs obtained by different methods should not be assumed to be directly interchangeable [17], [20], [25].

MIC and interpretive criteria

The MIC is the lowest tested concentration of an antifungal that meets the defined growth-inhibition endpoint under the specified laboratory method. MIC values should be reported in µg/mL, numerically equivalent to mg/L.

A high MIC indicates reduced in-vitro susceptibility. However, an MIC alone does not determine clinical outcome, and the same MIC may have different implications for different antifungal agents.

A critical interpretive limitation: CLSI has not established conventional species-specific clinical breakpoints for most antifungals against *C. auris*. CDC has published tentative thresholds based on expert opinion and information from related Candida species. These thresholds are useful for surveillance and clinical interpretation but are not equivalent to fully validated organism-specific clinical breakpoints [17].

The CDC's guidance updated on 1 October 2026 lists a *C. auris*-specific susceptible-only breakpoint for rezafungin in the relevant CLSI supplement. The tentative resistance thresholds for the other agents should be interpreted according to the current guidance rather than treated as universally validated clinical breakpoints [17].

MIC distribution and tentative resistance thresholds

The following thresholds are useful for presenting antifungal susceptibility findings in a systematic review. They reflect CDC guidance available as of October 2026 [17].

Antifungal agent	Class	Tentative MIC threshold (µg/mL)	Interpretation
Fluconazole	Azole	≥32	Resistant
Voriconazole	Azole	No established threshold	Interpret cautiously; no CDC tentative breakpoint
Itraconazole	Azole	No established threshold	Agent-specific interpretation required
Posaconazole	Azole	No established threshold	Agent-specific interpretation required
Amphotericin B	Polyene	≥2	Resistant
Anidulafungin	Echinocandin	≥4	Resistant
Caspofungin	Echinocandin	≥2	Resistant
Micafungin	Echinocandin	≥4	Resistant
Rezafungin	Echinocandin	See current CLSI	Use the applicable *C. auris*-

		supplement	specific susceptible-only criterion

Proposed evidence table: antifungal resistance studies

The following table summarizes representative evidence, including reported MICs and molecular mechanisms where these have been documented.

Antifungal agent/class	Testing method	Reported MIC or finding	Resistance mechanism
Fluconazole [8], [18], [20]	CLSI broth microdilution	Frequently elevated; resistant isolates may have MICs ≥32 to >128 µg/mL	ERG11 substitutions, including Y132F and K143R; efflux-related mechanisms
Voriconazole [18], [20]	CLSI broth microdilution	Variable MICs; elevated values in some fluconazole-resistant isolates	ERG11 alterations and other azole-resistance mechanisms
Amphotericin B [16], [17], [21]	Broth microdilution; gradient diffusion in selected studies	MIC ≥2 µg/mL meets the current CDC tentative resistance threshold	Alterations in ergosterol biosynthesis or membrane composition may contribute
Micafungin [17], [20]	CLSI broth microdilution	MIC ≥4 µg/mL meets the CDC tentative resistance threshold; resistant isolates may have substantially higher MICs	FKS1 hotspot substitutions, including S639F
Anidulafungin [17], [20]	CLSI broth microdilution	MIC ≥4 µg/mL meets the CDC tentative resistance threshold	FKS1-associated reduction in target susceptibility
Caspofungin [17], [20]	CLSI broth microdilution	MIC ≥2 µg/mL meets the CDC tentative resistance threshold	FKS1 mutations affecting β-1,3-D-glucan synthase
Multiple antifungal classes [23], [24]	Broth microdilution and complementary testing	Resistance to azoles, amphotericin B and echinocandins documented in individual isolates	Multiple resistance mechanisms; some isolates carry both ERG11- and FKS1-associated changes
Multiple antifungal classes [24]	Broth microdilution and molecular characterization	Pan-drug-resistant isolates reported, including resistance extending to flucytosine	Combined or sequential resistance mechanisms

Resistance surveillance and future directions

Effective surveillance requires the integration of phenotypic AFST, molecular resistance characterization and genomic epidemiology. Laboratories should monitor shifts in MIC distributions, identify isolates with elevated echinocandin MICs, and investigate potential outbreaks involving resistant strains [17], [12].

A standardized surveillance dataset should ideally include:

- Patient and isolate identifiers, with appropriate confidentiality safeguards.
- Specimen type and distinction between colonization and clinical infection.
- Country, hospital and clinical setting.
- Species identification method.
- AFST method and antifungal agents tested.

- MIC values and applicable interpretive criteria.
- ERG11 and FKS1 sequence variants, where available.
- Previous antifungal exposure and clinical treatment response.
- Relevant genomic information for suspected transmission clusters.

Future research should prioritize standardized MIC reporting, validation of organism-specific breakpoints, improved understanding of resistance mechanisms and clinical outcome studies linking MICs and molecular variants with treatment response. Particular attention should be given to emerging echinocandin resistance, pan-resistant isolates and the geographic variation of resistance-associated genotypes [12], [18], [22].

Current Laboratory Diagnostic Approaches for *C. auris*

Accurate laboratory identification of *C. auris* is essential for the timely diagnosis of invasive fungal infections, detection of asymptomatic colonization, implementation of infection-prevention measures and antifungal susceptibility testing. The organism presents a particular diagnostic challenge because conventional biochemical systems and routine phenotypic methods may misidentify it as other Candida species or unrelated yeasts [26], [27].

Current diagnostic approaches include conventional culture, biochemical identification systems, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS), real-time polymerase chain reaction (PCR), species-specific nucleic acid amplification assays and DNA sequencing. The choice of method depends on whether the objective is to identify an isolate, detect colonization directly from a screening specimen, diagnose infection from a clinical specimen or characterize the organism for epidemiological surveillance [26], [28].

The Centers for Disease Control and Prevention (CDC) identifies MALDI-TOF MS with an appropriate reference database as the most reliable routine approach for identifying cultured isolates, while real-time PCR is the preferred approach for colonization screening when available. Sequencing of the internal transcribed spacer (ITS) region or the D1/D2 region of the 28S ribosomal RNA gene provides an important confirmatory method [27], [28].

Conventional culture and colony morphology

Culture-based methods remain important in the diagnosis of *C. auris* because they allow recovery of viable organisms for subsequent species identification, antifungal susceptibility testing and molecular characterization. Clinical specimens may include blood, other normally sterile body fluids, wound specimens, respiratory specimens and urine, depending on the clinical presentation. Skin swabs are used principally to detect colonization rather than to establish invasive disease [27], [28].

C. auris can grow on routine fungal culture media, including Sabouraud dextrose agar. On chromogenic Candida media, colonies may appear white, pink, red or purple, depending on the medium and strain. Growth at elevated temperatures, including approximately 40–42°C, may assist laboratory investigation but is not sufficiently specific to establish the species identity [27].

Colony morphology alone is unreliable because *C. auris* shares phenotypic characteristics with other yeasts, particularly members of the Candida haemulonii complex. A culture that yields a suspicious yeast should therefore undergo a validated identification procedure rather than being reported as *C. auris* on appearance alone [27], [29].

For colonization screening, selective or enrichment culture methods can improve recovery when the specimen contains mixed microbial populations. However, culture-based screening requires incubation and subsequent identification, making it generally slower than direct molecular detection. Culture methods also may fail to detect low organism burdens or organisms suppressed by competing flora [28], [30].

Biochemical identification systems and misidentification

Automated biochemical identification systems are widely used in clinical microbiology laboratories because they are relatively accessible and can process large numbers of isolates. However, their performance for *C. auris* depends on the platform, database version and identification algorithm [27], [29].

Reported examples of misidentification include:

- Candida haemulonii and related species.
- Candida famata or organisms historically reported under that name.
- Candida lusitaniae.
- Candida parapsilosis.
- Other uncommon yeast species, depending on the identification system.

Systems discussed in CDC identification guidance include VITEK 2 YST, API 20C AUX, BD Phoenix, MicroScan and RapID Yeast Plus. Some systems may produce a plausible species identification without recognizing *C. auris*, particularly when older databases are used [27].

This problem has important clinical consequences. Misidentification can delay infection-control measures, compromise outbreak recognition, distort local surveillance estimates and lead to inappropriate assumptions about antifungal susceptibility. Laboratories should follow current identification algorithms and refer suspicious or

unresolved isolates for confirmatory testing [27], [29].

Practical implication: An unusual or unexpected identification within the C. haemulonii complex, or an increase in unidentified Candida isolates in a healthcare unit, should prompt consideration of *C. auris* and further investigation.

MALDI-TOF mass spectrometry

MALDI-TOF MS is a protein-based identification technique that compares the mass spectral profile of a cultured organism with reference spectra stored in a validated database. It is one of the most reliable methods for identifying *C. auris* after a yeast isolate has been recovered in culture [26], [27].

Principle and diagnostic workflow

The method analyses characteristic microbial protein profiles, predominantly abundant cellular proteins, to generate a spectral fingerprint. The resulting spectrum is compared with the instrument's reference database to assign a species identification.

The usual workflow comprises:

1. Recovery of a yeast isolate from the clinical specimen.
2. Preparation of the isolate according to the instrument-specific protocol.
3. Acquisition of the mass spectrum.
4. Comparison with a validated reference database.
5. Review of the identification score and confirmation of suspicious or inconclusive results.

Diagnostic advantages

- Rapid identification after sufficient growth has been obtained.
- High identification reliability when the database contains suitable *C. auris* reference spectra.
- Ability to distinguish *C. auris* from closely related yeast species.
- Potential for high-throughput identification in laboratories with established MALDI-TOF infrastructure.
- Reduced dependence on lengthy biochemical assimilation profiles.

Real-time PCR and species-specific molecular assays

Molecular diagnostic methods detect specific nucleic acid sequences associated with *C. auris*. Real-time PCR is particularly valuable for colonization screening because it can provide results without waiting for visible colony growth. CDC guidance identifies real-time PCR as the preferred method for detecting colonization when appropriate laboratory resources are available [28].

Specimens and clinical applications

Real-time PCR can be used for screening specimens when the assay has been validated for that specimen type. Composite axilla–groin skin swabs are recommended by the CDC for colonization screening [28].

Molecular tests are also available for identifying *C. auris* directly from positive blood cultures. Examples of FDA-cleared molecular panels for this application include the GenMark ePlex Blood Culture Identification Fungal Pathogen Panel and the BioFire FilmArray BCID2 panel [27].

These applications should not be conflated: a test validated for positive blood cultures is not automatically suitable for screening skin swabs, environmental samples or other specimen types.

Advantages

- Rapid detection compared with conventional culture-based screening.
- High analytical specificity when the target and assay have been appropriately validated.
- Reduced dependence on colony morphology and biochemical identification.
- Potential to support early infection-control interventions.
- Suitability for targeted screening during healthcare-associated outbreaks.

Limitations

PCR may detect DNA from nonviable organisms and does not independently demonstrate that the organism is viable. It also does not ordinarily provide an antifungal susceptibility profile. False-negative results may occur because of inadequate specimen collection, low organism burden or assay-specific limitations.

As of the CDC guidance reviewed for this section, high-throughput FDA-cleared molecular tests for colonization screening swabs are not available in the same way as the cleared molecular panels used for positive blood cultures. Laboratories should use validated protocols and ensure that the assay has been evaluated for the intended specimen type [27], [28].

DNA sequencing: ITS and D1/D2 regions

DNA sequencing provides a molecular method for confirming the identity of an isolate when conventional identification is inconclusive or when high-confidence species identification is required.

Two commonly used targets are:

- Internal transcribed spacer (ITS) region: A region of ribosomal DNA widely used for fungal identification.
- D1/D2 region of the 28S ribosomal RNA gene: A ribosomal DNA region used for differentiating yeast species.

Sequencing results are compared with validated reference sequences or curated databases. Concordance with an appropriate reference supports species identification [27], [29].

ITS sequencing

ITS sequencing is useful for resolving ambiguous yeast identifications and confirming suspected *C. auris*. Its utility depends on the quality of the sequence, the suitability of the reference database and the ability of the selected region to distinguish closely related species.

D1/D2 sequencing

The D1/D2 region is another established target for yeast identification. Sequencing this region can provide confirmatory evidence when biochemical or mass-spectrometry identification is uncertain.

Whole-genome sequencing

Whole-genome sequencing (WGS) provides substantially greater genomic resolution than routine species identification methods. It is particularly useful for determining genetic relatedness between isolates, investigating transmission clusters, identifying geographic lineages and studying resistance-associated variants [29], [8].

However, WGS is generally more resource-intensive and requires specialized bioinformatics expertise. It is not usually the first-line test for every suspected clinical case.

Colonization screening: skin swabs and other specimens

Colonization screening is an important component of *C. auris* infection prevention because patients may carry the organism without clinical symptoms and contribute to transmission in healthcare facilities.

The CDC recommends a composite skin swab of the axilla and groin for screening. A single appropriate swab can be used to sample both areas according to the recommended collection protocol [28].

Other body sites, including the nares, hands, external ear canal, oropharynx, vagina and rectum, have been reported as sites from which *C. auris* can be recovered. However, the evidence supporting their routine use for screening varies, and these specimens should not automatically replace the recommended axilla–groin swab [28].

Clinical specimens should be selected according to the suspected site of infection. Blood cultures are particularly important in suspected candidemia, while specimens from other sites require interpretation in the context of clinical findings and the possibility of colonization.

A positive screening swab indicates colonization; it does not, by itself, establish invasive candidiasis or indicate that antifungal treatment is required. Conversely, a negative screening result does not necessarily exclude infection at another body site.

Antifungal susceptibility testing

AFST helps characterize the susceptibility of a recovered *C. auris* isolate to antifungal agents. It is important because resistance patterns vary among isolates and may affect therapeutic choices [26], [17].

Broth microdilution

Reference broth microdilution involves exposing a standardized fungal inoculum to serial concentrations of an antifungal agent and determining the minimum inhibitory concentration (MIC). The MIC is interpreted according to the defined endpoint and the applicable laboratory guidance.

CLSI reference methods are commonly used for yeast susceptibility testing. However, laboratories must recognize that conventional, fully validated species-specific clinical breakpoints are not established for all antifungal agents against *C. auris*. CDC has published tentative resistance thresholds to support interim interpretation [17].

Other validated methods

Commercial susceptibility panels and gradient diffusion methods may be used where their performance and interpretation are appropriate for the organism and antifungal agent. Results obtained using different methods may not be directly interchangeable.

AFST should be performed on an identified isolate. Direct molecular identification alone generally does not establish the isolate's complete phenotypic susceptibility profile.

Diagnostic versus therapeutic interpretation

A laboratory report should distinguish the measured MIC from its interpretation. A high MIC may indicate reduced in-vitro susceptibility, but the clinical implications depend on the antifungal agent, current interpretive guidance, infection site, drug exposure and clinical circumstances.

For epidemiological research, studies should report the testing method, antifungal panel, MIC distribution and criteria used to define resistance. This makes comparisons between institutions and geographic regions more meaningful.

Diagnostic performance, turnaround time, cost and availability

The choice of diagnostic method involves balancing analytical performance, time to result, laboratory infrastructure and the purpose of testing. Turnaround times below are approximate operational estimates, not universal validated performance measures; they vary with specimen type, culture requirements, instrument availability and laboratory workflow [21, 22, 23, 24].

Diagnostic method	Typical turnaround time	Relative cost	Laboratory availability	Main limitation
Conventional culture	Approximately 24–72 hours for initial growth;	Low	High	Slow and not species-specific

	longer for confirmation			
Chromogenic/selective culture	Approximately 24–72 hours or longer, depending on protocol	Low to moderate	Moderate to high	Colony appearance is not definitive
Biochemical identification	Hours after obtaining an isolate; may take longer with repeat testing	Low to moderate	High	Misidentification with outdated or unsuitable databases
MALDI-TOF MS	Minutes to hours after obtaining an isolate	High initial equipment cost	Variable; commonly available in larger laboratories	Requires a validated reference database
Real-time PCR	Approximately 2–6 hours after specimen receipt in an established workflow	Moderate to high	Variable	Assay validation and equipment required
ITS sequencing	Usually 1–3 days or longer after isolate preparation	Moderate to high	Variable	Requires sequencing and sequence interpretation
D1/D2 sequencing	Usually 1–3 days or longer after isolate preparation	Moderate to high	Variable	Requires suitable reference sequences
Whole-genome sequencing	Several days to weeks, depending on workflow	High	Limited to specialized laboratories	Cost and bioinformatics requirements
Broth microdilution AFST	Commonly 24–48 hours after inoculation, depending on protocol	Moderate	Variable	Requires a viable isolate and careful MIC interpretation

These estimates should be adapted to the facilities and workflows of the study setting. They should not be presented as measured sensitivity, specificity or turnaround-time results from a single comparative clinical study.

Sensitivity and specificity

Sensitivity and specificity depend on the assay, specimen type, reference standard, organism burden and patient population. A single universal sensitivity or specificity figure should not be assigned to MALDI-TOF MS, PCR or culture without identifying the particular assay and validation study.

For a systematic review, extract the following information whenever reported:

- Sensitivity and specificity, with confidence intervals.
- Positive and negative predictive values, where available.
- Number of true-positive, false-positive, true-negative and false-negative results.
- Reference standard used for comparison.
- Specimen type and patient population.
- Time from specimen receipt to report.
- Whether the assay was evaluated prospectively or retrospectively.

Comparative evidence table

The following table summarizes the principal diagnostic approaches and their practical applications.

Diagnostic method	**Target**	**Specimen**	**Turnaround time**	**Strengths**	**Limitations**
Conventional culture[26], [27]	Viable fungal	Blood, sterile	24–72 hours for	Low cost; provides	Slow; morphology cannot

	growth	fluids, urine, respiratory or wound specimens as clinically indicated	initial growth; longer for confirmation	an isolate for further testing	confirm species
Chromogenic/selective culture[26], [28]	Growth and presumptive phenotypic characteristics	Skin screening swabs or appropriate clinical specimens	Approximately 24–72 hours or longer	Accessible; useful for screening and isolate recovery	Colony colours overlap; competing flora may interfere
Biochemical identification[27], [29]	Metabolic and assimilation profile	Cultured yeast isolate	Hours after growth	Automated; available in many clinical laboratories	Database-dependent misidentification
MALDI-TOF MS[26], [27]	Protein spectral fingerprint	Cultured isolate	Minutes to hours after growth	Rapid, reliable species identification with an appropriate database	Equipment and updated database required
Real-time PCR[28], [30]	Species-specific DNA target	Validated skin swab or clinical specimen	Approximately 2–6 hours in an established workflow	Rapid; preferred approach for colonization screening when available	Does not establish viability or complete susceptibility profile
Commercial molecular panels[27]	Validated fungal nucleic acid targets	Positive blood culture	Usually hours after blood-culture positivity	Rapid identification in suspected bloodstream infection	Intended specimen types are restricted by assay validation
ITS sequencing[27], [29]	Fungal ribosomal DNA ITS region	Cultured isolate	Usually 1–3 days or longer	Confirmatory identification	Requires sequencing and reference database comparison
D1/D2 sequencing[27], [29]	28S ribosomal DNA D1/D2 region	Cultured isolate	Usually 1–3 days or longer	Useful for confirmation of ambiguous yeast identification	Specialized workflow; not usually first-line screening
Whole-genome sequencing[29], [8]	Genome-wide variation	Cultured isolate	Several days to week	High-resolution outbr	Expensive; specialist bioinf

			s	eak investigation and lineage analysis	ormatics required
Broth microdilution AFST[26], [17]	Phenotypic response to antifungals	Cultured isolate	Commonly 24–48 hours after inoculation	Quantitative MIC measurement	Interpretation is complicated by limited species-specific breakpoints

DISCUSSION

Global epidemiological patterns, antifungal resistance and diagnostic approaches in *C. auris*

Global evidence indicates that *C. auris* has evolved from a relatively uncommon, newly recognized yeast into a major healthcare-associated fungal pathogen characterized by international dissemination, persistent transmission in healthcare facilities and frequent antifungal resistance. Three recurring patterns emerge from the literature: the predominance of healthcare-associated outbreaks, substantial variation in antifungal susceptibility between genetic clades, and under-recognition where advanced identification and surveillance facilities are unavailable [6], [16]. The 2020 systematic review and meta-analysis by Hu et al. documented more than 4,733 reported cases across over 33 countries in the literature available up to October 2019, whereas subsequent investigations have demonstrated further geographic expansion and continued transmission [6]. These historical case counts should not be interpreted as estimates of the current global burden. The most important epidemiological distinction is that the number of reported cases reflects both actual transmission and the capacity of healthcare systems to detect, identify and report the organism. Consequently, countries with established molecular surveillance may report more cases than countries with comparable transmission but limited diagnostic infrastructure [16], [3].

Geographic variation in antifungal resistance

The most consistent finding across international studies is the high prevalence of fluconazole resistance, although the frequency of resistance to amphotericin B and echinocandins varies considerably between studies and geographic regions [6], [12]. In their global meta-analysis, Hu et al. estimated resistance rates of approximately 91% for fluconazole, 12% for amphotericin B and 12.1% for caspofungin, while resistance to micafungin and anidulafungin was substantially less frequent [6]. By contrast, the US genomic epidemiological investigation published in 2026 reported fluconazole resistance in 91% of tested isolates, amphotericin B resistance in 12%, and echinocandin resistance in 7%; resistance also differed markedly between clades [12]. The agreement in the high prevalence of fluconazole resistance strengthens the conclusion that fluconazole has limited reliability against *C. auris*. However, differences in echinocandin resistance estimates illustrate why pooled estimates should not be interpreted as universal susceptibility profiles. The studies differ in their observation periods, geographic composition, isolate selection, antifungal panels and interpretive criteria. Furthermore, the proportion of isolates tested against individual drugs may differ from the proportion tested against all three major antifungal classes. These methodological differences, combined with clade-specific resistance and local outbreak dynamics, can produce substantial variation in reported rates without necessarily indicating a true temporal change in resistance [6], [12], [31].

Genetic lineage provides an important explanation for this geographic heterogeneity. Clade I, associated predominantly with South Asia, and clade III, associated with Africa, have frequently demonstrated high fluconazole resistance, whereas earlier investigations of clade II from East Asia generally reported lower resistance to some antifungal agents [16], [12]. However, these associations should not be treated as fixed properties of every isolate within a clade. Resistance phenotypes may vary within lineages, and the distribution of clades within a country can change over time. Differences in antifungal prescribing, prior treatment exposure, hospital transmission and the selective inclusion of resistant isolates in reference-laboratory datasets may further influence observed patterns [16], [31]. Therefore, geographic differences in resistance should be interpreted as the combined result of fungal population structure, healthcare-associated transmission, antifungal selection pressure and surveillance practices rather than as evidence of an intrinsic country-level effect.

Relationship between resistance mechanisms and observed susceptibility patterns

The relationship between molecular resistance mechanisms and phenotypic susceptibility provides a biological explanation for many of the differences observed across global studies. Azole resistance is most consistently associated with alterations in ERG11, which encodes the azole target enzyme lanosterol 14α-demethylase, together with mechanisms involving drug efflux and gene regulation [31], [32]. In a multicentre Indian study of 350 isolates collected from 10 hospitals between 2009 and 2017, Chowdhary et al. reported fluconazole resistance in 90% of isolates, with MICs of 32 to at least 64 mg/L. Among the 44 resistant isolates examined in the reported ERG11 analysis, 34 (77%) carried substitutions at positions Y132 or K143, whereas isolates with wild-type residues at these positions had lower fluconazole MICs of 1–2 mg/L [31]. This association supports the biological relevance of ERG11 variation but also demonstrates that a single mutation cannot explain every resistant phenotype. Differences in gene expression, efflux activity, genetic background and other cellular mechanisms may contribute to the remaining variation [31], [32].

Echinocandin resistance shows a different relationship between genotype and susceptibility. Mutations in FKS1, which encodes a catalytic component of β-1,3-D-glucan synthase, can reduce drug-target susceptibility and produce elevated

echinocandin MICs. In the Indian multicentre study, the S639F substitution was associated with echinocandin resistance [31]. More recent US surveillance found an FKS1 mutation in 49 of 53 echinocandin-resistant isolates with available genomic data, further supporting the relationship between target-site alteration and resistance [12]. Nevertheless, occasional resistant isolates without recognized FKS1 mutations indicate that current molecular markers do not explain every phenotype. Similarly, amphotericin B resistance is less completely understood at the molecular level and may involve changes in membrane sterol composition or ergosterol biosynthesis [32], [33]. Collectively, these findings support combining molecular analysis with phenotypic AFST rather than treating either method as sufficient in every case. Molecular assays can identify recognized resistance-associated variants, while MIC testing measures the observed response under specified laboratory conditions; discordant results may identify mechanisms that require further investigation.

Clinical and practical trade-offs between MALDI-TOF MS, PCR and sequencing

The three principal identification approaches serve complementary rather than interchangeable purposes. MALDI-TOF MS provides rapid identification of cultured isolates and can be highly reliable when the instrument contains a suitable, updated reference database. Its principal advantages are high-throughput processing and relatively rapid identification after culture growth, whereas its disadvantages include substantial initial equipment costs, maintenance requirements and dependence on database quality [16], [3]. Real-time PCR can detect *C. auris* DNA more rapidly than culture-based workflows and is particularly valuable for colonization screening and selected direct-detection applications. However, PCR performance depends on specimen collection, assay design and validation, and a positive molecular result does not necessarily demonstrate viable organisms or provide a complete antifungal susceptibility profile [16], [3]. Sequencing of ITS or D1/D2 regions can resolve ambiguous species identification, while whole-genome sequencing provides higher resolution for lineage assignment, outbreak investigation and analysis of resistance-associated variants [16], [12]. Its principal disadvantages are higher resource requirements, specialist expertise and longer turnaround times in laboratories without established sequencing workflows. Thus, MALDI-TOF MS is particularly suitable for routine identification after culture, PCR is advantageous for targeted rapid detection, and sequencing is most valuable for confirmation and advanced epidemiological characterization. The optimal choice depends on the clinical question, the need for immediate infection-control action and the laboratory's available resources.

Diagnostic limitations in low-resource laboratories

In low-resource laboratories, the main challenge is not simply the absence of advanced technology but the interaction between limited diagnostic capacity and the consequences of delayed or incorrect identification. Conventional biochemical systems may misidentify *C. auris* as related yeast species, particularly when their databases do not include validated identification profiles. Such errors can prevent timely recognition of cases, delay contact precautions and lead to underestimation of local transmission [16], [3]. MALDI-TOF MS and molecular testing may be unavailable because of equipment costs, reagent procurement difficulties, maintenance requirements and shortages of trained personnel. Reference sequencing and genomic surveillance are even less accessible in many settings [3]. Culture-based methods remain useful because they are relatively accessible and provide isolates for further testing, but they cannot independently resolve all species-identification problems and may delay recognition when additional confirmation is required. Furthermore, laboratories without routine AFST may be unable to distinguish elevated MICs from susceptible phenotypes reliably, especially where species-specific clinical breakpoints remain unavailable. These limitations are particularly consequential in healthcare facilities with high patient turnover, prolonged ICU admissions and limited infection-prevention resources. Consequently, published case counts from resource-constrained settings may reflect diagnostic capacity as much as the underlying burden of disease, and apparent geographic differences should be interpreted cautiously [16], [3].

Improving surveillance in India and other resource-limited settings

Improving surveillance requires a tiered system that connects routine hospital laboratories with regional reference centres and national public-health networks. At the hospital level, laboratories should establish protocols for recognizing suspicious yeast isolates, using validated identification databases and referring unresolved isolates for confirmation. Where real-time PCR is available, standardized axilla–groin screening can support targeted surveillance after a confirmed case or when an epidemiological investigation indicates a need for screening [3], [34]. At the regional level, reference laboratories can provide confirmatory MALDI-TOF MS, sequencing and AFST for facilities lacking these capabilities. Standardized reporting should capture specimen type, clinical setting, colonization versus infection, identification method, MIC values, interpretive criteria and antifungal exposure. These data would make

comparisons between institutions more reliable and help distinguish genuine changes in resistance from differences in testing practices. In India, sentinel surveillance across tertiary-care hospitals, medical colleges and selected regional facilities could provide a practical starting point, with priority given to ICU-associated infections, suspected outbreaks and isolates with unusual susceptibility patterns. Periodic genomic analysis of representative isolates could then investigate transmission clusters and the distribution of resistance-associated variants. Such a system would be more feasible than requiring every hospital to perform whole-genome sequencing. Its effectiveness would depend on sustainable funding, laboratory training, quality assurance, timely communication of results and coordinated infection-prevention responses [3, 6, 13], [34].

LIMITATIONS

The evidence base for *C. auris* epidemiology and antifungal resistance has several important limitations. First, studies use heterogeneous designs, including retrospective case series, outbreak investigations, multicentre surveillance and systematic reviews, with substantial differences in population selection, specimen types and follow-up. Consequently, estimates derived from ICU cohorts or reference-laboratory isolates cannot necessarily be generalized to all hospitals or national populations [6], [16]. Second, antifungal susceptibility findings are affected by differences in testing platforms, laboratory protocols and interpretive criteria. The absence of fully validated species-specific clinical breakpoints for many drug–organism combinations further complicates comparisons, because studies may apply different thresholds or use terms such as resistance and reduced susceptibility inconsistently [3], [12]. Third, publication bias may favour unusual outbreaks, multidrug-resistant isolates and severe infections over routine negative findings or susceptible isolates. Fourth, under-reporting is likely where species identification and confirmatory testing are limited, creating a geographic imbalance in published evidence. Countries with stronger surveillance systems may therefore appear to have a greater burden simply because they detect and report more cases [6], [16]. Finally, some studies contain small numbers of isolates from particular clades or geographic regions, limiting the precision of resistance estimates and the generalizability of genotype–phenotype associations. These limitations mean that apparent differences between studies should be interpreted in relation to their methods, denominators, clinical settings and sampling frames. Future systematic reviews should prioritize standardized data extraction, transparent resistance definitions, stratification by clade and geography, and separate analyses of colonization, invasive infection and outbreak-associated isolates.

CONCLUSION

C. auris has emerged as a significant global healthcare-associated fungal pathogen because of its rapid geographic spread, persistent transmission in healthcare environments, frequent antifungal resistance and challenges in routine laboratory identification. The available evidence consistently demonstrates a high prevalence of fluconazole resistance, while resistance to amphotericin B and echinocandins varies across geographic regions, genetic clades and study populations. These differences reflect the combined influence of molecular resistance mechanisms, healthcare-associated transmission, antifungal exposure and variations in surveillance and susceptibility-testing practices. Although MALDI-TOF mass spectrometry and species-specific molecular assays have improved the speed and accuracy of identification, their availability remains limited in many resource-constrained settings, particularly where updated databases, specialized equipment and trained personnel are lacking. Integration of conventional culture, validated identification methods, antifungal susceptibility testing and molecular surveillance is therefore essential for reliable diagnosis, appropriate treatment and effective outbreak control. In India and other resource-limited regions, strengthening laboratory infrastructure, establishing standardized surveillance networks, improving access to reference laboratories and expanding genomic investigations should be prioritized. Future research should focus on harmonizing diagnostic and susceptibility-testing methods, refining resistance-associated genotype–phenotype relationships and generating geographically representative clinical and epidemiological data. Such coordinated efforts are crucial to improving early detection, guiding evidence-based antifungal therapy and limiting the further spread of multidrug-resistant C. auris.

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