

The laboratory investigation, management, and infection prevention and control of *Candida auris*: a narrative review to inform the 2024 national guidance update in England

Christopher R. Jones^{1,†}, Claire Neill^{1,†}, Andrew M. Borman^{2,3}, Emma L. Budd¹, Martina Cummins⁴, Carole Fry¹, Rebecca L. Guy¹, Katie Jeffery^{5,6}, Elizabeth M. Johnson^{2,3}, Rohini Manuel⁷, Mariyam Mirfenderesky¹, Ginny Moore⁸, Bharat Patel⁷, Silke Schelenz⁹, Karren Staniforth¹, Surabhi K. Taori¹⁰ and Colin S. Brown^{1,11,*}

Abstract

The emergent fungal pathogen *Candida auris* is increasingly recognised as an important cause of healthcare-associated infections globally. It is highly transmissible, adaptable, and persistent, resulting in an organism with significant outbreak potential that risks devastating consequences. Progress in the ability to identify *C. auris* in clinical specimens is encouraging, but laboratory diagnostic capacity and surveillance systems are lacking in many countries. Intrinsic resistance to commonly used antifungals, combined with the ability to rapidly acquire resistance to therapy, substantially restricts treatment options and novel agents are desperately needed. Despite this, outbreaks can be interrupted, and mortality avoided or minimised, through the application of rigorous infection prevention and control measures with an increasing evidence base. This review provides an update on epidemiology, the impact of the COVID-19 pandemic, risk factors, identification and typing, resistance profiles, treatment, detection of colonisation, and infection prevention and control measures for *C. auris*. This review has informed a planned 2024 update to the United Kingdom Health Security Agency (UKHSA) guidance on the laboratory investigation, management, and infection prevention and control of *Candida auris*. A multidisciplinary response is needed to control *C. auris* transmission in a healthcare setting and should emphasise outbreak preparedness and response, rapid contact tracing and isolation or cohorting of patients and staff, strict hand hygiene and other infection prevention and control measures, dedicated or single-use equipment, appropriate disinfection, and effective communication concerning patient transfers and discharge.

DATA SUMMARY

Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

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Author affiliations: ¹HCAI, Fungal, AMR, AMU, and Sepsis Division, UK Health Security Agency, London, UK; ²UKHSA Mycology Reference Laboratory, National Infection Services, UKHSA South West Laboratory, Science Quarter, Southmead Hospital, Bristol, UK; ³MRC Centre for Medical Mycology, University of Exeter, Geoffrey Pope Building, Stocker Road, Exeter, UK; ⁴Department of Microbiology and Infection Control, Barts Health NHS Trust, London, UK; ⁵Oxford University Hospitals NHS Foundation Trust, Oxford, UK; ⁶Radcliffe Department of Medicine, University of Oxford, Oxford, UK; ⁷Public Health Laboratory London, Science Group, UK Health Security Agency, London, UK; ⁸Research and Evaluation, UK Health Security Agency, Porton Down, Salisbury, UK; ⁹Department of Microbiology, King's College Hospital NHS Foundation Trust, London, UK; ¹⁰Department of Medical Microbiology, NHS Lothian, Edinburgh, UK; ¹¹National Institute for Health Research Health Protection Research Unit (NIHR HPRU) in Healthcare Associated Infections and Antimicrobial Resistance, Imperial College London, London, UK.

***Correspondence:** Colin S. Brown, colin.brown@ukhsa.gov.uk; candidaauris@ukhsa.gov.uk

Abbreviations: AFLP, amplified fragment length polymorphism; CDC, Centers for Disease Control and Prevention (USA); CINAHL, Cumulative Index to Nursing & Allied Health; CNS, central nervous system; COTHI, South African Centre for Opportunistic, Tropical and Hospital Infections; COVID-19, Coronavirus SARS-CoV-2 virus; CSC, compressed sodium chloride; DNA, deoxyribonucleic acid; ECDC, European Centre for Disease Control; ECG, electrocardiogram; EPA, Environmental Protection Agency; GAFFI, Global Action For Fungal Infections; HIV, Human Immunodeficiency Virus; ICU, intensive care unit; IPA, isopropyl alcohol; IPC, infection prevention and control; ITS, internal transcribed spacers; LAMP, loop-mediated isothermal amplification; MALDI-TOF, matrix-assisted laser desorption/ionisation time-of-flight; MDR, multi-drug resistant; MIC, minimum inhibitory concentration; NICE, National Institute for Health and Care Excellence; PAHO, Pan American Health Organisation; PCR, polymerase chain reaction; PHAC, Public Health Agency of Canada; PPE, personal protective equipment; SNPs, single nucleotide polymorphisms; UK, United Kingdom; UKHSA, United Kingdom Health Security Agency; USA, United States of America; WGS, whole genome sequencing; WHO, World Health Organisation.

†These authors contributed equally to this work

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INTRODUCTION

Candida auris is an emerging fungal pathogen, first described in 2009 from the external auditory canal of a patient in Japan [1], but identified in retrospect as far back as 1996 [2]. It has now been detected worldwide, across 6 continents, and in over 60 countries [3]. The inaugural World Health Organization (WHO) fungal priority pathogen list considers *C. auris* a critical priority pathogen, highlighting its threat to global public health [4].

C. auris infections have been frequently reported from the bloodstream and seen in association with bone, central nervous system (CNS), and intra-abdominal infections [5]. It has additionally been isolated from wounds, ear and respiratory specimens, urine, bile, and jejunal biopsies [6]. Detection in surveillance swabs from the axilla and/or groin may indicate carriage rather than infection, with carriage posing a risk of transmission to others and of subsequent invasive infection. Worryingly, *C. auris* is highly transmissible between patients in healthcare settings and is associated with prolonged persistence in the environment [7, 8]. Of further concern, accurate identification requires specialised laboratory methods, which are not available in many countries, thus challenging outbreak detection and control efforts.

There have been six genetically distinct clades of *C. auris* discovered to date. This includes the South Asian clade, which was first detected in India and Pakistan (clade I), the East Asian clade first detected in Japan (clade II), the South African clade first detected in South Africa (clade III), the South American clade first detected in Venezuela (clade IV), and two further clades; one recently detected in Iran (clade V) [9] and a novel clade VI in Singapore [10]. However, depending on population movement, these clades can be detected elsewhere. Antifungal resistance to fluconazole is common, and patterns of resistance to other antifungal agents vary by clade and region. Invasive infections from multidrug-resistant (MDR) *C. auris* have been associated with high mortality, and concerns have been raised regarding transmission, persistent colonisation, and the need for effective disinfection. Moreover, active community spread of independent clusters of MDR *C. auris* has now been described in the USA [11, 12].

This critical-priority fungal pathogen is increasingly detected globally, including across Europe [13], and is considered endemic in some countries such as the USA [14], South Africa [15], and India [16]. Given the limited mycology diagnostic capacity in many countries, it is likely that *C. auris* may be more widespread than previously thought [17]. This review provides an update on epidemiology, the impact of the COVID-19 pandemic, risk factors, identification and typing, resistance profiles, treatment, colonisation, and infection prevention and control measures. This review informed the recent update to the United Kingdom Health Security Agency (UKHSA) guidance on the laboratory investigation, management, and infection prevention and control of *Candida auris*.

METHODS

To identify updates since a previous review of the *C. auris* literature [18], a search of articles was conducted, between 2019 and 2023, with snowballing for any other significant studies. This included Medline, Embase, Scopus, NICE Evidence Search, Global Health, and CINAHL, and was limited to publications in the English language. The search terms *Candida auris* and *C. auris* were used. Articles were deduplicated and excluded if there was no reference to *C. auris* or if they did not contain information relevant to the key areas of the review, as described above.

Emergence and spread of *C. auris*

C. auris was first detected in 2009 in Japan; however, the first retrospectively identified isolate dates back to 1996 [1, 19]. In the Republic of Korea, 15 patients with samples taken between 2006 and 2011 were found to have *C. auris*, all with a background of chronic otitis media and systemic antifungal use [20]. Following additional epidemiological investigation, it was proposed that intra- and interhospital clonal transmission had occurred [2]. In Europe the first imported case dates to 2007, a South Indian clade (clade I) isolate [21].

Despite these discoveries, retrospective analyses of international samples support that *C. auris* was a novel pathogen prior to 2009. From 15271 retrospectively analysed *Candida* isolates taken between 2004 and 2015, only 4 were found to be *C. auris*, all of which had been collected since 2009 [22]. A further retrospective analysis of unusual *Candida* species performed at the Mycology Reference Laboratory, UK Health Security Agency (UKHSA), failed to find any evidence of *C. auris* in the UK prior to 2013 [23].

The first reports of invasive fungaemia associated with *C. auris* were from a multicentre surveillance study of candidaemia conducted in the Republic of Korea in 2009, with one case dating back to 1996 from an incidental finding of unidentified yeasts in bloodstream isolates [19]. All cases had been hospitalised for at least 12 days before isolation of the organism. Invasive infections were also discovered in India dating back to 2009, initially in 12 inpatients from 2 hospitals in New Delhi, with clonal isolates suggesting interhospital transmission [24].

As of December 2023, *C. auris* has been identified in 61 countries across 6 continents (Fig. 1, Supplementary Material 1). In the USA, *C. auris* became notifiable in 2018, and reports of case detection subsequently increased more than twofold. Between January and December 2022, 2377 clinical cases had been identified across the USA and targeted screening had identified a further 5754

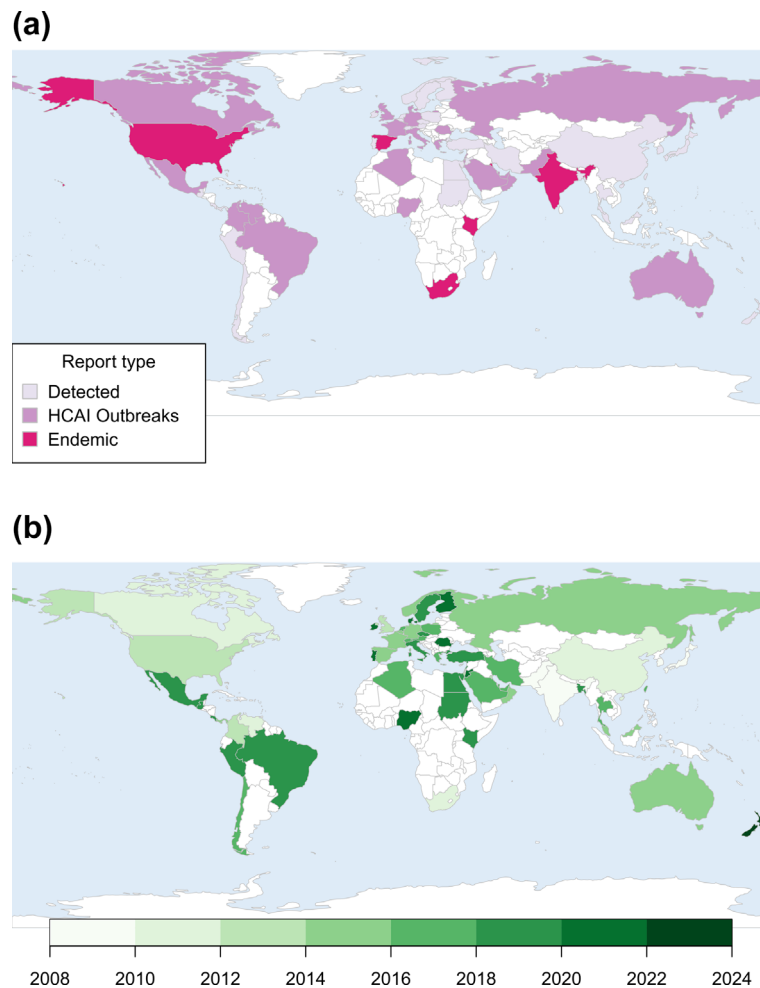


Fig. 1. Global epidemiology of *Candida auris*. (a) Countries where *C. auris* has been reported are presented and categorised by reported association with HCAI outbreaks or evidence of endemicity within a country. (b) Countries where *C. auris* has been reported are presented according to the year that the first case was detected. Note: there are many countries where *C. auris* has not yet been reported (white shading); however, this does not mean that it is not present within these countries. Cases of *C. auris* have been detected and reported in Reunion; however, this is not visible on the maps. Maps were prepared in R (v4.3.1) using the package rworldmap. See Supplementary Material 1 for a full list of countries and references used to produce these maps.

screening cases [25]. In response to a substantial rise in detection in European countries, a rapid risk assessment of *C. auris* in healthcare settings was updated by the European Centre for Disease Prevention and Control (ECDC) in 2018 [26], with a further risk assessment in 2022 following an outbreak in northern Italy. Additionally, a survey by the ECDC in 2020 reported ongoing variability in laboratory capacity and overall preparedness for *C. auris* detection [27].

C. auris represents a significant burden of disease in certain countries and has become endemic in several [15, 16]. In South Africa, the organism has been detected in almost 100 hospitals, causing large outbreaks, and is reportedly responsible for approximately 1 in 10 cases of candidaemia [15]. In India, *C. auris* has been implicated in 5% of candidaemia cases across 27 intensive care units [28]. Healthcare outbreaks have been reported from hospitals in several countries, with even more reporting sporadic cases (Fig. 1, Supplementary Material 1). These reports likely represent under-ascertainment of *C. auris* globally, given the challenges in laboratory identification and lack of laboratory infrastructure in some countries.

Community prevalence of *C. auris* remains unknown, and screening on admission to hospital is not routine practice. Following three large outbreaks in acute healthcare settings, a surveillance study in England was performed in 2017–18, involving screening of 998 admissions to 8 intensive care units across 3 major cities; all screens were found to be negative [29]. Another study in the USA found that carriage was only detected in people previously exposed to the hospital environment [30]. In areas where *C. auris* is endemic, however, community cases may be more prevalent. A recent study looking at *C. auris* amongst patients with

chronic respiratory disease in India found that 9.3% (3/32) of patients who were colonised with *C. auris* were colonised at the time of admission [8].

Genomic analysis and epidemiology

Whole-genome sequencing (WGS) analyses indicate that *C. auris* emerged simultaneously on several different continents, with four distinct clades across three continents; South Asian (clade I), East Asian (clade II), African (clade III), and South American (clade IV) [31]. A further genetically distinct clade has more recently been isolated in Iran from a 14-year-old girl who had never travelled abroad and was diagnosed with otomycosis, followed by a further fluconazole-resistant case distinct from the first [32, 33]. A sixth clade has just been reported in patients from Singapore and Bangladesh [10].

Each of the clades are separated by thousands of single-nucleotide polymorphisms (SNPs), but strains are highly clonal within each clade, with on average fewer than 70 SNPs separating any 2 isolates [34]. This, along with various geographical resistance mechanisms, supports the hypothesis of independent clonal expansion and evolution. Potential drivers for the emergence of the pathogen include increasing antifungal selection pressures in humans, animals, and the environment [31].

A retrospective analysis of 912 worldwide cases across 44 countries between 2009 and 2020 found the South Asian strain (clade I) to be the most prevalent, having been identified in 17 countries [35]. This was followed by the South African strain (clade III) found in eight countries [35]. Only five countries reported presence of the East Asian strain (clade II) and three of the South American strain (clade IV).

Experience in the UK

A retrospective analysis of historical isolates showed that the first identified *C. auris* isolates in the UK were in 2013, from blood cultures in unrelated patients [23]. In 2014, a single isolate from pleural fluid was recorded, and in 2015 there were 15 isolates identified, 9 of which were from sterile sites. Between 2013 and 2022, a total of 363 *C. auris* isolates were reported through laboratory surveillance in England, with 44 (12.1%) isolated from blood culture specimens [36]. During the COVID-19 pandemic, the number of isolates reported reduced substantially, probably reflecting the lack of new introductions from endemic areas due to extreme travel restrictions. However, cases have now begun to increase, and new outbreaks have been detected in acute healthcare settings. All clades except the South American clade and recently discovered clades V and VI have been identified to date in the UK. Sporadic introductions continue, many involving patients repatriated directly from international hospitals in endemic regions, including India, Qatar, Kuwait, Oman, Pakistan, and Kenya [37].

Previously, outbreaks have been identified in three separate public sector hospitals in England, each of which was prolonged and challenging to control [38–41]. The first was in a cardiothoracic centre between 2015 and 2016, involving 50 patients over a 16 month period. Most cases were confined to colonisation of skin sites or mucosa, and 44% ($n=22/50$) required antifungal therapy [39]. The second outbreak started in 2016 and involved 34 patients including 8 bloodstream infections. It was controlled within 13 months by an intensive screening and infection control approach maintaining a low mortality rate [40]. The third outbreak from 2015 to 2017 involved an intensive care setting, with 70 patients having been colonised or infected, linked to reusable axillary temperature probes, indicating environmental persistence and healthcare-associated transmission [38]. Despite a bundle of infection control interventions, the incidence of new cases was reduced only after removal of the temperature probes. All outbreak sequences formed a single genetic cluster within the *C. auris* South African clade (III), most likely due to a single undetected introduction in 2013.

C. auris and COVID-19

Early in the pandemic, patients with severe COVID-19 infection, including those in intensive care, were particularly vulnerable to bacterial and fungal co-infection [42–44]. With studies having independently identified that underlying respiratory illness and mechanical ventilation in intensive care are significant predictors of *C. auris* colonisation and infection, it was highlighted that the SARS-CoV-2 pandemic could provide conditions for widespread novel outbreaks of *C. auris* [23, 45].

Although the spread of resistant pathogens, including *C. auris*, has been observed amongst critically ill COVID-19 patients during peaks of the pandemic in certain countries, including Italy and the USA, this has not been the experience of all areas [46, 47]. In the UK, despite large numbers of patients in intensive care settings, *C. auris* outbreaks were not identified during the pandemic.

The Americas

The Pan American Health Organization (PAHO) released an epidemiological alert in 2021 in response to *C. auris* outbreaks during the COVID-19 pandemic [48]. In 2020, seven countries had documented cases of *C. auris*, mostly in patients with COVID-19, including Brazil, Guatemala, Mexico, Peru, the USA, Panama, and Colombia; *C. auris* had not been identified in four of these countries prior to the pandemic. Several outbreaks of *C. auris* have been identified in the USA in COVID-19 units of acute care hospitals, and new cases without links to known cases or healthcare abroad have also been identified across multiple states [47, 49]. In Brazil, two colonised patients were identified in a COVID-19 intensive care unit in December 2020 [50]. Screening

was undertaken and an outbreak was subsequently identified with 8/47 (17%) patients testing positive from the axilla, as well as positive findings from environmental screening. Digital thermometers demonstrated the highest rate of positive cultures from environmental screening, followed by bed rails, vital sign monitors/intravenous infusion pumps, and tray tables [50]. With travel restrictions during the pandemic and the absence of travel history among the colonised patients, it was hypothesised that the species was introduced several months before recognition of the first case [51].

In Guatemala, two isolates were detected on the same surgical unit, the first isolated in soft tissue and bone biopsies in a patient with osteomyelitis and the second from a surgical site infection, with neither case being COVID-19 related [48]. A hospital outbreak in Mexico also started with the identification of infection in a patient without COVID-19; however, it later spread to 12 patients within a COVID-19 intensive care unit, with 3 of 4 intensive care areas affected within 3 months [52]. Mortality was high (5/6; 83.3%) amongst patients with candidaemia.

In Peru, two patients with respiratory illness, one with latent TB and one with COVID-19 infection, were identified as having *C. auris* [48]. Both had a central venous catheter *in situ*, indwelling urinary catheter, and were on mechanical ventilation. In Panama, a significant association with COVID-19 was identified, with 124 isolates of *C. auris* having been identified prior to the PAHO epidemiological alert, of which 108 were in patients with COVID-19 infection [48]. In Colombia, 340 cases of *C. auris* were identified in 2020, several of which were in patients hospitalised with SARS-CoV-2 [48].

Europe

In Spain, *C. auris* became the most isolated *Candida* species from blood cultures after the start of the pandemic, associated with a large outbreak in a tertiary hospital that commenced in 2017 but worsened during the pandemic [53]. This was linked with overoccupancy in the intensive care unit, higher workload of healthcare workers, and poor compliance with infection prevention and control (IPC) measures. In Italy, the index case of *C. auris* was identified in 2019, and a nosocomial outbreak subsequently declared in patients hospitalised in intensive care with COVID-19 infection [46]. A single genetic lineage was identified, suggesting a point source. There was also a high rate of MDR organisms identified in patients with COVID-19 admitted to intensive care, in addition to *C. auris*. Again, concerns regarding IPC practices were raised and several patients required a high level of care, as well as longer hospital stays, and frequent use of broad-spectrum antibiotics [46]. In response to 277 cases associated with this outbreak, spread across at least 8 healthcare facilities in the Italian region of Liguria, and 11 cases in facilities in the neighbouring region of Emilia-Romagna, ECDC conducted a rapid risk assessment for the European Union [54].

Asia

In Lebanon, the first cases and subsequent outbreak of *C. auris* occurred in October 2020, with a total of 14 cases identified in critical care units over 13 weeks [55]. Two patients were identified with *C. auris* during their stay in COVID-19 intensive care after being admitted through the emergency department, with environmental contamination of the department remaining a possible source.

In India, an outbreak of *C. auris* was identified in an intensive care unit with COVID-19 patients [43].

This included 10 patients with *C. auris* bloodstream infection and a case fatality rate of 60% (6/10). Of note, this was an elderly and comorbid cohort with severe COVID-19 infection and the mortality directly attributable to *C. auris* is difficult to determine. Affected patients had been hospitalised in intensive care for prolonged periods of time (range 20–60 days), and all had indwelling invasive devices such as central venous and urinary catheters, and other risk factors such as the need for mechanical ventilation or steroid treatment and underlying chronic diseases, including diabetes mellitus and hypertension. However, four of the patients who died experienced persistent candidaemia despite micafungin therapy, with recurrence of *C. auris* detected in serial blood cultures.

Middle East

COVID-19 and *C. auris* co-infection was identified in the United Arab Emirates, where it was hypothesised that the increment in fungal infections was the result of acquisition in the hospital setting, and higher susceptibility of the patients, given treatment with broad-spectrum antibiotics and immunosuppressive therapies [56].

An epidemiological analysis of *C. auris* cases during the pandemic concluded that changes in practice were likely to have been relevant to the spread, including changes in prescribing practices, perceptions of appropriate use of personal protective equipment (PPE), including extended and excessive use, and an increase in agency staffing with varied levels of training and experience in PPE use and care of COVID-19 patients [57].

It has been concluded in several regions that the pandemic created an opportunistic environment for spread of *C. auris*, posing pressures such as overwhelmed intensive care units, suboptimal infection control practices, PPE use, temporary staffing, and inadequate training [55, 57, 58]. A previous review of the literature identified 36 cases of co-infection with *C. auris* and COVID-19, highlighting that most cases were males, with an age range of 25–86 years, and candidaemia was the predominant presentation [59]. Date of onset of infection post-admission ranged from 4 to 45 days, and most patients had significant underlying

comorbidities. Almost all had an indwelling urinary catheter, intravenous catheters, and were on broad-spectrum antibiotics, and most had received steroids. The mortality rate was 53% for 30 cases with documented outcomes, although the number of deaths directly attributable to *C. auris* is uncertain [59]. The high case fatality rate reported in several studies around the world is in contrast to the UK experience, where no fatalities directly attributable to *C. auris* have been reported to date. The reasons underlying this difference are poorly described but likely multifactorial, including factors related to the host, the pathogen, and the wider healthcare system.

Risk factors

Acquisition of *C. auris* is commonly associated with high-risk healthcare settings and particularly high-dependency contexts such as intensive care units, with findings of prospective screening on admission suggesting a high propensity for nosocomial acquisition [39, 60]. Patients may be colonised rapidly during their inpatient stay, some within 4 days [60]. A retrospective analysis of 912 cases worldwide found that a higher proportion of men (61.4%) were affected [13]. A population-based study in Spain also showed that incidence was higher in autumn and amongst the age group of 65–84 years [61].

Identified risk factors predisposing to *C. auris* infection are similar to those for other *Candida* species. These factors include severe underlying disease with immunosuppression, such as HIV and bone marrow transplantation, corticosteroid therapy, neutropenia, malignancy, those with chronic kidney disease or diabetes mellitus, a prolonged stay in ICU, mechanical ventilation, presence of a central-venous catheter or urinary catheter, prolonged exposure to broad-spectrum antibiotic or antifungal use, underlying respiratory illness, vascular surgery, or surgery within the previous 30 days [23, 45, 46, 62, 63].

Review of clinical characteristics associated with *C. auris* found that underlying diseases were high, but that kidney disease was the only significant risk factor for mortality in *C. auris*-infected patients [35]. Overall mortality with *C. auris* infection is reported as high in the literature, up to 40–60% worldwide, possibly due in part to severe underlying conditions in the at-risk populations, the MDR nature of the pathogen, and limited availability of certain antifungal drugs in some countries [22, 64, 65]. However, as mentioned above, the extent to which mortality is directly attributable to *C. auris* infection can be difficult to determine, especially in populations with a higher relative mortality in general, such as those in critical care settings.

Mechanical ventilation is an important risk factor. The rate of *C. auris* colonisation in residents of skilled nursing facilities caring for ventilated patients was found to be up to 10 times higher than the occurrence in nursing facilities without ventilator support [66]. Additional risk factors for colonisation in this cohort of residents in a ventilator-capable nursing facility include one or more acute care hospital visits in the prior 6 months (adjusted OR 4.2; 95% CI, 1.9–9.6), antibiotic treatment with carbapenems in the prior 90 days (aOR 3.5; 95% CI, 1.6–7.6), and systemic fluconazole in the prior 90 days (aOR 6.0; 95% CI, 1.6–22.6) [66].

C. auris can affect both adult and paediatric populations. Nosocomial infection in children has been reported largely under the age of 1 year and in those with certain medical conditions, including prematurity and malignancy, and in children with risk factors such as intravascular catheters and those receiving parenteral nutrition [67]. Transmission is also possible from a colonised mother to baby during delivery [3]. Clusters of infection have been identified in neonates, with risk of mortality associated with invasive disease [68–70].

Identification and typing

Identification of *C. auris* has been challenging using conventional laboratory diagnostic techniques [71]. Genetic analyses have established that *C. auris* is closely related to *C. haemulonii* and *Clavispora* (previously *Candida*) *lusitaniae* [34]. Other common misidentifications are described in Table 1. Isolates should therefore be identified to the species level. Options for species identification are available and include matrix-assisted laser desorption/ionisation time-of-flight (MALDI-TOF) mass spectrometry [72–74]. MALDI-TOF can reliably differentiate *C. auris* from other *Candida* species [75, 76]. Accuracy of this system is reliant on the spectra for the sample organism being present in the reference database and care being given to an appropriate extraction method.

A novel chromogenic agar, CHROMagar Candida Plus, appears promising in the identification and differentiation of *C. auris* from other *Candida* species, with *C. auris* colonies appearing as a light blue colour with a blue halo (Fig. 2), and obtaining a sensitivity and specificity of 100% at 36 h incubation [3, 74, 77, 78]. This is a promising alternative to conventional mycological media for the screening of patients who may be colonised or infected with *C. auris*. It has been reported that some strains of *C. parapsilosis* have consistent colonial morphology with *C. auris* on CHROMagar Candida Plus [79]. Therefore, it is recommended that full identification of suspect isolates is performed to confirm an identification of *C. auris* from CHROMagar Candida Plus.

The detection of *C. auris* has been enhanced by the development of molecular tests [80]. Turnaround times and diagnostic sensitivity are improved using molecular technologies, particularly for specimens with a high pre-test probability of *C. auris* colonisation or infection [3]. Molecular identification can be performed by sequencing various DNA loci in specific domains of ribosomal genes (18S rDNA, 28S rDNA or internal transcribed spacers ITS1, ITS2), by conventional or real-time polymerase chain reaction (PCR) and loop-mediated isothermal amplification (LAMP) [24, 34].

Table 1. Examples of misidentification of *C. auris* and the corresponding identification method used (table adapted from Elbaradei *et al.* [167] and Kaur *et al.* [63]). Previous yeast names are in parentheses (see [168])

Species misidentification	Method	Reference
<i>Candida albicans</i>	MicroScan	[97]
	Vitek-MS	[169]
<i>Diutina (Candida) catenulata</i>	BD Phoenix	[76]
	MicroScan	[76]
<i>Debaryomyces hansenii</i> (previously <i>Candida famata</i>)	Vitek-2 (software upgrade to version 8.01 includes <i>C. auris</i> . It is, however, recommended to confirm isolates identified as <i>C. haemulonii</i> and <i>C. duobushaemulonii</i> , <i>C. famata</i> , and <i>C. auris</i> by MALDI-TOF or DNA sequencing)	[24, 136]
	MicroScan	[76]
<i>Meyerozyma (Candida) guilliermondii</i>	MicroScan	[76]
<i>Candida haemulonii</i>	BD Phoenix	[76]
	Vitek-2 (software upgrade to version 8.01 includes <i>C. auris</i> . It is, however, recommended to confirm isolates identified as <i>C. haemulonii</i> and <i>C. duobushaemulonii</i> , <i>C. famata</i> , and <i>C. auris</i> by MALDI-TOF or DNA sequencing)	[19, 24, 75, 76, 136, 170]
	Vitek-MS	[171]
<i>Clavispora (Candida) lusitanae</i>	MicroScan	[76]
	Vitek-MS	[171]
<i>Candida parapsilosis</i>	MicroScan	[76]
	RapID Yeast Plus	[172]
<i>Candida sake</i>	Api 20C AUX	[24, 171]
<i>Candida tropicalis</i>	MicroScan	[97]
<i>Rhodotorula glutinis</i>	Api 20C AUX	[19, 76]
<i>Candida duobushaemulonii</i>	Vitek-2 (software upgrade to version 8.01 includes <i>C. auris</i> . It is, however, recommended to confirm isolates identified as <i>C. haemulonii</i> and <i>C. duobushaemulonii</i> , <i>D. hansenii</i> , and <i>C. auris</i> by MALDI-TOF or DNA sequencing)	[136]
<i>Lachancea (Saccharomyces) kluyveri</i>	API ID32C	[173]

The identification of *C. auris* using traditional phenotypic and biochemical methods is challenging, and these have largely been superseded by proteomics and molecular methods [3]. Commercial biochemical identification systems commonly used in clinical microbiology laboratories may be unreliable for *C. auris* identification if their system has not been updated to include *C. auris*, due to phenotypic similarities with other species [76, 81]. Checking the system for *C. auris* detection ability is therefore required.

Molecular typing of *C. auris* can be performed using a variety of methods, including sequencing of rDNA loci (D1/D2 or ITS regions) to differentiate between the major phylogeographical clades [82]. Further delineation for outbreak analysis requires higher resolution methods, which can be performed by WGS analysis and typing by amplified fragment length polymorphism (AFLP) [3, 34]. International networks are being established to improve genomic capacity and develop analysis pipelines that will support ability to detect outbreaks, identify introductions, and characterise transmission of fungal infections [83, 84].

In low-resource settings, the accurate identification of *C. auris* using the methods described above may not be readily achievable. A cross-sectional study exploring the current state of clinical mycology in Africa surveyed 40 institutions across 21 countries and found that few laboratories reported having the capacity to correctly identify *C. auris* [17]. One alternative approach that takes advantage of the salt and thermal tolerance of *C. auris* involves a selective modified Sabouraud agar; when supplemented with 10% NaCl and dulcitol instead of dextrose as a carbon source and cultured for 72 h at 42°C, the tentative identification of

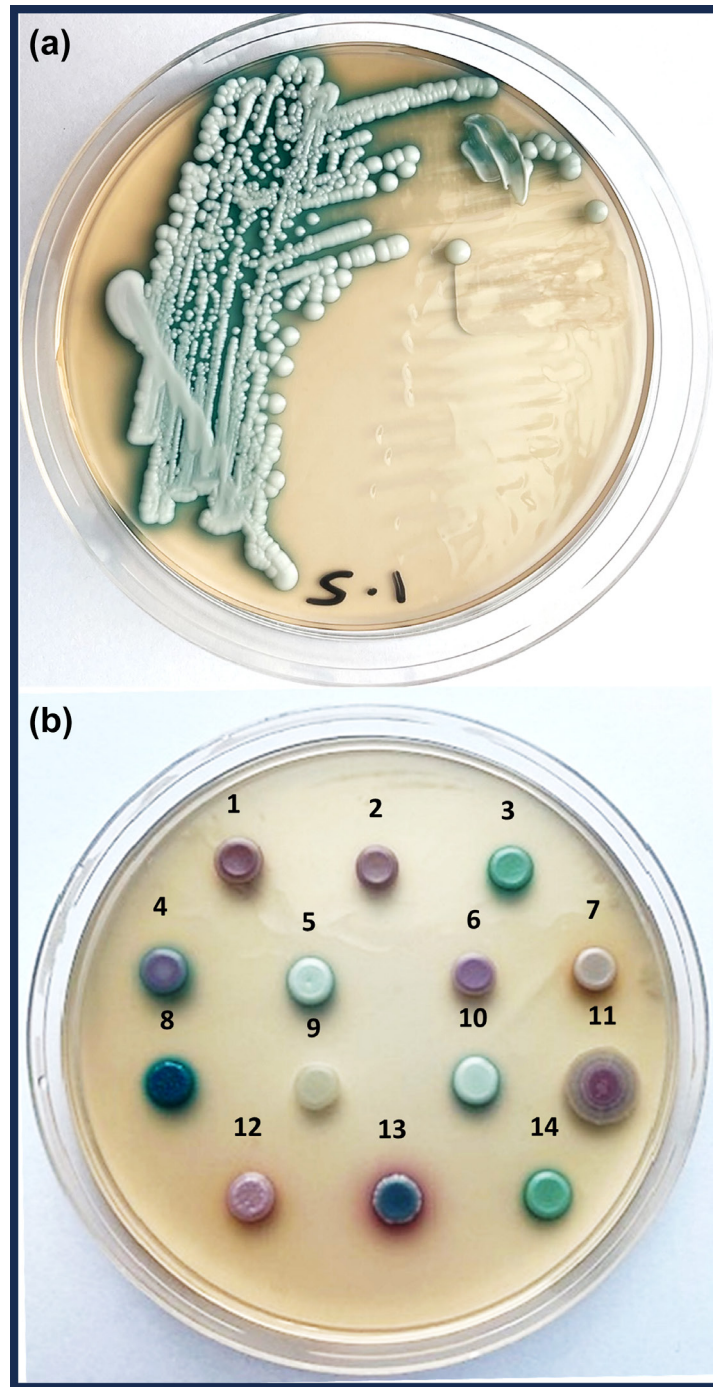


Fig. 2. (a) *Candida auris* (clade II) isolate streaked onto a plate of Chromagar Candida Plus with distinctive pale cream colony and diffusing blue halo. (b) Comparative colonial appearances of common species of *Candida* and allied yeast genera spotted onto Chromagar Candida Plus. Clinical isolates tested were: 1, *Nakaseomyces glabratus* (ex-*Candida glabrata*); 2, *Saccharomyces cerevisiae*; 3, *Candida albicans*; 4, *Candida parapsilosis*; 5, *Candida auris* (clade II); 6, *Clavispora lusitanae* (ex-*Candida lusitanae*); 7, *Meyerozyma guilliermondii* (ex-*Candida guilliermondii*); 8, *Trichosporon asahii*; 9, *Pichia cactophila* (ex-*Candida inconspicua*); 10, *Candida auris* (clade III); 11, *Pichia kudriavzevii* (ex-*Candida krusei*); 12, *Meyerozyma caribbica* (ex-*Candida fermentati*); 13, *Candida tropicalis*; 14, *Candida dubliniensis*. Plates incubated for 36 h at 35 °C. Image provided by Andrew M. Borman.

C. auris is possible [85, 86]. However, even with a tentative diagnosis of *C. auris*, access to effective therapy is a further challenge. Although sustained advocacy by organisations such as Global Action For Fungal Infections (GAFFI) has raised the profile of fungal infections and supported the addition of echinocandins to the WHO essential medicines list [87], in 2021 the survey by Driemeyer *et al.* [17] found that only 5–22.5% of respondents from African institutions reported having access to an echinocandin.

Since their inclusion in the WHO essential medicines list availability is increasing [88, 89], but there are still substantial gaps. An emphasis on the sustainable development of laboratory capacity and workforce in many low-resource settings around the world is essential; not just for fungal infections such as *C. auris* but across the spectrum of pathogens to support antimicrobial stewardship initiatives; enhance the surveillance of antimicrobial resistant infections at local, regional, and national levels; and most importantly improve patient outcomes.

Antifungal resistance

C. auris shows high minimum inhibitory concentrations (MICs) for various antifungal agents. Fluconazole resistance is widespread but not universal; however, this is generally not a therapeutic option for most clinical cases [90]. Additionally, many *C. auris* isolates also show high MICs for newer azoles such as voriconazole, although this varies in a clade-specific manner [91], likely indicating the ineffectiveness of these agents for clinical cases [90]. Clinical cut-off values have, however, not yet been described.

In a collection of *C. auris* isolates ($n=54$) from five continents, 41% were found to be resistant to two or more antifungal classes [31]. Resistance to echinocandins remains uncommon; however, with widespread use of the drug as first-line therapy, it has been increasingly identified [92]. Pan-resistant strains have also been described [12] and, worryingly, evidence of their transmission between healthcare facilities is emerging [93].

Antifungal drug resistance is thought to be an acquired as well as a shared trait, with the potential to develop over time when fungi are exposed to antifungals [94, 95]. The emergence of echinocandin resistance, detected by WGS and phenotypically, within an ongoing prolonged outbreak is concerning [96]. *C. auris* has an intrinsic ability to exhibit or develop resistance very rapidly even while the patient is still undergoing treatment, which is why it is essential to use the antifungals at the right time and at the correct dosage [34]. Antifungal susceptibility testing is therefore recommended for all *C. auris* isolates.

Clinical characteristics and complications

The clinical spectrum associated with *C. auris* ranges from asymptomatic colonisation to invasive candidiasis, most commonly as healthcare-associated candidaemia [71]. Other healthcare-associated infections reported include intravascular catheter infection, urinary tract and respiratory infection, meningitis, osteomyelitis, surgical site infection, and otomastoiditis [39, 92, 97–99]. *C. auris* colonises the axillae, groin, nares, respiratory, and urinary tract of hospitalised patients [6, 100]. Intestinal colonisation occurs less frequently but has been associated with *C. auris* urinary infections [101]. Colonisation reportedly results in invasive infections in up to 5–10% of individuals, with mechanical ventilation and placement of invasive devices identified as two major risk factors [6, 102]. The proportion developing invasive infection may vary according to underlying patient risk factors, IPC measures, diagnostic capabilities, and institutional familiarity with *C. auris* outbreaks.

The ability for rapid colonisation of skin and high transmissibility within the healthcare setting can result in prolonged and serious outbreaks [38, 39]. Transmission can occur via contact with contaminated items or from affected individuals. Screening of the patient environment has yielded *C. auris* isolates with identical fingerprinting patterns to patient isolates, suggesting shedding of *C. auris* by colonised patients [6]. Contaminated equipment identified during outbreak investigations has included thermometers, temperature probes, pulse oximeters, blood-pressure cuffs, a cloth lanyard, and a call bell [50, 51, 103, 104]. The duration of patient colonisation remains uncertain; however, a recent study demonstrated that most patients with *C. auris* colonisation did not have detectable *C. auris* 12 months after discharge to the community setting [21].

Invasive disease can be associated with high mortality in patients within an intensive care setting [63], although the proportion of deaths directly attributable to *C. auris* is difficult to determine. Indeed, this has not been the experience in all settings (e.g. the UK), as described above. Infection can be complicated by multidrug resistance, with bloodstream infections being more difficult to treat, and risks of complications, including spondylodiscitis, endocarditis, and ventriculitis, and clinical syndromes including otomycosis and otomastoiditis [2, 99, 105]. Involvement of skin, respiratory, urogenital, and abdominal sites has also been reported [106], including vulvovaginal candidiasis in the context of immunosuppression [107].

Different clades are associated with different clinical presentations and appear to display differences in virulence in animal models [108, 109]. A comparison study of *in vivo* pathogenicity of the four initial *C. auris* clades in a neutropenic bloodstream infection murine model found that the highest overall mortality was observed for the South American clade (96%), followed by the South Asian (80%), South African (45%), and East Asian (44%) clades [109]. The most virulent isolates appear to exhibit pathogenicity comparable to that of *Candida albicans*. In an immunocompetent mouse model, *C. auris* was demonstrated to be highly virulent, but less virulent than *C. albicans* [110].

In relation to patterns of infection, South Asian, and South American clades are commonly associated with bloodstream infections (47–76%), whereas the South African clade is associated with a higher percentage of urinary tract infections or colonisation (38%) [111]. Isolates from the East Asian clade are associated with otitis externa, which also show higher genetic diversity compared to other clades and may indicate an older natural population [111]. All except for the East Asian clade (and more recently discovered Iranian and sixth clades) have been linked to outbreaks causing invasive infections [63].

Bloodstream infections can be followed by complications such as infective endocarditis and spondylodiscitis. Recurrence has also been reported [105]. Case fatality associated with invasive infection is variably reported. In the USA, among patients with blood isolates of *C. auris*, 30 day mortality was reported as 39% and 90 day mortality as 58% [112]. In a UK outbreak, however, no mortality was directly attributed to *C. auris* infection [39].

Treatment considerations

Antifungal agents commonly used to treat *Candida* infections include fluconazole and the echinocandins. *C. auris* isolates are commonly resistant to fluconazole; however, resistance to the other antifungal agents is variable.

Therapeutic recommendations for *C. auris* include echinocandin monotherapy as empirical treatment prior to the results of susceptibility testing, considering common resistance patterns identified to date [34, 113, 114]. Although reports of echinocandin- and pan-resistant isolates are increasing, in regions where most strains continue to remain susceptible it is reasonable for echinocandins to remain as first-line treatment [114]. Patients should, however, be monitored for clinical improvement, with follow-up cultures and susceptibility testing, as the organism can develop resistance quickly, including during treatment [115]. If the urinary tract or CNS is involved, dual therapy may be necessary, as some antifungal classes (including echinocandins) do not have bioavailability in either urine or the CNS.

Evidence is lacking about the most appropriate therapy for pan-resistant strains, where resistance to all three major classes of antifungals has been identified (echinocandins, amphotericin B, and azoles) [116]. There is *in vivo* evidence of inhibition of pan-resistant strains through combinations of two antifungal drugs using fixed concentrations, with an effective response noted from flucytosine combinations with amphotericin B, azoles, or echinocandins [117]. There is additional *in vitro* evidence supporting combination therapy against *C. auris* with caspofungin in combination with posaconazole [118] or anidulafungin in combination with manogepix (the active moiety of fosmanogepix) or flucytosine [119]. A novel echinocandin, rezafungin, appears to be promising from *in vitro* studies, including in subsets of echinocandin-resistant *C. auris*, and this is currently undergoing phase 3 trials [120, 121]. Fosmanogepix, a first-in-class antifungal with a novel mechanism of action available in intravenous and oral formulations, has shown potential both *in vitro* and in phase 2 studies [122, 123]. Both persistent and recurrent *C. auris* bloodstream infections have been documented; animal studies and *in vitro* investigations suggest that micafungin-based combination therapies are promising in this context [113, 124, 125].

Infection prevention and control

C. auris is able to grow at higher temperatures than many other fungi and is able to tolerate high salt concentrations [6]. These are important characteristics in its ability to persist in the environment and to survive for long periods of time, creating opportunities for colonisation and transmission. There are additional challenges associated with rapid acquisition and prolonged colonisation, particularly in nosocomial settings, leading to an increased risk of contamination and transmission for weeks or months following exposure [67, 92].

Whole-genome sequencing analyses of isolates from patients and their environment have shown that *C. auris* can contaminate surfaces at varying distances from the patient's bed, including items not frequently touched and those distant from the patient [126, 127]. Outbreak investigations have isolated the organism from environmental samples, including those taken from a mattress, bedside table, bed rail, chair, window sill, and from the air [39]; from bed surfaces and equipment such as ventilators, a temperature probe, ECG leads [60]; pulse oximeters [38]; and the floor, patient bedside trolleys, air conditioners, a bed sheet, pillow, mobile phone, oxygen mask, and an intravenous pole [8]. Biomedical products and equipment should therefore be single use; reusable equipment should ideally be single-patient use and left in the patient's room, to ensure thorough terminal decontamination at the time of discharge [128].

Infection prevention and control measures are crucial to reducing transmission. Many of the major world health organisations have published guidance and recommendations regarding the isolation of patients, contact precautions, and cleaning of equipment and environments in contact with *C. auris*, including the UK, the USA, Europe, South Africa, Australia, and Canada (Table S1 [36, 48, 114, 129–135]).

Identification of *C. auris* and screening

C. auris can be transmissible whether an individual is infected or colonised, and thus infection control precautions are the same [136]. Screening is widely recommended where transmission has occurred, or where there are close contacts of confirmed *C. auris* cases [3]. Active screening of exposed and potentially exposed patients followed by strict infection control measures has been successful in outbreak management [137]. Evidence suggests that frequently encountered sites for colonisation and therefore for screening include the axilla and groin [60, 112]. Screening sites also shown to culture positive for *C. auris*, and that may be relevant, include the rectum, pharynx, urine, nose, mouth, ear, vascular catheter-exit site, and wounds [105, 112, 128]. A recent study has demonstrated that adding nasal swabs to composite axilla and groin swabs may yield additional positive isolates, with 25% of positive findings being on nasal culture alone [112].

The benefits of routine screening of healthcare workers remain unclear; however, screening in outbreak investigations has identified some positive findings [39, 126]. *C. auris* has been found on the skin, hands, and nares of care providers and healthcare workers, and screening may therefore be initiated if risk factors are identified [3, 138, 139].

The duration of colonisation with *C. auris* remains unknown, although it can be protracted whilst patients remain in healthcare settings [136]. Screening methodologies may not identify all sites of colonisation and deisolation of contacts therefore remains challenging in this setting. An outbreak investigation reported having deisolated close contacts after three consecutive negative screens, whereupon one contact subsequently tested positive again and so weekly screening until discharge was introduced [39]. In response to high rates of detection in New York, a pilot case management programme for people colonised with *C. auris* who were discharged to a community setting was implemented. Although long-term colonisation was observed in some, serial *C. auris* assessments found that approximately two-thirds of patients colonised as inpatients and discharged to a community setting did not remain colonised indefinitely [140]. The time taken for patients to become serially negative was 8.6 months (IQR 5.7–10.8) and for patients who became serially negative, the median time to the first negative was 4.7 months (IQR 3.5–7.5).

Transmission-based precautions

Given the evidence around transmission risk associated with *C. auris*, patients colonised or infected should be cared for in a single room with contact precautions, preferably with their own bathroom. A flagging system indicating the isolation should also be visible at the entry of the room and patients should have an alert on their medical records to ensure appropriate isolation on transfer and readmission [134]. Verbal and written communication of infection transmission risk is essential for inter-facility patient transfer, including to community healthcare settings. If single room occupancy is not possible, patients with *C. auris* should as a minimum be cohorted, ideally with single-patient-use commodes and single-use equipment. The importance of hand hygiene should be reinforced with healthcare workers. Hand washing with soap and water, alcohol-based, or chlorhexidine-based hand rub, have all been shown to be effective in eliminating *C. auris* from hands [60, 90]. Transmission-based precautions are recommended, including personal protective equipment (PPE) and single-use items, for the duration of the stay in a healthcare facility [136]. PPE including gloves and a long-sleeved gown is recommended for use in contacts with patients with *C. auris* or their environment [26, 128, 135].

Special precautions are advised when cleaning or exposed to body fluids in *C. auris*-affected areas. The CDC recommend transmission-based precautions and enhanced barrier precautions that are similar to those for other MDR organisms [135]. Ideally, patients should only be moved for necessary medical procedures and should be last on the list to allow for a thorough clean following, with minimum staff involved [136].

Effective contact precautions are especially important in the context of invasive lines in high-risk cohorts, due to the ability of fungal cells to adhere to the device, and their growth occurring in the form of a biofilm [63].

Disinfection of environmental surfaces

C. auris can resist certain disinfectants and is well adapted to nosocomial environments, having the potential to remain viable on vertical surfaces in the immediate environment of colonised patients for up to 28 days [60, 86]. Additionally, the organism has been cultured from bedding for up to 7 days [60]. *C. auris* can colonise and persist on surfaces for longer than *C. albicans*, and has shown a prolonged metabolic activity [141]. Furthermore, the minimum time taken to acquire *C. auris* from a patient or their immediate environment is 4 h or less, further reinforcing the importance of rigorous IPC measures [39].

Evidence supporting effective products and methods for disinfection of environmental surfaces contaminated by *C. auris* remains limited due to challenges with comparison of studies, with many applying different experimental techniques, the results of which also cannot readily be compared or directly translated to efficacy in real-world scenarios.

Chlorine-based disinfectants, in the form of sodium hypochlorite (NaOCl) and sodium dichloroisocyanurate, are commonly used in the healthcare setting for disinfection, especially for MDR organisms, and these have been the most investigated in relation to *C. auris* [142]. A chlorine-based disinfectant was noted to be effective during an outbreak in the UK in 2015 (1000 p.p.m. Chlor-Clean, Guest Medical), having been used in the daily routine disinfection of the patient area and equipment, with a 10000 p.p.m. chlorine-based product (Haz-Tab, Guest Medical) having been used for terminal cleaning followed by further disinfection with hydrogen peroxide vapour [39]. *In vitro* studies have also investigated the efficacy of chlorine-based products. A chlorine-based disinfectant (Haz-Tab 1000 p.p.m. chlorine) was tested against different clinical isolates of *C. auris* with an exposure time of 5 min and all isolates had at least a 4.5 log₁₀ reduction in growth [143]. A further study evaluated chlorine-based products at 1000 p.p.m. (Chlor-Clean) and 10000 p.p.m. (Haz-Tab) against clinical isolates of *C. auris* and other *Candida* species, finding that *C. auris* isolates were effectively killed at all concentrations with a minimum of 3 min contact time [144].

Similar studies also concluded that sodium hypochlorite (NaOCl) with concentrations of 1000 p.p.m. or higher were effective in eradicating *C. auris* [128, 135, 145]. In relation to effectiveness on surfaces, following application of 1 and 2% NaOCl to four different surfaces (stainless steel, ceramic, plastic, and glass) for a 10 min contact time, complete eradication of *C. auris* was reported on all surfaces [60]. A further study then investigated the efficacy of NaOCl at 1000 and 10000 p.p.m. on cellulose matrix,

stainless steel, and polyester surfaces inoculated with clinical isolates of *C. auris* [145]. At all concentrations, NaOCl demonstrated significant killing on all substrates at contact times of 5 and 10 min; however, among all the materials tested, complete eradication was only achieved on cellulose substrates. Several commercially available products containing NaOCl have also been found to be ineffective against dry biofilms containing *C. auris* [146].

The efficacy of peracetic acid has been tested on stainless steel, polymer, and cellulose surfaces. Peracetic acid at 2000 p.p.m. was found to have significant killing activity against *C. auris*; however, as with NaOCl, complete eradication was achieved on cellulose matrix but not with steel or plastic [145]. As *C. auris* can survive on plastic surfaces, for efficient removal it has been recommended that peracetic acid be added to NaOCl, peracetic acid (3500 p.p.m.) and sodium dichloroisocyanurate (1000 p.p.m.) having been effective against dry biofilms containing *C. auris* [146].

There are some data regarding the effectiveness of hydrogen peroxide in disinfectant and vaporised form, but with lower levels of supportive evidence [144, 147]. Application of hydrogen peroxide vapour appeared to be promising for environmental decontamination in an *in vitro* study, based on a hospital outbreak [144, 148]. This is currently recommended as a potential additional safety measure for manual cleaning and disinfection regimes, rather than replacing other regimes [149]. A study of 0.5 and 1.4% hydrogen peroxide solutions found that they were also effective [147].

Quaternary ammonium compounds are commonly used disinfectants in healthcare settings, but the overall evidence regarding their efficacy for *C. auris* is conflicting, and their use is therefore currently discouraged [150, 151].

Further studies highlighted that application in accordance with recommended concentrations and contact times was essential for efficacy with glutaraldehyde, phenols, hydrogen peroxide, and ethanol [60, 150]. Two per cent glutaraldehyde and 5% phenol were found to be effective on multiple surfaces when the recommended contact times of 20 and 60 min, respectively, were used [60]. Alcohol (29.4% Purell Healthcare Surface Disinfectant) did show some killing activity but was not as effective as chlorine-based disinfectants or hydrogen peroxide [147].

The evidence regarding the effectiveness of ultraviolet-C (UV-C) light is conflicting [150, 152]. Several UV surface disinfection devices have been tested for efficacy against *C. auris*. These UV systems vary significantly, and each requires individual verification. Reports that found UV to be effective typically quote log reductions between 2.48 and 5.5 for cycle times between 10 and 20 min [153]. This will vary depending on the UV emitter being tested, the distance between the emitter and the contaminated surface, the presence or absence of additional soil, exposure time, the angle of incident radiation, and the degree of shadowing. Other studies have concluded that *C. auris* is significantly less susceptible to killing by UV-C in comparison to other *Candida* species [154], with one study stating the *C. auris* is not effectively killed by standard UV-C disinfection [155] and other studies highlighting strain variability [148, 156]. Data are limited and the parameters for effective disinfection with UV-C are not yet well understood.

The CDC currently recommends use of Environmental Protection Agency (EPA)-registered hospital-grade disinfectants that are effective against *Clostridium difficile* spores; primarily chlorine-based products [135]. This has been supported by outbreak management success with stopping transmission [157] and by further investigation of chlorine-based disinfection, concluding effectiveness with appropriate application [143]. If EPA-registered hospital-grade disinfectants are unavailable, suggested alternatives include hydrogen peroxide 0.5–1.4%, or quaternary ammonium compounds supplemented with isopropyl alcohol and/or ethyl alcohol. The ECDC recommends terminal cleaning using disinfectants and methods with certified antifungal activity, including chlorine-based disinfectants (at a concentration of 1000 p.p.m.), hydrogen peroxide or others. The Public Health Agency of Canada (PHAC) and South African Centre for Opportunistic, Tropical and Hospital Infections (COTHI) interim recommendations include 'regular' and terminal cleanings with a chlorine-releasing agent at 1000 p.p.m., and COTHI suggests the addition of hydrogen peroxide vapour, when feasible. The Pan American Health Organization/World Health Organization (PAHO/WHO) recommends cleaning with soap and water followed by disinfection with 0.1% bleach. PAHO/WHO recommend 'high activity' compounds, such as sodium hypochlorite, hydrogen peroxide (and vaporised), and peracetic acid. The Australian Society of Infectious diseases recommends the use of products that claim to have sporicidal activity for disinfection (e.g., ≥1000 p.p.m. bleach, peracetic acid or accelerated hydrogen peroxide).

Terminal cleaning and disinfection of the environment remain an essential element of the IPC precautions required to prevent transmission of *C. auris* from infected or colonised patients. Whilst antimicrobial surfaces and coating will never replace environmental decontamination, this is an area of active research. One example of this is a fast-acting, permanent antimicrobial surface made of compressed sodium chloride (CSC). Pilot data indicate at least 99% reduction of *C. auris* in 1 min. There are *in vitro* data suggesting that *C. auris* strains can be killed on contact when exposed to caspofungin that is reformulated as a covalently bound surface layer on glass and plastic [158]; however, the risk of selecting resistant strains is uncertain and an important concern.

Patient decolonisation

Several studies have investigated products for decolonisation in relation to *C. auris*, with effectiveness having been demonstrated for chlorhexidine gluconate, povidone-iodine, and alcohol [39, 60, 143]. The benefits of decolonisation and subsequent

recolonisation risk remain unclear. There are therefore no protocols for decolonisation of patients with *C. auris*; an international expert working group were unable to make recommendations due to a lack of evidence [128].

Chlorhexidine gluconate is widely used in healthcare for handwashing, pre-procedure skin preparation, intravascular catheter exit-site care, oral care for prevention of ventilator-associated pneumonia, and whole-body bathing [159]. It is effective for the decolonisation of *Candida* species, although there is little evidence specific to *C. auris* [142]. One study demonstrated that chlorhexidine gluconate (<0.02% with a contact time of 24 h) was effective in inhibiting growth of planktonic cells and biofilms of clinical isolates of *C. auris* [160]. With concentrations between 0.125 and 1.5%, and a 3 min contact time, growth of *C. auris* was inhibited and increased efficacy was seen at 3 and 30 h [144]. However, *C. auris* had consistently higher minimal inhibition concentrations (MICs) when compared to other *Candida* species tested. A study of 2% chlorhexidine gluconate used alone failed to eliminate *C. auris* with a contact time of 2 min. The addition of 70% isopropyl alcohol (IPA), however, did reduce all six strains of *C. auris* to undetectable levels within 2 min, suggesting that a chlorhexidine/IPA disinfectant could reduce colonisation if applied appropriately [143]. This may cause skin damage, and further evidence is required.

Patients can remain colonised despite daily chlorhexidine washes [39, 60]. Decolonisation may, however, reduce *C. auris* bioburden on the skin surface of affected individuals, potentially reducing the risk of transmission [39, 92]. The evidence suggests challenges result from persistent colonisation. It remains uncertain if this relates to reinfection from the environment or reduced susceptibility to chlorhexidine. In one study, two patients with persistent colonisation in the groin suffered from diarrhoea, which may have contributed to persistence [60].

In a facility where *C. auris* was endemic, integrating microbial genomic and epidemiological data revealed occult *C. auris* colonisation of multiple body sites not commonly targeted for screening [161]. High concentrations of chlorhexidine were associated with suppression of *C. auris* growth but not with deleterious perturbation of commensal microbes. A murine model study observed that *C. auris* can enter the dermis without causing overt histopathological signs of inflammation, expanding into deeper tissues, which could make decolonisation challenging [162]. This could also explain the reoccurrence of *C. auris* in patients who had serial negative swabs. Interestingly, chlorhexidine antiseptic protected against colonisation of *C. auris* on mouse skin.

A study investigating the *in vitro* yeasticidal activity of povidone-iodine against *C. auris*, compared to *C. albicans* and *Candida glabrata*, was encouraging. The growth of all clinical *C. auris* isolates was inhibited at concentrations between 0.07 and 1.25%, which is below many of the commercially available concentration of 10%, with a minimum contact time of 3 min [144].

A murine skin colonisation model was used to test fungal burden reduction following treatment with 1% terbinafine or 1% clotrimazole in a proprietary Advanced Penetration Technology formulation. This found that both treatments significantly reduced fungal burden compared to that in control groups [163]. Compounds with antimicrobial activity have been assessed for efficacy in antifungal decolonisation, including triclosan, boric acid, and zinc oxide, which can be used for long periods of time without an abrasive skin effect. Antifungal activity of boric acid and triclosan was demonstrated against multiple *Candida* species, including a clade of *C. auris* [164].

CONCLUDING REMARKS

Candida auris is increasingly detected at widespread geographical locations, and prevalence is likely to be greater than what is currently reported. Detection has improved in many countries known to be affected by *C. auris*. The organism should be considered when unidentified or unusual *Candida* species are isolated from patients who fail to respond to empirical antifungal therapy. Colonisation and infection have been primarily detected in high-dependency settings to date, and important risk factors include intravascular catheters and mechanical ventilation. The COVID-19 pandemic therefore had an extremely detrimental impact in several countries worldwide, with widespread transmission and *C. auris* co-infection frequently associated with overcrowding, inadequate PPE use, and suboptimal IPC practice. This was also associated with high mortality, although that directly attributable to *C. auris* cannot be established. Accurately evaluating prevalence outside of high-dependency settings has been a challenge and requires further study to inform guidance.

It is clearly difficult to completely prevent transmission following identification of someone colonised with *C. auris*, even whilst employing strict IPC and isolation techniques, but control has been demonstrated [137, 165, 166].

Although treatment of asymptomatic colonisation is not currently widely recommended in the literature, this requires further review. Potential benefit to individuals and implications for wider transmission has been suggested; however, efficacy is uncertain. The duration patients can remain colonised, and thus how long patients should be isolated after first detection, is also uncertain. Continuing isolation for the duration of an inpatient stay has been recommended, given the risk of transmission. Although the CDC recommend deisolation following two consecutive negative screening swabs, this criterion is uncommonly met and recolonisation is possible.

Antifungal susceptibility testing should be performed on all isolates, and empirical treatment with an echinocandin is recommended whilst awaiting results. The development of increasingly resistant strains is, however, a concern. Future strategies include

Box 1. Priority research questionsEpidemiology

- What is the global burden of disease associated with and attributable to *Candida auris*?
- Can wastewater sequencing support *Candida auris* surveillance?
- Is there an ecological niche and is this relevant for human transmission?

Pathogenesis

- How does the skin microbiome influence *Candida auris* colonisation, and vice versa?

Antimicrobial resistance

- What are the optimal clinical breakpoints for *Candida auris*?

Diagnosis

- What strategies best support the implementation of fungal diagnostics in resource-constrained settings?
- Can point-of-care rapid diagnostics reliably inform outbreak investigations?

Treatment

- What is the most effective treatment for *Candida auris* infections at sites that are poorly penetrated by echinocandins (e.g. urinary tract, central nervous system)?
- When should combination therapy be used and which agents?

Infection prevention and control

- Why does *Candida auris* colonisation persist for so long?
- Is *Candida auris* colonisation any different from that of other MDR pathogens, such as MRSA, ESBLs, CPEs?
- Which virulence factor(s) determine the ability of *Candida auris* to persist in the healthcare environment?
- What is the optimal disinfection regimen for daily and terminal cleaning?
- How can we improve the design of multi-use equipment and the hospital environment to facilitate cleaning and reduce the environmental burden of infectious pathogens?

combination therapies and novel and alternative treatments, such as natural compounds (e.g. rocaglates), photodynamic therapy, and novel triazoles (e.g. PC945), but these require further evaluation. Antimicrobial stewardship plays a key role in limiting unnecessary antimicrobial use and ensuring appropriate case management.

A multidisciplinary response is needed to control *C. auris* transmission in a healthcare setting and should emphasise outbreak preparedness and response, rapid contact tracing and isolation, cohorting of patients and staff, hand hygiene, strict IPC, dedicated or single-use equipment, appropriate disinfection, transfers, and discharge.

Despite intensive research efforts over the past decade, several unanswered questions regarding *C. auris* remain [18] Box 1.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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