# Candida auris

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## Continuing Education Activity

*Candida auris* (*C auris*) is an emerging multidrug-resistant yeast that poses a significant global health threat. First identified in 2009, this pathogen has now been isolated in over 60 countries across 6 continents. Unlike other Candida species, *C auris* demonstrates a unique ability to colonize the skin, persist on surfaces for prolonged periods, and spread efficiently within healthcare facilities. This course reviews its association with outbreaks, high mortality rates of 30% to 60%, and frequent misidentification in clinical laboratories, which has prompted the World Health Organization to classify it as a “critical priority” fungal pathogen. Participants will gain an in-depth understanding of accurate detection, timely diagnosis, and appropriate therapeutic selection for *C auris,* which is essential for treatment, as options remain limited due to variable resistance patterns across antifungal classes.

This activity examines the most recent evidence on the epidemiology, transmission, diagnosis, and management of *C auris*. Yeast identification methods employed by laboratories can misidentify *C auris* as other yeasts when using traditional biochemical methods, making the detection and control of this pathogen difficult. The transmission of *C auris* occurs in nosocomial settings, even in those implementing infection prevention and control measures. This activity for healthcare professionals is designed to enhance the learner's understanding of *C auris*'s resistance mechanisms, emerging treatment approaches, infection control strategies, and competence in optimizing diagnostic accuracy, as well as implementing interprofessional therapeutic decision-making and infection prevention practices to enhance patient safety and reduce healthcare-associated outbreaks.

**Objectives:**

- Identify the epidemiology of *Candida auris* infections.
- Differentiate *Candida auris* from other Candida species using evidence-based laboratory diagnostic methods.

- Apply infection prevention and control measures to reduce nosocomial transmission of *Candida auris*.
- Collaborate with an interprofessional team to develop strategies for improving care coordination to reduce *Candida auris* infections and improve patient outcomes.

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**Introduction**

*Candida auris* (*C auris*), a multidrug-resistant yeast, has emerged as a significant global concern among fungal pathogens. This organism readily colonizes the skin and demonstrates a strong association with nosocomial infections in healthcare environments. Its capacity to trigger outbreaks linked to high mortality rates led the World Health Organization (WHO) to classify it within the "critical priority group" of the fungal priority pathogen list.[WHO.Fungal Priority Pathogens List.2022] First identified as a novel *Candida* species in 2009, *C auris* has since been reported in 61 countries across 6 continents as of 2023.[1]

Although *C auris* frequently colonizes the skin, the organism can also cause invasive infections associated with mortality rates ranging from 30% to 60%.[2] This yeast qualifies as a multidrug-resistant species, showing variable resistance patterns to many antifungal agents typically used for other *Candida* infections. The rising prevalence of both infection and colonization in recent years reflects several contributing factors, including the ability of *C auris* to persist on skin and abiotic surfaces for extended periods,[3] efficient transmission within healthcare facilities,[4] frequent diagnostic challenges with misidentification,[5] and high resistance rates across multiple antifungal classes.[6]

Laboratory yeast identification methods often misclassify *C auris* as other yeast species, complicating both detection and control efforts.[5] Transmission most commonly occurs in nosocomial settings, even where infection prevention and control measures are actively enforced. In the United States, *C auris* is designated as a nationally notifiable pathogen, enabling systematic public health monitoring and targeted containment of its spread.

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**Etiology**

*C auris* is a yeast species belonging to the genus *Candida*, which gets its name from the Latin word *auris* ("ear"), as it was first isolated from the external ear canal of a patient in a Japanese hospital in 2009.[7] Analysis of the yeast genomic DNA revealed a distinct

species with a close phylogenetic profile to *Candida ruelliae*, *Candida haemulonii*, *Candida duobushaemulonii*, and *Candida pseudohaemulonii*.[7][8]

*C auris* is a budding yeast with cells that may be single, in pairs, or in groups. The cells are ovoid, ellipsoidal, or elongate, and measure 2.5 to 5 µm in size. *C auris* rarely forms hyphae or pseudohyphae, nor does it form germ tubes.[9] However, growth under high-salt stress, eg, on yeast extract, tryptone, and dextrose plus 10% NaCl, and depletion of heat-shock proteins can induce pseudohyphae-like forms.[10][11] *C auris* strain grows well at 40 °C but shows slow growth at 42 °C.[7] The colonial growth of *C auris* in culture medium varies depending on the medium. On Sabouraud agar, *C auris* produces smooth, white to cream-colored colonies.[12] On CHROMagar, colonies of *C auris* may display multiple color morphs ranging from pale to dark pink and rarely beige.

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**Epidemiology**

*C auris* likely evolved from a plant saprophyte to become a human pathogen after adapting to higher environmental temperatures.[13] Genetic analysis of *C auris* isolates has demonstrated the following 6 distinct geographical clades:

- Clade 1 (South Asian)
- Clade II (East Asian)
- Clade III (South African)
- Clade IV (South American)
- Clade V (Iran)
- Clade VI (Singapore) [14][15]

Additional clades may yet be discovered. Genetic differences between the clades are suggestive that *C auris* emerged independently in the aforementioned geographic locations.

The prevalence of *C auris* infection globally is unknown and likely underreported due to the lack of commercially available diagnostic methods and resemblance to other phenotypically related *Candida* species.[16] A study queried the international SENTRY Antifungal Surveillance Program that sought to identify 15,271 candidemia isolates collected between 2004 and 2015 from 152 international medical centers (Asia, Europe, Latin America, and North America). The study revealed that no *C auris* isolates were identified before 2009, indicating that the prevalence of *C auris* was rare before this

time.[17] Further surveillance studies of misidentified samples recovered from South Korea in 1996, 2004, and 2006, as well as Pakistan in 2008, later detected *C auris*.[18] These studies suggest that *C auris* emerged before 2009, although the rapid global spread occurred afterward. As of December 2023, *C auris* had been identified in 61 countries across all continents except Antarctica.[1]

In the United States, the Centers for Disease Control and Prevention (CDC) reported 4,514 new clinical cases of *C auris* in 2023, with continued year-over-year increases in case counts since the first reported case in 2016. Between 2016 and 2023, a total of 10,788 clinical cases were reported.[CDC.Tracking Candida Auris.2024] Epidemiologic information from these cases suggests that most strains were introduced from abroad and that these strains belonged to the clades of *C auris* originating from Clade I (South Asian) and Clade IV (South American).[12][19][20] While the isolates belong to distinct clades that originated abroad, most cases of the infection were acquired in the United States within a healthcare setting, demonstrating clonal nosocomial transmission.[21]

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## Pathophysiology

### Transmission

*C auris* spreads efficiently from person to person.[22] Unlike most other *Candida* species, which typically cause infection through the host's own microflora, *C auris* transmission often occurs through direct acquisition from another individual. This pathogen does not function as a resident commensal organism and rarely inhabits the human gastrointestinal tract, a characteristic that differentiates it from many *Candida* species.[23] *C auris* shows a marked affinity for colonizing the skin, particularly in the axilla and groin. Colonization can develop within days to weeks of exposure, and invasive infection may follow within days to months.[6] Once established, colonization may persist for many months or even indefinitely.[24] Individuals colonized with *C auris* often shed the fungus into their surroundings despite appearing asymptomatic, underscoring the need to identify colonized patients before placement of indwelling devices or surgical procedures. These individuals may transmit the organism to other patients and abiotic surfaces.

Environmental contamination plays a crucial role in the transmission process. Patients may shed *C auris* onto surfaces and fomites, including hallways, chairs, beds, windowsills, counters, electrocardiogram leads, blood pressure cuffs, infusion pumps, and ventilators.[24] Shared multiuse equipment, eg, temperature probes and pulse oximeters, may act as reservoirs.[25] Laboratory studies have shown that *C auris* can survive on moist or dry surfaces for up to 7 days,[26] with some cells remaining viable for as long as 4 weeks

and culturable for up to 2 weeks following colonization.[27] Strict adherence to isolation protocols and contact precautions plays a vital role in preventing nosocomial transmission of *C auris*.

**Virulence Factors**

Genetic studies of *C auris* have revealed that a substantial percentage of its genes are involved in central metabolism, a common trait among pathogenic *Candida* species that enables adaptation in diverse environments.[28] *C auris* has numerous virulence attributes that resemble *C albicans*, eg, enzyme secretion, nutrient acquisition, siderophore-based iron acquisition, tissue invasion, 2-component histidine kinase system, and pathways involved in cell wall modeling.[29][30] Virulence factors may be strain-dependent. A study of 16 *C auris* isolates revealed varying levels of phospholipase and proteinase production.[31]

*C auris* has also demonstrated the ability to evade the immune response. In a comparison study between *C albicans* and *C auris*, neutrophils preferentially targeted and killed *C albicans*.[32] This same study demonstrated that *C auris* evaded neutrophil attack and the innate immune response, a finding similar to another study that showed greater recognition and the ability to stimulate cytokine release and phagocytosis in *C albicans* compared to *C auris*.[33] *C auris* also has an expanded family of proteases and lipases that facilitate tissue invasion and acquisition of nutrients, leading to pathogenicity.[34] In addition, *C auris* possesses a unique surface colonization factor, Scfl, which is crucial for adhesion to both biological and inert surfaces, as well as for biofilm formation and virulence.[35] Importantly, *C auris* forms robust biofilms on both biological and abiotic surfaces, eg, medical and prosthetic devices, which contributes to its persistence on surfaces and nosocomial transmission.[35][36]

In vitro studies have shown that *C auris* isolates may be aggregating or nonaggregating. The failure of *C auris* to release daughter cells after budding results in a large aggregation of cells that is difficult to disrupt by detergent vortexing or detergent.[37] The property of aggregating strains is thought to promote survival in hospital environments. However, in vivo models have shown that the nonaggregating isolates exhibit more pathogenicity than aggregating isolates and have greater pathogenicity than *C albicans*.[31][37] The thermotolerance of *C auris*, which grows optimally at 37 °C and survives in temperatures up to 42 °C, also helps certain strains persist in hospital environments. This thermotolerance is largely governed by the calcineurin and Ras/cAMP/PKA pathways.[38][39]

**Resistance Factors**

The primary factor contributing to the high mortality rates from *C auris* infection is its ability to develop resistance to multiple antifungal agents.[31] Biofilm formation enables the sequestration of drugs within the extracellular matrix, conferring antifungal tolerance observed in many *Candida* species.[40] A recent study showed that matrix sequesters nearly 70% of the available triazole antifungal due to its rich mannan-glucan polysaccharides.[41] Although *C auris* forms less complex and robust biofilms compared to other *Candida* species (eg, *C albicans), C auris* can form high-burden biofilms in specific environments (eg, on skin), contributing to colonization and environmental persistence.[42] One study found that *C auris* isolates with biofilms were not susceptible to any antifungal agent, including fluconazole, echinocandins, and polyenes, compared to planktonic *C auris* isolates, which were only resistant to fluconazole.[43] Genetic studies of *C auris* have revealed expansions of genes associated with drug resistance and multidrug efflux.[8] Resistance to azoles and echinocandins is mediated by mutations in the genes encoding the lanosterol 14-alpha-demethylase (*ERG11*) gene and drug target 1,3-beta-glucan synthase (FSK1), respectively.[44] Efflux pumps (eg, the ATP-binding cassette (ABC)) and major facilitator superfamily (MFS) also play a role in azole resistance.[30][45]

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**History and Physical**

The clinical presentation of *C auris* infection is similar to that of other *Candida* species. *C auris* has been isolated from different body sites, including the nose, pharynx, sputum, lungs, pleural cavity, heart, blood, liver, abdominal cavity (peritoneal fluid), rectal or stool culture, urine, vagina, bone, axilla, groin, wounds/surgical tissue, pus, ear, and brain.[46][47] C *auris* colonization is thought to be uncommon in healthy individuals who have not recently been hospitalized. In a study in Bangladesh of 800 newly hospitalized individuals without recent healthcare exposure within the previous 3 months, skin swabs from the axilla and groin isolated no *C auris*.[48]

Isolates from nonsterile body sites (eg, the genitourinary tract, skin and soft tissues, and lungs) likely represent colonization rather than actual infection.[12] Any indwelling devices, eg, venous catheters, ports, urinary catheters, and prosthetic devices, should be examined for erythema, tenderness, and purulent material.

Clinical conditions include bloodstream infections (candidemia), as well as infections of the lungs, kidneys, liver, skin, ears, and urinary tracts.[49][50] Compared to other *Candida* species, which are typical commensals of the gastrointestinal tract and not typically associated with nosocomial transmission, *C auris* has been shown to thrive on the

skin.[6] *C auris* forms a multilayer biofilm that proliferates best in the milieu that mimics sweaty axillary skin conditions.[51] Individuals colonized with *C auris* are a source of transmission to others. Colonization may occur within a few hours to a few days of exposure, and invasive infections may develop within days to months after initial colonization.[6]

Risk factors associated with *C auris* infections are similar to those of other *Candida* species.[47] These risk factors include:

- Presence of a central venous catheter
- Indwelling urinary catheter
- Immunosuppressive state (eg, human immunodeficiency virus, hematologic malignancy, solid tumors, transplant recipients, neutropenia, chemotherapy, and corticosteroid therapy)
- Diabetes mellitus
- Chronic kidney disease
- Exposure to broad-spectrum antibiotics or previous exposure to antifungal agents within 30 days
- Concomitant bacteremia or candiduria
- Parenteral nutrition
- Blood transfusion
- Hemodialysis
- Surgery within 30 days
- Admission to intensive care units

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## Evaluation

### Laboratory Identification Studies

The evaluation of suspected *C auris* infection begins with obtaining a clinical specimen from the site of infection. Traditional diagnostic methods for candidemia and invasive candidiasis rely on positive blood cultures. In patients with focal signs of infection, a biopsy should be collected when feasible for staining, culture, and histopathologic assessment. These conventional approaches, however, demonstrate limited sensitivity. Blood cultures

for invasive candidiasis yield a sensitivity of approximately 50%, with even lower detection rates in patients harboring deep-seated infections without candidemia.[52]

Accurate identification of *C auris* presents considerable challenges for clinical laboratories. Standard microbiology methods frequently misidentify the organism, leading to diagnostic delays. Phenotypic characterization, such as the morphology and pigmentation of colonies in culture, combined with the organism's ability to grow at high temperatures up to 42 °C and in saline-rich environments, may aid in distinguishing *C auris* from other *Candida* species. Despite this, phenotypic traits alone cannot provide definitive identification. Common diagnostic platforms often misclassify *C auris* as *C haemulonii* or other yeast species, including *C famata, C guilliermondii, C lusitaniae, C parapsilosis, C sake, Saccharomyces cerevisiae*, and *Rhodotorula glutinis*.[53]

**Molecular studies**

Molecular identification methods are the standard-of-care testing for a definitive diagnosis of *C auris*. Many biochemical methods and automated testing methods commonly misidentify *C auris* for other *Candida* species, notably *C haemulonii* and other yeast species.[54] Nonculture-based methods (eg, the beta-D-glucan (BDG) assay) have a sensitivity of approximately 73% in diagnosing candidemia overall, but with a lower sensitivity of 51% for diagnosing candidemia from *C auris*.[55] The most accurate identification is achieved using devices equipped with matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) and appropriate reference databases, which can differentiate *C auris* from other *Candida* species.[56]

Accurate identification relies on the spectra of the sample organisms. This results in the misidentification of *C auris* as *C albicans* and *C haemulonii* by MALDI-TOF MS.[57] Once spectra are added to the MALDI-TOF MS database, the labeling of *C auris* to the species level becomes accurate, although the distinction between geographic strains depends on the number of spectra for each clade in the library.[58] Other molecular methods have also been developed and are based on sequencing the D1-D2 region of the 28s rDNA or the internal transcribed region (ITS) of the rDNA.[16][59] An automated molecular test using competitive DNA hybridization and electrochemical detection can rapidly distinguish 15 fungal pathogens, including *C auris*. A multi-center study demonstrated that this testing method has 100% sensitivity and specificity for *C auris*, *C dubliniensis*, *C famata*, and *C krusei,* and was able to distinguish between other *Candida* species, including *C glabrata*, *C lusitaniae*, *C albicans*, *C tropicalis*, and *C parapsilosis*.[60] The more accurate, reliable, and rapid forms of molecular tests are not always available in all facilities, which makes the diagnosis, management, and infection control efforts challenging.

A range of molecular techniques, including amplified fragment length polymorphism (AFLP), have been utilized for the typing of *C auris* isolates. The role of AFLP analysis in the demarcation of the geographical clusters has been demonstrated.[61]

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**Treatment / Management**

The most challenging aspect of managing invasive *C auris* infections is the level of drug resistance and the ability of *C auris* to develop drug resistance to the 3 main classes of antifungals, as previously discussed. A study from India investigated the susceptibility patterns of 350 *C auris* isolates and showed that 90% were resistant to azoles (fluconazole), 8% were resistant to polyene (amphotericin B), and 2% were resistant to echinocandins (anidulafungin and micafungin).[44] The study showed that overall, 25% of isolates were multidrug-resistant, and 13% of the isolates were multi-azole-resistant.[44] The CDC breakpoint analysis of isolates in the United States revealed exceptionally high minimal inhibitory concentrations (MICs) for azoles, echinocandins, polyenes, and nucleoside analogs.[62]

In vitro investigations reveal that the synergistic use of antifungals has shown promising initial results for the combination treatment of voriconazole and micafungin in multiresistant isolates. However, this was not observed in other combinations of echinocandins and azoles.[63]

Concrete documentation of standardized therapeutic options for *C auris* infection remains absent. Most cases require individualized management, directed by antifungal susceptibility testing. Expert consultation with an infectious disease specialist provides critical guidance in optimizing treatment decisions. Antifungal therapy should begin only when clinical disease is present. Management should be avoided in patients merely colonized with *C auris* when isolates originate from noninvasive sites (eg, the respiratory tract, urine, or skin).[46]

The CDC has published tentative guidelines for initial therapy.[46] Concerns about resistance to triazole antifungal agents and amphotericin B have led to the recommendation of using echinocandins as empirical treatment before the availability of specific susceptibility testing results.[64][65] Adults and children older than 2 months may be started on echinocandin therapy with caspofungin or micafungin. Anidulafungin may be used in adults; however, this medication is not approved for use in children and should be avoided in this age category. Monitoring for clinical improvement, repeating blood cultures to ensure clearance of fungemia, and repeating susceptibility testing should be conducted, as resistance to echinocandins may develop. In clinically unresponsive patients, liposomal

amphotericin B may be considered as an alternative. In neonates and infants younger than 2 months, the initial choice of antifungal therapy is amphotericin B deoxycholate, followed by liposomal amphotericin B. Echinocandins are not recommended for the treatment of *C auris* infection of the central nervous system, given their poor uptake.

Effective management of candidemia requires more than timely antifungal therapy. Removal of central venous catheters or other indwelling devices, along with prompt drainage of infectious collections, represents essential steps in care. Persistent positive blood cultures should prompt a thorough search for metastatic foci, including endocarditis, suppurative thrombophlebitis, or abscess formation. Nonneutropenic patients with candidemia should receive a dilated ophthalmologic examination within the first week of diagnosis, while neutropenic patients should undergo the same evaluation 1 week after recovery from neutropenia to screen for endophthalmitis, chorioretinitis, and vitritis.

Repeat blood cultures must be obtained daily or every other day until candidemia clears. The Infectious Disease Society of America recommends continuing antifungal therapy for 2 weeks after blood cultures remain negative in patients without metastatic complications.[66] Emerging antifungal agents offer future therapeutic options for *C auris* infection, including the first-in-class agent fosmanogepix, the novel triazole opelconazole, the second-generation echinocandin rezafungin, and the triterpenoid antifungal ibrexafungerp.[67]

Prevention of invasive infection in colonized individuals involves minimizing the entry of the organism into sterile body sites. Ensuring appropriate use of medical devices (eg, central venous catheters, indwelling urinary catheters) and maintenance of tracheostomy sites is needed. Continuous assessment of the need for such invasive lines and tubes, followed by prompt removal, is a basic strategy to mitigate the risk of introducing organisms to sterile sites. Patients undergoing surgical procedures should have meticulous skin preparation with an alcohol-based agent.[46]

The site of infection plays a crucial role in the choice of antifungals for invasive infections. Echinocandins do not have adequate penetration into many sites (eg, cerebrospinal fluid), as previously discussed, because of their high molecular weight, and the very inactive drug could be recovered from urine.[68] The use of amphotericin B, with the potential addition of 5-flucytosine, has been recommended for treating urinary tract infections.[68] For central nervous system disease, as with other candidal infections, an empirical combination of amphotericin B and 5-flucytosine has shown some success, with therapy tailored according to sensitivity testing results.[69]

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## Differential Diagnosis

The differential diagnoses of *C auris* include invasive fungal infections caused by other species of *Candida* and comprise 95% of all invasive fungal infections, including:

- *Candida albicans*
- *Candida glabrata*
- *Candida tropicalis*
- *Candida paratropicalis*
- *Pichia kudriavzevii* [70]

Other differentials include:

- Aspergillosis
- Bacterial sepsis
- Cryptococcosis
- Septic shock

While these pathogens are more common, infections with *C auris* typically occur in severely ill individuals in healthcare settings such as intensive care units and long-term care settings. Additional risk factors include immunosuppression, prolonged or frequent hospitalizations, extensive or long-term use of antimicrobial agents, and the presence of invasive medical devices. These risk factors also increase the likelihood of individuals contracting other infections, although they do not specifically increase the risk of *C auris* infection.

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## Prognosis

The mortality rate of invasive infections associated with *C auris* is comparatively higher than that of other *Candida* species, with mortality rates ranging from 30% to 60%.[2]. The variable mortality rate data may be due to several factors, including the extent of the infection, age, associated risk factors, and comorbid conditions. Infections have been reported in preterm infants and older adults.[47] Pediatric populations have shown a higher likelihood of survival compared to older populations.[71] Early identification of *C auris* and

prompt treatment with appropriate antifungal regimens are associated with higher survival rates.[18]

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**Complications**

The complications of invasive *C auris* infection vary depending on the extent of the infection, host comorbidities, and resistance patterns. While the most common presentation of *C auris* infection occurs as bloodstream infection (candidemia), it may spread hematogenously to seed different organs and cause multiorgan dysfunction. Conversely, a localized infection may eventually become an overwhelming bloodstream infection and have further complications such as sepsis, multi-organ system failure involving the kidneys, heart, lungs, eyes, brain, liver, and spleen, and ultimately death.

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**Deterrence and Patient Education**

Given the high rates of transmissibility and antifungal resistance patterns, *C auris* has been declared a public threat by the CDC. In June 2016, the CDC announced to general clinicians, infection control clinicians, laboratories, and public health authorities about *C auris*, making all cases in the United States reportable.[12] The CDC has outlined various aspects of infection control and prevention of *C auris*. In 2022, the WHO identified *C auris* as 1 of 4 fungal pathogens in the "critical priority group."[WHO.Fungal Priority Pathogens List.2022]

Good hand hygiene is the fundamental component of infection control. Healthcare personnel should adhere to standard hand hygiene principles to prevent the spread of *C auris*.[24] The preferred alcohol-based hand rubs are effective against *C auris*, as are chlorhexidine hand rubs when hands are not visibly soiled.[72] Visibly soiled hands should be washed with soap and water. Contact precautions, including the use of gowns and gloves, should be followed. Gloves do not substitute for hand hygiene.

The recommendations for infection control of *C auris* are adapted from infection control strategies for *Clostridium difficile* infections and other multidrug-resistant organisms, which have demonstrated rapid nosocomial spread. Infection control is applied to both infected and colonized individuals since both pose a risk of transmission.

Transmission-based precautions are implemented in acute care hospitals, long-term acute care hospitals, and nursing homes, including skilled nursing facilities with ventilator units. In the acute care setting, contact precautions are recommended, and in skilled nursing facilities, either contact precautions or enhanced barrier precautions are used. Contact

precautions include the use of gloves and gowns by healthcare personnel, single-room placement, and grouping patients with only *C auris* infection or colonization in cohorts in nonsingle-occupancy rooms or specific hospital wings.[20]

Notably, patients with *C auris* should not be grouped with those having other multidrug-resistant organisms, excluding *C auris*. Patients may remain colonized with *C auris* for months, even after the treatment and resolution of an acute infection. Patients are recommended to stay on contact precautions for the entire duration of their hospitalization. The CDC does not recommend routine assessment for colonization. However, patients with a prolonged hospital stay or residing in nursing homes may be screened 3 months after the last *C auris*-positive test, provided they are no longer on antifungal therapy for at least 1 week or have received topical antiseptics for 48 hours. Contact precautions may be discontinued if the patient has had 2 negative colonization tests at least 1 week apart.

*C auris* may persist in the healthcare environment on a variety of surfaces.[26] Environmental disinfection of the patient's room and other areas where care is received should be performed daily. Equipment shared between patients should be thoroughly cleaned and disinfected. Fungicidal products that are effective against other *Candida* species and quaternary ammonia compounds may not necessarily be effective against *C auris*.[73] Ultraviolet light, commonly used for environmental disinfection, appears to be ineffective against *C auris*.[74] In vitro studies have demonstrated that sodium hypochlorite and hydrogen peroxide are effective against *C auris*.[72][73][75] Environmental studies have found that rooms cleaned with sodium hypochlorite and hydrogen peroxide vapor were effective.[20][76]

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**Enhancing Healthcare Team Outcomes**

*Candida auris* is an emerging multidrug-resistant fungal pathogen that poses a critical global health threat. Unlike other Candida species, it thrives on skin, persists in healthcare environments, and spreads easily in clinical settings. Its high mortality rates, challenges in laboratory identification, and resistance to multiple antifungal classes make it a unique and urgent concern. The World Health Organization has listed *C auris* as a priority pathogen, and its management requires rapid recognition, evidence-based treatment, and strict infection prevention strategies. Effective response depends on coordinated efforts across healthcare disciplines and continuous epidemiological monitoring.

Physicians, advanced practitioners, and infectious disease specialists must collaborate closely with laboratory technicians to ensure accurate diagnosis, timely reporting, and

tailored antifungal therapy. Nurses, pharmacists, and infection prevention officers play vital roles in monitoring patients, managing therapies, ensuring environmental disinfection, and preventing the spread of infections within facilities. Open interprofessional communication and precise record-keeping strengthen coordination and patient safety. Engagement with epidemiologists, both within institutions and globally, supports surveillance and outbreak control. By sharing information, aligning strategies, and coordinating care, healthcare teams improve outcomes, reduce transmission, and protect both individual patients and community health.

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