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Research note

Acquired fluconazole resistance and genetic clustering in *Diutina* (*Candida*) *catenulata* from clinical samples

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ABSTRACT

Objectives: *Diutina* (*Candida*) *catenulata* is an ascomycetous yeast isolated from environmental sources and animals, occasionally infecting humans. The aim of this study is to shed light on the *in vitro* antifungal susceptibility and genetic diversity of this opportunistic yeast.
Methods: Forty-five *D. catenulata* strains isolated from various sources (including human and environmental sources) and originating from nine countries were included. Species identification was performed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry and confirmed via internal transcribed spacer ribosomal DNA barcoding. *In vitro* antifungal susceptibility was determined for seven systemic antifungals via the gradient strip method after 48 hours of incubation at 35°C using Etest® (Biomérieux) or Liofilchem® strips. Isolates exhibiting fluconazole minimal inhibitory concentrations (MICs) of ≥8 μg/mL were investigated for mutations in the *ERG11* gene. A novel microsatellite genotyping scheme consisting of four markers was developed to assess genetic diversity.
Results: MIC ranges for amphotericin B, caspofungin, micafungin, isavuconazole, and posaconazole were 0.19–1 μg/mL, 0.094–0.5 μg/mL, 0.012–0.064 μg/mL, 0.003–0.047 μg/mL, and 0.006–0.032 μg/mL, respectively. By comparison, a broad range of MICs was noted for fluconazole (0.75 to >256 μg/mL) and voriconazole (0.012–0.38 mg/L), the higher values being observed among clinical strains. The Y132F amino acid substitution, associated with azole resistance in various *Candida* species (*C. albicans*, *C. tropicalis*, *C. parapsilosis*, and *C. orthopsilosis*), was the main substitution identified. Although microsatellite typing showed extensive genetic diversity, most strains with high fluconazole MICs clustered together, suggesting human-to-human transmission or a common source of contamination.
Discussion: The high rate of acquired fluconazole resistance among clinical isolates of *D. catenulata* is of concern. In this study, we highlight a link between the genetic diversity of *D. catenulata* and its antifungal

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resistance patterns, suggesting possible clonal transmission of resistant isolates. **Céline Nourrisson, Clin Microbiol Infect 2022;▪:1**

Introduction

Diutina catenulata (formerly *Candida catenulata*) is an ascomycetous yeast occasionally responsible for invasive fungal diseases, mostly fungaemia, in humans [1–3]. Outside the clinical setting, this species has been reported to be present in various animals, dairy products, dust, surface water, and soil samples [4–7].

Yet, little is known about the *in vitro* susceptibility profile of this opportunistic species to systemic antifungals; however, case reports and surveillance programmes have reported variable susceptibility to azoles [1–3,8]. *D. catenulata* strains with high minimal inhibitory concentrations (MICs) for azoles have also been reported from animals in various countries, raising concern owing to the risk of spreading to clinical settings and environment [5–7].

At Clermont-Ferrand Hospital, France, *D. catenulata* was first identified in 2016 as a cause of fungaemia. This case, followed by the isolation of additional clinical strains at our hospital, prompted us to gain further insight into the antifungal susceptibility and genetic diversity of this opportunistic species.

Methods

Fungal strains

Forty-five *D. catenulata* strains collected from nine different countries were analysed: 24 clinical strains, including 17 strains from four French hospitals (Table S1), and 21 strains from veterinary and environmental sources, including the genome sequenced WY3-10-4 strain [4] (Table S2).

Identification

Species identification was performed using the VITEK® matrix-assisted laser desorption/ionization time-of-flight mass spectrometry instrument (database v3.2.0, BioMérieux) and confirmed via internal transcribed spacer ribosomal DNA barcoding.

In vitro antifungal susceptibility testing

MICs were determined at a single laboratory after 48 hours of incubation at 35°C using Etest® strips (BioMérieux) for fluconazole (FLU), voriconazole, posaconazole, amphotericin B, caspofungin, and micafungin and Liofilchem® strips (Liofilchem) for isavuconazole. Except for amphotericin B (full inhibition), MICs were read at 80% growth inhibition according to the manufacturer's recommendations. *Candida parapsilosis* ATCC 22019 and *Candida krusei* ATCC 6258 were used as controls strains.

ERG11 sequencing

Strains exhibiting FLU MICs of ≥8 μg/mL were subjected to *ERG11* sequencing (see primers in Table S3).

Microsatellite typing

A search for possible microsatellite sequences was conducted using Tandem Repeats Finder (https://tandem.bu.edu/trf/trf.html) with WY3-10-4 as a reference (BioProject PRJNA421257) [4,9]. New gene sequences were deposited in GenBank (accession numbers ON312106 to ON312285) (Table S3). Raw microsatellite profiles were imported into the R package (release 4.0.3) for UPGMA (Unweighted Pair Group Method with Arithmetic mean) dendrogram construction based on microsatellite repeat number using hclust with ward.D2 aggregation method. The discriminatory power of this typing scheme was determined using the Hunter index [10].

Ethics

This study was recorded in the Nantes Hospital by the local's Data Protection Officer under the following reference: TS005.BIO.AP.2019_17.

Results

We first diagnosed *D. catenulata* fungaemia at our hospital in 2016 in a patient with Hodgkin lymphoma. This strain (CLE2101) exhibited a high FLU MIC (≥256 μg/mL using Etest®). According to the medical records, this patient worked as a cattle breeder and consumed raw dairy products collected from his cows. Since then, 12 additional isolates have been identified at our centre. To gain further knowledge on this rare opportunistic yeast, 32 additional *D. catenulata* strains, previously isolated and stored in different laboratories and originating from different countries and various sources (24 clinical isolates), were investigated (Tables S1 and S2).

In vitro antifungal susceptibilities of the 45 strains to seven antifungals are given in Fig. 1. A wide range of MIC values were observed for FLU (0.75 to ≥256 μg/mL). All strains with FLU MICs of ≥8 μg/mL were clinical strains (n = 8/24). For 50% of them, voriconazole MICs ranged from 0.19 to 0.38 μg/mL. Two strains (CLE9871-1 and CLE9871-2) collected from a single sample/patient displayed distinct FLU susceptibility profiles (1.5 and ≥ 256 μg/mL, respectively). All strains displayed low MICs for the remaining antifungals.

The Y132F (A395T) substitution was identified in four of the eight strains with FLU MICs of ≥8 μg/mL (Fig. 2). One strain (CLE0951) harboured a previously undescribed *ERG11* mutation (C1389G, I463M) and displayed a FLU MIC of 16 μg/mL. The remaining strains displayed a wild-type *ERG11* sequence.

The best four putative microsatellite targets (MS7, MS14, MS55, and MS479) identified by bioinformatics analyses and experimental tests were selected to set up a microsatellite typing scheme (Table S3). Overall, this method highlighted 22 haplotypes distributed across 11 clades (H-index = 0.881) (Fig. 2). Some strains clustered according to their geographical location (clade IX), whereas other clades grouped together strains from distant geographical locations (clades III and VI) and/or sources (clade VIII). The two strains collected from the same patient were distributed in two distant clades. Three of the four Y132F strains clustered together (clade VII). All three isolates were collected at Clermont-Ferrand Hospital 15 months apart from different patients.

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(A)

Antifungal	MIC range (μg/mL)	Mode (μg/mL)	MIC_{50} (μg/mL)	MIC_{90} (μg/mL)
Fluconazole	0.75-≥256	3	3	48
Voriconazole	0.012-0.38	0.032-0.047	0.032	0.125
Posaconazole	0.006-0.032	0.012	0.012	0.023
Isavuconazole	0.003-0.047	0.012	0.012	0.023
Amphotericin B	0.19-1	0.5	0.5	0.75
Caspofungin	0.094-0.5	0.19	0.19	0.38
Micafungin	0.012-0.064	0.023	0.023	0.032

(B)

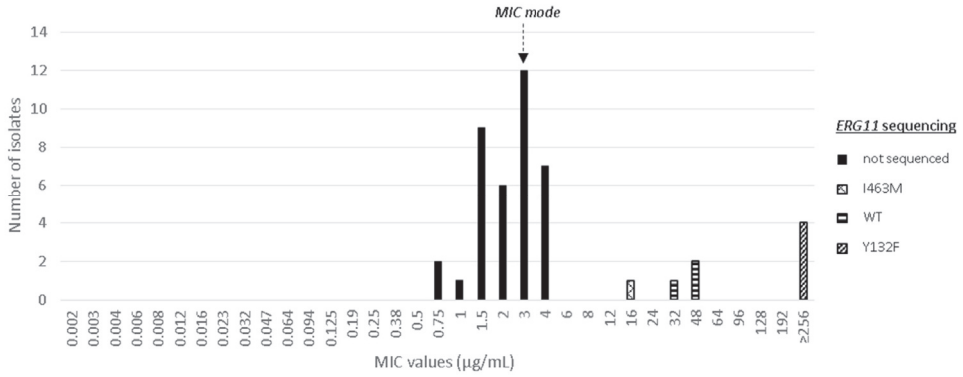

Fig. 1. *In vitro* susceptibility of *Diutina catenulata* to seven systemic antifungals by Etest® ($n = 45$). (a) Minimal inhibitory concentration (MIC) ranges, mode, MIC_{50}, and MIC_{90}; (b) Fluconazole MIC distribution and *ERG11* sequencing findings. WT, wild type.

Discussion

This collection, which, to the best of our knowledge, is the largest collection to be investigated so far, allowed us to highlight the variable FLU susceptibility of this rare opportunistic yeast. Interestingly, the elevated FLU MICs observed in clinical strains (33%) suggest that FLU could not be the best therapeutic option and prompt MIC testing in patients with invasive disease. A recent study also reported elevated FLU MICs among *D. catenulata* clinical isolates from a surveillance programme in the People's Republic of China [3]. Interestingly, the same study reported some isolates with reduced susceptibility to echinocandins, which was not observed in our study. Regarding the mechanisms explaining the variable FLU susceptibility, we observed that all strains with FLU MICs of ≥256 μg/mL carried the Y132F amino acid substitution in *ERG11*, which is commonly associated with FLU resistance in various *Candida* species [11]. However, alternative mechanisms (*ERG11* overexpression and active efflux) may be involved because some of our strains with elevated MICs showed a wild-type *ERG11* sequence.

Interestingly, the search for medical records was unsuccessful in identifying previous azole exposure in the eight patients with high FLU MICs. This might support possible patient-to-patient transmission of resistant strains or an environmental contamination source. The potential contribution of environmental fungicides to azole resistance in medically relevant yeasts has been discussed elsewhere [12]. This paradigm raises concern because another finding of our work is that behind apparent genetic diversity, our microsatellite scheme unveiled clonal clusters. Strikingly, all three clinical strains carrying the *ERG11* Y132F mutation were isolated between 2016 and 2017 from three distinct azole-naïve patients admitted at the same hospital. This strongly reminds the scenario reported with *C. parapsilosis* in European hospitals [13,14]. The presence of genotypically indistinguishable strains from different origins may also suggest that *D. catenulata* can cross borders between different sources. However, these conclusions must take into account the suboptimal discriminatory power of this first microsatellite scheme specifically developed for *D. catenulata*, which would benefit from being improved with additional markers.

Although the use of the Etest® method might be viewed as a limitation of this study, this choice can be explained not only by the good agreement of this method with reference microdilution assays when testing medically relevant yeasts but also by its practicability in the clinical laboratory, which explains its increasing use [15–17]. Although no clinical breakpoint is available, regardless of the method used, the bimodal distribution observed for FLU as well as the observation of *ERG11* mutations in strains with the highest MICs raise the possibility of acquired resistance in this rare species.

To conclude, this study provides evidence for the presence of *ERG11* amino acid substitution associated with high FLU MICs (≥8 μg/mL) among *D. catenulata* clinical strains, suggesting acquired

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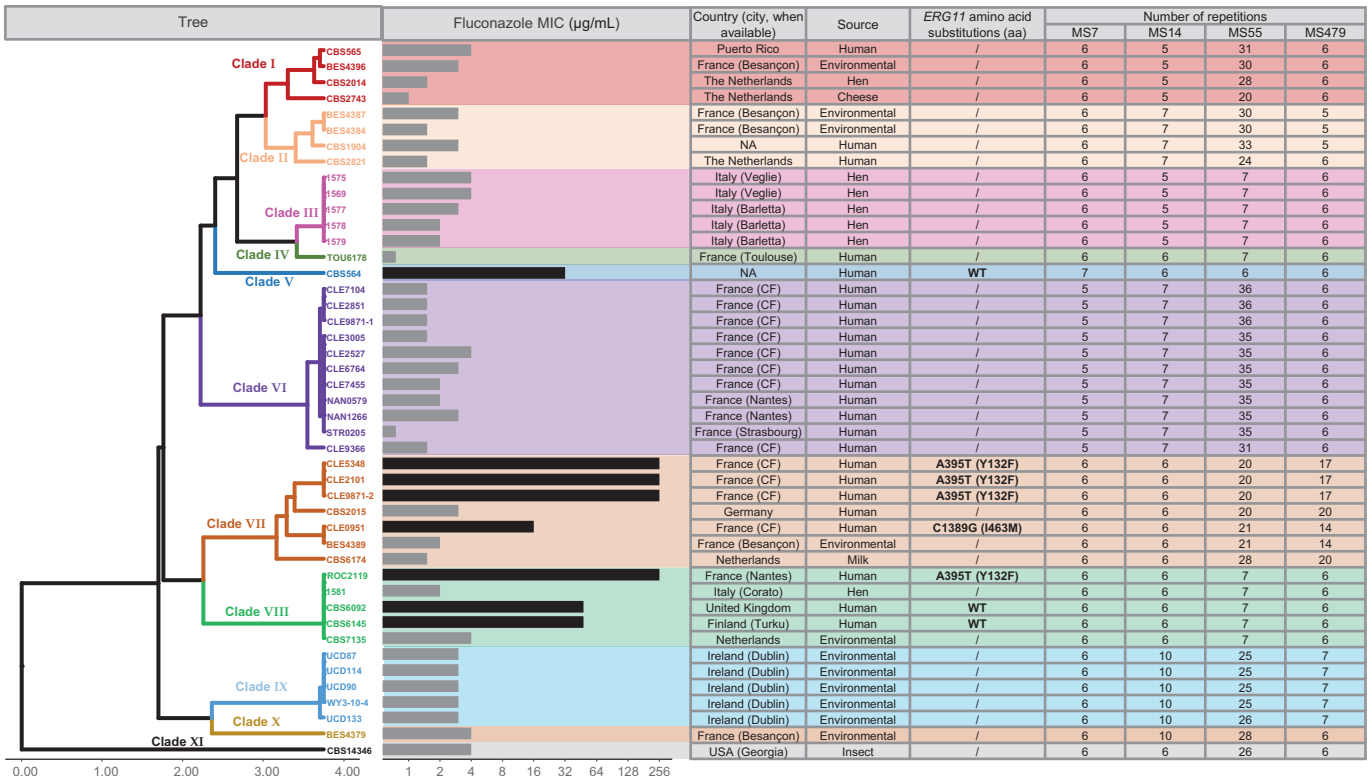

Tree	Fluconazole MIC (µg/mL)	Country (city, when available)	Source	*ERG11* amino acid substitutions (aa)	Number of repetitions			
					MS7	MS14	MS55	MS479
CBS565		Puerto Rico	Human	/	6	5	31	6
BES4396		France (Besançon)	Environmental	/	6	5	30	6
CBS2014		The Netherlands	Hen	/	6	5	28	6
CBS2743		The Netherlands	Cheese	/	6	5	20	6
BES4387		France (Besançon)	Environmental	/	6	7	30	5
BES4384		France (Besançon)	Environmental	/	6	7	30	5
CBS1904		NA	Human	/	6	7	33	5
CBS2821		The Netherlands	Human	/	6	7	24	6
1575		Italy (Veglie)	Hen	/	6	5	7	6
1569		Italy (Veglie)	Hen	/	6	5	7	6
1577		Italy (Barletta)	Hen	/	6	5	7	6
1578		Italy (Barletta)	Hen	/	6	5	7	6
1579		Italy (Barletta)	Hen	/	6	5	7	6
TOU6178		France (Toulouse)	Human	/	6	6	7	6
CBS564		NA	Human	**WT**	7	6	6	6
CLE7104		France (CF)	Human	/	5	7	36	6
CLE2851		France (CF)	Human	/	5	7	36	6
CLE9871-1		France (CF)	Human	/	5	7	36	6
CLE3005		France (CF)	Human	/	5	7	35	6
CLE2527		France (CF)	Human	/	5	7	35	6
CLE6764		France (CF)	Human	/	5	7	35	6
CLE7455		France (CF)	Human	/	5	7	35	6
NAN0579		France (Nantes)	Human	/	5	7	35	6
NAN1266		France (Nantes)	Human	/	5	7	35	6
STR0205		France (Strasbourg)	Human	/	5	7	35	6
CLE9366		France (CF)	Human	/	5	7	31	6
CLE5348		France (CF)	Human	**A395T (Y132F)**	6	6	20	17
CLE2101		France (CF)	Human	**A395T (Y132F)**	6	6	20	17
CLE9871-2		France (CF)	Human	**A395T (Y132F)**	6	6	20	17
CBS2015		Germany	Human	/	6	6	20	20
CLE0951		France (CF)	Human	**C1389G (I463M)**	6	6	21	14
BES4389		France (Besançon)	Environmental	/	6	6	21	14
CBS6174		Netherlands	Milk	/	6	6	28	20
ROC2119		France (Nantes)	Human	**A395T (Y132F)**	6	6	7	6
1581		Italy (Corato)	Hen	/	6	6	7	6
CBS6092		United Kingdom	Human	**WT**	6	6	7	6
CBS6145		Finland (Turku)	Human	**WT**	6	6	7	6
CBS7135		Netherlands	Environmental	/	6	6	7	6
UCD87		Ireland (Dublin)	Environmental	/	6	10	25	7
UCD114		Ireland (Dublin)	Environmental	/	6	10	25	7
UCD90		Ireland (Dublin)	Environmental	/	6	10	25	7
WY3-10-4		Ireland (Dublin)	Environmental	/	6	10	25	7
UCD133		Ireland (Dublin)	Environmental	/	6	10	26	7
BES4379		France (Besançon)	Environmental	/	6	10	28	6
CBS14346		USA (Georgia)	Insect	/	6	6	26	6

Fig. 2. Representation of the phylogenetic tree associated with fluconazole minimal inhibitory concentrations (MICs) and epidemiological data of 45 strains of *Diutina catenulata*. Each clade was symbolized by a different branch colour. Dark MIC bars highlighted fluconazole MICs of ≥8 µg/mL. '/' indicates 'not applicable' (fluconazole MICs of <8 µg/mL). aa, amino acids; CF, Clermont-Ferrand; NA: not available; WT, wild type.

resistance. More data and *in vivo* experiments would be useful to correlate MICs with clinical outcome. The isolation of genetically indistinguishable strains, including FLU-resistant strains from azole-naïve patients, raised questions about the transmission routes. Further investigations relying on whole-genome sequencing followed by comparative genomics would contribute to clarify these points.

Author contributions

CN, PP, and FM conceptualized the study, developed the methodology, and performed the formal analysis. CN, MM, RAL, ER, VB, and FM conducted the investigation. CN, MM, RAL, FH, FG, CC, GB, SC, MS, PLP, PP, and FM collected the resources. PP and FM curated the data. CN and FM wrote the original draft of the article. MM, RAL, FH, FG, CC, GB, SC, MS, PP, and FM wrote, reviewed, and edited the article. CN, PP, and FM performed visualization. FM supervised the study.

Transparency declaration

The authors declare that they have no conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cmi.2022.09.021.

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