MEDWIN PUBLISHERS
Committed to Create Value for Researchers

Open Access Journal of Microbiology & Biotechnology
ISSN: 2576-7771

Candida Species Survival Strategies: New Approaches

Dias VC*

Department of Parasitology, Microbiology and Immunology, Federal University of Juiz de Fora, Brazil

***Corresponding author:** Vanessa Cordeiro Dias, Department of Parasitology, Institute of Biological Sciences, Federal University of Juiz de Fora, Juiz de Fora, s/n – University Campus Bairro São Pedro - ZIP Code: 36036-330, Brazil, Tel: +55 (32) 2102-3213; Email: vancdias2@hotmail.com

Review Article

Volume 7 Issue 2

Received Date: April 26, 2022

Published Date: May 17, 2022

DOI: 10.23880/oajmb-16000226

Abstract

The incidence of fungal infections has increased significantly in recent years, motivated especially by COVID-19. *Candida* yeasts are responsible for most of these infections. This commensal yeast is easily found in the oral mucosa, gastrointestinal tract, urogenital tract, skin and mucous membranes of healthy individuals. Under certain circumstances, when there is a disruption of the biological balance, there may be an increase in the multiplication and invasion of host tissues by these microorganisms. The expression of certain virulence factors such as polymorphism, adhesins, proteases and phospholipases and the formation of microbial biofilms, facilitate tissue penetration and confer greater pathogenicity to these yeasts. This mini-review provides information on the current state of knowledge about virulence, including antifungal resistance of *Candida* species.

Keywords: *Candida*; Survival; Virulence; Resistance

Introduction

Candida species, e.g., *Candida albicans, Candida glabrata, Candida dubliniensis*, and *Candida parapsilosis*, are colonizers of human host. Under certain circumstances these species can cause infections ranging from superficial to life-threatening disseminated candidiasis [1,2]. However, discovered in 2009 in Japan, the species *C. auris*, which carries numerous virulence factors, such as multiple resistances to antifungal agents, does not have, so far, scientific proof of colonization in humans, only infection [3]. Many factors contribute to the occurrence of fungal infections, in particular those caused by *Candida.* It is possible to list: cancer, co-infections (COVID-19), changes in the immune system (whether acquired or congenital), invasive medical procedures, catheters, mechanical ventilation, prolonged, hyperglycemia, treatment with extensive antimicrobial factors, prolonged use of antimicrobials, among others [4]. The success of *C. albicans*, the most prevalent and best studied *Candida* species, as both human pathogen and commensal depends on its genetic, biochemical, and morphological flexibility which facilitates adaptation to a wide range of biotic and abiotic environments.Furthermore, in many host niches *Candida* cells coexist with members of the human microbiota. The resulting fungal–bacterial interactions have a major influence on the success of *C. albicans* as commensal and also influence disease development and outcome [1].

The ability of *Candida* species to infect the host is supported by multiple virulence factors, which include the ability to transition morphologically between yeast forms and hyphae (polymorphism), formation of an extracellular polymer matrix (biofilm/slime) and the expression of adhesins and several hydrolytic enzymes, such as hemolysins, proteases and phospholipases [5-7]. The polymorphism guarantees the fungus survival in stressful conditions in the environment (osmotic changes, dehydration and temperature changes), protects the cell from the immune defense of the

host and is responsible for its adhesion in living tissues [8]. Adhesins are also involved in the process of adhering to the host tissue, facilitating colonization, penetration and invasion [8]. Growing in biofilm, microorganisms may present resistance to antifungals used in medical routine, contributing to therapeutic failure [7]. Hydrolytic enzymes promote the rupture of the host cell membrane, facilitating invasion to the host cell, including mucous membranes and blood vessels. They also act by avoiding the host's immune response [9].

Among clinical isolates of *Candida*, antifungal resistance is an important virulence factor. Although some species, such as *C. krusei*, present natural or intrinsic resistance to antifungals used in routine medicine, an increase in acquired resistance to these drugs has been observed [2]. This phenomenon may be associated with formation of biofilms, hindering or preventing the action of antifungals, presence of genes encoding enzymes capable of blocking the pharmacological action of these drugs, production of efflux pump, alteration of drug binding target, among others [7-10]. However, this therapeutic failure observed in vivo may also be related to other factors, such as: natural selection; indiscriminate/irrational use of antifungals; inaccurate laboratory diagnosis; in addition to prescribing inappropriate drug therapy [11].

Conclusion

Given the above, it is extremely important to know the factors and mechanisms of pathogenicity in *Candida* species, because these microorganisms are capable of causing infections ranging from superficial to systemic and life-threatening. As knowledge about pathogenicity in *Candida* increases, so does the possibility of prevention, and thus opportunities are designed to develop diagnostic and therapeutic tools.

References

1. Polke M, Hube B, Jacobsen ID (2015) Candida survival strategies. Adv Appl Microbiol 91: 139-235.

2. Silva S, Negri M, Henriques M, Oliveira R, Williams DW, et al. (2012) *Candida glabrata*, *Candida parapsilosis* and *Candida tropicalis*: biology, epidemiology, pathogenicity and antifungal resistance. FEMS Microbiol Rev 36(2): 288-305.

3. Du H, Bing J, Hu T, Ennis CL, Nobile CJ, et al. (2020) *Candida auris:* Epidemiology, biology, antifungal resistance, and virulence. PLoS Pathog 16(10): e1008921.

4. Silva DL, Lima CM, Magalhães VCR, Baltazar LM, Peres NTA, et al. (2021) Fungal and bacterial coinfections increase mortality of severely ill COVID-19 patients. J Hosp Infect 113: 145-154.

5. Nicholls S, MacCallum DM, Kaffarnik FAR, Selway L, Peck SC, et al. (2011) Activation of the heatshock transcription factor Hsf1 is essential for the fullvirulence of the fungal pathogen *Candida albicans*. Fungal Genet Biol 48(3): 297-305.

6. Pfaller MA, Diekema DJ (2007) Epidemiology of invasive candidiasis: a persistent public health problem. Clin Microbiol Rev 20(1): 133-163.

7. Baillie GS, Douglas LJ (2000) Matrix polymers of Candida biofilms and their possible role in biofilm resistance to antifungal agents. J Antimicrob Chemother 46(3): 397-403.

8. Talapko J, Juzbaši´c M, Matijevi´c T, Pustijanac E, Beki´c S, et al. (2021) *Candida albicans*—The Virulence Factors and Clinical Manifestations of Infection. J Fungi (Basel) 7(2): 79.

9. Wibawa T (2016) The role of virulence factors in *Candida albicans* pathogenicity. J Med Sci 48(1): 58-68.

10. León MGFD, Castro RH, Cuevas EC, Coronel IHG, Aceituno VAV, et al. (2021) Candida glabrata Antifungal Resistance and Virulence Factors, a Perfect Pathogenic Combination. Pharmaceutics 13 (10): 1529.

11. Ha JF, Italiano CM, Heath CH, Shih S, Rea S, et al. (2011) Candidemia and invasive candidiasis: a review of the literature for the burns surgeon. Burns 37(2): 181-195.