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Retrospective Identification and Characterization of *Candida dubliniensis* Isolates among *Candida albicans* Clinical Laboratory Isolates from Human Immunodeficiency Virus (HIV)-Infected and Non-HIV-Infected Individuals

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Fungal opportunistic infections, and in particular those caused by the various *Candida* species, have gained considerable significance as a cause of morbidity and, often, mortality. The newly described species *Candida dubliniensis* phenotypically resembles *Candida albicans* so closely that it is easily misidentified as such. The present study was designed to determine the frequency at which this new species is not recognized in the clinical laboratory, to determine the patient populations with which *C. dubliniensis* is associated, to determine colonization versus infection frequency, and to assess fluconazole resistance. Over a 2-year period, 1,251 isolates that were initially identified as *C. albicans* by a hospital clinical laboratory were reevaluated for *C. dubliniensis* by inability to grow at 45°C, colony color on CHROMagar Candida medium, coaggregation assay with *Fusobacterium nucleatum*, and sugar assimilation profiles (API 20C AUX yeast identification system). A total of 15 (1.2%) isolates from 12 patients were identified as *C. dubliniensis*. Ten of the patients were found to be immunocompromised (these included patients with human immunodeficiency virus infection or AIDS, cancer patients receiving chemotherapy, and patients awaiting transplantation). Thirteen isolates were highly susceptible to fluconazole (MIC, <0.5 μg/ml). Three isolates from one patient, genotypically confirmed as the same strain, showed variable susceptibility to fluconazole. The first isolate was susceptible, whereas the other two isolates were dose-dependent susceptible (MIC, 16.0 μg/ml). These data confirm the close association of *C. dubliniensis* with immunocompromised states and that increased fluconazole MICs may develop in vivo. This study emphasizes the importance of screening germ-tube-positive yeasts for the inability to grow at 45°C followed by confirmatory tests in order to properly identify this species.

The continuing AIDS epidemic, malignancies, and aggressive chemotherapeutic interventions have created an extremely vulnerable population of immunocompromised patients who are highly susceptible to a variety of microbial infections, including fungal infections (1, 4, 5, 16, 28, 31). Antifungal drug resistance and the emergence of novel species and species previously not associated with human disease as potential pathogens have also greatly contributed to the drastic increase in fungal infections (2, 24, 29). Although the true clinical significance of the newly identified species *Candida dubliniensis* is not yet fully known, it is already established as an opportunistic pathogen due to its association with recurrent oral infections as well as its implication in cases of superficial and systemic disease in immunocompromised individuals (2, 11, 27). In addition to being a significant cause of candidosis, *C. dubliniensis* was found to be easily induced to develop resistance to fluconazole in vitro, a phenomenon which may have resulted in its emergence (15, 16). Few studies have been performed to investigate virulence factors of *C. dubliniensis*; however, preliminary data so far have suggested that isolates of this species produce higher levels of proteinase and are more adherent to buccal epithelial cells than *Candida albicans* isolates (10, 29). One recently observed characteristic exhibited by *C. dubliniensis* is that cells grown at 37°C on Sabouraud dextrose agar (SDA) coaggregate in vitro with the oral anaerobic bacterium *Fusobacterium nucleatum* (ATCC 49256), whereas *C. albicans* cells grown under the same conditions fail to do so (8). This binding of *C. dubliniensis* to *F. nucleatum* may play an important role in the colonization ability of *C. dubliniensis* in the oral cavity (8, 13, 14). In addition to its potential clinical implications, this intergeneric coaggregation between *F. nucleatum* and *C. dubliniensis* has been used to develop a rapid and specific assay to discriminate between *C. dubliniensis* and *C. albicans* isolates (8). Most recently, electron microscopic studies comparing the cell walls of *C. dubliniensis* and *C. albicans* revealed major ultrastructural differences between these two species that allow *C. dubliniensis* to express constant cell surface hydrophobicity (7).

As *C. dubliniensis* continues to gain importance as a significant opportunistic pathogen and in light of its predisposition for fluconazole resistance, it has become important for laboratories to screen for this species in clinical specimens. Until this species is fully accepted as a clinically important *Candida* species by clinical laboratories, *C. dubliniensis* isolates will continue to be identified as *C. albicans* and the efforts to investigate the epidemiology and the true clinical significance of this new species will continue to be hampered (26, 29). To that end, the investigation described here was initiated to document the prevalence of *C. dubliniensis* presumptively identified as *C. albicans* in the clinical laboratory. In addition, the study aimed to determine the presence of fluconazole resistance among clinical isolates, to test for strain stability among multiple isolates from the same patient, and to provide some epidemio-

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logical and clinical data regarding the patients from whom these *C. dubliniensis* isolates were recovered.

A total of 1,251 isolates of *C. albicans*, recovered between January 1998 and January 2000 by the Clinical Microbiology Laboratories at the University of Maryland Hospital, were evaluated. The isolates were originally identified by their ability to produce germ tubes and chlamydospores on Tween 80-oxgall-caffeic acid agar (Remel, Lenexa, Kans.). For this study, all isolates were screened for their ability to grow on SDA (Difco Laboratories, Detroit, Mich.) at 45°C for 48 h. Isolates that failed to grow at 45°C were retested for germ tube production in serum after 3 h of incubation at 37°C. Germ-tube-positive isolates negative for growth at 45°C were then incubated at 37°C on SDA for 24 h for the *F. nucleatum* coaggregation (CoAg) assay, were streaked on the chromogenic medium CHROMagar Candida (CHROMagar, Paris, France) for evaluation of colony color after 48 h of incubation at 37°C, and were tested for sugar assimilation. Isolates confirmed as *C. dubliniensis* were then evaluated for fluconazole susceptibility.

The CoAg ability of the presumptive *C. dubliniensis* isolates with the anaerobic oral bacterium *F. nucleatum* ATCC 49256 was tested as described previously (8). *C. dubliniensis* type strain CD36 and *C. albicans* ATCC 18804 were used as positive and negative controls, respectively.

Assimilation patterns with a variety of carbohydrate substrates were determined by using the API 20C system (bioMerieux Vitek, Inc., Hazelwood, Mo.) according to the manufacturer's instructions.

In vitro fluconazole susceptibility was determined by a macrobroth dilution susceptibility assay carried out according to the method outlined in The National Committee for Clinical Laboratory Standards document M27-A (17). The MIC was recorded as the highest concentration of drug demonstrating at least 80% reduction in turbidity when compared to the positive control tube. Interpretation of results was performed according to the guidelines of Rex et al. (22). A MIC at 48 h of $\leq$8 μg/ml was taken to indicate a sensitive organism. A MIC of $\geq$16 to 32 μg/ml was taken to indicate a dose-dependent susceptible organism. A MIC of $\geq$64 μg/ml was taken to indicate a resistant organism.

In order to test for strain stability in patients with multiple isolates, EK using pulsed-field gel electrophoresis was used. The preparation of intact DNA in agarose plugs was performed by the method of King et al. (9). All isolates meeting the phenotypic criteria of *C. dubliniensis*, a *C. albicans* control, and the *C. dubliniensis* type strain (CD36) were tested as previously described (6).

Epidemiological and clinical information for patients from whom *C. dubliniensis* isolates were recovered was obtained by review of patients' hospital records, with the approval of the University of Maryland.

Of the 1,251 yeast isolates initially identified as *C. albicans*, 15 failed to grow at 45°C. All 15 isolates were confirmed as germ tube positive by repeat testing. In addition, they all produced abundant chlamydospores on corn meal agar; chlamydospores were often attached in triplet or pair arrangement at the end of short, hyperbranching pseudohyphae. All of the 15 isolates produced the characteristic dark green colony color on CHROMagar Candida medium, identical to the color produced by the *C. dubliniensis* type strain CD36 (courtesy of Derek Sullivan) used for comparison.

Sugar-assimilation profiles obtained for 14 of the 15 isolates generated an API 20C biocode (6172130), which corresponded to a very good identification of *C. dubliniensis*. One isolate gave a biocode of 6152130, which also corresponded to a very good identification for *C. dubliniensis*. Multiple isolates recovered from the same patients had identical biocodes. None of the 15 isolates of *C. dubliniensis* assimilated α-methyl-D-glucoside or xylose, the two key sugars that have been used to differentiate *C. dubliniensis* from *C. albicans* (19, 23).

TABLE 1. Characteristics of the *C. dubliniensis* clinical isolates

Isolate[a]	Fluconazole	
	In vitro activity (μg/ml)	Sensitivity
CD-102	0.25	Susceptible
CD-106	0.5	Susceptible
CD-249	<0.125	Susceptible
CD-266	<0.125	Susceptible
CD-316	0.5	Susceptible
CD-598	0.25	Susceptible
CD-798	0.25	Susceptible
CD-839	0.25	Susceptible
CD-993	0.25	Susceptible
CD-1001	0.25	Susceptible
CD-1009	0.5	Susceptible
CD-1063	<0.125	Susceptible
CD-1095	<0.125	Susceptible
CD-1249	16.0	Dose-dependent susceptible
CD-1250	16.0	Dose-dependent susceptible

[a] All isolates were negative for growth at 45°C, produced a dark green colony color on CHROMagar, had API profiles of 6172130 and 6152130, and were 4+ by CoAg assay.

A 4+ visual coaggregation reaction with *F. nucleatum* was observed with all 15 *C. dubliniensis* patient isolates grown at 37°C. No coaggregation occurred with the 37°C-grown *C. albicans* ATCC strain used as a negative control.

Thirteen of the 15 isolates were sensitive (MIC, $\leq$0.5 μg/ml), whereas two isolates recovered from the same patient were dose-dependent susceptible (MIC, 16.0 μg/ml) (Table 1).

Eight to nine DNA bands, including a chromosome-sized band of <1 Mb, were obtained for the five *C. dubliniensis* isolates recovered from two patients by pulsed-field gel electrophoresis, consistent with the reference strain CD36. The variation in the mobilities of the DNA molecules yielded three different EK patterns with the five isolates. Two isolates from the same patient (patient 8; Table 3) had very similar EK patterns, differing only by one band. However, three isolates from the second patient (patient 5; Table 2) had an identical EK pattern which was different from that of the first patient.

Chart review of the patients from whom *C. dubliniensis* isolates were recovered revealed that 10 of the 12 patients were immunocompromised (Tables 2 and 3). Five patients had human immunodeficiency virus (HIV) infection or AIDS, whereas seven patients were HIV negative. *C. dubliniensis* was most commonly isolated from oral or respiratory specimens. None of the patients had evidence of candidal infection at the time of collection.

In this investigation, 15 of 1,251 (1.2%) isolates initially identified as *C. albicans* by the University of Maryland Clinical Microbiology Laboratory were found to be *C. dubliniensis*. The inability of isolates to grow at 45°C proved to be a simple and reliable method for presumptive identification of *C. dubliniensis* (20). However, for proper identification of this species in the clinical laboratory, we propose that all germ-tube-positive yeast isolates that fail to grow at 45°C on SDA be confirmed by a 3-min CoAg assay or by carbohydrate assimilation profiles with the API yeast identification system.

Ten of the 12 patients were confirmed to be clinically immunocompromised; six patients were HIV positive (three of

TABLE 2. Characteristics of HIV-positive patients with *C. dubliniensis* isolated

Patient	Isolate	Isolate source	Sex/age	Diagnosis on admission	Underlying illnesses	Risk factors	Fluconazole therapy
1	CD-249	BRWSH[a]	Female/40	AIDS, CD4[b] = 120, VL[c] > 750,000	Erythema in oropharynx, renal failure	Prednisone, Bactrim (trimethoprim-sulfamethoxazole), broad-spectrum antibiotics	Unknown
2	CD-266	Sputum	Female/45	HIV+, CD4 = 685, VL = 8,755	Pneumonia	Bactrim	Unknown
3	CD-316	BRWSH	Male/41	AIDS, CD4 = 6	Oral thrush, pneumonia	Prednisone, intravenous Bactrim	Diflucan
4	CD-1063	Sputum	Male/46	HIV+, CD4 = 445, VL = 113,619	Pneumonia, end-stage renal failure	Broad-spectrum antibiotics	Unknown
5	CD-1095, CD-1249, CD-1250	BRWSH	Male/30	AIDS, CD4 = 17, VL > 750,000	Kaposi's sarcoma, PCP,[d] cryptococcal meningitis, perianal herpes simplex virus infection	Broad-spectrum antibiotics	Diflucan

[a] Bronchial washing.
[b] CD4, count/mm^3.
[c] VL, viral load (copies/ml).
[d] PCP, *Pneumocystis carinii* pneumonia.

whom had AIDS) and three were receiving chemotherapy for treatment of cancer. Although there was no evidence of candidemia present in these patients, the recovery of *C. dubliniensis* from the cancer patients confirms the report of Meis et al. (12) describing the presence of this species among non-HIV-positive immunocompromised individuals. Two patients were awaiting organ transplantation, one had undergone abdominal surgery and was intubated, and one, other than having been recently hospitalized for pneumonia and having hypoalbuminemia (which could be an indication of malnourishment), had no evident cause for immunosuppression.

Thirteen of the 15 isolates, including two from the same patient (patient 8; Table 3), were highly susceptible to fluconazole (MICs, ≤0.5 μg/ml) (Table 1). Interestingly, although the first of the three isolates recovered from the other patient (patient 5; Table 2) with multiple isolates was highly susceptible to fluconazole (MIC, <0.125 μg/ml), the MICs for the remaining two isolates obtained 3 months later, 1 week apart, were higher, and both isolates were dose-dependent susceptible (MIC, 16.0 μg/ml). The patient's chart indicated prior fluconazole therapy. This observation supports the in vitro development of resistance seen in *C. dubliniensis* isolates following exposure to fluconazole, as reported by Moran et al. (15, 16). This is also supported by strain typing data with electrophoretic karyotyping. Isolates from the two patients with multiple isolates had two EK patterns, each pattern identical for all isolates from the same patient and not found in other patients, demonstrating strain stability among isolates from the same patient.

The 15 isolates came from a variety of body sites; however, the majority were recovered from oral or respiratory sources and none were recovered from blood or deep tissue. In addition, none of the patients had clinical evidence of infection, indicating that the 12 patients were most likely colonized, although infections have been documented by other studies.

TABLE 3. Characteristics of non-HIV-positive patients with *C. dubliniensis* isolated

Patient	Isolate	Isolate source	Sex/age	Diagnosis on admission	Underlying illness or factor	Risk factors	Fluconazole therapy
6	CD-102	Stool	Female/50	Hodgkin's lymphoma, metastatic breast cancer	Prosthetic heart valve	Chemotherapy	Unknown
7	CD-106	Oral	Female/48	Non-Hodgkin's lymphoma	—[a]	Chemotherapy	Unknown
8	CD-993	Unknown	Male/66	Multiple myeloma	*Pseudomonas aeruginosa* bacteremia	Chemotherapy, neutropenia	Unknown
	CD-1009	Oral					
9	CD-598	Ear	Female/66	Recurrent endocarditis	Unknown	Awaiting organ transplant	Unknown
10	CD-1001	Urine	Female/54	End-stage renal failure	Diabetes, urine culture *Candida glabrata*	Awaiting organ transplant	Unknown
11	CD-798	Urine	Female/56	Motor vehicle accident	Subarachnoid hemorrhage, abdominal surgery with skin graft	Tracheostomy, urinary catheter, nasogastric tube	Unknown
12	CD-839	Urine	Female/27	Tylenol toxicity	Morbid obesity	History of pneumonia, hypoalbuminemia	Unknown

[a] —, patient died.

C. dubliniensis has been most frequently implicated in cases of recurrent mucosal candidosis in HIV-infected individuals and most recently in an unusual form of linear gingival erythematous candidosis (3, 24–27, 30). The role of *C. dubliniensis*, however, is not limited to oral candidosis. In 1999, a report by Meis et al. (12) described three cases of candidemia due to *C. dubliniensis* in HIV-negative patients with chemotherapy-induced immunosuppression. Although these cases constitute the first documented evidence of systemic involvement by *C. dubliniensis*, prior cases of candidemia may have been mistakingly attributed to *C. albicans*, the result of the close phenotypic resemblance between these two species. This is well illustrated by a recent survey by Odds et al. (18) that examined the prevalence of *C. dubliniensis* among a large stock collection of yeast isolates originally identified as *C. albicans*, dating back to the early 1970s. Among the 2,589 yeast isolates examined in that survey, 52 isolates were reidentified as *C. dubliniensis*. These findings show that *C. dubliniensis* isolates from clinical material predate the AIDS epidemic and that this species has been isolated in the past from HIV-negative subjects.

In a clinical review of six cases of *C. dubliniensis* in HIV-positive patients, Meiller et al. (11) associated *C. dubliniensis* with AIDS progression, as demonstrated by high viral loads. Most recently, 11 *C. dubliniensis* isolates were recovered from 11 HIV-negative hospitalized patients, confirming the association of this species with colonization and infection in populations other than the HIV infected (21).

Despite the close phenotypic resemblance between *C. dubliniensis* and *C. albicans*, discriminating between these two species in the clinical laboratory should no longer be a difficult task due to the availability of simple and rapid tests that provide reliable identification of *C. dubliniensis*.

As the immunocompromised population continues to grow in numbers, choice of proper medical management and adequate therapy becomes all the more paramount. The recognition of *C. dubliniensis* is necessary not only for therapeutic purposes, but also as a means to determine the incidence and prevalence of this species among the various susceptible populations, as well as to determine its role in disease, especially in invasive and systemic infections.

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REFERENCES

1. **Boerlin, P., F. Loerlin-Petzold, C. Durussel, M. Addo, J.-L. Pagani, J.-P. Chave, and J. Bille.** 1995. Cluster of oral atypical *Candida albicans* isolates in a group of human immunodeficiency virus-positive drug users. J. Clin. Microbiol. **33:**1129–1135.
2. **Coleman, D., M. Rinaldi, K. Haynes, J. Rex, R. Summerbell, E. Anaissie, A. Li, and D. Sullivan.** 1998. Importance of *Candida* species other than *Candida albicans* as opportunistic pathogens. Med. Mycol. **36**(Suppl. 1)**:**156–165.
3. **Coleman, D. C., D. J. Sullivan, D. E. Bennett, G. P. Moran, H. J. Barry, and D. B. Shanky.** 1997. Candidiasis: the emergence of a novel species, *Candida dubliniensis*. AIDS **11:**557–567.
4. **Gilfillan, G. D., D. J. Sullivan, K. Haynes, T. Parkinson, D. C. Coleman, and N. A. R. Gow.** 1998. *Candida dubliniensis*: phylogeny and putative virulence factors. Microbiology **144:**829–838.
5. **Hazen, K.** 1995. New and emerging yeast pathogens. Clin. Microbiol. Rev. **8:**462–478.
6. **Jabra-Rizk, M. A., A. A. M. A. Baqui, J. I. Kelley, W. A. Falkler, Jr., W. G. Merz, and T. F. Meiller.** 1999. Identification of *Candida dubliniensis* in a prospective study of patients in the United States. J. Clin. Microbiol. **37:** 321–326.
7. **Jabra-Rizk, M. A., W. A. Falkler, Jr., W. G. Merz, J. I. Kelley, A. A. M. A. Baqui, and T. F. Meiller.** 1999. *Candida dubliniensis* and *Candida albicans* display surface variations consistent with observed intergeneric coaggregation. Rev. Iberoam. Micol. **16:**8–14.
8. **Jabra-Rizk, M. A., W. A. Falkler, Jr., W. G. Merz, J. I. Kelley, A. A. M. A. Baqui, and T. F. Meiller.** 1999. Coaggregation of *Candida dubliniensis* with *Fusobacterium nucleatum*. J. Clin. Microbiol. **37:**1464–1468.
9. **King, D., J. Rhine-Chalberg, M. A. Pfaller, S. A. Moser, and W. G. Merz.** 1995. Comparison of four DNA-based methods for strain delineation of *Candida lusitaniae*. J. Clin. Microbiol. **33:**1467–1470.
10. **McCullough, M., B. Ross, and P. Reade.** 1995. Characterization of genetically distinct subgroup of *Candida albicans* strains isolated from oral cavities of patients infected with human immunodeficiency virus. J. Clin. Microbiol. **33:**696–700.
11. **Meiller, T. F., M. A. Jabra-Rizk, A. A. M. A. Baqui, J. I. Kelley, V. I. Meeks, W. G. Merz, and W. A. Falkler, Jr.** 1999. Oral *Candida dubliniensis* as a clinically important species in HIV-seropositive patients in the United States. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. **88:**573–580.
12. **Meis, J. F. G. M., M. Ruhnke, B. E. De Pauw, F. C. Odds, W. Siegert, and P. E. Verweij.** 1999. *Candida dubliniensis* candidemia in patients with chemotherapy-induced neutropenia and bone marrow transplantation. Emerg. Infect. Dis. **5:**150–153.
13. **Moore, W. E. C., L. V. Holdeman, E. P. Cato, et al.** 1985. Comparative bacteriology of juvenile periodontitis. Infect. Immun. **48:**507–519.
14. **Moore, W. E. C., L. V. Holdeman, E. P. Cato, R. M. Smibert, J. A. Burmeister, and R. R. Ranney.** 1982. Bacteriology of moderate (chronic) periodontitis in mature human adults. Infect. Immun. **41:**510–515.
15. **Moran, G. P., D. Sanglard, S. M. Donnelly, D. Shanley, D. J. Sullivan, and D. C. Coleman.** 1998. Identification and expression of multidrug transporters responsible for fluconazole resistance in *Candida dubliniensis*. Antimicrob. Agents Chemother. **42:**1819–1830.
16. **Moran, G. P., D. J. Sullivan, M. C. Henman, C. E. McCreary, B. J. Harrington, D. B. Shanley, and D. C. Coleman.** 1997. Antifungal drug susceptibilities of oral *Candida dubliniensis* isolates from human immunodeficiency virus (HIV)-infected and non-HIV-infected subjects and generation of stable fluconazole-resistant derivatives in vitro. Antimicrob. Agents Chemother. **41:**617–623.
17. **National Committee for Clinical Laboratory Standards.** 1997. Reference method for broth dilution antifungal susceptibility testing of yeasts. Approved standard M27-A. National Committee for Clinical Laboratory Standards, Wayne, Pa.
18. **Odds, F. C., L. Van Nuffel, and G. Dams.** 1998. Prevalence of *Candida dubliniensis* isolates in a yeast stock collection. J. Clin. Microbiol. **36:**2869–2873.
19. **Pincus, D. H., D. C. Coleman, W. R. Pruitt, A. A. Padhye, I. F. Salkin, M. Geimer, A. Bassel, D. J. Sullivan, M. Clarke, and V. Hearn.** 1999. Rapid identification of *Candida dubliniensis* with commercial yeast identification systems. J. Clin. Microbiol. **37:**3533–3539.
20. **Pinjon, E., D. Sullivan, I. Salkin, D. Shanley, and D. Coleman.** 1998. Simple, inexpensive, reliable method for differentiation of *Candida dubliniensis* from *Candida albicans*. J. Clin. Microbiol. **36:**2093–2095.
21. **Polacheck, I., J. Strahilevitz, D. Sullivan, S. Donnelly, I. F. Salkin, and D. C. Coleman.** 2000. Recovery of *Candida dubliniensis* from non-human immunodeficiency virus-infected patients in Israel. J. Clin. Microbiol. **38:**170–174.
22. **Rex, J. H., M. A. Pfaller, J. N. Galgiani, M. S. Bartlett, A. Espinel-Ingroff, M. A. Ghannoum, M. Lancaster, F. C. Odds, M. G. Rinaldi, T. J. Walsh, and A. L. Barry.** 1997. Development of interpretive breakpoints for antifungal susceptibility testing: conceptual framework and analysis of in vitro/in vivo correlation data for fluconazole, intraconazole, and *Candida* infections. Clin. Infect. Dis. **24:**235–247.
23. **Salkin, I. F., W. R. Pruitt, A. A. Padhye, D. Sullivan, D. Coleman, and D. H. Pincus.** 1998. Distinctive carbohydrate assimilation profiles used to identify the first clinical isolates of *Candida dubliniensis* recovered in the United States. J. Clin. Microbiol. **36:**1467.
24. **Sullivan, D., D. Bennett, M. Henman, P. Harwood, S. Flint, F. Mulcahy, D. Shanley, and D. Coleman.** 1993. Oligonucleotide fingerprinting of isolates of *Candida* species other than *C. albicans* and of atypical *Candida* species from human immunodeficiency virus-positive and AIDS patients. J. Clin. Microbiol. **31:**2124–2133.
25. **Sullivan, D., and D. Coleman.** 1997. *Candida dubliniensis*: an emerging opportunistic pathogen. Curr. Top. Med. Mycol. **8:**15–25.
26. **Sullivan, D., and D. Coleman.** 1998. *Candida dubliniensis*: characteristics and identification. J. Clin. Microbiol. **36:**329–334.
27. **Sullivan, D., K. Haynes, J. Bille, P. Boerlin, L. Rodero, S. Lloyd, M. Henman, and D. Coleman.** 1997. Widespread geographic distribution of oral *Candida dubliniensis* strains in human immunodeficiency virus-infected individuals. J. Clin. Microbiol. **35:**960–964.
28. **Sullivan, D., T. Westerneng, K. A. Haynes, D. Bennett, and D. Coleman.** 1995. *Candida dubliniensis* sp. nov.: phenotypic and molecular characterization of a novel species associated with oral candidiasis in HIV-infected individuals. Microbiology **141:**1507–1521.
29. **Sullivan, D. J., G. Moran, S. Donnelly, S. Gee, E. Pinjon, B. McCartan, D. B. Shanley, and D. C. Coleman.** 1999. *Candida dubliniensis*: an update. Rev. Iberoam. Micol. **16:**72–76.
30. **Velegraki, A., and M. Logotheti.** 1998. Presumptive identification of an emerging yeast pathogen: *Candida dubliniensis* (sp.nov.) reduces 2,3,5-triphenyltetrazolium chloride. FEMS Immunol. Med. Microbiol. **20:**239–241.
31. **Velegraki, A., O. Nicolatau, M. Theodoridou, G. Mostrou, and N. J. Legakis.** 1999. Pediatric AIDS-related linear gingival erythema: a form of erythematous candidiasis. J. Oral Pathol. Med. **28:**178–182.