



Knowledge, Attitude and Predisposing Risk Factors of Non-Albican Vulvovaginal Candidiasis among Symptomatic Women in Port Harcourt

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Authors' contributions

This work was carried out in collaboration between both authors. Authors KTW and JAI designed the study, wrote the protocol and first draft of the manuscript while author JAI performed the statistical analysis, managed the analyses of the study and the literature search. Both authors read and approved the final manuscript.

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ABSTRACT

Background: The profile of non *Candida albicans* has risen greatly as a causative agent of Vulvovaginal candidiasis. However, little is known about the predisposing factors or the contributory attitude of those affected. This has made prevention and control of these agents difficult and for a long time, had been neglected because of the assumption that *Candida albicans* causes all or almost all cases of Vulvovaginal candidiasis.

Aim: To determine the knowledge, attitude and risk factors predisposing female patients to the acquisition of Non-*Candida albicans* vulvovaginitis in Port Harcourt using questionnaires, mycological culture technique, and germ tube testing.

Materials and Methods: High vaginal swab (HVS), demographic data and epidemiological risk factors were collected from 247 respondents with symptomatic vulvovaginal Candidiasis. Germ tube testing method was used to differentiate between albicans and non *Candida albicans*. The data was analyzed using the SPSS version 20. Association between variables was compared by using the

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Chi-square (χ^2) test and level of significant was set at $P < 0.05$.

Results: The prevalence of non *Candida albicans* among symptomatic women with Vulvovaginal Candidiasis was 76.68%, the notable associated risk factors elicited from the study were inappropriate antifungal used which was found among 56.03% of the total non *Candida albicans* positive respondents this was statistically significant another was poor health seeking behavior, as much as 41.0% of the positive respondent treated their previous VVC at patent medicine store also was significant statistically $P < 0.005$ while the poor knowledge of etiology of VVC was also outrageously high with as much as 50.86% of positive respondent believed VVC was a toilet infection, while 20.68% have no knowledge of the etiology and 10.34%, 8.62%, 9.48% respectively attributed VVC etiology to sexual activity, poor hygiene and ovulation.

Conclusion: The associated risk factors of this high prevalence of non *Albican candida* in the study are common practice in our environment, hence there is need for coordinated health education to create adequate awareness to mitigate against this rising profile of non *Candida albicans* in our environment.

Keywords: Vulvovaginal candidiasis; *Candida albicans*; non *Candida albican*; Port Harcourt.

1. INTRODUCTION

Vulvovaginal Candidiasis (VVC) is referred to as signs and symptoms of inflammation of the vulva and vagina in the presence of *Candida* species (*Candida albicans* and non *Candida albicans*) and in the absence of other infectious etiology [1]. However, *Candida* species especially the *Candida albicans* can be isolated in the vaginal tracts of 20 to 30% of healthy asymptomatic women at any single point in time and in up to 70% of women if followed up over a 1-year period [2]. *Candida* species as normal flora are incapable of causing infection except when enabling environment is created. Such environment includes distortion of the host's defenses especially cellular defense, the body physiology or displacement of other normal flora [3]. *Candida* infection most often occurs when the balance between colonization and the host defense is temporarily disturbed, hence infection such as VVC, which can be sporadic or often associated with the presence of a known risk factor are common in such a situation [2]. Generally, VVC is classified into uncomplicated and complicated. Uncomplicated VVC is characterized by sporadic or infrequent occurrence of mild to moderate disease caused mainly by *C. albicans* in immunocompetent women, while the complicated VVC includes VVC caused by non-*C. albicans*, those associated with pregnancy or other concurrent conditions such as uncontrolled diabetes or immunosuppression and recurrent VVC (RVVC). [2].

Historically, *Candida albicans* has been implicated in all cases of VVC [4], however, over the last decade, there have been various

documented evidences demonstrating increase in the frequency of VVC caused by non- *Candida albicans* species [5,6]. Many of these studies showed inverse in incidences of the etiology of VVC in favour of non-*C. albicans* [7] Which supported the assertions of Powell *et al* that non albican yeast caused clinically significant in approximately half of patient with VVC [8] A worldwide review study demonstrated a sharp decline in incidence of *Candida albicans* in VVC from 90% to 70% or even less over the years [9]. In North America, many European and Asian countries, several studies have shown a general shift from predominance of *C. albicans* in VVC to non-*Candida albicans*, notably *C. glabrata*, *C. parapsilosis*, *C. tropicalis* and *C. krusei* [10,11,12]. This shift has been attributed to both host and behavioral factors such as uncontrolled diabetes, prolong antibiotic exposure, contraceptive use and glucocorticoids [13] Deorukhkar *et al.* [14], who also reported 63.4% prevalence of non *Albicans candida* in VVC quoted indiscriminate and empirical antimycotic drugs use as a notable risk factor, 63.3% of non *candida albicans* was also reported in a separate study by Obisesan *et al* with predominant *C glabrata*, which was theorized to have been due breakthrough vaginal infection in women receiving long term low-dose fluconazole maintenance prophylaxis [15].

VVC due to non *C. albicans* is classified as recurrent VVC because treatment is seemingly difficult as the common empirical therapy for the treatment of VVC due to *Candida albicans* could either only suboptimally suppress most species of non-*Candida albicans* or outrightly has no effect, as most of the commonly isolated species of non-*Candida albicans* are known to have

intrinsic or acquired resistant to these common empirical treatment such as fluconazole [1,16].

Several factors have been associated with this change in the epidemiology of *Candida* species in VVC however, indiscriminate and empirical use of broad-spectrum antimicrobials is most commonly identified [17].

This study aimed at identifying risk factors that predispose to the emergence of non-*Candida albicans* in our setting, the attitude and knowledge of the patients about the infections caused by these organisms. The ethical approval for the study was obtained from the University of Port Harcourt.

2. METHODOLOGY

A total of two hundred and forty-seven (247) symptomatic women with clinical details or diagnosis suggestive of vulvovag in it is who reported to the department of medical microbiology department laboratory, University of Port Harcourt teaching hospital, Port Harcourt for High vaginal swab (HVS) collection, were recruited concurrently for this descriptive cross-sectional study. All sexually active symptomatic women of reproductive age from the outpatient clinic of the hospital were included in the study while exclusion criteria includes women on their monthly menstruation, women who are virgins and those who declined consent The minimum sample size required for the study was 247 (given a prevalence of 79.9%) [15] based on sample size calculation given below [18]

$$N=Z^2(p)(q)/d^2$$

A well structured questionnaire and laboratory procedure were used to obtained all relevant data for the study The questionnaire comprises three sections; socio-demographic information, knowledge and practice of infections and its management. Appropriate samples were high vaginal swab (HVS) and this was collected according to the recommended standard protocol [19] by first explaining the procedure and then obtaining informed consent from each of the respondents. The procedure was done with the aid of sterile speculum and swab stick, all samples collected were immediately sent for analysis which included microscopy, culture and germ tube test. The HVS samples were inoculated on Sabouraud dextrose agar (SDA) plate and incubated aerobically at 35-37°C for 24-48 hours.

The agar plates were examined for visible growth after the incubation period, however, any plate that yielded no growth after this incubation period was incubated for additional 2-3 days before discarding as negative [20]. Identification of yeast was done by regarding the colonial morphology of culture isolates on sabouraud dextrose agar, Gram staining and Germ tube for categorization of *Candida* species into *Candida albicans* and non *Albicans candida*.

2.1 Germ Tube Test

The procedure is as follows. Some colonies of the isolated yeast cell was inoculated in human serum in a test tube and incubated at 35-37°C for about 3 hours. A drop of the incubated serum was placed on a grease-free microscope slide and covered with a coverslip and examined under the microscope with x40 objective magnifications for the presence of germ tube [21] *Candida albicans* is germ tube test positive. From the study, all gem tube negative *Candida* species were regarded as non-*C. albicans*.

A well-structured questionnaire was administered to each patient who gave consent to be recruited for the study, the questionnaires were self-administered and each was retrieved before HVS collection but two of the patients who initially gave consent declined HVS collection.

3. RESULTS

A total of 182 candida species were isolated in the study, 64% of this was non- *C. albicans* and 36% belong to the *C. albicans* specie which represented 116 and 66 of the total respondents respectively.

From table 1 shows that 114 of the total respondents do not usually complete their medications for the treatment of previous vulvovaginitis, of this, 65 were positive for non *Albican candida* species representing 46.15% and 56.03% of the total *Candida* and non-*C. albicans* respectively isolated from the study and was statistically significant $P<0.05$

However, only 21.57% of the respondents who routinely complete their medication for previous infection were positive for non-*C. albicans* species and was also statistically significant $P<0.05$ while 65.57% of the isolated *Candida* from respondents who didn't respond to the question were positive for non- *C. albicans*. This was not statistically significant.

Analysis of knowledge and risk factors of candida infections with aid of questionnaires showed that only 24.6% of the respondents who visited clinic/ hospital for treatment for previous VVC were positive for non- *C. albicans* while as much as 41.0% of those access care

from patent medicine stores were positive, both were statistically significant $P < 0.05$. While the other 34.4% of the positive respondents didn't use any form of orthodox treatment, this was however not significant statistically $P < 0.05$ Table 2.

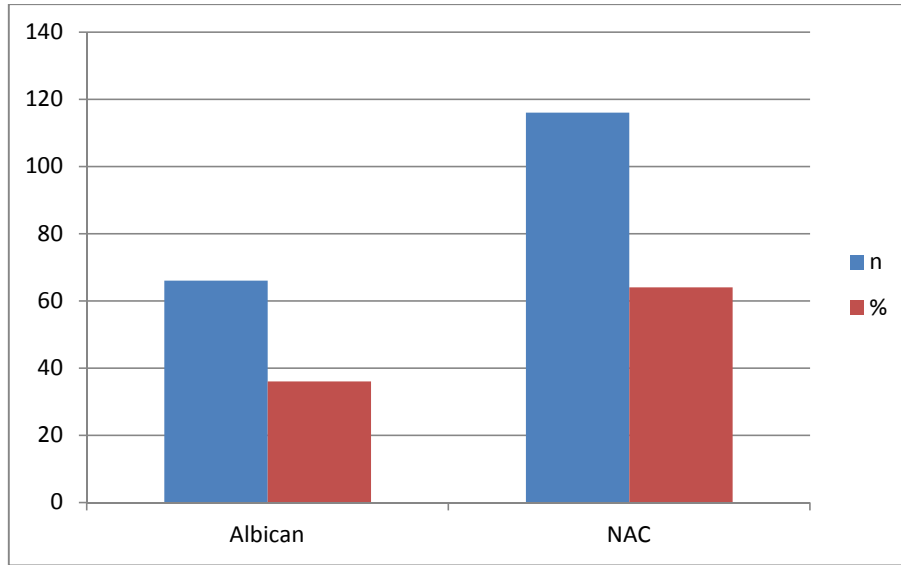


Fig. 1. Percentage and number of *Candida albicans* and non *Candida albicans* isolated

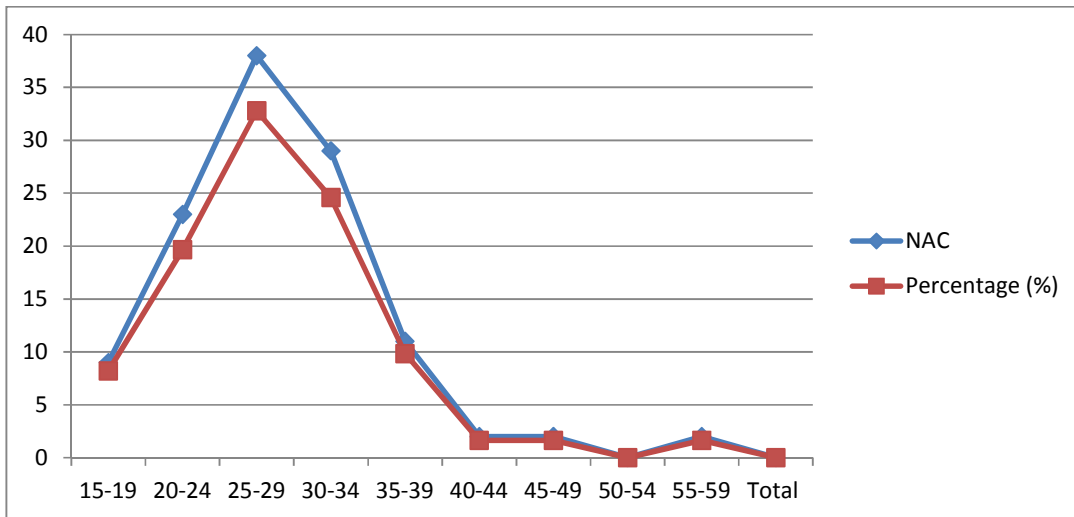


Fig. 2. The relationship of non *Candida albicans* with age of respondents

Table 1. Attitude to medication usage among respondents

Responses	Total respondents	non- <i>C. albicans</i>	<i>Albican candida</i>	χ^2 (p-value)
Complete dosage	51(20.77)	11(9.48%)	28(42.86)	14.18 (0.0001)*
Incomplete dosage	114(46.15)	65(56.03)	17(25.71)	8.11 (0.0044)*
No response	82(33.07)	40(34.48)	21(31.43)	0.08 (0.7462)**

χ^2 : Chi-square statistic. *difference between the groups is statistically significant ($p < 0.05$)

**difference is not statistically significant ($p > 0.05$)

Table 2. Health seeking behavior among respondents

Location	Respondents (n, %)	Positive cases (n, %)	NAC	<i>Candida albicans</i>	χ^2 (p-value)
Hospital	114 (46.56)	74 (40.6)	28 (24.6)	45 (68.18)	17.38 (<0.0001)*
Patent medicine store	66(26.72)	57 (31.3)	48 (41.0)	9(13.64)	7.37 (0.0061)*
No treatment	66 (26.72)	51 (28.1)	40 (34.4)	12 (18.18)	3.28 (0.0698)**
Total	247 (100.0)	182 (100.0)	116 (100.0)	66 (100.0)	

χ^2 : Chi-square statistic. *difference between the groups is statistically significant ($p < 0.05$)

**difference is not statistically significant ($p > 0.05$)

Table 3. Perceived sources of infection among respondents

Response	Primary (n, %)	Secondary (n, %)	Tertiary (n, %)	Total (n, %)
Toilet	8 (33.3)	33 (61.1)	18 (47.36)	59 (50.86)
Sexual activity	1 (3.7)	7 (12.96)	4 (10.53)	12 (10.34)
Poor hygiene	0 (0.0)	5 (9.26)	5 (13.16)	10 (8.62)
Ovulation	0 (0.0)	4 (7.41)	7(18.42)	11 (9.48)
Don't know	15 (63.0)	5 (9.26)	4 (10.53)	24 (20.68)
Total	24 (100.0)	54 (100.0)	38 (100.0)	116 (100.0)

On the knowledge of the source of the candida infections among respondents positive for non-*C. albicans* infections, it was found that 50.86% of the respondents believed that candida infections are caused by poor toilet hygiene while 10.34%, 9.48%, 8.864% respectively attributed the infections to sexual activity, ovulation and poor personal hygiene. 20.68% of the positive respondents don't know the source of the infection, of these, 63% had primary level of education while 9.26% and 10.53% of them had secondary and tertiary level of education respectively. For the total respondents positive for candida infection, 20.6% had primary level of education while 46.5% and 32, 7% has secondary and tertiary level of education respectively Table 3.

4. DISCUSSIONS

Recently, *Candida* species have become major pathogens of numerous human infections not only in immune suppressed individuals but also in many cases of immune competent individuals.

In recent past, *Candida albicans* was regarded as the sole pathogen responsible for all yeast infections [17] but currently several studies have shown increasing prevalence of the non-*C. albicans* species [22,1].

In our study, 63.73% of the total *Candida* species isolated was non-*C. albicans* species, while only

36.26% was *Candida albicans*. This finding is in accordance with the studies done by Mohanty et al. [23], Kumari et al. [24] and Sachin et al. [16] with a prevalence of 64.8%, 67.6% and 66.3% respectively. This further supports the assertions that there is a shift from the initial predominant *Candida albicans* as the sole pathogen of vulvovaginal candidiasis to non *Albican candida* species [15]. This observations may be due to widespread self medication of the canter antifungal agents leading to suboptimal dosages and acquisition of resistant among *Candida* species [25]. In the age- non- *C. albicans* prevalence distributions, the modal age range of prevalence was 20-24 years representing 32.79% closely followed by age range 24-29 years with prevalence of 24.59%. This observations was similar to the finding of Onourah et al. [26] who reported incidence of 55% for age range of 20- 30years, this high distribution of non-*C. albicans* within this age group however was probably due to the fact that women in this group are more sexually active, more likely to self-medicate antifungal in suboptimal doses and use various hormonal contraceptives to prevent pregnancy thereby reducing their vaginal immunity [26]. Also in this study, there was notable decreased prevalence in the extremes of ages of the studied population which was similar to the observations of Obisesan et al. [15]. This is may be due to premenopausal or menopausal changes with attendant less sexual activities and reduction in

the use of hormonal contraceptives with resultant decrease level of estrogen and corticoids and thus increased vaginal immunity [26]. One limitation of the study was the use of only germ tube test to differentiate *Candida albican* from non *Candida albicans*, this may have erroneously increased the percentage of *Candida albican* reported as up to 2-3% of all germ tube positive candida species have been identified as *C. dubliniensis* [27].

There was general poor level of awareness about vulvovaginal candidiasis among the women, especially with regard to modes of transmission, health seeking behaviors and attitude to treatment. From the study, it was observed in assessment of the knowledge of the mode of transmission that as much as 53.08% of the respondents believed VVC are toilet infection, this agreed partly with finding of Jumbo et al who had 58.20% in a similar study. When the level of awareness of the etiology of VVC was compared with level of education, 55.08% of the respondents who believed VVC are a toilet infection had secondary level of education while 33 % had tertiary level of education. Though, majority of the respondent have good level of education, yet exhibited high level of ignorance with regard to the etiology of candida infections. This is likely the reflections of the general societal myth about the subject.

Analysis of the health seeking behavior of respondents shown that 40.98% of the positive cases accessed treatment from patent medicine stores for previous infections while 34.43% did not use any orthodox medication and is statistically significant 7.37 (0.0061) at $P < 0.05$. This probably had led to suboptimal dosage of medication or outright wrong medication creating room for selective pressure or resistant [25] In addition, it was also observed that as much as 56.03% of the respondents positive for NAC species were those who usually don't complete their medications and it was also statistically significant $P < 0.05$. This corroborated the report of Sardi et al. [17] that inappropriate use of antifungal predisposes to the emergence of non-*C. albicans*.

5. CONCLUSIONS

Poor health-seeking behaviors', inappropriate and unregulated antimicrobial usage had been identified over the years and in our present study as pivot to the emergence and continuous cause of increasing prevalence of non *Candida albicans*

So there is need for coordinated effort toward awareness creation aiming at attitudinal change among the general public and appropriate government policy for antimicrobial regulation and antibiotics stewardship at all level of health care service delivery.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Pappas PG, Kauffman CA, Andes D, Benjamin DK, Calandra TF, Edwards JE, et al. Clinical practice guidelines for the management of candidiasis; Update by the Infectious Diseases Society of America. *Clinical infectious Diseases*. 2009;48(5): 503-53.
2. Achkar JM, Fries BC, Candida infections of the genitourinary tract, *Clin Microbiol Rev*. 2010;23:253-273.
3. Torn Volk's, *Candida albicans*, cause of most "yeast infections" in humans' *Fungus of the Month*; 1999. Available:<http://TomVolkFungi.net>
4. Singh SI, Treatment of vulvovaginal candidiasis. *Clin Rev*. 2003;136(9):26-30.
5. Ringdahl E. Treatment of recurrent vulvovaginal candidiasis. *Am Fam Physician*. 2000;61(11):3306-3312.
6. Ray D, Goswami R, Banejee U, Dadhwal V, Goswami D, Mandel P, et al. Prevalence of *Candida glabrata* and its response to boric acid vaginal suppositories in comparison with oral fluconazole in patients with diabetes and vulvovaginal candidiasis. *Diabetes Care*. 2007;30(2):312-317.
7. Lass-Flörl C. The changing face of epidemiology of invasive fungal disease in Europe. *Mycoses*. 2009;52(3):197-205.
8. Powell AM, Gracely E, Nyirjesy P. Non-*Albican candida* vulvovaginitis; Treatment experience at a tertiary care vaginitis center. *Journal of Lower Genital Tract Disease*. 2016;20(1):85-89.
9. Hassan MHA, Ismail MA, Shoreit AMMAM. Prevalence of vaginal infection by multidrug resistant *Candida* Species among different ages in Egypt. *AJMR*. 2017;5(4):78-85.
10. Wang FJ, Zhang D, Liu ZH, Wu WX, Bai HH, Dong HY. Species distribution and *in vitro* antifungal susceptibility of

- vulvovaginal *Candida* isolates in China. Chinese Medical Journal 2016;129(10): 1161-65.
11. Chen S, Slavin M, Nguyen Q,Marriott D, Playford EG, Ellis D, et al. Active surveillance of candidemia, Australia. Emerg Infect Dis. 2006;12(10):1508-1516.
 12. Nyirjesy P, Sobel JD. Genital mycotic infections in patients with diabetes. Postgraduate Medicine. 2013;125(3):33-46.
 13. Nnadi DC, Singh S, The prevalence of genital *Candida* species among pregnant women attending antenatal clinic in a tertiary health center in North-West Nigeria, Sahel Med J. 2017;20(1)33-37.
 14. Deorukhkar SC, Saini S, Mathew S, Non-albicans candida infection: An emerging threat; 2014. Available:<http://dx.doi.org/10.1155/201416159585>.
 15. Obisesan OJ, Olowe OA, Taiwo SS, Phenotypic detection of genitourinary candidiasis among sexually transmitted disease clinic attendees in Ladoke Akintola University Teaching hospital, Osogbo, Nigeria, Journal of Environmental and Public Health. 2015;401340. Available:<http://dx.doi.org/10.1155/2015/401340>
 16. Deorukhkar SC, Saini S. Laboratory approach for diagnosis of candidiasis through ages, int. J. curr. Microbiol. App. Sci. 2014;3(1):206-218.
 17. Sardi JC, Scorzoni L, Bernardi T, Fusco-Almeida AM, Mendes Giannini MJ. *Candida* species: Current epidemiology, pathogenicity, biofilm formation, natural antifungal products and new therapeutic options. Journal of Medical Microbiology. 2013;62(1):10-24.
 18. Araoye MO. Sample size determination. Research methodology with statistics for health and social science. Ilorin, Nathadex publishers. 2004;2:115-121.
 19. BSOP 28, investigation of genital tract associated specimen, issue no 4.1 issue date 03:05:05 issue by standard unit evaluation and standard laboratory page 2-33,ref no BSOP 28i4. 2011;142-151.
 20. Naglik JR, Challacombe SJ, Hube B. *Candida albican* secreted aspartate proteinase in virulence and pathogenesis microbe molel Bio Rev. 2003;67(3):40-423.
 21. Menza N. Wanyoike W. Muturi WM. Prevalence of vaginal candidiasis and determination of the occurrence of *Candida* species in pregnant women attending the ante-natal clinic of Thika District Hospital, Kenya. Open Journal of Medical Microbiology. 2013;3(4):1-9.
 22. Miceli MH, Diaz JA, Lee SA. Emerging opportunistic yeast infections. The Lancet Infectious Disease. 2011;11:142-151.
 23. Mohanty S, Xess I, Hasan F, Kapil A, Mittal S, Tolosa JE, Prevalence and susceptibility to fluconazole of *Candida* species causing vulvovaginitis. Indian Journal of Medical Research. 2007;126(3): 216–219.
 24. Kurnara V, BenerjeeT, KumarP, Pandey S. Tilak R. Emergence of non *Albican candida* among candida vulvovaginitis cases and study of their potential virulence factors, from a tertiary center, North India. Indian J Pathol Microbiol. 2013;56(2):144-7.
 25. Magill SS, Shields C, Sears CL, Choti M, Merz WG. Triazole cross-resistance among *Candida* spp. case report, occurrence among bloodstream isolates and implications for antifungal therapy. J Clin Microbiol. 2006;44:529-35.
 26. Onuorah S, Obika I, Okafor U. Prevalence of *Candida* species among vaginitis symptomatic pregnant women attending ante-natal clinic of Anambra State University Teaching Hospital, Awka, Nigeria. Bioengineering and Bioscience. 2015;3(2):23-27.
 27. Moran GP, Coleman DC, Sullivan DJ: *Candida albican* versus *Candida dubliniensis*. Why is *C. albicans* more pathogenic? IJM; 2012. Available:<http://dx.doi.org/10.1155/2012/205921>

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