As a clinical mycologist, one of the most frequent questions I get asked by clinical technicians is, “Do we still need to distinguish *Candida dubliniensis* from *Candida albicans*?” When I ask the questioner why they separate them, the usual response is that *C. dubliniensis* is often resistant to fluconazole. When *C. dubliniensis* was first described in 1995 (11), AIDS was epidemic and highly active antiretroviral therapy (HAART) was just becoming available. Because of these factors, mucocutaneous candidiasis was commonly seen and fluconazole prophylaxis was often prescribed. Many of the first strains of *C. dubliniensis* from HIV-positive patients exhibited elevated fluconazole MIC values or the ability to develop fluconazole resistance under drug pressure in vitro (5, 6). Despite this, the original discoverers of *C. dubliniensis* reported that it remained fairly susceptible to fluconazole (9).

To try to put this question to rest, we tested 42 isolates of *C. dubliniensis* for susceptibility to fluconazole. Isolates were collected as part of a population-based candidemia surveillance conducted by the CDC in Atlanta and Baltimore. Isolates were identified as *C. dubliniensis* by a Luminex-based assay (4) or by sequencing of the D1/D2 region of the ribosomal DNA (rDNA). Isolates were tested by broth microdilution using the methodology of Clinical and Laboratory Standards Institute standard M27-A3 (3). The MIC of these 42 isolates was 0.25 μg/ml, and the MIC was 0.5 μg/ml, with only two isolates (4.8%) having a MIC value above 0.5 μg/ml, both of which had a MIC value of 16 μg/ml. The two isolates with elevated MIC values would not recently have been considered resistant, but by applying the new *C. albicans* species-specific breakpoints (7) would now be considered resistant. To try this question to rest, we tested 42 isolates of *C. dubliniensis* for susceptibility to fluconazole. Isolates were collected as part of a population-based candidemia surveillance conducted by the CDC in Atlanta and Baltimore. Isolates were identified as *C. dubliniensis* by a Luminex-based assay (4) or by sequencing of the D1/D2 region of the ribosomal DNA (rDNA). Isolates were tested by broth microdilution using the methodology of Clinical and Laboratory Standards Institute standard M27-A3 (3). The MIC of these 42 isolates was 0.25 μg/ml, and the MIC was 0.5 μg/ml, with only two isolates (4.8%) having a MIC value above 0.5 μg/ml, both of which had a MIC value of 16 μg/ml. The two isolates with elevated MIC values would not recently have been considered resistant, but by applying the new *C. albicans* species-specific breakpoints (7) would now be considered resistant. Two other very recent studies had similar conclusions. In their 10-year global *Candida* surveillance, Pfaller and colleagues (8) found that only 2.6% (3.9% by new *C. albicans* species-specific breakpoints) of 310 *C. dubliniensis* isolates were resistant to fluconazole. In a 6-year national surveillance of fungemia in Denmark, Arenstrup and colleagues (1) found only 3.1% fluconazole resistance among 65 clinical isolates of *C. dubliniensis*. This does not mean that there is never a reason to determine species. Certainly in an outbreak setting, it is important to know the species for epidemiological purposes, and it is important for surveillance trends. There are some commercially available systems that adequately identify *C. dubliniensis*, and these may be useful in centers with a high HIV-positive prevalence (2). However, for a recurrent isolate, it would be much more efficacious to perform susceptibility testing on the isolate than to identify it as *C. dubliniensis*, especially in light of the fact that *C. albicans* is considered to be the more virulent pathogen (10). The additional biochemical testing or molecular assay costs the additional biochemical testing or molecular assay costs the clinical laboratory time and money and could slow the initiation of effective empirical therapy by the belief that fluconazole cannot be used for *C. dubliniensis* infections. Therefore, my answer to the question of whether it is necessary to distinguish *C. dubliniensis* from *C. albicans* in the clinical microbiology laboratory is usually, “No.”

The findings and conclusions of this letter are those of the author and do not necessarily represent the views of the Centers for Disease Control and Prevention.

REFERENCES


Shawn R. Lockhart* Mycotic Diseases Branch Centers for Disease Control and Prevention Atlanta, Georgia 30333

*Phone: (404) 639-2569 Fax: (404) 315-2376 E-mail: gyi2@cdc.gov

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