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The Epidemiology and Prevention of *Candida auris*

Graham M. Snyder[1] · Sharon B. Wright[2]

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Abstract

Purpose of Review *Candida auris* has recently emerged as a pathogen with the potential for nosocomial transmission and outbreaks. The aim of this review is to summarize the global dissemination of this pathogen, characterize patient and facility characteristics associated with infection and outbreaks, and outline evidence to support interventions to prevent of transmission in the healthcare setting.

Recent Findings *C. auris* has emerged separately in four clades, with international spread within a decade of its first identification and report. Acquisition and infection have predominantly been identified as healthcare-associated events. The presence of invasive devices, intensive care, and broad-spectrum antibiotic and antifungal use may be important risk factors for the development of infection due to *C. auris*. Nosocomial transmission is likely associated with colonization density and suboptimal infection prevention practices. The optimal strategy for reducing transmission from the environment requires further study.

Summary *Candida auris* is a recently emerging fungal pathogen that may cause nosocomial infections and outbreaks. Based on observed transmission patterns and interventions, key prevention measures outlined in the review include case finding and surveillance, hand hygiene, and environmental disinfection.

Keywords Infection prevention and control · Transmission · Outbreak · Emerging pathogen

Introduction

Candida auris is an emerging yeast that to date has been described primarily as a healthcare-associated pathogen. In the decade since the pathogen has first been reported, it has been identified from clinical and surveillance isolates throughout the world.

The reasons for its emergence remain unexplained. In some countries, the incidence remains rare or not yet described while other settings have observed *C. auris* constituting an alarming proportion of *Candida* isolates. Perhaps enigmatically, *C. auris* has not appeared to have emerged in relationship with the most common candidal pathogens that cause > 90% of invasive candidiasis in humans: *Candida albicans*, *Candida glabrata*, *Candida tropicalis*, *Candida parapsilosis*, and *Candida krusei* [1]. Rather, it is closely related to but genetically distinct from the rarer healthcare-associated pathogens *Candida ruelliae*, *Candida haemulonii*, *Candida pseudohaemulonii*, *Candida duobushaemulonii*, and *Candida heveicola* [2]. Furthermore, phylogenetic and genomic analyses have progressively demonstrated four distinct clades; substantial inter-clade variation and intra-clade homogeneity suggest the species emerged independently in multiple geographic regions (South Asia, South Africa, South America, and East Asia) [2–5, 6••].

C. auris exhibits various attributes that should signal concern to the infectious disease clinician, hospital epidemiologist, and antimicrobial steward: potential to cause nosocomial outbreaks, substantial mortality among patients with invasive infection, difficulty in identifying the pathogen, frequent multidrug-resistance, and persistence in the environment.

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✉ Graham M. Snyder
snydergm3@upmc.edu

[1] Department of Infection Prevention and Control, University of Pittsburgh Medical Center, 3601 5th Avenue, Falk Medical Building, Suite 150, Pittsburgh, PA 15213, USA

[2] Division of Infection Control/Hospital Epidemiology, Silverman Institute of Health Care Quality and Safety, Beth Israel Deaconess Medical Center, 330 Brookline Ave, Mailstop SL-435, Boston, MA 02215, USA

Therefore, an understanding of the epidemiology of its spread and methods to prevent transmission are of immediate importance to all providers. In this review, we will summarize the global dissemination of this pathogen, characterize patient and facility characteristics associated with infection and outbreaks, and outline evidence to guide prevention of transmission in the healthcare setting.

Expanding Geography and Epidemiology

First reported in 2009, the earliest known *C. auris* isolate was obtained in 2006 from an infection of the external ear canal of a 70-year-old woman in Tokyo [2]. Subsequently, the rising global prominence of *C. auris* has been described in case reports, case series, surveys, and cohort studies.

Table 1 summarizes key features of these reports. Several Indian studies suggest a substantial prevalence in that country: among 27 geographically diverse intensive care units, 5.6% (52/918) of nosocomial candidemia episodes were due to *C. auris* (2011–2012) [14], a single-center case series reported 6.1% (15/245) of yeast isolates from blood cultures and other sterile site specimens were attributed to *C. auris* (2011–2013) [12], and a single-center study among all patients with candidemia found 17.5% (20/144) of isolates were due to *C. auris* (2012–2017) [42]. Among 695 candida bloodstream isolates submitted to a Kuwaiti reference laboratory, 14 (2.0%) were speciated as *C. auris* and the annual proportion increased over a 4-year period from 2014 to 2017: 0.5% (1/183), 2.4% (4/165), 1.8% (3/171), and 3.4% (6/175) [50].

Whether or not other regions of the world might expect a rising frequency of *C. auris* infections over time remains to be determined, as data on the prevalence of *C. auris* among *Candida* species are less well-studied in other regions and among other clades of *C. auris*. A single-center study in the UK published in 2016 reported 1 of 2246 (0.04%) of screened patients admitted during an outbreak were positive for *C. auris* [19]. A 2017 study that queried the international SENTRY Antimicrobial Surveillance Program reported on the 15,271 *Candida* isolates from 2004 to 2015 [4]. Only 0.03% (4/15,275) were speciated as *C. auris*, three of which occurred in 2013 or later, supporting both the recent emergence and isolated nature of described outbreaks [4].

Public health authorities have provided descriptive reports of the emergence of this pathogen. A June 2016 clinical alert from the US Centers for Disease Control and Prevention (CDC) brought to attention potential challenges in identifying the pathogen and was accompanied by a recommendation to speciate *Candida* isolates from sterile body sites (and from non-sterile sites in certain situations warranting enhanced surveillance) [58, 59]. By 2018, the CDC reported 493 confirmed and 30 probable cases from 12 states, 899 patients with colonization from six of those states, and three states contributing over 95% of the confirmed cases [60]. Some, but not all, cases in the USA have been traced to patients who previously received medical care in countries with reported *C. auris* cases, and phylogenetic testing suggests importation of cases from all four identified clades with subsequent local transmission [6••]. Similar epidemiologic patterns have been observed in the European Union and UK. In April 2018, the European Centre for Disease Prevention and Control reported the first cases from 2013, with a surge in *C. auris* reports in 2016 (290 cases of infection or colonization) and 2017 (303 cases). Cases had been observed in six countries, with 98% of cases from two countries reporting nosocomial outbreaks [61•]. Detailed analysis of outbreak isolates from the UK has also suggested importation from at least two different clades with local transmission [5, 52•]. Synthesizing the international prevalence and public health data, reports of infections due to *C. auris* are increasing and associated with clonal spread.

Reports have demonstrated a range of mortality associated with *C. auris* (Table 1), and there exists no high-quality study of attributable mortality. The most informative data comes from a single-center outbreak of 70 patients infected or colonized with *C. auris* and controls with negative active surveillance screens and no history of *C. auris* [52•]. The study found no substantial difference in 30-day mortality between cases and controls: 16.7% (11/66) and 15.7% (52/331), among cases and controls respectively, and the 90-day mortality among cases and controls was 20.3% (13/64) and 19.9% (44/221) [52•]. The studies described in Table 1 analyzing more than ten patients report mortality rates associated with *C. auris* ranging from 27.5 to 60.0% [4, 11, 12, 18, 21, 24, 50, 51].

Case ascertainment for determining prevalence and attributable mortality requires reliably identifying the pathogen. Automated susceptibility panels have misidentified *C. auris* as a variety of organisms depending on the panel used. For example, in a 2016 survey of isolates from six hospitals in Columbia with five different biochemical identification systems, organisms were misidentified as *C. haemulonii*, *C. albicans*, *C. tropicalis*, and *Candida famata* [21]. In a study using a CDC-prepared fungal panel and four biochemical identification systems, the pathogen was misidentified as *Rhodotorula glutinis*, *Candida catenulata*, *Candida guilliermondii*, *Candida lusitaniae*, and *C. parapsilosis* [62]. Infectious disease clinicians should partner with their microbiology colleagues to ensure that *C. auris* is appropriately identified from clinical specimens [63]. Additional methods including matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS) and polymerase chain

reaction (PCR) technologies may allow for more rapid identification of the pathogen [64–66].

Risk Factors for Acquisition and Disease

Many studies have described characteristics of patients colonized or infected with *C. auris*; however, few have systematically investigated risk factors for acquisition. As part of an outbreak investigation, Eyre and colleagues performed a case-control study in which 66 case patients were admitted to the intensive care unit (ICU) prior to diagnosis of *C. auris*, including seven with invasive infections [52•]. A total of 361 controls were selected from patients admitted to the same ICU with no prior history of *C. auris* infection and negative surveillance screens. In multivariate models, factors associated with development of colonization or infection included use of axillary temperature monitoring (odds ratio, 95% confidence interval (OR, 95% CI), 6.80, 2.96–15.63) and receipt of any antifungal (10.34, 1.64–65.18). However, the frequency of antifungal exposure was low in both groups (three in each received fluconazole) [52•]. In a smaller single-center study in an outbreak setting, 15 patients admitted to ICUs and subsequently colonized or infected with *C. auris* over an approximately 20-month period were matched 2:1 to controls based on time of hospitalization and admission ward. Although the authors do not provide effect estimates for variables included in the multivariate model, tetracycline (minocycline or tigecycline) exposure and diarrhea were significantly associated with *C. auris* acquisition [48].

A cohort study of patients with *C. auris* bloodstream infections ($N = 52$) in 27 Indian ICUs were compared with 1087 patients from the same population with candidemia due to non-*C. auris* organisms [24]. The final multivariate model identified the following significant variables associated with *C. auris* infection (OR, 95%CI): prior antifungal exposure (2.8, 1.64–4.86), vascular surgery (2.3, 1.00–5.36), public-sector hospital (2.2, 1.25–3.87), ICU location in northern India (2.1, 1.17–3.84), underlying respiratory disease (2.1, 1.31–3.60), urinary catheter (1.9, 1.11–3.42), and low APACHE II score on admission (0.8, 0.81–0.96) [24].

It is difficult to draw conclusions about factors contributing to *C. auris* infections from these available studies dominated by local circumstances such as outbreak conditions. However, findings from uncontrolled case series may generate hypotheses regarding factors contributing to the development of *C. auris* disease (Box) [4, 11, 12, 20, 21, 29, 46, 50, 51]. Cases are described in age groups from neonatal to elderly. Medical devices are commonly described potential risk factors: four of nine series report universal central venous catheter use, and the other five report use in 42–94% of cases [4, 11, 12, 20, 21, 29, 46, 50, 51], and urinary catheter use is described in five studies, ranging in frequency of use from 61 to 95% of cases [4, 11, 12, 21, 46]. While descriptions of cases in ICUs are common—and may be collinear with device utilization—mechanical ventilation is only described in three studies, occurring in 33–73% of cases [21, 46, 51]. Additional potential risk factors have been less frequently described including: recent surgery in > 50% of *C. auris* cases in four of six studies describing this factor [4, 11, 12, 21, 29, 46]; parenteral nutrition in six studies, ranging in frequency of 0–80% among cases [11, 12, 20, 21, 29, 46]; and immunosuppression, malignancy, end-stage renal disease and hemodialysis, and diabetes mellitus in a minority of cases.

Two case series describe co-carriage[1] with multidrug-resistant bacterial pathogens: among five nosocomial cases observed in Oman, three patients were co-colonized with carbapenem-resistant *Klebsiella pneumoniae* and one patient was co-colonized with *Stenotrophomonas maltophilia* [29]; and among three cases in Singapore, all three were co-colonized with carbapenem-resistant *Enterobacteriaceae* (two each with NDM-1 and OXA-232 resistance genes) [47]. Given the substantial overlap in factors associated with *C. auris* carriage and well-studied risk factors for multidrug-resistant bacterial pathogens, facilities should consider the risk of multidrug-resistant bacterial pathogens in situations for which *C. auris* screening is performed [67, 68].

Box. Factors associated with *Candida auris* colonization and infection.

Admission to a facility with *C. auris* cases
Broad-spectrum antibiotic and antifungal use
Concomitant conditions: recent surgery, parenteral nutrition
Intensive care unit admission
Invasive devices: central venous catheters, indwelling urinary catheters

Antifungal resistance, including multidrug resistance, is an important characteristic of *C. auris*. Detailed studies of minimum inhibitory concentrations have been published [3, 69, 70], as have studies identifying genotypic mechanisms of resistance [4, 22, 70]. Several studies demonstrate the high frequency of antifungal resistance observed from clinical isolates (Table 2). Therefore, exposure to antifungals would be expected a priori to be a risk factor for development of colonization and infection. Exposure to antibacterial agents might also be hypothesized to confer risk of developing *C. auris* infection [71]. In contrast with the infrequent with the infrequent antifungal exposure (specifically, fluconazole) among cases in the outbreak investigation reported by Eyre and colleagues (5% of cases and 0.8% of controls) [52•], eight of the nine case series described in this section report more frequent exposure to antifungals—prior receipt was

[1] In this review, "carriage" refers to studies not differentiating between colonization and infection.

Table 1 Findings of published studies describing *Candida auris* cases, outbreaks, and cohort studies

Year(s) organism isolated	Country	Study design	Number of cases	Frequency (% (cases/total))	Measure of frequency	Mortality	Reference
2006	Japan	Case report	1	–		–	[2]
2004–2006	South Korea	Case series	15	–		–	[7]
2006	South Korea	Case series	15	–		–	[8]
1996, 2009	South Korea	Case series	3	–		66.7%	[9]
2011	India	Case series	2	–		–	[10]
2009–2011	India	Case series	12	–		50.0%	[11]
2011–2013	India	Case series	12	6.1% (15/245)	Yeast isolates	33.3%	[12]
2012–2013	South Africa	Case series	4	–		–	[13]
2011–2012	India	Cohort study	52	5.6% (52/918)	ICU candidemia episodes	–	[14]
NR	India	Case report	1	–		–	[15]
2014	Kuwait	Case report	1	–		–	[16]
NR	India	Case series	5	–		100.0%	[17]
2012–2013	Venezuela	Outbreak investigation	18	–		27.8%	[18]
2015–2016	UK	Outbreak investigation	50	0.04% (1/2246)	Patient admission screening	0.0%	[19]
2013–2016	USA	Case series	7	–		57.1%	[20]
2016	Columbia	Case series	17	–		35.2–38.4%	[21]
2008–2015	(Various)	Case series	54	0.03% (4/15271)	*Candida* isolates	58.5%	[4]
2014	Israel	Case series	6	–		40.0%	[22]
2013–2017	USA	Case series	122	11.5% (45/390)	Case contact screening	–	[23]
2011–2012	India	Cohort study	52	–		27.0–41.9%	[24]
2017	Canada (ex India)	Case report	1	–		–	[25]
2013	South Korea	Case report	1	–		–	[26]
2017	Oman	Case series	2	–		50.0%	[27]
NR	USA	Case report	1	–		–	[28]
2016–2017	Oman	Case series	5	–		60.0%	[29]
2017	India	Case series	3	–		–	[30]
2016	Panama	Case series	9	–		22.2–77.8%	[31]
2017–2018	Saudi Arabia	Case series	3	–		33.3%	[32]
NR	USA	Case report	1	–		–	[33]
2013–2017	Europe	Case series	620	–		–	[34]
NR	Japan	Case report	1	–		–	[35]
2013–2015	Columbia	Case series	3	–		67.7%	[36]
2016–2017	Israel (ex South Africa)	Case series	2	–		0.0%	[37]
2014–2017	Kuwait	Case series	56	–		–	[38]
2016–2017	Columbia	Case series	123	–		–	[39]
2017	Switzerland (ex Spain)	Case report	1	–		–	[40]
2017	United Arab Emirates	Case report	1	–		–	[41]
2012–2017	India	Cohort study	45	17.5% (20/114)	*Candida* isolates	–	[42]
NR	India	Case report	1	–		–	[43]
NR	China	Case report	1	–		–	[44]
NR	Malaysia	Case report	1	–		–	[45]
2016–2017	Spain	Outbreak investigation	140	–		41.4%	[46]
2012, 2016	Singapore (ex India/Bangladesh)	Case series	3	–		50.0%	[47]
2011–2017	China	Case series	15	–			[48]

Table 1 (continued)

Year(s) organism isolated	Country	Study design	Number of cases	Frequency (% (cases/total))	Measure of frequency	Mortality	Reference
2018	Austria	Case report	1	–		–	[49]
2015–2017	Kuwait	Case series	17	2.01% (14/695)	Candidemia isolates	60.0%	[50]
2013–2017	USA	Outbreak investigation	112	10.7% (61/572)	Inpatient case contact screening	27.5–58.1%	[51]
2015–2017	UK	Outbreak investigation	70	9.3% (267/2872) 2.9 cases/100 patient-days at risk	Inpatient-days of screening Inpatient acquisition rate	16.7–20.3%	[52•]
2012–2016	South Africa	Case series	1692	–		–	[53]
NR	USA	Case report	1	–		–	[54]
2013–2017	USA	Case series	385	–		–	[6]
2018	China	Case series	2	–		100.0%	[55]
NR	Iran	Case report	1	–		–	[56]
2015	Australia (ex Kenya)	Case report	1	–		–	[57]
2015–2016	Columbia	Case series	NR	71.4% (5/7)	Case contact screening	–	[39]

Note: Definitions of mortality may vary between studies; for studies with > 1 measure of mortality, ranges are provided. Mortality not quantified for case reports; of ten case report publications reporting mortality, six (60.0%) reported patient death

"–" data not applicable or not reported; *ICU*, intensive care unit; *NR*, not reported

observed in 32–72% of cases (and a combined total of 86 of 192 cases (45%)) [4, 11, 12, 21, 29, 46, 51]. Of the seven case series describing antibiotic use, four report all case patients being exposed to broad-spectrum antibiotics and the remainder report exposure frequencies of 75–88%; details of the specific antibiotics are not provided [11, 12, 21, 29, 46, 50, 51]. These data leave the relationship between antimicrobial exposure and *C. auris* acquisition and infection unresolved.

Prevention of Transmission

Patient Carriage, Surveillance, and Decolonization

Though *Candida* species are common causative pathogens of healthcare-associated infections, they are uncommonly transmitted in the healthcare setting [72, 73]. Both inter- and intra-facility transmission of *C. auris* has been demonstrated. Inter-facility transmission patterns have been less-well described compared with outbreaks within a facility, but evidence of outbreaks involving acute care and long-term care facilities within an urban region [51], transmission within regions [23, 39], and within countries [34] all suggest healthcare networks are important for transmission dynamics [74, 75]. Eight of the case series described in the prior section reported the duration of admission prior to *C. auris*, with median admission duration ranging from 9.5 to 52 days and only a small proportion of cases being community-acquired, suggesting the high frequency of and predilection for nosocomial transmission [11, 12, 20, 21, 29, 46, 50, 52•].

The results of studies performing patient surveillance screening, including as part of outbreak investigations, help to paint a picture of sources of transmission. As part of the measures to control a 50-case outbreak in the UK, investigators screened 2246 inpatient admissions and found only one (0.04%) positive for *C. auris*. Although the screened patients' prior healthcare exposure is not described, this estimate of colonization in the general population is the best currently available [19]. In the only study reporting screening of *C. auris*-positive patient family and visitor contacts, none of four screened contacts were positive [39].

Two studies in the USA describe results of screening healthcare contacts of patients positive for *C. auris*. In a case series of all 122 infections occurring in the USA between June 2016 and May 2017, 390 "close" contacts of 77 patients in three states were screened for colonization with *C. auris* and found a 12% colonization frequency (45/390) [23]. A similar rate of transmission to contacts was identified in a single-city, multifacility outbreak that included a total of 112 patients, including 51 (46%) with clinical isolates and 61 identified by surveillance [51]. Screening among 572 patients identified 61 (11%) carriers, and screening among patients in long-term care facilities demonstrated higher frequency of colonization (13%) than screening among patients in acute care hospitals (8%) [51]. These studies suggest a community reservoir may be unlikely; transmission to contacts in the healthcare setting is a significant risk that may depend on colonization density.

Table 2 Select studies describing the frequency of antifungal resistance among *Candida auris* isolates

Country, study setting (years)	Sample size	Frequency of resistance to select antifungals							Reference
		Fluconazole (%)	Voriconazole (%)	Echinocandin (%)	Amphotericin B (%)	Flucytosine (%)	≥2 drug classes	≥3 drug classes	
India, 10 acute care hospitals (2009–2017); clinical isolates	350	90.3	14.9	2.0	7.8	16.0	25.1	2.0	[70]
USA, multistate survey (2013–2017); clinical and surveillance isolates	99	88.9	–	6.1	33.3	–	39.4	–	[6]
Columbia, outbreaks in 4 hospitals (2015–2016); clinical, surveillance, and environmental isolates[a]	85	12.9	0	0	30.6	–	0	0	[39]
UK, single-center outbreak (2015–2017); clinical and surveillance isolates	79[b]	100	97.5	0	17.7	0	17.7	0	[52•]
Multination survey (2008–2015), clinical isolates	54	92.6	53.7	7.4	35.2	5.6	40.7	3.7	[4]
USA, single region outbreak (2016–2017); clinical isolates	51	98.0	–	0	29.4	–	25.5	0	[51]

Note: Not all drugs/drug classes tested in each study are reported here

"–" data not reported

[a] Data abstracted from published supplementary table. Amphotericin B-resistant isolates were observed in only two of four hospitals

[b] Eighty isolates were tested for susceptibility to voriconazole

The above studies employed various screening strategies including: composite axilla and groin with or without nasal swab [23, 51]; axilla, perineum, and urine [37]; and nose, axilla, groin, tracheostomy, wound, and urine [52•]. Comparative data of screening strategy by body site is lacking, including among these studies. In the study by Tsay et al., among 184 patients screened by nasal swab in addition to composite axilla and groin swab, only 2 (1%) were positive from the nares and both were also positive on axilla/groin testing [23]. A report of small outbreaks at four Columbian hospitals demonstrated a transmission to five of seven (71%) screened in-hospital contacts. The screening strategy for contact patients in this study included ears (1/7 patients were positive at this site), nose (1/7), mouth (0/7), axilla (2/7), groin (2/7), and rectum (2/7). Four patients were positive at only one site (axilla, groin, rectum) and one patient was positive at four sites (ear, nose, axilla, rectum) [39]. Given this limited data, the optimal strategy remains to be determined, although the axilla and groin are favored body sites for screening.

One study of a large single-center outbreak provides the most detailed information to date of active surveillance screening. Eyre and colleagues performed 9153 screening swabs from the nose, tracheostomy, axilla, groin, urine, and wounds (if present) from 900 patients over 2872 patient days of screening, including on admission, weekly to thrice weekly, and at discharge, depending on the unit [52•]. Investigators found 267 (9%) of the 2872 screening events demonstrated *C. auris*, from 62 (7%) of the 900 patients, for a rate of 2.9 acquisitions per 100 ICU patient days at risk [52•]. The median duration of carriage was 61 days (or 82 days, depending on the definition of loss of carriage) [52•], a duration that is consistent with qualitative reports elsewhere of prolonged carriage [51].

Two studies provide information on the sensitivity of surveillance cultures. In the Eyre study, samples repeated within 2 days inferred a 78% sensitivity of the sampling method [52•]. Chow and colleagues found that same-day isolates, which may provide an approximate estimate of sensitivity, were closely related supporting a high sensitivity of surveillance swabs [6••]. The test characteristics and reproducibility of sampling warrants further investigation, and non-culture-based PCR methodologies may facilitate surveillance in the endemic and epidemic setting [76, 77].

Very little is published on effectiveness of *C. auris* decolonization strategies that may include chlorhexidine washes, nasal treatment, and oral nystatin [19]. In vitro studies of disinfectants for patient use have demonstrated efficacy of chlorhexidine (0.125–1.5%), povidone iodine (0.07–1.25 and 10%), and chlorhexidine (2%) in isopropyl alcohol (70%) [78, 79], though the effect of chlorhexidine without alcohol has not been consistently demonstrated [79], and in biofilm models, povidone iodine and chlorhexidine may demonstrate reduced effectiveness [80].

Hand Hygiene, Contact Precautions, and Healthcare Worker Carriage

Hand hygiene and contact precautions (the use of gown and gloves as a component of transmission-based precautions) may be important tools for prevention based on the mode of transmission of healthcare-associated pathogens. No data concerning the impact of hand hygiene and contact precautions to reduce transmission of *C. auris* specifically have been published. However, their effectiveness has been inferred based on data from other organisms [81, 82], and improving hand hygiene adherence [46, 52•] and the use of contact precautions [19, 23, 33, 46, 52•] have been described as components of *C. auris* outbreak control. One study investigated the efficacy of various hand hygiene products on eradicating growth of *C. auris* on volunteers' fingertips after inoculation with the organism. Cultures following hand hygiene interventions with soap and water, 70% alcohol-based hand rub, or 70% alcohol plus 0.5% chlorhexidine gluconate hand rub demonstrated no growth [30].

As part of an outbreak investigation, screening of healthcare workers for pathogen carriage may identify individuals as potential routes of transmission, either as the initial source or an intermediate one, particularly through ineffective hand hygiene. Several studies describing healthcare workers screening have demonstrated very low frequencies of carriage. During an outbreak investigation, Schelenz et al. found only 1 of 258 (0.4%) of healthcare workers positive for carriage using agar impression plates of hands, as well as nose, axilla, groin, and throat swabs. The one positive screen was positive on nares swab only [19]. In a large single-center outbreak, Ruiz-Gaitan found none of the 101 surgical intensive care unit healthcare workers screened by hand and ear canal sampling were positive for *C. auris* carriage. Similarly, in a very small cluster of two patients, Chen found none of five tested healthcare workers were positive for hand carriage [46, 55]. Two studies have detected healthcare worker hand carriage, however: a single-center investigation of three *C. auris* bloodstream infections over a 3-month period identified 4/145 (3%) healthcare workers from two of four units were positive for hand carriage [30], and a report from three hospitals as part of a country-wide outbreak found two of six (33%) healthcare workers tested positive for hand carriage [39].

In most situations, healthcare worker screening for carriage of *C. auris* is likely to be of low yield. Instead, the focus on reducing transmission risk may be based on prevention strategies employed for other multidrug-resistant organisms including maximizing hand hygiene adherence and the use of gowns and gloves. The optimal disinfectant product for hand hygiene and the impact of contact precautions in the prevention of transmission of *C. auris* remain to be quantified.

Environmental Contamination

Given the epidemiologic evidence that nosocomial transmission is more common than community transmission of *C. auris*, it is important to consider contamination of the healthcare environment and mitigate its role in transmission. Two in vitro studies have demonstrated the pathogen's ability to survive on surfaces for at least 7–28 days [83, 84]. Sampling of the patient environment demonstrated recovery of *Candida* species from dry surfaces similar to methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant enterococci, and significantly greater recovery of *Candida* species than these pathogens from moist patient environments, suggesting a greater potential for *C. auris* to survive in the environment than other pathogens [84]. In a third study, potential persistence of *C. auris* in the patient environment was demonstrated using 3 cm^2 samples of hospital linens kept at room temperature, where a standardized inoculum was recovered up to seven but not eight or greater days [30].

Studies in the hospital setting show variable contamination of surfaces and equipment with *C. auris*. Several studies investigating the role of the environment in the setting of one or two cases of *C. auris* infection demonstrated negative or predominantly negative environmental cultures [33, 37, 40, 55, 56]. Other case reports describe environmental testing as frequently or universally positive, and subsequently negative or termination of cases after cleaning with a hypochlorite-based disinfectant with or without ultraviolet or hydrogen vapor no-touch disinfection [19, 20, 23]. The difference in the reported frequency of environmental contamination may be due to colonization density, unit design, or patterns of shared equipment use.

For example, as part of a single-center investigation related to three *C. auris* infections, open-bay ICUs where patients were cared for most frequently demonstrated fomite contamination (9% (17/189 samples) and 10% (7/68 samples)) while less-frequented ICU and neurosurgical units demonstrated no contamination (from 10 and 37 samples, respectively) [30]. At three sampling periods (separated by 2–3 months) during a 128-case single-center outbreak, *C. auris* was "rarely detected in the general environment or air" but cultured from axillary and skin surface temperature probes, pulse oximeters, and patient hoist devices [52•]. In a regional multifacility outbreak, environmental samples of high-touch surfaces and fomites were undertaken "whenever possible" at 20 facilities, of which 16 facilities (75%) identified at least one positive environmental culture. The authors provide detailed findings from the 781 samples and several findings merit highlight: 8% (62/781) of all samples were positive; surfaces and equipment within the room were more commonly contaminated than equipment outside the room (12 vs.

Table 3 Summary of key interventions recommended (or to be considered) by select governmental agencies to prevent transmission of *Candida auris*

Agency (country/region)	Active surveillance population	Hand hygiene	Isolation	Transmission-based precautions	Environmental disinfection	Additional special measures	Reference
Centers for Disease Control and Prevention (USA)	Contacts of newly identified case patients. Patients with an overnight stay in a healthcare facility outside of the USA in the previous year	Alcohol-based hand rub, or soap and water if hands are visibly soiled	Single room or cohorting with another patient with *C. auris*	Standard and contact precautions, for the duration of colonization, perhaps indefinitely	Use a disinfectant active against *Clostridioides difficile* spores	Minimize the number of care providers	[91]
Public Health England (UK)	Patients admitted from affected hospitals within the UK or from hospitals in countries reporting outbreaks. Close contacts in intensive care settings or contacts of patients prior to implementation of isolation procedures	Soap and water followed by alcohol-based hand rub	Single room or cohorting for colonized or infected patients or pending screening from high-risk areas	Contact precautions	Post-discharge terminal cleaning with sodium hypochlorite disinfectant, with or without no-touch disinfection	Single-use medical equipment; chlorhexidine skin washes for critically ill patients, mouth gargle with chlorhexidine, and topical nystatin and terbinafine at key sites	[92]
European Centre for Disease Prevention and Control (Europe)	Patients recently admitted or transferred from hospitals with detected *C. auris* case. Close contact patients	–	Single room or cohorting	Contact precautions	Post-discharge terminal cleaning with chlorine-based disinfectants, hydrogen peroxide, or other disinfectants with fungicidal activity	Staff cohorting. Single-use equipment or cohorting of equipment among cases	[61•]
Centre for Opportunistic, Tropical and Hospital Infections (South Africa)	Routine screening on admission not recommended	Soap and water followed by alcohol-based hand rub	Single room or cohorting	Standard and contact precautions	Environmental cleaning with a chlorine-based disinfectant and consider hydrogen peroxide vapor for no-touch disinfection after terminal cleaning	Off-unit procedures should be scheduled for last case of the day, followed by thorough cleaning	[93]

5%, respectively); bathroom surfaces and curtains were contaminated in > 25% of samples; few surfaces (totaling 52 samples) never demonstrated contamination; and PCR was positive when culture was negative in only 19 (2%) samples [51]. In a similar fashion, Escandon and colleagues categorized sampled surfaces into four zones sequentially further from the patient and with less direct patient and provider contact, from zone 1 comprising the patient bed and adjacent environment to zone 4 comprising adjacent bathroom and hallway surfaces [39]. The total surface contamination rate was 11% (37 of 322 surfaces), with decreasing frequency with distance: zone 1, 16% (14/85); zone 2, 14% (11/76); zone 3, 12% (7/58), and zone 4, 5% (5/103). Also of note, there was variability in the contamination rate among the four hospitals, including low frequencies (0% (0/107) and 3% (2/59)) and high frequencies (18% (12/68) and 26% (23/88)), and so trends by zone are difficult to observe by hospital [39]. These data suggest that some degree of environmental contamination may be present in most cases, and more likely associated with surfaces that have the greatest patient contact.

Few reports describe disinfection strategies, and no reports provide comparative data. Several studies describe effective room cleaning with a sodium hypochlorite-based cleaner, with or without no-touch disinfection [19, 20, 23]. Another case report without subsequent transmission events describes the use of paracetic acid/hydrogen peroxide surface disinfection for daily cleaning and terminal disinfection, supplemented by ultraviolet disinfection for post-discharge room cleaning and relocating all patients once to a freshly terminally cleaned room [33]. Increased frequency of daily room cleaning may be required to effectively reduce environmental contamination [19, 46].

Since both disinfection of the environment and reusable medical equipment are critical in reducing transmission of *C. auris*, product selection may play a critical role in transmission from environmental source. In vitro studies have demonstrated the efficacy of a variety of disinfectants in planktonic and dry surface biofilm models, including 5% phenol, 2% glutaraldehyde, paracetic acid (2000 ppm), paracetic acid (1200 ppm) + hydrogen peroxide (< 1%) + acetic acid, hydrogen peroxide (0.5%, 1.4%, and vaporized), hydrogen peroxide (11%) + silver nitrate, sodium hypochlorite (0.39–2% or 1000 ppm), and a quaternary ammonium product [30, 78, 79, 85, 86]. The demonstrated efficacy may be dependent upon the hospitability of the surface [85]. Furthermore, in one study acetic acid (> 5%), ethyl alcohol 29.4%, and ammonium chloride products demonstrated diminished efficacy [86], and in another study using dry surface biofilm transferability models, wipe-based disinfectants including sodium hypochlorite, sodium dichloroisocyanurate, paracetic acid, chlorine dioxide, and benzalkonium chloride products all showed variables degrees of inefficacy [87]. These findings suggest the *in vitro* susceptibility of *C. auris* to disinfectants that in practice may have diminished effectiveness. As data on the germicidal activity of disinfectants becomes available [88•], future studies are needed to characterize optimal environmental disinfection strategies in clinical practice. Emerging data clarifying the role for ultraviolet disinfection warrants further investigation [89, 90].

Governmental Recommendations for Prevention of *C. auris*

Several governmental organizations, including in countries that have encountered large outbreaks, have provided guidance for the prevention of *C. auris*. Salient features of these recommendations are summarized on Table 3. All guidelines emphasize the importance of isolation and/or cohorting of the patient, hand hygiene, contact precautions, and environmental cleaning. Areas of discrepancy include method of hand hygiene (single method or soap and water followed by alcohol-based hand rub), active surveillance strategy (identification of patients at high risk of importing the pathogen), and methods of environmental disinfection (the use of an alternative to chlorine-based disinfectant and consideration of no-touch disinfection technology).

Conclusions

Over the last decade, *C. auris* has emerged independently in four clades and spread internationally, with varying prevalence by region. Identified risk factors for acquisition and infection include the use of invasive devices, admission to intensive care units, recent surgery, administration of parenteral nutrition, and broad-spectrum antibiotic and antifungal use. Cases have predominantly been attributed to healthcare-associated transmission, with inferential data to suggest colonization density and the contaminated environment as an important contributors to transmission risk. Optimal infection prevention strategies require further study but for now include meticulous hand hygiene, contact precautions, and close attention to environmental cleaning.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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