ORIGINAL ARTICLE

A *Candida auris* Outbreak and Its Control in an Intensive Care Setting

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N Engl J Med 2018;379:1322-31.
DOI: 10.1056/NEJMoa1714373
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ABSTRACT

BACKGROUND

Candida auris is an emerging and multidrug-resistant pathogen. Here we report the epidemiology of a hospital outbreak of *C. auris* colonization and infection.

METHODS

After identification of a cluster of *C. auris* infections in the neurosciences intensive care unit (ICU) of the Oxford University Hospitals, United Kingdom, we instituted an intensive patient and environmental screening program and package of interventions. Multivariable logistic regression was used to identify predictors of *C. auris* colonization and infection. Isolates from patients and from the environment were analyzed by whole-genome sequencing.

RESULTS

A total of 70 patients were identified as being colonized or infected with *C. auris* between February 2, 2015, and August 31, 2017; of these patients, 66 (94%) had been admitted to the neurosciences ICU before diagnosis. Invasive *C. auris* infections developed in 7 patients. When length of stay in the neurosciences ICU and patient vital signs and laboratory results were controlled for, the predictors of *C. auris* colonization or infection included the use of reusable skin-surface axillary temperature probes (multivariable odds ratio, 6.80; 95% confidence interval [CI], 2.96 to 15.63; P<0.001) and systemic fluconazole exposure (multivariable odds ratio, 10.34; 95% CI, 1.64 to 65.18; P=0.01). *C. auris* was rarely detected in the general environment. However, it was detected in isolates from reusable equipment, including multiple axillary skin-surface temperature probes. 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. The sequenced isolates from reusable equipment were genetically related to isolates from the patients.

CONCLUSIONS

The transmission of *C. auris* in this hospital outbreak was found to be linked to reusable axillary temperature probes, indicating that this emerging pathogen can persist in the environment and be transmitted in health care settings. (Funded by the National Institute for Health Research Health Protection Research Unit in Healthcare Associated Infections and Antimicrobial Resistance at Oxford University and others.)

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Candida auris is an emerging, multidrug-resistant pathogen that has recently been associated with outbreaks worldwide, often in intensive care units (ICUs).[1,2] It was described in 2009 after isolation from the ear canal of a Japanese patient[3] and was reported as a cause of bloodstream infection in South Korea in 2011.[4] Whole-genome sequencing has revealed genetically distinct clades of the fungus on different continents and subcontinents, including East Asia, South Asia, South America, and southern Africa.[5] Reidentification of four *C. auris* isolates from an international collection of 15,271 invasive candida isolates collected from 2004 onward supports the emergence of *C. auris* as a relatively recent clinical problem.[5] This paradox of a relatively old species with phylogenetically distinct geographic clades simultaneously causing newly recognized disease among patients worldwide is incompletely understood. It may relate to changes in the natural environmental niche of the organism, changing antifungal prophylaxis and treatment, changing approaches to diagnosis and to the identification of species, or changes in health care environments.

In Europe, *C. auris* has been identified in the United Kingdom,[6] Spain,[7] Norway,[8] and Germany.[9] A large outbreak involved 72 patients between April 2015 and November 2016 and was centered around a cardiothoracic ICU in London.[10,11]

After alerts in June 2016 in the United States[12] and the United Kingdom,[13] a look-back exercise at Oxford University Hospitals NHS Foundation Trust identified 4 patients who were colonized and 5 patients who were infected with *C. auris* between February 2, 2015, and October 16, 2016; of these 9 patients, 8 had been in the neurosciences ICU before diagnosis. All the *C. auris* isolates were identified prospectively. A patient and environmental screening program was introduced on October 24, 2016.

Here, we report the epidemiology of *C. auris* in this setting. We describe the risk factors for colonization, colonization duration, rates of invasive infection, the molecular epidemiology of the outbreak, and the infection-control measures that were undertaken.

METHODS

SETTING

Oxford University Hospitals NHS Foundation Trust consists of four teaching hospitals (1225 beds) and provides secondary and tertiary care to Oxfordshire, United Kingdom (population, approximately 600,000), and the surrounding region. Our neurosciences ICU has 16 beds, 13 in an open-plan configuration plus three side rooms with 1 bed each, with approximately 650 admissions per year. We studied the patients who were colonized or infected with *C. auris* between February 2, 2015, and August 31, 2017, after a look-back exercise in which records from January 1, 2012, onward were reviewed (see the Supplementary Appendix, available with the full text of this article at NEJM.org).

ETHICS

Patient and environmental screening was undertaken as part of routine infection control in the hospital. Ethics approval was not required for the sequencing of isolates. Deidentified electronic health records were analyzed with approval from the Oxfordshire Research Ethics Committee and the national Confidentiality Advisory Group.

PATIENT AND ENVIRONMENTAL SCREENING

Routine *C. auris* screening began on October 24, 2016. Patients underwent screening on admission to the neurosciences ICU, weekly, and on discharge. Screening involved culture of swab specimens from the nose, axilla, groin, tracheostomy (if present), and wounds, as well as urine culture. The frequency of screening was amended to three times per week for axilla and groin swabs from January 1, 2017, to July 5, 2017, to detect colonization earlier, with a complete screen performed on admission and discharge. Weekly screening of patients was also conducted in the adjacent neurosciences ward. Patients continued to undergo screening in the same way after a swab specimen was found to be positive. Isolates were identified with the use of matrix-assisted laser desorption ionization–time of flight (MALDI-TOF), and antifungal susceptibility testing was undertaken by broth microdilution (see the Supplementary Appendix). Various methods for environmental screening were used, focusing on sampling of high-touch areas and reusable devices (see the Supplementary Appendix).

DEFINITIONS

We conducted a case–control study to identify risk factors for *C. auris* colonization and infection in patients admitted to the neurosciences ICU. The case patients were any patients who had been ad-

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mitted to the neurosciences ICU before *C. auris* colonization or infection (i.e., a *C. auris*–positive screen or clinical isolate). Controls were patients who had never been colonized or infected with *C. auris* and who had been admitted to the neurosciences ICU before they had one or more negative *C. auris* screening results — that is, patients who had been at risk in the neurosciences ICU but did not become colonized or infected.

WHOLE-GENOME SEQUENCING

We sequenced available first and last *C. auris* isolates obtained from each patient and the environmental isolates stored by our microbiology laboratory. Samples from multiple time points, as well as multiple samples from the same time point, were sequenced in a random subgroup of patients. Six to 12 colonies from primary culture plates were subcultured together before DNA extraction for sequencing. Whole-genome sequencing was performed with the use of Illumina MiSeq, with reads mapped to an outbreak-specific reference sequence generated by long-read sequencing of one isolate with the Oxford Nanopore MinION. Sequences were compared with the use of single-nucleotide polymorphisms (SNPs), maximum-likelihood phylogenies, and Bayesian time-scaled phylogenies (see the Supplementary Appendix).[14] Because sequences were generated by pooling 6 to 12 fungal colonies, we used a previously described probabilistic method[15] to determine when sequence mixtures were present.

STATISTICAL ANALYSIS

Factors that were associated with the first *C. auris* colonization or infection per patient were identified with the use of multivariable logistic regression with backward elimination (exit $P>0.1$), allowing for nonlinear effects of continuous factors and interactions (see the Supplementary Appendix). Potential risk factors were assessed in the 90 days before the first *C. auris* isolate in case patients and the last negative screening result in controls.

RESULTS

CASE PATIENTS

A total of 70 patients were identified as being colonized or infected with *C. auris* between February 2, 2015, and August 31, 2017 (Fig. 1A); 66 patients (94%) had been admitted to the neurosciences ICU before the diagnosis, with a median stay of 8.4 days in the ICU before diagnosis (interquartile range, 4.6 to 13.4). Three other patients had been admitted to the adjacent neurosciences ward before diagnosis. The final patient had had no exposure to the neurosciences ward or ICU; the diagnosis in this patient was made in 2015, which predated most cases.

Invasive *C. auris* infections developed in 7 patients: 4 had candidemia, and 3 had central nervous system device-associated meningitis (1 with candidemia); an orthopedic-device infection was found in the patient without exposure to the neurosciences ward or ICU. Five infections occurred before patient screening started. There were no invasive infections noted after November 2016. One patient with an invasive infection died 229 days after the collection of the last invasive isolate from that patient, with subsequent sterile blood and cerebrospinal fluid cultures; the death was therefore judged to not be attributable to *C. auris* infection.

PATIENT SCREENING

Culture was performed on 9153 screening swabs obtained from patients, representing 2872 unique patient-days of screening among 900 patients (Fig. 1B). Of the 2872 screening swabs from a given day and patient, 267 (9.3%) yielded one or more *C. auris* isolates, in 62 unique patients (8 case patients who were colonized or infected were identified before the screening period from clinical samples). The acquisition rate during the screening period was 2.9 cases per 100 neurosciences ICU inpatient–days at risk.

One or more negative *C. auris* screening results without any positive results were found in 838 patients; 363 of these patients had been admitted to the neurosciences ICU before their last negative screening result. The electronic patient records for 2 patients were incomplete; these patients were excluded from the analysis, which left 361 controls for the determination of risk factors for *C. auris* acquisition in the neurosciences ICU.

RISK FACTORS FOR COLONIZATION OR INFECTION

The median age was 52 years (interquartile range, 42 to 64) among the 66 case patients and 56 years (interquartile range, 44 to 67) among the 361 controls; 44 case patients (67%) and 188 controls (52%) were male. The most common primary diagnoses among both case patients and controls were trauma and intracranial bleeding (Table S1 in the Supplementary Appendix).

In multivariable models, the risk of colonization

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or infection initially increased with length of stay in the neurosciences ICU before declining again among patients with longer stays (P=0.001) (Table 1, and Fig. S1 in the Supplementary Appendix). Similarly, the risk of colonization was greatest in association with high-normal to moderately elevated neutrophil counts (P=0.01) (Fig. S2 in the Supplementary Appendix). The risk of colonization or infection was also associated with any skin-surface axillary temperature monitoring with the use of reusable probes (odds ratio, 6.80; 95% confidence interval [CI], 2.96 to 15.63; P<0.001). These temperature probes were used in 57 case patients (86%) and 122 controls (34%). There was some evidence that the risk of colonization or infection was higher among patients with lower serum albumin levels (P=0.06), a higher body temperature (P=0.08), and higher serum sodium levels (P=0.07). Systemic fluconazole treatment was also associated with an increased risk (odds ratio, 10.3; 95% CI, 1.64 to 65.2; P=0.01), although only 3 case patients (5%) received antifungal agents before colonization or infection.

ENVIRONMENTAL SCREENING AND INFECTION-CONTROL RESPONSE

A total of 128 environmental samples were obtained in November 2016, February 2017, and April 2017. *C. auris* was rarely detected in the general environment or air (one settle plate was found to be positive). However, the organism was detected from reusable patient-monitoring equipment (axillary temperature probes and a pulse oximeter) and a patient hoist (Table S2 in the Supplementary Appendix). All skin-surface temperature probes (Fig. S3 in the Supplementary Appendix) were withdrawn from use on April 11, 2017. However, they came back into use during the annual leave of a senior nurse, and acquisitions of infection and colonization continued (Fig. 1). All probes were comprehensively withdrawn from the neurosciences ICU on April 24, 2017, and cultured — five that had been in recent use and five that had been in storage. *C. auris* was isolated from four probes that are presumed to be those that had recently been in use. No other candida species were isolated from any probe. After the removal of the temperature probes, four additional cases were identified up to the end of the study (August 31, 2017), the last on July 17, 2017. Additional measures for the prevention and control of infection were implemented (Table S3 in the Supplementary Appendix).

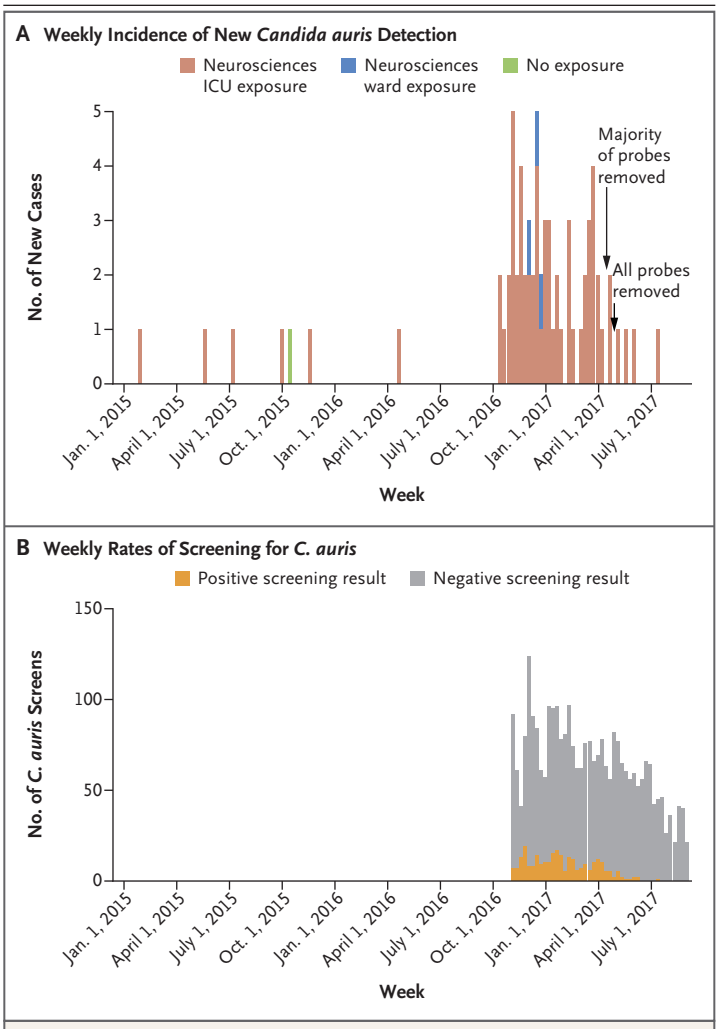


Figure 1. Detection of *Candida auris* and Rates of Screening.

In Panel A, red indicates patients who had had exposure to the neurosciences intensive care unit (ICU) before diagnosis of *C. auris* infection or colonization, blue indicates patients who had had exposure to the neurosciences ward but not to the neurosciences ICU, and green indicates patients who had had exposure to neither unit. The timing of the removal of reusable temperature probes is shown. The data in Panel B are deduplicated to unique patient screening days — that is, in instances in which multiple swabs were obtained from a single patient on the same day, this is represented as a single data point, shown as positive if any of the swabs were positive. A full list of infection-control interventions is provided in Table S3 in the Supplementary Appendix.

ANTIFUNGAL SUSCEPTIBILITY TESTING

The first isolate from each patient and all the invasive isolates underwent antifungal susceptibility testing; 79 of 79 (100%), 78 of 80 (98%), and 66 of 73 (90%) isolates were resistant to fluconazole, voriconazole, and posaconazole, respectively, on the basis of breakpoints established for *C. albicans* (Table S4 in the Supplementary Appendix). A total of 14 of 79 isolates (18%) were ampho-

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Table 1. Multivariable Predictors of *Candida auris* Colonization.*

Variable	Controls (N=361)	Case Patients (N=66)	Univariable Analysis		Multivariable Analysis	
			Odds Ratio (95% CI)	P Value	Odds Ratio (95% CI)	P Value
Median ICU stay before diagnosis (IQR) — days†	1.8 (0.7–6.6)	8.4 (4.6–13.4)				
Length of ICU stay before diagnosis				<0.001		0.001
1 day			Reference		Reference	
3 days			3.89 (2.38–6.36)		2.24 (1.30–3.86)	
5 days			7.37 (3.65–14.89)		2.97 (1.35–6.53)	
10 days			12.68 (5.38–29.88)		2.78 (1.02–7.54)	
20 days			6.75 (2.78–16.40)		0.69 (0.22–2.19)	
Axillary temperature monitoring — no. (%)	122 (34)	57 (86)	12.41 (5.94–25.90)	<0.001	6.80 (2.96–15.63)	<0.001
Median blood sodium level (IQR) — mmol/liter	139.3 (137.1–141.1)	141.4 (138.5–143.6)	1.20 (1.09–1.31)	<0.001	1.10 (0.99–1.22)	0.07
Median neutrophil count (IQR) — cells/mm^3†	8600 (6600–10,900)	9600 (7300–10,900)				
Neutrophil count				0.003		0.01
4000 cells/mm^3			Reference		Reference	
7000 cells/mm^3			2.18 (1.40–3.41)		2.21 (1.30–3.76)	
10,000 cells/mm^3			4.41 (1.84–10.59)		4.72 (1.64–13.59)	
15,000 cells/mm^3			1.17 (0.37–3.71)		1.69 (0.45–6.42)	
Median body temperature (IQR) — °C	36.5 (36.3–36.9)	36.9 (36.6–37.3)	2.44 (1.78–3.35)‡	<0.001	1.43 (0.96–2.14)‡	0.08
Any antifungal treatment — no. (%)§	3 (1)	3 (5)	5.68 (1.12–28.79)	0.04	10.34 (1.64–65.18)	0.01

* Complete data were available for all factors shown for all 66 case patients and 361 controls. Only factors in the multivariable model are shown; more complete information about the characteristics of case patients and controls, as well as the univariable odds ratios for all factors, including sex, age, primary diagnosis, emergency admission status, invasive ventilation, central venous access, albumin level, potassium level, creatinine level, hemoglobin level, heart rate, respiratory rate, blood pressure, and use of broad-spectrum antibiotics, are provided in Table S1 in the Supplementary Appendix. CI denotes confidence interval, ICU intensive care unit, and IQR interquartile range.

† There was a nonlinear relationship between risk of colonization and duration of stay in the neurosciences ICU and between risk of colonization and neutrophil count (details are provided in Table S1 in the Supplementary Appendix).

‡ The odds ratio per 0.5°C increase in body temperature is shown.

§ All the patients who had received treatment with an antifungal agent received fluconazole only.

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tericin-resistant. No micafungin or flucytosine resistance was identified.

SURVIVAL AND CARRIAGE DURATION

The crude mortality rate was similar among case patients and controls. Among patients for whom data on 30-day vital status were ascertainable at the end of the study, 30-day mortality was 17% (11 of 66) among case patients and 16% (52 of 331) among controls (P=0.85 by Fisher's exact test). Among those for whom 90-day vital status was ascertainable, 90-day mortality was 20% (13 of 64) and 20% (44 of 221), respectively (P=1.00).

A total of 60 case patients (58 colonized and 2 infected) had screening samples sent on at least 1 day, with a median number of distinct screening days per patient of 7 (interquartile range, 4 to 13; range, 1 to 30) (Fig. S4 in the Supplementary Appendix). The axilla was often colonized first: among case patients, the first positive screening result was from the axilla in 22 of 60 patients (37%), from another site (groin or urine) in 21 of 60 (35%), and from both the axilla and one or more other sites in 17 of 60 (28%); on subsequent screening days, these sites were positive in 34 of 207 (16%), 66 of 207 (32%), and 107 of 207 (52%), respectively (P<0.001).

To estimate the sensitivity of a single screen, we considered patients who underwent screening twice within 2 days, assuming that loss of colonization was minimal within this time window: 62 of 79 screening samples (78%) obtained 1 to 2 days after a positive screen were positive. Because a single screen was imperfectly sensitive, we defined clearance of colonization as either two or three consecutive negative screening results, treating death while colonized as a competing risk. The median duration of carriage among patients remaining alive was 61 days (interquartile range, 33 to not estimable) when two consecutive negative screening results were used to define clearance of colonization and was 82 days (interquartile range, 37 to not estimable), when three consecutive negative results were used (Fig. 2A, and Figs. S5 and S6 in the Supplementary Appendix). After a positive screening result, 175 of 234 next screening results (75%) were positive; after one, two, and three negative screening results, 23 of 49 (47%), 7 of 21 (33%), and 1 of 12 (8%) next screening results, respectively, were positive (Fig. 2B, and Table S5 in the Supplementary Appendix).

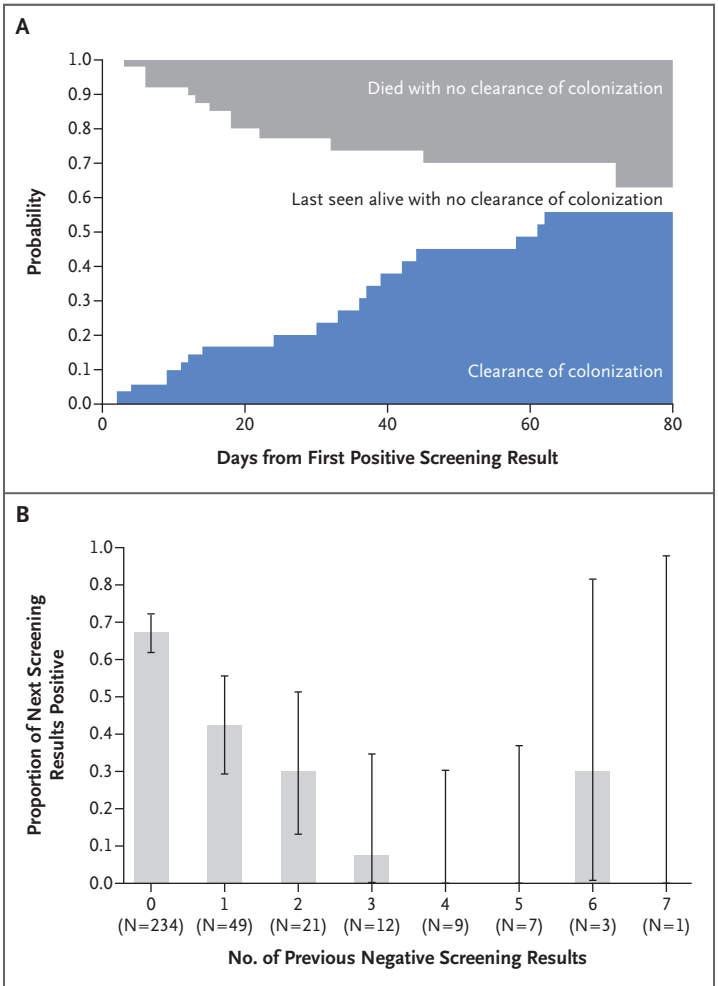


Figure 2. Duration of *C. auris* Colonization.

Panel A shows the proportion of patients with clearance of *C. auris* colonization according to the number of days since their first positive screening result, with death without clearance treated as a competing risk. Because any one screen was imperfectly sensitive, clearance of colonization was defined as two consecutive negative screening results, timed from the day of the first negative screening result. Of the 21 patients whose colonization was cleared, 7 had a subsequent relapse (details, including whole-genome sequence comparisons, are provided in Table S5 in the Supplementary Appendix). A graph constructed under an alternative definition of three consecutive negative screening results is provided in Figure S5 in the Supplementary Appendix; of the 11 patients whose colonization was cleared according to this definition, 2 had a relapse. Panel B shows the proportion of next screening results found to be positive according to the number of consecutive previous negative screening results in the same patient. I bars indicate the 95% confidence intervals calculated from exact binomial distributions.

SEQUENCE ANALYSIS

A total of 78 isolates were available for whole-genome sequencing: 72 screening or invasive isolates from 37 patients, plus 6 environmental iso-

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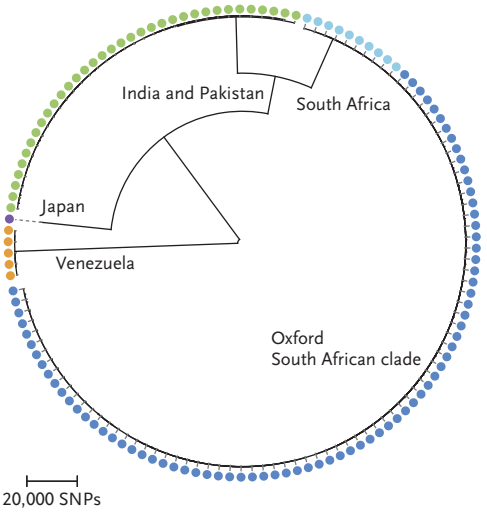


Figure 3. Maximum-Likelihood Phylogeny Comparing Outbreak Sequences with a Previously Sequenced Global Collection.

Shown are 78 outbreak sequences as compared with previously sequenced strains from Lockhart et al.,[5] plus four additional Indian isolates.[16] SNP denotes single-nucleotide polymorphism.

lates from five temperature probes and one hoist. All sequences fell within the South African *C. auris* clade (Fig. 3). The rate of *C. auris* evolution was 5.75 mutations per genome per year (95% highest posterior density interval, 4.49 to 7.11) (Fig. S7 in the Supplementary Appendix). The sequences formed a single subclade, estimated to have emerged in April 2013 (95% highest posterior density interval, August 2012 to December 2013).

We identified 40 unique sequences that differed from another sequence by at least 1 SNP. When a probabilistic method was used to identify isolates containing mixtures of these unique sequences, we found that 52 of 78 samples (67%) had no evidence of mixed colonization or infection within the 6 to 12 colonies sequenced, and 26 of 78 (33%) contained 2 of the unique sequences; 7 mixed sequences differed by 5 or fewer SNPs, 7 by 6 to 14 SNPs, and 12 by 30 or more SNPs (Table S6 in the Supplementary Appendix). Allowing for the mixed colonizations or infections, 104 sequences were identified in the 78 patient or environmental samples (Fig. 4, and Fig. S8 in the Supplementary Appendix). Three temperature-probe samples were also found to have mixed colonization.

Sequences from isolates obtained from reusable patient equipment were found throughout the phylogenetic tree of sequenced patient isolates (Fig. 4), including close matches between patient and temperature-probe samples; for example, Patients 24 and 32 had mixed colonization similar to that found on Temperature Probes 1 and 2. Conversely, transmission between patients in nearby beds could not explain the transmission pattern (Fig. 4). There was no evidence that patients with closely genetically related sequences were likely to be in nearby beds (P=0.34 for trend) (Fig. S9 in the Supplementary Appendix).

We investigated whether mixed colonization was likely to have resulted from simultaneous acquisition of multiple strains or from serial acquisitions of strains over time. Considering sequences that differed from each other by more than 5 SNPs as distinct genotypes, we found that 8 of 37 patients (22%) had mixed colonization or infection at their first positive screening result, as compared with 9 of 35 (26%) at any subsequent time point. There was no evidence that samples that were obtained closer in time to the first positive sample were more likely to be mixed than those obtained later (P=0.62 by rank-sum test).

DISCUSSION

We report an outbreak of *C. auris* colonization and infection in our neurosciences ICU. The most compelling explanation for the sustained *C. auris* transmission that we observed was persistence of the organism on reusable equipment — in particular, on skin-surface axillary temperature probes. Current recommended infection-control procedures for *C. auris* outbreaks include patient contact isolation and enhanced cleaning with chlorine-based products.[8,13] In addition, we implemented "decluttering" to facilitate cleaning, reduced bedside equipment, and removed fans and forced-air convection blankets. Despite these intensive measures, the outbreak was prolonged.

In our neurosciences ICU, continuous temperature monitoring with a skin-surface temperature probe in the axilla was part of routine care for patients who received mechanical ventilation and those undergoing temperature monitoring for neuroprotective management. These reusable probes were cleaned between patients with the use of wipes containing quaternary ammonium compound, which was the accepted custom and practice but differed from the manufacturer's instructions for use (see the Supplementary Appendix).

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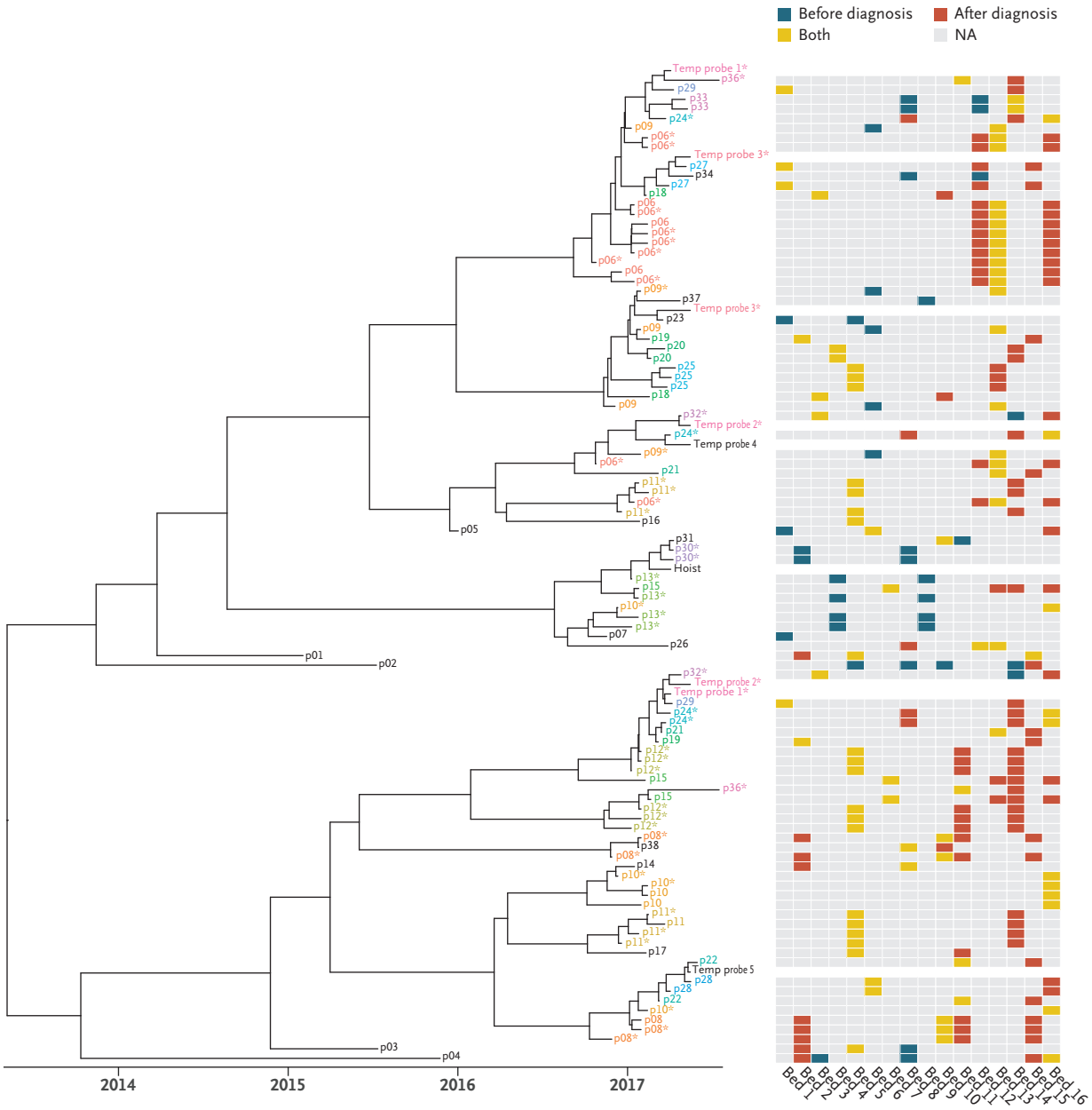


Figure 4. Time-Scaled Bayesian Phylogeny of 104 Unique Outbreak Sequences Obtained from 78 Isolates.

Samples from patients are labeled with a p, and environmental samples are labeled with their source (temperature [temp] probe or hoist). Samples with an asterisk (*) denote mixed infections detected within a single isolate pool obtained from 6 to 12 colonies. The locations of patients' beds before and after the date of each patient's first positive sample are shown on the right. Within the neurosciences ICU, beds are arranged in a circular layout, such that bed 1 is adjacent to beds 16 and 2, bed 2 is adjacent to beds 1 and 3, and so on. Beds 1 through 13 are in an open-plan configuration, and beds 14 through 16 are in separate side rooms. NA denotes not applicable.

The temperature probes are difficult to clean with wipes, with a two-layer rubber sheath protecting the distal end of the wire adjacent to the sensor (Fig. S3 in the Supplementary Appendix). In addition, a recent study has shown that quaternary ammonium compounds have relatively poor activity against all candida species.[17]

Several lines of evidence support the role of these temperature probes in the transmission of *C. auris*. Controlling for length of stay in the neu-

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rosciences ICU, patient vital signs and laboratory results, and previous use of antifungal agents, the use of axillary temperature probes increased the odds of *C. auris* colonization or infection by a factor of 6.80; these temperature probes were used in 86% of cases. Although previous treatment with fluconazole was also a strong risk factor, only 5% of case patients had been exposed. Antifungal agents have previously been reported to increase the risk of invasive *C. auris* infection.[18] In a finding that supported the role of axillary temperature probes in transmission, when a diagnosis was first made, patients were more likely to be initially colonized in the axilla or at all screened sites than they were to have isolated colonization in the groin or urine. In addition, *C. auris* was cultured from several temperature probes but was not widely found in the general environment or air. Whole-genome sequencing placed temperature-probe isolates throughout the phylogenetic tree of isolates from the outbreak, which suggested widespread mixing of isolates from probes and patients. The spatial proximity of beds in the ICU could not explain the pattern of transmission observed on whole-genome sequencing. Finally, removal of the temperature probes from use at least partially controlled the outbreak. However, even after this intervention was implemented, *C. auris* was not completely eliminated, and cases continued to be diagnosed, albeit at a lower rate. This probably in part reflects the survival of this organism in the hospital environment, particularly on plastic[19] and moist surfaces.[20] Of note, we did not identify any other candida species from our temperature-probe cultures.

This outbreak of *C. auris* colonization and infection probably arose from a single introduction of the South African *C. auris* clade into Oxford around mid-2013 from outside the United Kingdom (Fig. S7 in the Supplementary Appendix). On the basis of Public Health England surveillance data, no other U.K. hospital had reported similar isolates before this outbreak.

Antifungal susceptibility testing revealed resistance to fluconazole, voriconazole, and posaconazole; resistance to amphotericin was also found in 18% of cases. The emergence of *C. auris* in our neurosciences ICU resulted in an increase in invasive infections due to candida species overall, from approximately 1 per year from 2010 through 2014 to 7 in 18 months (February 2015 through July 2016). However, invasive infections did not develop in most colonized patients. In addition, there were no deaths directly attributable to *C. auris* infection, and there was no excess 30-day or 90-day crude mortality associated with colonization. There were no invasive infections after November 2016; this finding may be related to the introduction of single-dose micafungin prophylaxis for surgical procedures in colonized patients (Table S3 in the Supplementary Appendix).

On the basis of the analysis of repeated screening samples obtained within a period of no more than 2 days, we estimate the sensitivity of a single *C. auris* screen to have been 78%. No specific attempts were made to decolonize patients; however, all the patients were routinely bathed with 2% chlorhexidine washcloths. Given this approach and the definition of loss of colonization as two or three consecutive negative screening results, the median duration of colonization was approximately 2 to 3 months, with only a small proportion of patients testing positive after three negative screening results. Because only two patients had invasive infection during the serial screening period, we could not determine whether their colonization duration differed from those of other patients. Persistence of skin colonization lasting 1 to 3 months and environmental contamination lasting up to 3 months was also described in a report of seven cases from the United States.[21]

Our study is limited by the fact that we did not store and sequence all the strains involved in the outbreak; 37 of 70 patients (53%) had at least one sequence obtained, since the significance of this organism only became apparent over time. In addition, for pragmatic reasons, our screening strategy changed during the study, which may have altered ascertainment. Our study was not sufficiently large to determine risk factors for invasive infection (of which there were seven cases) in colonized patients.

In conclusion, on the basis of our investigation of an outbreak of *C. auris* infection in a neurosciences ICU, survival in the environment appeared to facilitate the persistence and transmission of this organism. Our results indicate that reusable patient equipment may serve as a source of health care–associated outbreaks of infection with *C. auris*.

The views expressed in this article are those of the authors and not necessarily those of the National Health Service, the National Institute for Health Research (NIHR), the Department of Health, or Public Health England.

Supported by the NIHR Health Protection Research Unit (HPRU) in Healthcare Associated Infections and Antimicrobial

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Resistance at Oxford University, in partnership with Public Health England, and by the NIHR Biomedical Research Centre Oxford. Dr. Eyre is an NIHR Clinical Lecturer and Robertson Foundation Fellow, and Drs. Peto and Crook are NIHR Senior Investigators.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

We thank the members of the independent steering committee of the NIHR HPRU: John Coia, Neil French, Charis Marwick, and Mike Sharland.

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