

# UC Irvine

## UC Irvine Previously Published Works

### Title

Rapid Environmental Contamination With *Candida auris* and Multidrug-Resistant Bacterial Pathogens Near Colonized Patients

### Permalink

<https://escholarship.org/uc/item/4mt824cz>

### Authors

Sansom, Sarah E  
Gussin, Gabrielle M  
Schoeny, Michael  
et al.

### Publication Date

2023-12-06

### DOI

10.1093/cid/ciad752

### Copyright Information

This work is made available under the terms of a Creative Commons Attribution License, available at <https://creativecommons.org/licenses/by/4.0/>

Peer reviewed

# Rapid Environmental Contamination With *Candida auris* and Multidrug-Resistant Bacterial Pathogens Near Colonized Patients

Sarah E. Sansom,<sup>1,a</sup> Gabrielle M. Gussin,<sup>2,a</sup> Michael Schoeny,<sup>3</sup> Raveena D. Singh,<sup>2</sup> Hira Adil,<sup>4</sup> Pamela Bell,<sup>1</sup> Ellen C. Benson,<sup>1</sup> Cassiana E. Bittencourt,<sup>5</sup> Stephanie Black,<sup>4</sup> Maria Del Mar Villanueva Guzman,<sup>1</sup> Mary Carl Froilan,<sup>1</sup> Christine Fukuda,<sup>1</sup> Karina Barsegyan,<sup>2</sup> Ellen Gough,<sup>1</sup> Meghan Lyman,<sup>6</sup> Jinal Makhija,<sup>1</sup> Stefania Marron,<sup>1</sup> Lydia Mikhail,<sup>7</sup> Judith Noble-Wang,<sup>8</sup> Massimo Pacilli,<sup>4</sup> Robert Pedroza,<sup>2</sup> Raheeb Saavedra,<sup>2</sup> D. Joseph Sexton,<sup>6</sup> Julie Shimabukuro,<sup>5</sup> Lahari Thotapalli,<sup>1</sup> Matthew Zahn,<sup>7</sup> Susan S. Huang,<sup>2,b</sup> and Mary K. Hayden<sup>1,b</sup>

<sup>1</sup>Division of Infectious Diseases, Rush University Medical Center, Chicago Illinois, USA; <sup>2</sup>Division of Infectious Diseases, University of California, Irvine School of Medicine, Irvine California, USA; <sup>3</sup>College of Nursing, Rush University Medical Center, Chicago Illinois, USA; <sup>4</sup>Disease Control Bureau, Chicago Department of Public Health, Chicago Illinois, USA; <sup>5</sup>Department of Pathology and Laboratory Medicine, University of California, Irvine School of Medicine, Irvine California, USA; <sup>6</sup>Mycotic Diseases Branch, Centers for Disease Control and Prevention, Atlanta Georgia, USA; <sup>7</sup>Division of Epidemiology and Assessment, Orange County Health Care Agency, Santa Ana, California, USA; and <sup>8</sup>Division of Healthcare Quality Promotion, Centers for Disease Control and Prevention, Atlanta Georgia, USA

**Background.** Environmental contamination is suspected to play an important role in *Candida auris* transmission. Understanding speed and risks of contamination after room disinfection could inform environmental cleaning recommendations.

**Methods.** We conducted a prospective multicenter study of environmental contamination associated with *C. auris* colonization at 6 ventilator-capable skilled nursing facilities and 1 acute care hospital in Illinois and California. Known *C. auris* carriers were sampled at 5 body sites followed by sampling of nearby room surfaces before disinfection and at 0, 4, 8, and 12 hours after disinfection. Samples were cultured for *C. auris* and bacterial multidrug-resistant organisms (MDROs). Odds of surface contamination after disinfection were analyzed using multilevel generalized estimating equations.

**Results.** Among 41 known *C. auris* carriers, colonization was detected most frequently on palms/fingertips (76%) and nares (71%). *C. auris* contamination was detected on 32.2% (66/205) of room surfaces before disinfection and 20.5% (39/190) of room surfaces by 4 hours after disinfection. A higher number of *C. auris*-colonized body sites was associated with higher odds of environmental contamination at every time point following disinfection, adjusting for facility of residence. In the rooms of 38 (93%) *C. auris* carriers co-colonized with a bacterial MDRO, 2%–24% of surfaces were additionally contaminated with the same MDRO by 4 hours after disinfection.

**Conclusions.** *C. auris* can contaminate the healthcare environment rapidly after disinfection, highlighting the challenges associated with environmental disinfection. Future research should investigate long-acting disinfectants, antimicrobial surfaces, and more effective patient skin antiseptics to reduce the environmental reservoir of *C. auris* and bacterial MDROs in healthcare settings.

**Keywords.** *Candida auris*; environmental contamination; colonization; disinfection.

*Candida auris* is an emerging multidrug-resistant yeast of increasing concern [1–3]. The U.S. Centers for Disease Control and Prevention “2019 Antibiotic Resistance Threats Report” lists

*C. auris* as an urgent threat, the first fungal pathogen to receive this designation [3]. Unlike other pathogenic yeasts, *C. auris* causes large, intractable outbreaks in healthcare settings [4–7]. Five percent to 10% of persons colonized with *C. auris* are estimated to develop invasive infections, which can be resistant to antifungal agents and are associated with high mortality [8, 9]. Healthcare-associated spread is promoted by the ability of *C. auris* to colonize human skin, shed into the environment, persist on inanimate surfaces for prolonged periods, and resist routine disinfectants [5, 10–13]. In the United States, long-term acute care hospitals and ventilator-capable skilled nursing facilities (SNFs) have become focal points of *C. auris* spread, with colonization prevalence in some facilities exceeding 70% [14–16]. *C. auris* continued to spread during the coronavirus disease 2019 pandemic, with notable expansion both in post-acute and acute care hospitals (ACHs) worldwide [17–22].

Understanding how *C. auris* colonization contributes to environmental contamination is critical to inform infection

Received 22 September 2023; editorial decision 26 November 2023; published online 6 December 2023

<sup>a</sup>Authors contributed equally to this work.

<sup>b</sup>Authors contributed equally to this work.

Correspondence: S. E. Sansom, University Infectious Diseases, Rush University Medical Center, 600 S. Paulina St, Ste. 143, Chicago, IL 60612, USA (sarah\_e\_sansom@rush.edu); G. M. Gussin, Division of Infectious Diseases, University of California, Irvine School of Medicine, 100 Theory, Suite 120, Irvine, CA 92617, USA (gussing@hs.uci.edu).

Clinical Infectious Diseases®

© The Author(s) 2023. Published by Oxford University Press on behalf of Infectious Diseases Society of America.

This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs licence (<https://creativecommons.org/licenses/by-nc-nd/4.0/>), which permits non-commercial reproduction and distribution of the work, in any medium, provided the original work is not altered or transformed in any way, and that the work is properly cited.

For commercial re-use, please contact journals.permissions@oup.com

<https://doi.org/10.1093/cid/ciad752>

prevention, control, and outbreak response. We conducted a prospective multicenter study of SNF residents and ACH patients who were colonized with *C. auris*. We evaluated factors affecting the speed, magnitude, and scope of contamination of high-touch environmental surfaces in participant rooms that could contribute to spread of *C. auris*. Specifically, we measured time to environmental contamination to determine whether more frequent cleaning of high-touch objects should be recommended to reduce transmission risk from *C. auris* carriers.

## METHODS

### Study Design

We conducted a prospective, multicenter study of known *C. auris* carriers and their rooms from June 2021 through April 2022 in Chicago, IL, and Orange County, CA. *C. auris* carriage was defined as culture detection of *C. auris* from any screening or clinical specimen within the past 30 days (Figure 1). The project was conducted under local public health authority and deemed exempt from human subject research oversight under regulation 45 CFR 46.102(I)(2). Participant characteristics, medical history, and antimicrobial exposure within the prior 14 days were ascertained from medical records using a standardized data collection form.

Study sites in Chicago, IL, included one 28-bed ventilator-capable isolation unit within a 244-bed SNF and any inpatient ward within Rush University Medical Center (RUMC), a 676-bed tertiary-care ACH. Project staff members identified SNF study participants by routine periodic point-prevalence *C. auris* skin screening cultures collected by the local health department. Eligible ACH study participants were identified by daily review of *C. auris* clinical cultures and/or history of positive culture reported to the Illinois XDRO Registry ([www.xdro.org](http://www.xdro.org)) (Supplementary Table 1).

In Orange County, CA, residents of 5 ventilator-capable SNFs were evaluated for eligibility by point-prevalence *C. auris* skin screening performed within each facility for routine *C. auris* surveillance (Supplementary Table 1). Mean licensed beds per facility was 134 (range, 99–202).

### Body Site Sampling

Known *C. auris* carriers were cultured for *C. auris* and bacterial multidrug-resistant organisms (MDROs) at the bilateral nares, axillae, inguinal creases, palms/fingertips, and perianal skin. Samples were collected immediately before room disinfection. Flocked swabs (FLOQSwabs, Copan, Murrieta, CA) were used to sample a 5 × 5 cm<sup>2</sup> area from each body site, then placed in Amies transport medium with neutralizer.

### Environmental Surface Disinfection

Participant rooms were cleaned daily by facility environmental services (EVS) staff according to each facility's routine procedure

using facility-approved cleaning products (Supplementary Table 2). Floors were wet mopped daily in the ACH and at varying intervals in the SNFs. For the study described here, routine daily cleaning was followed immediately by targeted disinfection of high-touch surfaces by study staff using hydrogen peroxide wipes (Clorox Healthcare, EPA-approved for *C. auris* EPA registration #67619-25).

### Environmental Surface Sampling

We conducted an initial pilot study of 10 surfaces inside and 10 surfaces outside of participant rooms to identify which environmental surfaces were most likely to be contaminated with *C. auris*. The results of the pilot study were used to identify the most commonly contaminated sites to include in the full study. Environmental surfaces were cultured for *C. auris* and bacterial MDROs using premoistened sponge-sticks (3M Sponge-Stick with neutralizing buffer; 3M, St. Paul, MN) immediately before disinfection, and at 0 (immediately following high-touch surface disinfection), 4, 8, and 12 hours after disinfection (Figure 1). Sponge-sticks were processed using the stomacher method, as described previously [23].

### Sample Processing, Culture Methods and Organism Identification

Body site and environmental samples underwent semiquantitative cultivation for *C. auris* and other MDROs including methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant Enterococcus (VRE), extended-spectrum  $\beta$ -lactamase-producing Enterobacterales (ESBL), and carbapenem-resistant Enterobacterales (CRE) at RUMC or University of California, Irvine Hospital laboratories as described previously [12, 15, 24].

Briefly, for *C. auris*, aliquots (100  $\mu$ L) of Amies transport medium were inoculated directly onto CHROMagar Candida plates (Becton Dickinson) and incubated at 37 °C for at least 48 hours and up to 7 days. A second 100- $\mu$ L aliquot was inoculated into Salt Sabouraud Dulcitol Broth and incubated at 40 °C for 7 days; cloudy broth cultures were subsequently inoculated onto CHROMagar Candida plates [13] and incubated for up to 7 days.

For bacterial MDROs, aliquots (100  $\mu$ L) were inoculated directly onto agar plates and incubated at 37 °C for 18–24 hours. Agar media used included CHROMID MRSA (bioMérieux, Marcy l'Etoile, France) or Spectra MRSA (Remel, Lenexa, KS, USA) or BBL CHROMagar MRSA (Becton Dickinson, Heidelberg, Germany) for MRSA isolation; Spectra VRE (Remel) for VRE isolation; CHROMagar Orientation with ESBL supplement (CHROMagar, Paris, France) or MacConkey agar with cefpodoxime disk (2  $\mu$ g/mL) for isolation of ESBL-producing Enterobacterales; CHROMagar mSuperCARBA (CHROMagar, Paris, France) or MacConkey agar with meropenem disk (2  $\mu$ g/mL) for isolation of CRE. A second 100- $\mu$ L aliquot was inoculated into 5 mL of Thioglycolate Broth and incubated for 18–24 hours; cloudy broth cultures were subsequently inoculated onto the same

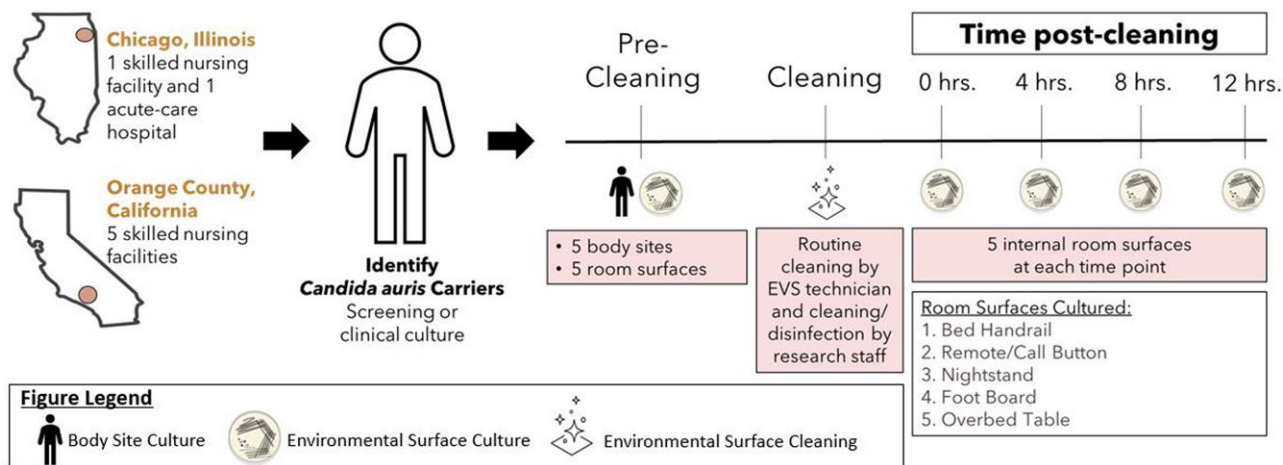


Figure 1. Sampling workflow overview.

selective agar plates and incubated for 18–24 hours. Culture workflows are illustrated in [Supplementary Figure 1](#).

#### Data Analysis

Probability of surface contamination by *C. auris* and bacterial MDROs over the course of 12 hours after effective disinfection was analyzed. Effective disinfection was defined as no growth of *C. auris* from an environmental surface at the immediate postdisinfection timepoint (time 0); environmental surfaces with growth of *C. auris* immediately after disinfection were excluded from analysis. Odds of surface contamination after disinfection were calculated using multilevel generalized estimating equations (GEE) models in which timepoints (4, 8, and 12 hours after disinfection) were nested within environmental surfaces, and environmental surfaces were nested within participant rooms. The facility was modeled as a fixed-effect covariate. Additional covariates included body site colonization with *C. auris* measured as either (1) the number of colonized body sites or (2) average semiquantitative culture value using a 6-point ordinal scale, as measured by: no growth, growth in broth only, 1+, 2+, 3+, or 4+ growth on directly inoculated agar plate. Using similar models, we assessed the impact of facility type (SNF or ACH) and number of body sites colonized with bacterial MDROs on *C. auris* room surface contamination. For all calculations, statistical significance was defined as 2-tailed  $P < .05$ .

## RESULTS

#### Cohort Characteristics

Forty-five known *C. auris* carriers were enrolled. Clinical characteristics of participants are shown in [Table 1](#). Presence of invasive devices was high (>70% with  $\geq 1$  device), most participants were bedbound (56%), and most were housed

with roommates (62%). Exposure to antibacterial agents (37%) was observed more frequently than exposure to antifungal agents (7%) in the 14 days before study enrollment.

#### Body Site Colonization

*C. auris* was cultured from  $\geq 1$  body site from 41/45 participants on the day of enrollment, and 128/205 (62%) participant body site cultures grew *C. auris*. Four participants did not have *C. auris* detected from any body site on the day of enrollment. These participants included: (1) a female participant admitted to the RUMC medical intensive care unit (ICU) with documented history in the Illinois XDRO Registry [25] of positive *C. auris* surveillance culture approximately 1 month before ICU admission; (2) a male participant admitted to the RUMC cardiac ICU with *C. auris* infective endocarditis and blood cultures growing *C. auris* on the day of enrollment; (3) a male participant admitted to a SNF who was enrolled approximately 6 weeks after a positive composite surveillance culture from the axillae, inguinal creases, and nares; and (4) a male participant admitted to a SNF who was enrolled approximately 4 months following a positive composite surveillance culture from the axillae and inguinal crease. This experience prompted us to perform additional point prevalence surveys that ensured a shorter time from positive surveillance culture to study enrollment.

Among the five body sites tested, *C. auris* was detected most commonly from the palms/fingertips (76%) and the nares (71%). *C. auris* was detected less often on perianal skin (54%), axillae (56%), and inguinal creases (56%) ([Figure 2A](#)). *C. auris* detection was higher using combinations of body sites compared with single body sites ([Table 2](#)). The proportion of *C. auris* carriers that would be detected with current screening recommendations was 83% (bilateral axillae and inguinal creases) [27].

**Table 1. Clinical Characteristics Among *Candida auris*-Colonized Participants**

Variable	SNF (N = 31)		ACH (N = 14)	
	Orange County, CA (n = 21)	Chicago, IL (n = 10)	Chicago, IL (n = 14)	Overall (N = 45) <sup>a</sup>
Positive body site for <i>C. auris</i> on day of enrollment	21	8 <sup>b</sup>	12 <sup>b,d</sup>	41
Male, n (%)	13 (62)	5 (50)	8 (57)	26 (58)
Age, mean $\bar{y}$ ( $\pm$ SD)	65 ( $\pm$ 14)	58 ( $\pm$ 11)	61 ( $\pm$ 15)	62 ( $\pm$ 14)
Participant lucid, n (%)	8 (38)	3 (30)	6 (43)	17 (38)
Invasive devices, n (%)				
Feeding tube	14 (67)	9 (90)	7 (50)	30 (67)
Tracheostomy	14 (67)	9 (90)	5 (36)	14 (31)
Ventilator	9 (43)	5 (50)	2 (14)	16 (36)
Wound(s)	15 (71)	7 (70)	13 (93)	35 (78)
Urinary catheter	5 (24)	5 (50)	3 (21)	13 (29)
Multiple room occupancy, n (%)	19 (90)	9 (90)	0 (0)	28 (62)
Bedbound status, n (%)	5 (24)	9 (90)	11 (79)	25 (56)
Incontinence, n (%)				
Urinary incontinence	18 (86)	8 (80)	10 (71)	36 (80)
Fecal incontinence	19 (90)	8 (80)	11 (79)	38 (84)
Bath in past 24 h, n (%)	20 (95)	7 (70) <sup>c</sup>	12 (86) <sup>c</sup>	39 (87) <sup>c</sup>
Chlorhexidine gluconate used	20 (100)	2 (29)	11 (92)	33 (85)
Antimicrobial exposure, n (%)				
Systemic antibiotic, prior 14 d	5 (14)	1 (10)	10 (71)	16 (36)
Systemic antifungal, prior 14 d	0 (0)	0 (0)	4 (29)	4 (9)

Abbreviations: ACH, acute care hospital; SNF, skilled nursing facility.

<sup>a</sup>Forty-five participants were enrolled, of which 41 grew *C. auris* on the day of sample collection.

<sup>b</sup>There were 4 participants enrolled that did not have a positive body site culture on the day of enrollment.

<sup>c</sup>Bathing information was not available for 2 participants.

<sup>d</sup>Three of 14 ACH patients had  $\geq 1$  positive clinical culture for *C. auris*, including urine (1), blood (1), and abscess (1).

### Pilot Evaluation

Of 10 environmental surfaces tested after disinfection within 12 participant rooms, *C. auris* was detected most frequently on surfaces close to the participant, including the bed handrail (42%), overbed table (42%), television remote/call button (25%), footboard (25%), and nightstand (17%). These 5 environmental surfaces were retained for sampling in the full study. Environmental surfaces outside of participants' rooms were rarely contaminated with *C. auris* and were not cultured in the full study (Supplementary Table 3).

### Environmental Contamination After Disinfection

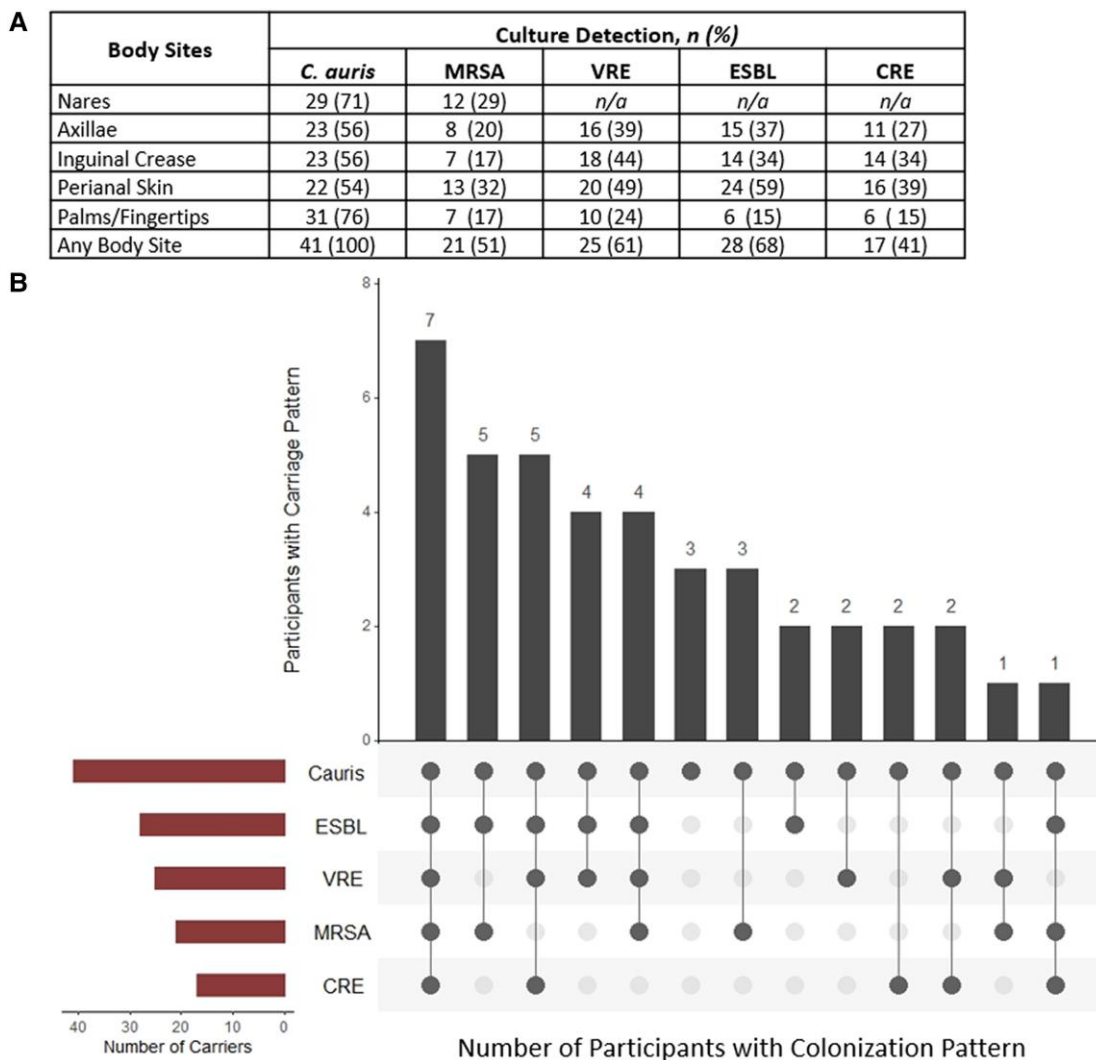
Next, we assessed time to environmental surface contamination, comparing immediate postdisinfection cultures (0 hours) to those collected at 4, 8, and 12 hours after room disinfection. Environmental surface contamination before disinfection was high in both SNF and ACH facilities (SNF 43/145 surfaces [30%] versus ACH 23/60 surfaces [38%];  $P = .659$ ). Room surfaces with growth of *C. auris* in culture immediately after disinfection (SNF 7/145 [4.8%] and ACH 6/60 [10%]) were excluded from time to contamination analyses. Time to contamination was rapid, with 39/190 (20.5%) room surfaces contaminated by 4 hours after disinfection. The proportion of contamination remained stable between 4 and 12 hours after disinfection. At all timepoints, the microbial load on surfaces

remained below predisinfection levels (Figure 3). The percentage of surfaces contaminated after disinfection was higher in the ACH compared with SNFs (SNF 12% vs ACH 34% by 12 hours;  $P < .001$ ) (Supplementary Figure 2).

### Risk Factors for Environmental Contamination

In our GEE models, having a higher number of *C. auris*-colonized body sites was associated with higher odds of environmental contamination at every time point following surface disinfection, adjusting for facility of residence and time. Quantifying *C. auris* bioburden at each body site using a 6-point ordinal scale did not improve prediction of environmental contamination; thus, body site positivity was expressed as a binary variable (positive or negative culture result). GEE model results were stratified by facility type (ACH or SNF) because of a significant interaction between facility type and number of colonized body sites in predicting surface contamination ( $P = .03$ ). The odds of environmental contamination increased 1.40 to 2.16 times for each additional body site that was culture-positive (ACH: odds ratio, 2.16 [95% confidence interval, 1.63–2.88],  $P < .001$ ; SNF: odds ratio, 1.40 [95% confidence interval, 1.07–1.84],  $P = .015$ ).

Although there was no difference in mean bioburden of *C. auris* between participants in the 2 facility types as measured by proportion of body sites positive or density of colonization



**Figure 2.** Colonization patterns of *C. auris* and bacterial multidrug-resistant organisms (MDROs) among known *Candida auris* carriers. *A*, Composite patterns of *C. auris* and bacterial MDRO body colonization among 41 *C. auris* carriers. Nares was not tested for VRE, ESBL, or CRE. *B*, Patterns of *C. auris* and bacterial MDRO co-colonization visualized with UpSetR [26]. The set size corresponds to the frequency of colonization for each subject. Abbreviations: CRE, carbapenem-resistant Enterobacterales; ESBL, extended-spectrum beta-lactamase-producing Enterobacterales; MRSA, methicillin-resistant *Staphylococcus aureus*; VRE, vancomycin-resistant Enterococcus.

at body sites, ACH participants had higher rates of bedbound status (83% vs 45%,  $\chi^2 P = .024$ ), intravascular catheter use (75% vs 24%,  $P = .002$ ), systemic antibiotic exposure (75% vs 21%,  $P = .001$ ), and systemic antifungal exposure (25% vs 0%,  $P = .005$ ) compared with SNF residents.

#### Bacterial MDRO Co-colonization and Environmental Contamination

Among *C. auris* carriers, body co-colonization with bacterial MDROs (ie, MRSA, VRE, ESBL, CRE) was common, with at least 1 bacterial MDRO detected in 93% (38/41) of *C. auris*-colonized participants (Figure 2A and 2B). There were 28 ESBL (68%), 25 VRE (61%), 21 MRSA (51%), and 17 CRE (41%) co-colonized participants identified. Among participants with confirmed body co-colonization with each respective MDRO, environmental contamination with MRSA or VRE after disinfection

was common across all tested surfaces (Figure 3); contamination with the Gram-negative MDROs was less common. We did not observe an association between bacterial MDRO co-colonization and odds of *C. auris* room contamination.

#### DISCUSSION

Environmental contamination is suspected to play an important role in *C. auris* transmission in healthcare facilities. Although contamination of the healthcare environment has been well described for bacterial pathogens [23, 28, 29], information for *C. auris* is limited. Here, we report that contamination resulting from *C. auris* occurs rapidly after disinfection of the immediate environment near known *C. auris* carriers. Most environmental contamination occurred within 4 hours of

**Table 2. *Candida auris* Screening by Body Site Among Known *C. auris* Carriers<sup>a</sup>**

Body Site Combinations	Axilla	Inguinal Crease	Nares	Palms and Fingertips	Perianal Skin	<i>C. auris</i> Detected (n = 41) (% ± 95% CI) <sup>d</sup>
One body site	✓	...	...	...	...	23 (56 ± 15%)
	...	✓	...	...	...	23 (56 ± 15%)
	...	...	✓	...	...	29 (71 ± 14%)
	...	...	...	✓	...	31 (76 ± 13%)
	...	...	...	...	✓	22 (54 ± 15%)
Two body sites <sup>b</sup>	✓	✓	...	...	...	34 (83 ± 12%)
	✓	...	✓	...	...	35 (85 ± 11%)
	✓	...	...	✓	...	33 (80 ± 12%)
	✓	...	...	...	✓	33 (80 ± 12%)
	...	✓	✓	...	...	34 (83 ± 12%)
	...	✓	...	✓	...	35 (85 ± 11%)
	...	✓	...	...	✓	28 (68 ± 14%)
	...	...	✓	✓	...	36 (88 ± 10%)
	...	...	✓	...	✓	33 (80 ± 12%)
...	...	...	✓	✓	36 (88 ± 10%)	
Three body sites <sup>c</sup>	✓	✓	✓	...	...	39 (95 ± 7%)
	✓	✓	...	✓	...	37 (90 ± 9%)
	✓	✓	...	...	✓	35 (85 ± 11%)
	✓	...	✓	✓	...	37 (90 ± 9%)
	✓	...	✓	...	✓	38 (93 ± 8%)
	✓	...	...	✓	✓	38 (93 ± 8%)
	...	...	✓	✓	✓	39 (95 ± 7%)
...	✓	...	✓	✓	39 (95 ± 7%)	
Four body sites <sup>d</sup>	✓	✓	✓	✓	...	41 (100%)
	✓	✓	✓	...	✓	39 (95 ± 7%)
	✓	✓	...	✓	✓	38 (93 ± 8%)
	✓	...	✓	✓	✓	41 (100%)
Five body sites	✓	✓	✓	✓	✓	41 (100%)

<sup>a</sup>Initial body site screening approaches used to establish carrier status are outlined in [Supplementary Table 1](#).

<sup>b</sup>A composite swab of bilateral axillae and inguinal regions is currently recommended by the Centers for Disease Control and Prevention for *C. auris* screening [27].

<sup>c</sup>Value of adding nares, palms/fingertips, and perianal skin together with bilateral axillae and inguinal crease swab cultures is shown. Additional combinations with >90% colonization detection shown.

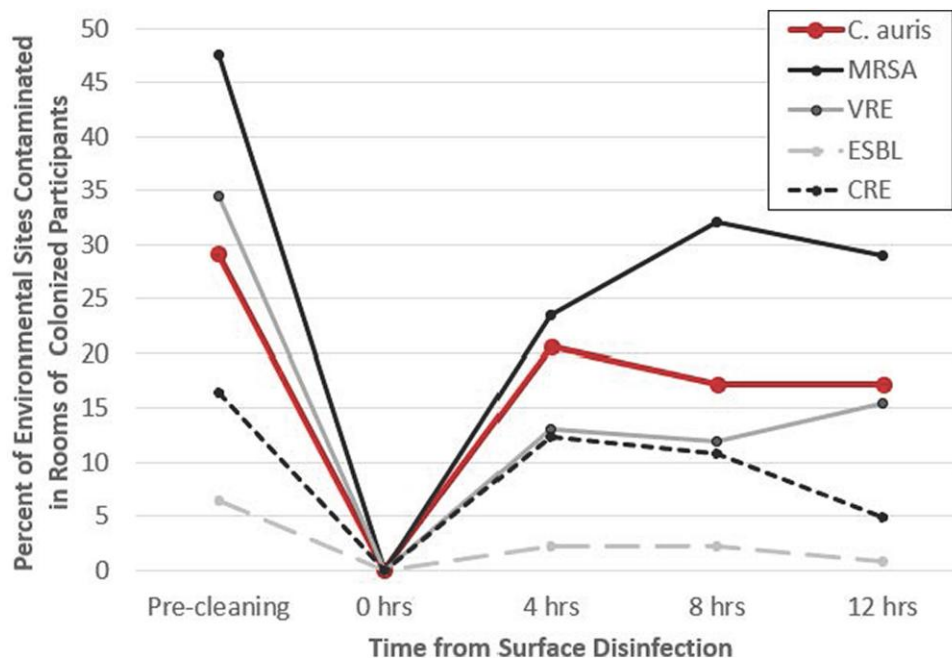
<sup>d</sup>Value of adding 2 additional body sites together with bilateral axillae and inguinal crease swab cultures is shown. Additional combinations with 100% colonization detection are also shown.

disinfection, suggesting that twice-daily or even 3 times-daily cleaning would not suffice to mitigate contamination that could lead to *C. auris* spread. Our prior work in SNFs demonstrated that cleaning fidelity was often poor, with <25% of high-touch surfaces adequately cleaned each day [30]. Practical limits to cleaning frequency could potentially be overcome with the use of long-acting disinfectants or antimicrobial surfaces. In the meantime, traditional infection prevention strategies, including assiduous hand hygiene and barrier precautions (eg, gowns, gloves), should be used for *C. auris* containment.

The positive correlation between *C. auris* environmental contamination and the number of colonized body sites provides another potential target for prevention. Notably, nearly all *C. auris* carriers were co-colonized with high-priority bacterial MDROs that also shed heavily into the nearby environment. In terms of the speed and magnitude of environmental contamination, *C. auris* behaved most similarly to MRSA. *C. auris* and MRSA share a propensity to colonize the skin and nares and to persist in the healthcare environment for prolonged

periods [1, 31]. Given these similarities, efforts to reduce shedding and spread by suppressing body-surface bioburden may be warranted. Although routine chlorhexidine bathing has been an effective component of MRSA prevention bundles [32], the effect of chlorhexidine skin antiseptics in controlling *C. auris* has not been proven [8, 14, 15]. However, we have previously demonstrated that chlorhexidine concentrations sufficient to reduce *C. auris* skin colonization [15] can be achieved with adequate attention to bathing technique [33]. Taken together, these findings highlight the need for novel solutions such as more broadly effective antiseptic bathing products to reduce both body-surface colonization and subsequent environmental contamination by multiple pathogens.

We found that *C. auris* carriers often had *C. auris* present on the hands and nares. These body sites are easily accessible and may be additive to or potentially replace currently recommended screening sites that target the axillae and inguinal creases. Addition of the nares yielded 12% additional capture and addition of palms/fingertips yielded 7% additional capture beyond



**Figure 3.** Time to environmental surface contamination with *Candida auris* and bacterial multidrug-resistant organisms (MDRO). Patterns of *C. auris* and MDRO environmental contamination by time point in relation to room disinfection are shown as composite of contaminated environmental surfaces at each time point. For each bacterial organism, surface contamination is evaluated among persons who were co-colonized with *C. auris* and that organism. There were 28 ESBL (68%), 25 VRE (61%), 21 MRSA (51%), and 17 CRE (41%) co-colonized participants identified. Abbreviations: CRE, carbapenem-resistant Enterobacterales; ESBL, extended-spectrum beta-lactamase-producing Enterobacterales; MRSA, methicillin-resistant *Staphylococcus aureus*; VRE, vancomycin-resistant Enterococcus.

an axillae/inguinal sample [27]. This supports prior work by our group [15] and others demonstrating the value of screening multiple body sites to detect *C. auris* colonization [5, 9, 16, 34, 35]. Beyond informing screening recommendations, high rates of colonization of the hands and nares reveal 2 additional targets for potential intervention; future work should evaluate the impact of enhanced hand hygiene and nasal decolonization for *C. auris* carriers. Because antiseptic bathing does not impact nasal carriage, we anticipate that nasal decolonization may be an important component for reducing *C. auris* carriage and preventing infection.

The finding of greater environmental surface contamination in acute care versus SNFs was unexpected. Confidence in the generalizability of this finding is limited because of the enrollment of only 1 ACH. In exploratory analyses, we observed that acute care participants were more likely to have higher medical complexity and to have recent exposure to systemic antimicrobial agents than were SNF participants. We hypothesize that the higher medical acuity and antibiotic exposure in the acute care participants may be associated with greater environmental surface contamination, possibly because of increased patient contact with medical staff (eg, higher care needs), with staff potentially acting as an intermediary for contamination of the patient environment. Exposure to antibiotics may also decrease the abundance of indigenous

commensal bacterial species on patients' skin and provide a favorable environment for increase in the abundance of *C. auris*.

This study has both strengths and limitations. Despite a small sample size of 41 patients, our conclusions regarding the speed and burden of environmental contamination are strengthened by frequent sampling of multiple environmental surfaces. Moreover, the 7 participating facilities span distinct geographic regions with historically different *C. auris* clade types (California: clade III; Illinois: clade IV [7, 36, 37]), expanding the generalizability of our findings. Furthermore, in contrast to prior studies, we evaluated *C. auris* colonization and environmental contamination under nonoutbreak conditions and in the context of bacterial MDRO co-colonization; our findings highlight the need for broad strategies that simultaneously address multiple pathogens. We did not monitor the activity within patient rooms or interactions with healthcare workers during each 12-hour study period; further work is needed to examine the impact of specific high-contact activities on *C. auris* environmental contamination. We also note that the effect of repeated surface sampling on the recovery of *C. auris* is not known and may have altered culture detection at later time points. However, this limitation does not obscure the primary findings that most contamination occurs within 4 hours after disinfection.



*C. auris* rapidly contaminates the healthcare environment, often together with multidrug-resistant bacterial pathogens, near colonized patients. The current findings extend our understanding of the impact of *C. auris* body colonization on environmental contamination [6, 12, 14, 15], which may serve as an intermediary for transmission. Importantly, our findings highlight the critical need to develop and implement broadly effective interventions that will impact multiple pathogens simultaneously. The speed of environmental contamination after surface disinfection highlights the challenges associated with managing environmental *C. auris* contamination. Use of long-acting disinfectants and/or antimicrobial surfaces, if available, may offer more durable and pragmatic solutions compared with high-frequency room cleaning. Additionally, strategies that have successfully reduced body-surface bioburden of bacterial MDROs deserve further investigation for their potential to reduce *C. auris*. In the meantime, traditional infection control and prevention measures, including environmental cleaning and disinfection in concert with hand hygiene and barrier precautions, should continue to be prioritized to mitigate spread of *C. auris* and other MDRO threats.

### Supplementary Data

Supplementary materials are available at *Clinical Infectious Diseases* online. Consisting of data provided by the authors to benefit the reader, the posted materials are not copyedited and are the sole responsibility of the authors, so questions or comments should be addressed to the corresponding author.

### Notes

**Acknowledgments.** The authors thank the participating facilities and patients for making this work possible.

**Financial support.** This work was supported by the Centers for Disease Control and Prevention Foundation (Project Number 1085.1-CDC Proj 047 to M. K. H. and S. S. H.) and the Centers for Disease Control and Prevention Epicenters Program (U54CK000607 to M. K. H.).

**Disclaimer.** The findings and conclusions of this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

**Potential conflicts of interest.** G. M. G., R. D. S., R. S., R. P., S. S. H., and M. K. H. have the following disclosures: Conducted studies where participating healthcare facilities received contributed antiseptic and cleaning products from Medline Industries, Inc., and Xttrium Laboratories. Companies had no role in the design, conduct, or analysis of these studies. The remaining authors have no conflicts to declare. K. B. reports other financial or nonfinancial interests: Conducted studies where participating hospitals/nursing homes received cleaning and antiseptic product from Medline Industries (payment to institution). G. M. G. also reports the following grants or contracts to institution: NIAID 5F31AI172386-02: Endemic and Emerging Multidrug Resistant Organisms in Nursing Homes: A Neglected Clinical Setting and 1P01AI172725: MDRO Carriage, Transmission, Sequelae, and Prevention in Nursing Homes. M. K. H. reports a position as unpaid volunteer position as President SHEA Board of Trustees and the following grants or contracts to institution: CDC BAA 75D301-19-67835 Evaluating emergence of resistance and changes in clinical pathogens following introduction of chlorhexidine bathing. S. S. H., R. S., J. S., and R. D. S. report grants or contracts to institution: 1P01AI172725: MDRO Carriage, Transmission, Sequelae, and Prevention in Nursing Homes. S. E. S. reports the following grants or contracts to institution: NIH—1R01AI175227-01 (Subaward from University

of Michigan) and Cohn Fellowship Intramural Career Development Grant (Rush University Medical Center); travel stipend to cover travel costs to present at IDWeek 2022 from Society for Healthcare Epidemiology of America. All other authors report no potential conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

### References

- Forsberg K, Woodworth K, Walters M, et al. *Candida auris*: the recent emergence of a multidrug-resistant fungal pathogen. *Med Mycol* **2019**; 57:1–12.
- Vallabhaneni S, Kallen A, Tsay S, et al. Investigation of the first seven reported cases of *Candida auris*, a globally emerging invasive, multidrug-resistant fungus—United States, May 2013–August 2016. *Am J Transplant* **2017**; 17:296–9.
- CDC. Antibiotic resistance threats in the United States. Atlanta, GA: U.S. Department of Health and Human Services, CDC, **2019**.
- Tsay S, Welsh RM, Adams EH, et al. Notes from the field: ongoing transmission of *Candida auris* in health care facilities—United States, June 2016–May 2017. *MMWR Morb Mortal Wkly Rep* **2017**; 66:514–5.
- Adams E, Quinn M, Tsay S, et al. *Candida auris* in healthcare facilities, New York, USA, 2013–2017. *Emerg Infect Dis* **2018**; 24:1816–24.
- Eyre DW, Sheppard AE, Madder H, et al. A *Candida auris* outbreak and its control in an intensive care setting. *N Engl J Med* **2018**; 379:1322–31.
- Chow NA, Gade L, Tsay SV, et al. Multiple introductions and subsequent transmission of multidrug-resistant *Candida auris* in the USA: a molecular epidemiological survey. *Lancet Infect Dis* **2018**; 18:1377–84.
- Schelenz S, Hagen F, Rhodes JL, et al. First hospital outbreak of the globally emerging *Candida auris* in a European hospital. *Antimicrob Resist Infect Control* **2016**; 5:35.
- Southwick K, Ostrowsky B, Greenko J, et al. A description of the first *Candida auris*-colonized individuals in New York State, 2016–2017. *Am J Infect Control* **2022**; 50:358–60.
- Ruiz-Gaitan A, Moret AM, Tasiias-Pitarch M, et al. An outbreak due to *Candida auris* with prolonged colonisation and candidaemia in a tertiary care European hospital. *Mycoses* **2018**; 61:498–505.
- Huang X, Hurabielle C, Drummond RA, et al. Murine model of colonization with fungal pathogen *Candida auris* to explore skin tropism, host risk factors and therapeutic strategies. *Cell Host Microbe* **2021**; 29:210–221.e6.
- Sexton DJ, Bentz ML, Welsh RM, et al. Positive correlation between *Candida auris* skin-colonization burden and environmental contamination at a ventilator-capable skilled nursing facility in Chicago. *Clin Infect Dis* **2021**; 73:1142–8.
- Welsh RM, Bentz ML, Shams A, et al. Survival, persistence, and isolation of the emerging multidrug-resistant pathogenic yeast *Candida auris* on a plastic health care surface. *J Clin Microbiol* **2017**; 55:2996–3005.
- Pacilli M, Kerins JL, Clegg WJ, et al. Regional emergence of *Candida auris* in Chicago and lessons learned from intensive follow-up at 1 ventilator-capable skilled nursing facility. *Clin Infect Dis* **2020**; 71:e718–e25.
- Proctor DM, Dangana T, Sexton DJ, et al. Integrated genomic, epidemiologic investigation of *Candida auris* skin colonization in a skilled nursing facility. *Nat Med* **2021**; 27:1401–9.
- Huang X, Welsh RM, Deming C, et al. Skin metagenomic sequence analysis of early *Candida auris* outbreaks in U.S. nursing homes. *mSphere* **2021**; 6:e0028721.
- Prestel C, Anderson E, Forsberg K, et al. *Candida auris* outbreak in a COVID-19 specialty care unit—Florida, July–August 2020. *MMWR Morb Mortal Wkly Rep* **2021**; 70:56–7.
- Lyman M, Forsberg K, Reuben J, et al. Notes from the field: transmission of pan-resistant and echinocandin-resistant *Candida auris* in health care facilities—Texas and the District of Columbia, January–April 2021. *MMWR Morb Mortal Wkly Rep* **2021**; 70:1022–3.
- de Almeida JN J, Francisco EC, Hagen F, et al. Emergence of *Candida auris* in Brazil in a COVID-19 intensive care unit. *J Fungi* **2021**; 7:220.
- Villanueva-Lozano H, Trevino-Rangel RJ, Gonzalez GM, et al. Outbreak of *Candida auris* infection in a COVID-19 hospital in Mexico. *Clin Microbiol Infect* **2021**; 27:813–6.
- Hanson BM, Dinh AQ, Tran TT, et al. *Candida auris* invasive infections during a COVID-19 case surge. *Antimicrob Agents Chemother* **2021**; 65:e0114621.
- Lyman M, Forsberg K, Sexton DJ, et al. Worsening spread of *Candida auris* in the United States, 2019 to 2021. *Ann Intern Med* **2023**; 176:489–95.
- Tanner WD, Leecaster MK, Zhang Y, et al. Environmental contamination of contact precaution and non-contact precaution patient rooms in six acute care facilities. *Clin Infect Dis* **2021**; 72(Suppl 1):S8–S16.
- McKinnell JA, Singh RD, Miller LG, et al. The SHIELD Orange County Project: multidrug-resistant organism prevalence in 21 nursing homes and long-term acute care facilities in southern California. *Clin Infect Dis* **2019**; 69:1566–73.

25. Trick WE, Lin MY, Cheng-Leidig R, et al. Electronic public health registry of extensively drug-resistant organisms, Illinois, USA. *Emerg Infect Dis* **2015**; 21:1725–32.
26. Conway JR, Lex A, Gehlenborg N. Upsetr: an R package for the visualization of intersecting sets and their properties. *Bioinformatics* **2017**; 33:2938–40.
27. Centers for Disease Control and Prevention. Screening for *Candida auris* colonization. Available at: <https://www.cdc.gov/fungal/candida-auris/c-auris-screening.html>. Accessed 24 August 2022.
28. Shams AM, Rose LJ, Edwards JR, et al. Assessment of the overall and multidrug-resistant organism bioburden on environmental surfaces in healthcare facilities. *Infect Control Hosp Epidemiol* **2016**; 37:1426–32.
29. Cadnum JL, Pearlmutter BS, Jencson AL, et al. Microbial bioburden of inpatient and outpatient areas beyond patient hospital rooms. *Infect Control Hosp Epidemiol* **2022**; 43:1017–21.
30. Murphy CR, Eells SJ, Quan V, et al. Methicillin-resistant *Staphylococcus aureus* burden in nursing homes associated with environmental contamination of common areas. *J Am Geriatr Soc* **2012**; 60:1012–8.
31. Du H, Bing J, Hu T, Ennis CL, Nobile CJ, Huang G. *Candida auris*: epidemiology, biology, antifungal resistance, and virulence. *PLoS Pathog* **2020**; 16:e1008921.
32. Popovich KJ, Aureden K, Ham DC, et al. SHEA/IDSA/APIC practice recommendation: strategies to prevent methicillin-resistant *Staphylococcus aureus* transmission and infection in acute-care hospitals: 2022 update. *Infect Control Hosp Epidemiol* **2023**; 44:1039–67.
33. Rhee Y, Hayden MK, Simms AT, et al. Impact of measurement and feedback on chlorhexidine gluconate bathing among intensive care unit patients: a multicenter study. *Infect Control Hosp Epidemiol* **2023**; 44:1375–80.
34. Biswal M, Rudramurthy SM, Jain N, et al. Controlling a possible outbreak of *Candida auris* infection: lessons learnt from multiple interventions. *J Hosp Infect* **2017**; 97:363–70.
35. Zhu Y, O'Brien B, Leach L, et al. Laboratory analysis of an outbreak of *Candida auris* in New York from 2016 to 2018: impact and lessons learned. *J Clin Microbiol* **2020**; 58:e01503-19.
36. Karmarkar EN, O'Donnell K, Prestel C, et al. Rapid assessment and containment of *Candida auris* transmission in postacute care settings-Orange County, California, 2019. *Ann Intern Med* **2021**; 174:1554–62.
37. Arensman K, Miller JL, Chiang A, et al. Clinical outcomes of patients treated for *Candida auris* infections in a multisite health system, Illinois, USA. *Emerg Infect Dis* **2020**; 26:876–80.