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MEDICAL MYCOLOGY TRAINING NETWORK

Outbreak of superbug *Candida auris*: Asian scenario and interventions

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Outbreak of superbug *Candida auris*: Asian scenario and intervention required by laboratories

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2009-2017 ....

Intensive care unit closed after new deadly superbug emerges in the UK

BMJ 2016;355:i5978 doi: 10.1136/bmj.i5978 (Published 7 November 2016)

Hospital transmitted *Candida auris* infections confirmed in the US

Michael McCarthy

Candida auris: a cause for concern?

Fungus with "super bug" qualities found in 44 cases in New York State

By Dr. Jordan Guerrein

How to Protect Yourself From the Candida Auris Fungal Infection

Cases of this rare yeast superbug are on the rise

By Hollie Levine

C. auris: Why reason of concern?

MDR clonal strains that are nosocomially transmitted

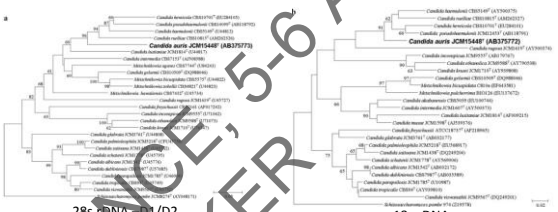
When it appeared? Where? How it spread?

Candida auris – first appeared

ORIGINAL ARTICLE *Microbiol Immunol* 2009; 53: 41–44

Candida auris sp. nov., a novel ascomycetous yeast isolated from the external ear canal of an inpatient in a Japanese hospital

Kazuo Satoh[1,2], Koichi Makimura[1,3], Yayoi Hasumi[1], Yayoi Nishiyama[1], Katsuhisa Uchida[1] and Hideyo Yamaguchi[1]

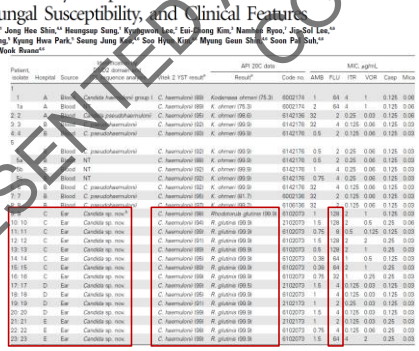


28s rDNA –D1/D2 18s rDNA

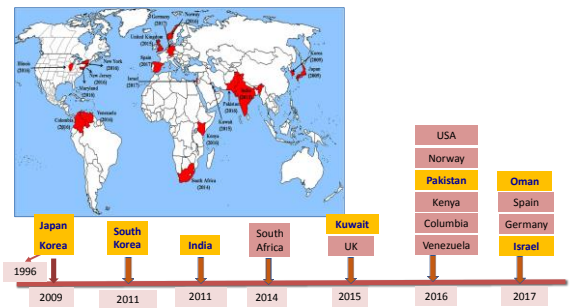
Clinical Infectious Diseases 2009; 48:e57–61

Candida haemulonii and Closely Related Species at 5 University Hospitals in Korea: Identification, Antifungal Susceptibility, and Clinical Features

Mi-Na Kim,[1] Jong Hee Shin,[1,a] Heungsup Sung,[1] Kyungwon Lee,[1] Eui-Chong Kim,[1] Namhee Ryoo,[1] Jin-Sol Lee,[1,a] Seok-Ho Jung,[2] Kyung Hwa Park,[3] Seung Jung Kee,[3,a] Soo Hyun Kim,[4] Myung Geun Shin,[4] Soon Pyo Suh,[4] and Dong Wook Ryang[4,a]



Emergence of *C. auris* strains in 5 continents



Chakrabarti A, *et al.* Intensive Care Med 2015; 41: 285

Chowdhary A, *et al.* J Hosp Infect 2016; 94: 209

Challenges !!

- ?True burden
  - Emerging reports
  - Exact burden unclear
- outbreaks in healthcare settings
  - Nosocomial spread
  - Environmental reservoir unknown
  - *C. haemulonii, C. famata, C. sake, S. cerevisiae, R. glutinis, C. lusitaniae, C.guilliermondii, C. parapsilosis*
- Identification
  - Misidentified by Vitek 2, API20C-AUX, Auxacolor, Phoenix and Microscan
  - T2 Candida panel may miss its identification
  - Accurate: Molecular sequencing (ITS or LSU region) or MALDI-TOF
- Multidrug resistance
  - ?epidemiological cutoff values (ECVs) or clinical breakpoints not defined
  - High rate of resistance to antifungals
- High mortality rate
  - Persistent candidemia
  - 33-72%

Burden, outbreaks, epidemiology

Prevalence *C. auris* candidemia

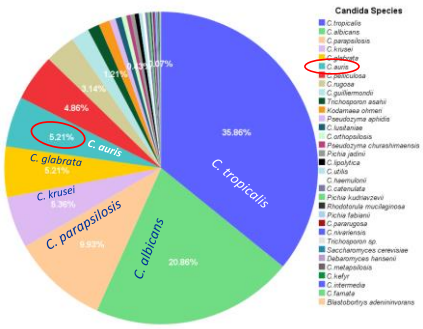
- 0.3% in South Africa (numerator & denominator not known) (Magobo *et al.* Emerg Infect Dis 2014; 20: 1250)
- 38% of Hospital-acquired candidemia in Kenya (Okinda *et al*, ECCMID: May 2014: Barcelona, Spain, Poster)
- 5% (3/60 paediatric), 30% (9/27 adult candidemia) in India (Chowdhary *et al.* Emerg Infect Dis 2013; 19: 1670)

➡ small numbers of cases in undefined populations

- 5.3% (74/1400 candidemia cases) in Indian ICUs (5th cause of candidemia in Indian ICUs) (Chakrabarti A, *et al.* Intensive Care Med 2015; 41: 285)

➡ Largest number of cases

Candida species isolated during Indian ICU study



Chakrabarti *et al.* Intensive Care Med 2015; 41: 285

Unique features from largest series: Indian ICUs

- 70.3% adults (median age 39 years)
- Higher in public-sector hospitals (62.2% vs 37.8%; P<0.001)
- Duration of ICU stay prior to candidaemia diagnosis significantly longer (median 25 days vs 15 days, P<0.001)
- High prior antifungal exposure (fluconazole in majority)
- Presence of a central venous line not significantly associated
- Duration of central line in days significantly higher (median 10.5, IQR 5–27 days)
- 30 day crude & attributable mortality of 41.9% & 27% respectively

Rudramurthy S, *et al.* J Antimicrob Chemother 2017; 72: 1794

Unique features from largest series: Indian ICUs

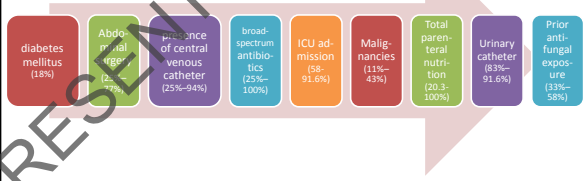
- Significant risk factors in Indian ICUs
  - public-sector hospital (P<0.006)
  - underlying respiratory illness (P<0.002)
  - vascular surgery (P<0.048)
  - prior antifungal exposure (P<0.001)
  - multiple interventions (P<0.007)

Patients with sepsis, undergoing invasive management for longer periods & exposed to antifungal agents

Investigate for *C. auris* candidemia

Rudramurthy S, *et al.* J Antimicrob Chemother 2017; 72: 1794

Risk factors in other studies



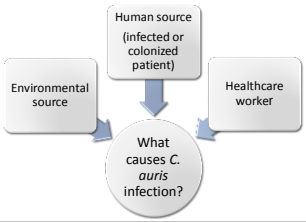
J Hosp Infect. 2016 Nov;94(3):209-212
Southern African Journal of Infectious Diseases 2016; 31(3):69–70

C. auris infection – other than blood-stream

- First isolation of *C. auris* was from the external ear canal of a 70-year-old woman in Japan (Microbiol Immunol 2009; 53: 41)
- Chronic otitis media - in Korea (2004-2006) reported 15 cases (Clin Infect Dis 2009; 48: e57)
- Vulvovaginitis -a young woman in India (J Infect Dev Countries 2015; 9: 435)
- Fatal pericarditis in an Indian patient with end stage liver disease (JMM Case Rep doi:.10.1099/jmmcr.0.T00018)
- In London outbreak – isolated from sternal wound (Antimicob Res Infect Control 2016; 5: 35)
- UTI & lung infections - ?

Why call it nosocomial spread?

- 0.04% (1/2246 patients screened at admission) in the UK (Antimicrob Resis Infect Control 2016: 5: 35)
- Persistent colonization of *C. auris* multiple body-sites of patients, carriage by healthcare workers, & presence in environment leading to high transmissibility & protracted outbreaks



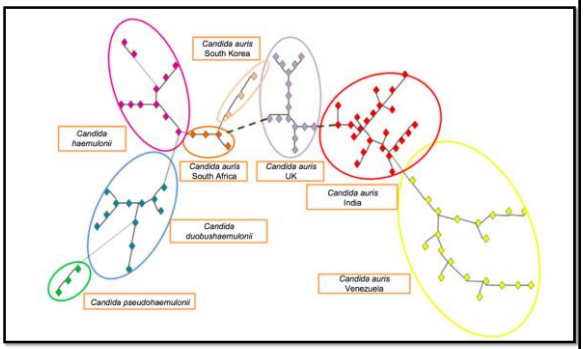
? Single source origin

Clonal origin

- AFLP & MLST suggest clonal strains in different countries
- Clustering of cases - indicating geographical strain variability
  - India, Kuwait & South Africa similar but differ from Japanese & Korean isolates
- Near simultaneous & independent emergence on different continents

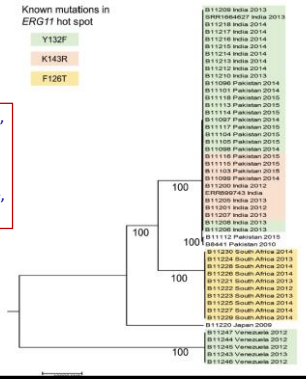
Chowdhary *et al.* PLoS Pathog 2017 13(5): e1006290
Rudramurthy *et al.* J Antimicrob Chemother 2017;72: 1794

AFLP-derived minimum spanning tree of *Candida auris*



Sharma & Upadhyay. Infect Drug Resist 2017; 10: 155

Whole genome sequencing of 54 isolates



- 4 clades: South Asia, South Africa, South America, East Asia
- Minimal difference within same geographical region
- Suggest simultaneous emergence, rather than spread

Lockhart SR, *et al.* Clin Infect Dis 2017; 64: 134

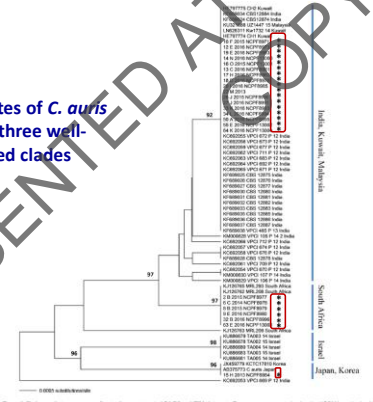
Isolates of the emerging pathogen *Candida auris* present in the UK have several geographic origins

Medical Mycology, 2017, 55, 1–0
doi:10.1093/mmy/myw147

Andrew M. Borman*, Adrien Szekely and Elizabeth M. Johnson

Isolate	Hospital	Site	Isolation Date	Town/City	NCPF No.
2	B	CSF	27 May 2015	Oxfordshire	NCPF 8977
6	C	Pleural fluid	10 June 2014	Central London	NCPF 8978
8	B	Pustule swab	2 Feb. 2015	Oxfordshire	NCPF 8979
9	E	Blood culture	16 May 2016	Slough	NCPF 8980
10	F*	Wound swab	19 May 2015	Bedford	NCPF 8971
12	E	Not stated	10 Jan. 2016	Slough	NCPF 8973
13	C	Sputum	11 May 2015	Central London	NCPF 8981
14	N*	Groin swab	14 June 2016	Chichester	NCPF13000
15	H	Not stated	20 June 2013	Central London	NCPF 8984
16	O*	Not stated	15 May 2015	Exeter	NCPF13001
17	H	Blood culture	10 April 2016	Central London	NCPF 8982
18	D*	Groin swab	19 May 2016	NW London	NCPF 8976
19	E	Femoral line	24 Aug. 2016	Slough	NCPF8983
20	I*	Wound swab	8 July 2016	Taunton	NCPF 8985
22	M	Not stated	13 Nov. 2013	Maidstone	Not viable
26	J	Wound swab	14 Dec. 2015	Stoke	NCPF 8990
27	J	Pleural fluid	15 Feb. 2016	Stoke	NCPF 8991
32	B	Blood culture	22 April 2016	Oxfordshire	NCPF 8996
33	K	Blood culture	30 July 2016	SE London	NCPF 8993
34	L	Swab	26 July 2016	Cardiff	NCPF 8994
50	A	Swab	14 April 2016	SW London	NCPF 8995
55	E	Urine	22 Aug. 2016	Slough	NCPF13002
63	F#	Not stated	2 Sep. 2016	Slough	NCPF 13003
64	E	Blood culture	29 Aug. 2016	SE London	NCPF13004

UK isolates of *C. auris* fall into three well-supported clades



Identification & characteristics of *C. auris*

Laboratory testing & misidentification – *C. auris*

Method	Comment
API-20C	Identify as *Rhodotorula glutinis, Candida sake, Saccharomyces cerevisiae*
Vitek - 2	Identify as *Candida haemulonii, Candida famata*
BD Phoenix	Identify as *Candida haemulonii*
Microscan	Identify as *C. famata, C. guilliermondii, C. lusitaniae, C. parapsilosis*
MALDI	**Can identify *C. auris* after improvement of data base** Before improvement – we updated the data base on our own (Ghosh *et al*. Clin Microbiol Infect. 2015; 21: 372-378)
DNA sequencing	**D1-D2 domain** of large subunit can identify correctly

***C. auris* could grow at 42° C, but failed to grow in presence of 0.01% or 0.1% cycloheximide**

Phenotypic identification of *C. auris*

	Growth by strain and type of media				
	Sabouraud Broth Base			Yeast Nitrogen Base	
Candida sp.	Dextrose	Dulcitol	Mannitol	Dulcitol	Mannitol
C. auris South Asia	+	+	+	+	+
C. auris Africa	+	+	+	+	+
C. auris S America	+	+	+	+	+
C. auris East Asia	+	+	+	+	+
C. glabrata	+	-	-	-	-
C. albicans	-	-	-	-	-
C. doubushaemulonii	-	-	-	-	-
C. haemulonii	-	-	-	-	-
C. parapsilosis	-	-	-	-	-
C. tropicalis	-	NA	NA	NA	NA

JCM Accepted Manuscript Posted Online 26 July 2017
J. Clin. Microbiol. doi:10.1128/JCM.00921-17

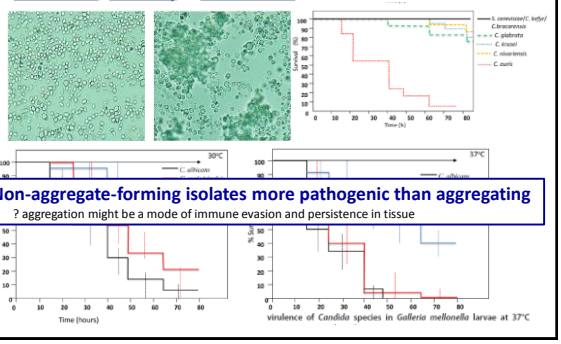
In vitro properties

- Thermotolerance, growing optimally at 37°C & viability up to 42°C, salt tolerance, & cell aggregation into large, difficult-to-disperse clusters (hyphae absent)
- Adhere to polymeric surfaces, form biofilms, & resist antifungal agents
- *C. auris* biofilms significantly thinner (50% thickness of *C. albicans* biofilm) (Larkin E, *et al.* Antimicrob Agents Chemother. 2017 Apr 24, online)
- Minimal ability to adhere to silicone elastomer (a representative catheter material) relative to *C. albicans*

mSphere. 2016 Jul-Aug; 1(4): e00189-16.

Comparative Pathogenicity of United Kingdom Isolates of the Emerging Pathogen *Candida auris* and Other Key Pathogenic *Candida* Species

Andrew M. Borman, Adrien Szekely, and Elizabeth M. Johnson



Non-aggregate-forming isolates more pathogenic than aggregating

? aggregation might be a mode of immune evasion and persistence in tissue

Biofilm-Forming Capability of Highly Virulent, Multidrug-Resistant *Candida auris*

Leighann Sherry, Gordon Ramage, Ryan Kean, Andrew Borman, Elizabeth M. Johnson, Malcolm D. Richardson, Riina Rautemaa-Richardson

Emerging Infectious Diseases
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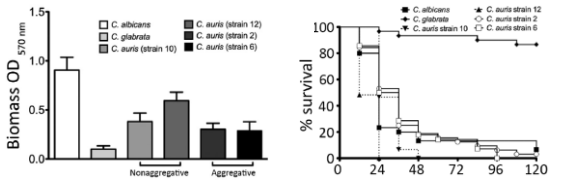
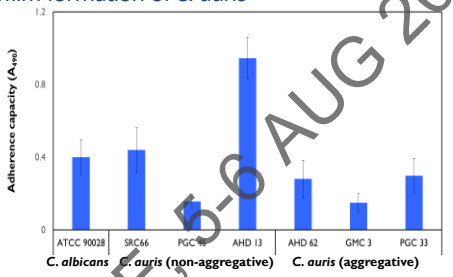


Biofilm formation of *C. auris*



Genome

- Size approximately 12.3 Mb
- Large percentage of genes devoted to central metabolism
- Cell wall modelling and nutrient acquisition, histidine kinase-2 component systems, iron acquisition, tissue invasion, enzyme secretion, multidrug efflux
- Weak phospholipase activity (majority of isolates being non-phospholipase producers)
- ATP-binding cassette (ABC) & major facilitator superfamily (MFS) transporter families along with drug transporters

Chatterjee S, *et al.* BMC Genomics 2015; 16:686
Sharma C, *et al.* Genome Announc. 2015; 3:pii: e00722

Drug resistance & therapy

Drug resistance reported till 2016

Reference	No of isolates tested	Method of susceptibility	MIC Range (µg/mL) FLU	VRC	AMB	CAS	5-FC
Satoh et al[21] (2009)	1	Not mentioned	2	0.03	–	–	0.5
Kim et al[8] (2009)	15	Etest method	2–128	0.03–2	0.38–1.5	0.125–0.25	–
Lee et al[22] (2011)	6	CLSI (2008)	2–128	0.03–1	0.5–1	0.06	–
Sarma et al[23] (2012)	15	Vitek 2 compact YST (MIC50/90)	64/64	1/2	8/16	–	1/1
Chowdhary et al[24] (2013)	12	CLSI (2008)	16–64	0.125–0.25	0.25–1	0.125–0.5	0.06–0.125
Chowdhary t al[24] (2013)	15	CLSI (2008)	64	0.5–4	0.25–1	0.25–1	0.25–64
Khillan et al[25] (2014)	4	CLSI (2008)	>64	0.06–0.125	0.125–0.5	1	0.125–4
Shallu Kathuria et al[13] (2016)	90	CLSI (2008)	4–>64	<0.03–16	0.125–8	0.125–8	<0.125–>64
Schelenz et al[26] (2016)	50	Sensititre YeastOne	>256	–	0.5–2	0.06–0.25	0.06–0.12
Sharma et al[14] (2016)	5	CLSI (2008)	≥64	0.125–16	0.25–4	0.25–8	0.125–64

Distribution of MIC of *C. auris* isolates (n=90)

Data tested	Test methods	No. of isolates at MIC (µg/mL) <0.03	0.03	0.06	0.13	0.25	0.5	1	2	4	8	16	>16	MIC (µg/mL) MIC50	MIC90
AMB	CLSI-BMD						2	16	23	35	4	6	4	1	4
	Vitek 2							1			48	41		8	16
	Etest	5		1	4	25	54		1					0.5	1
CAS	CLSI-BMD					1	29	27	25	1	4	3		0.5	1
	Vitek 2					21	34	28		7				0.5	4
	Etest	9	1	9	22	33	5	4		7				0.25	2
VRC	CLSI-BMD		1	4	7	8	18	17	18	6	3	3	5	1	8
	Vitek 2			3	5	12	28	16	14	10	2			1	4
	Etest	1		3	2	8	15	36	12	3	7		3	1	16

Sharma & Upadhyay. Infect Drug Resist 2017; 10: 155

Drug resistance reported in 2017

Antifungal	MIC Range, µg/mL	MIC_{50}, µg/mL	MIC_{90}, µg/mL
Fluconazole	4–256	128	256
Voriconazole	0.03–16	2	8
Itraconazole	0.125–2	0.5	1
Posaconazole	0.06–1	0.5	1
Caspofungin	0.03–16	0.25	1
Anidulafungin	0.125–16	0.5	1
Micafungin	0.06–4	0.25	2
Flucytosine	0.125–32	0.125	0.5
Amphotericin B	0.38–4	1	2

Abbreviations: MIC, minimum inhibitory concentration; MIC_{50}, MIC for 50% of isolates; MIC_{90}, MIC for 90% of isolates

- **Resistance to fluconazole – 93%, voriconazole - 54%, AmB – 35%, Echinocandins – 7%**
- **41% ≥ 2 classes**

Lockhart SR, *et al.* Clin Infect Dis 2017; 64: 134

Drug resistance menace in Asian countries

Fluconazole	• 90% resistant
Voriconazole	• Elevated MICs in 50% of isolates
Amphotericin B	• variable susceptibility; 15%–30% of the isolates exhibit high (>2 µg/ml) MICs
Echinocandin	• 2%–8% resistant
MDR	• 50% resistant to ≥2 antifungal classes
All classes resistant	• 4%
Indian ICUs	• Fluconazole 58.1% (R), amphotericin B (13.5%), Caspofungin 9.5% (high MIC);16.2% MDR

Rudramurthy *et al.* J Antimicrob Chemother 2017; 72: 1794

Chowdhary *et al.* PLoS Pathog 2017 13(5): e1006290.

Chakrabarti *et al.* Intensive Care Med 2015; 41: 285

Biofilm-Forming Capability of Highly Virulent, Multidrug-Resistant *Candida auris*

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Plantonic susceptibility profile

Drug	Planktonic MIC* Strain 2	Strain 6	Strain 10	Strain 12
Fluconazole	>32	>32	>32	>32
Voriconazole	8	8	32	1
Caspofungin	32	32	2	>32
Micafungin	0.5	≤0.0625	<0.06	≤0.0625
Liposomal amphotericin B	0.25	0.25	0.5	1
Amphotericin B	0.25	0.25	0.5	0.5
Chlorhexidine, %	≤0.02	≤0.02	≤0.02	≤0.02

*Values are mg/L except as indicated. All MIC tests were performed on 3 independent occasions, showing identical results each time.

Biofilm susceptibility profile

Drug	Sessile MIC* Strain 2	Strain 6	Strain 10	Strain 12
Fluconazole	>32	>32	>32	>32
Voriconazole	>32	>32	>32	>32
Caspofungin	>32	>32	>32	>32
Micafungin	>32	>32	0.25	>32
Liposomal amphotericin B	2	8	16	16
Amphotericin B	2	4	2	4
Chlorhexidine, %	≤0.02	≤0.02	≤0.02	≤0.02

Mechanism of drug resistance

- Resistance probably inducible under antifungal pressure with rapid mutational changes
  - Single copies of ERG3, ERG11, FKS1, FKS2and FKS3 genes present
  - Alterations at azole-resistance codons of ERG11 in *C. auris* isolates substitutions (strongly associated with country-wise–specific geographic clades)
  - Significant portion of genome encodes ABC and MFS transporter families along with drug transporters

Chatterjee S, *et al.* BMC Genomics 2015; 16:686
Sharma C, *et al.* Genome Announc. 2015; 3:pii: e00722

Therapeutic options

- No consensus exists for optimal treatment
- Echinocandins remain the first-line therapy for *C. auris* infection
  - Caspofungin shown to be inactive against *C. auris* biofilms
- Flucytosine (MIC50, 0.125–1 µg/ml) in renal tract or UTI
- Posaconazole (range, 0.06–1 µg/ml) & isavuconazole (range, <0.015–0.5 µg/ml) show excellent in vitro activity against *C. auris*
- New drugs- SCY-078 & pulmocide exhibit potent antifungal activity against *C. auris* isolates

AAC Accepted Manuscript Posted Online 5 June 2017
Antimicrob. Agents Chemother. doi:10.1128/AAC.00791-17

Pharmacodynamic Optimization for Treatment of Invasive *Candida auris* Infection

Lepak AJ, Zhao M, Berkow EL, Lockhart SR, Andes DR

Table. Nine select *Candida auris* strains used in the studies including country of origin, antimicrobial susceptibility results, and 24-hour total drug PK/PD target exposures in the murine invasive candidiasis model.

Strain	Country of Origin	96 h Growth in Untreated Controls (CFU/kidney)	Fluconazole		Micafungin			Amphotericin B	
			MIC (mg/L)	24 h Stasis AUC/MIC	MIC (mg/L)	24 h Stasis AUC/MIC	24 h 1 log kill AUC/MIC	MIC (mg/L)	24 h Stasis Cmax/MIC
B11804	Colombia	2.17	2	51.2	0.5	48.1	120.3	0.5	0.87
B11801	Colombia	2.86	16	26.3	1	32.9	49.9	2	NA
B11799	Colombia	2.08	16	36.3	2	18.5	92.1	0.5	1.29
B11221	South Africa	1.85	128	6.3	1	47.6	140.6	0.38	0.52
B11211	India	1.97	256	NA	4	NA	NA	1.5	0.69
B11785	Colombia	2.32	8	34.1	0.5	59.4	119.2	1.5	1.50
B11220	Japan	1.04	4	5.0	0.125	286.5	674.4	0.38	NA
B11203	India	2.13	256	NA	0.25	117.0	376.4	4	0.51
B11404	Pakistan	1.71	256	4.1	0.25	134.3	536.8	1	2.13
Median				**26.3**		**53.7**	**130.5**		**0.87**
Std dev				**18.5**		**87.9**	**235.3**		**0.60**

NA, endpoint not achieved

was a fluconazole AUC/MIC of 26, amphotericin B Cmax/MIC of 0.9, and micafungin AUC/MIC of 54. The micafungin PD targets for *C. auris* were ≥20-fold lower than other *Candida* species in this animal model. Clinically relevant micafungin exposures produced the most killing among the three classes.

Prevention & control of outbreak

Prevention of spread

- Problem – we do not know the source
- Admission screening for yeast carriage
- Isolation or cohorting of patients with dedicated nursing staff in separate areas, contact precaution & notify any positive case
- Epidemiological investigation, complemented by cross-sectional patient screening & environmental sampling
- Skin decontamination and oral gargles with chlorhexidine-containing mouth wash, & use of topical nystatin & terbinafine for cannula entry sites
- Environmental cleaning - chlorine & hydrogen peroxide products
- Hand hygiene compliance, maximal sterile barriers upon insertion & use of chlorhexidine for skin disinfection

Environmental Surfaces in Healthcare Facilities are a Potential Source for Transmission of *Candida auris* and Other *Candida* Species

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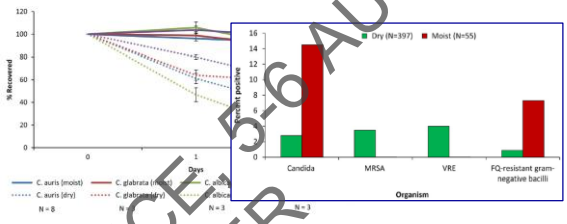

Survival on dry steel disks & moist non-nutrient agar

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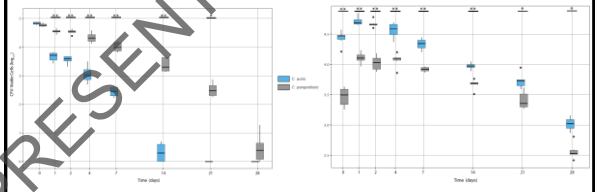
Survival, Persistence, and Isolation of the Emerging Multidrug-Resistant Pathogenic Yeast *Candida auris* on a Plastic Healthcare Surface

Rory M. Welsh[a#], Meghan L. Bentz[a], Alicia Shams[b], Hollis Houston[b], Amanda Lyons[b], Laura J. Rose[b], Anastasia P. Litvintseva[a#]

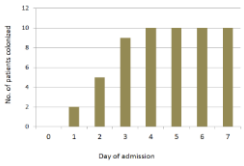


Survival measured by colony forming unit

Survival measured by esterase activity

Surveillance of *C. auris* in hospital

- Colonization of the patients in trauma ICU
  - None of the patients are colonized at the time of admission



Days of acquisition of *C. auris*

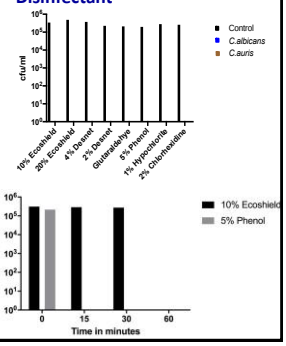
- Persistence of *C. auris* in hospital environment
  - **Hands** of healthcare workers
  - Contamination of bed surface, certain equipment like ventilator, temperature probes & ECG leads
  - *C. auris* can **persist on blankets or linen at least 7d**

How to get rid of *C. auris* from hospital environment?

- **Colonization of patients**
  - Chlorhexidine body wash
  - Oral nystatin tablets
- **Hand wash**

S. no	Group 1 (control)	Group-2 (Soap & water)	Group-3 (Alcohol + chlorhexidine)	Group-4 (70% Alcohol)
1	Confluent growth	2 colonies	No growth	No growth
2	Confluent growth	No growth	No growth	No growth
3	Confluent growth	No growth	No growth	No growth

- **Disinfectant**



CDC recommendation

- Placing patients with *C. auris* colonization / infection in single rooms
- Standard contact precautions by healthcare personnel
- Weekly screens for recurrence of colonization for patients admitted for prolonged duration
- First-line therapy remains an echinocandin although susceptibility testing is recommended
- Resistance surveillance is recommended in patients who are infected or colonized and are on antifungals

http://www.cdc.gov/fungal/diseases/candidiasis/candida-auris-alert.html

Strict vigilance of *C. auris*

- CDC
  - (http://www.cdc.gov/fungal/diseases/candidiasis/candida-auris-alert.html)
- Public Health England (PHE), London
  - https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/537174/Guidance_Candida__auris.pdf
- European Centre for Disease Prevention and Control (ECDC), Europe (http://ecdc.europa.eu/en/publications/Publications/Candida-in-healthcare-settings_19-Dec-2016.pdf)

When you should think you are dealing with *C. auris*?

- If the patient is from ICU or high-dependency area
- Transferred from another hospital after a long stay
- Multiple intervention & prior antifungal exposure
- If one identify in a commercial system -*Candida haemulonii, Candida famata, C. guilliermondii, C. lusitaniae, C. parapsilosis, Rhodotorula glutinis, Candida sake, Saccharomyces cerevisiae*
- If the *Candida* appears to be resistant to fluconazole & high MIC to voriconazole

***C. auris* could grow at 42˚ C, but failed to grow in presence of 0.01% or 0.1% cycloheximide. Ferment dextrose, dulcitol, mannitol**

Gaps in knowledge

- Is it a jump from Japan & Korea to India or we missing the isolates in other Asian countries?
- Whether it existed earlier than 1996?
- Why it is independently, almost simultaneously, emerged in so many places worldwide?
- Clonal or variation exist?
- Why it exhibits high level of antifungal resistance?
- Need to study source of agent & transmission mechanism
- Best therapeutic options

THANK YOU