

From Commensal to Pathogen: A Review of the Virulence Mechanisms of *Candida albicans* in Oral Candidiasis

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Abstract

Candida albicans is a predominant fungal commensal of the oral cavity that normally maintains a balanced relationship with the host without causing tissue damage. However, alterations in local and systemic host conditions can disrupt this equilibrium, enabling *C. albicans* to transition from a commensal organism to an opportunistic pathogen and cause oral candidiasis. This review aims to provide a comprehensive overview of the major virulence mechanisms that facilitate this commensal-to-pathogen transition, with particular emphasis on adhesion, morphological switching, biofilm formation, epithelial invasion, and host tissue damage. Initial adherence to oral epithelial surfaces is mediated by fungal adhesins, particularly the agglutinin-like sequence proteins and hyphal wall protein 1. Subsequent yeast-to-hyphal transition enhances adhesion, biofilm development, and tissue invasion through highly regulated polarized hyphal growth. Biofilm formation further promotes fungal persistence and resistance to antifungal agents and host immune responses. Epithelial invasion occurs through induced endocytosis and active penetration and is facilitated by fungal invasins and secreted hydrolytic enzymes, particularly secreted aspartyl proteinases. Understanding the coordinated interplay of these virulence mechanisms provides insight into the pathogenesis of oral candidiasis.

Keywords: *Candida albicans*, Oral candidiasis, Commensal, Virulence factors, Biofilm, pathogenicity.

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INTRODUCTION

Candida species are opportunistic fungal organisms that commonly exist as harmless commensals within the normal microbiota of the oral cavity, gastrointestinal tract and other mucosal surfaces. Among them, *Candida albicans* is the most prevalent and pathogenic species associated with oral candidiasis. Although oral colonization is common and occurs in a large proportion of healthy individuals, carriage alone does not invariably result in disease.[1]

Under normal circumstances, a dynamic balance exists between the *Candida albicans*, the resident microbial flora, and host defense mechanisms,

allowing *Candida albicans* to persist without causing tissue damage.[1]

However, alterations in the local or systemic host environment can disrupt this balance and promote the transition of *Candida albicans* from a commensal organism to an opportunistic pathogen. Predisposing factors such as denture use, broad-spectrum antibiotic therapy, corticosteroid inhalers, smoking, xerostomia, diabetes mellitus, immunosuppressive therapy, chemotherapy and human immunodeficiency virus (HIV) infection increase susceptibility to candidal overgrowth and infection. Once favorable conditions arise, *Candida albicans* can proliferate and colonize mucosal surfaces, leading to oral candidiasis. [1,2]

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Virulence Factors of *Candida albicans*

The transition of *Candida albicans* from a harmless commensal organism to an opportunistic pathogen is largely mediated by a diverse array of virulence factors. These factors not only facilitate colonization, survival and persistence within the host but also enable tissue invasion, host cell damage and evasion of immune responses.[3]

Major virulence factors include:

- Adhesion to host surfaces
- Morphological switching between yeast and hyphal forms
- Expression of adhesins and invasins
- Biofilm formation
- Secretion of hydrolytic enzymes

Figure 1 illustrates the transition of *Candida albicans* from a commensal organism to a pathogen

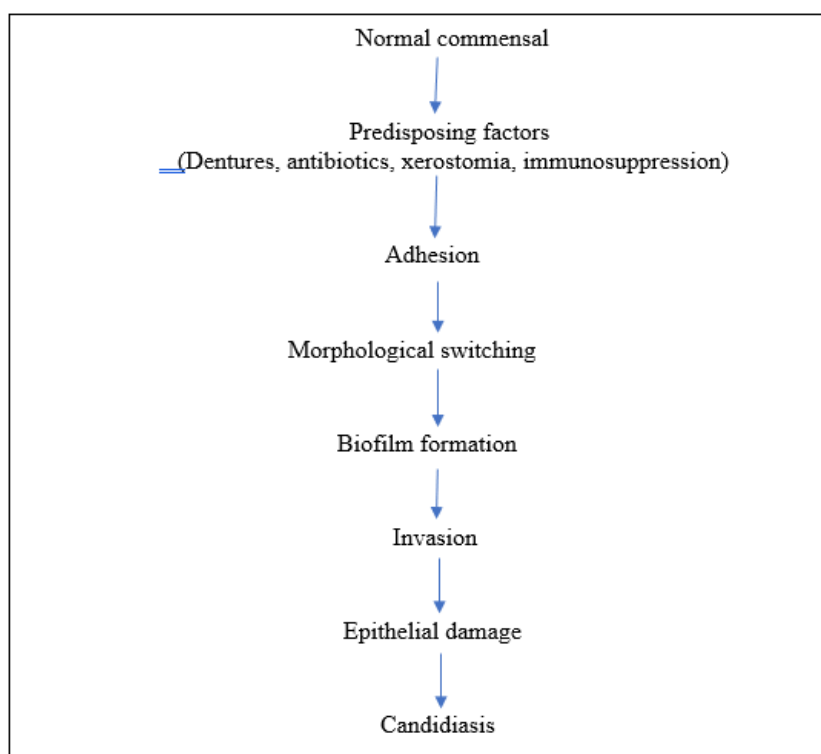


Figure 1: Schematic representation of the transition of *Candida albicans* from a commensal organism to an opportunistic pathogen. Under favourable predisposing conditions, *Candida albicans* adheres to host surfaces, undergoes morphological switching, forms biofilms, invades host tissues, and induces epithelial damage, ultimately leading to candidal infection

Adhesion to Epithelial Cells

Adhesion to epithelial cells is considered the initial and one of the most important steps in the pathogenesis of *Candida albicans* infection. The ability of *Candida* to remain attached to oral epithelial cells enables colonization, survival, and progression toward tissue invasion and infection. For successful colonization and persistence within the oral cavity, it must overcome natural host defense mechanisms such as salivary flow and swallowing, which help remove loosely attached microorganisms from mucosal surfaces. [3,4]

Adhesion is a complex and multifactorial process involving interactions between fungal cell wall components and epithelial cell surface receptors. These interactions may be non-specific, such as hydrophobic and electrostatic forces, or specific, mediated through specialized fungal adhesins. [3,4]

Factors Associated with Adhesion

Candida albicans possesses a diverse group of surface proteins known as adhesins, which facilitate attachment to other *Candida* cells, microorganisms, abiotic surfaces (dentures or other prosthesis) and host cells. In both its commensal and pathogenic states, the organism must initially adhere to host cells or ligands through these surface adhesins in order to establish colonization and infection. [3,4]

Among these, the most important include the agglutinin-like sequence (ALS) family of adhesins and hyphal wall protein 1 (Hwp1).

Agglutinin-Like Sequence (ALS) Family

The ALS family consists of eight glycosylphosphatidyl inositol (GPI)-linked cell surface glycoproteins, namely Als1–7 and Als9, which mediate attachment to epithelial cells, host ligands and other

biological as well as abiotic surfaces. Of these, Als3 is considered particularly important because it is predominantly expressed on the hyphal surface and contributes significantly to epithelial adherence during infection. [5,6]

Hyphal Wall Protein 1 (Hwp1)

Hyphal wall protein 1 (Hwp1) is another important adhesin expressed mainly on the surface of candidal hyphae. Hwp1 functions as a substrate for mammalian transglutaminase enzymes, enabling covalent attachment between *Candida* hyphae and epithelial cells. Studies have shown that strains lacking HWP1 exhibit reduced virulence, highlighting its significant role in candidal adhesion and pathogenesis. [4,7,8]

Morphological Switching

Candida albicans is a polymorphic fungus capable of existing in multiple morphological forms including budding yeast cells, pseudohyphae, and true hyphae. Among these forms, yeast and hyphae are most commonly associated with infection and possess distinct roles in candidal pathogenicity. [4,9]

In response to environmental and host cues, *Candida albicans* can switch from the yeast form to filamentous hyphal growth, which facilitates epithelial penetration and tissue invasion. This reversible transition between yeast and hyphal forms is termed dimorphism and is regarded as one of the major virulence factors of *Candida*.

The yeast form is mainly associated with initial attachment and dissemination, whereas the hyphal form is more invasive and plays a major role in epithelial penetration and tissue destruction. The transition from yeast to hyphal growth occurs in response to various host environmental stimuli that activate multiple regulatory signaling pathways involved in hyphal development. [4,6]

An important environmental signal involved in hyphal induction is contact sensing. Upon contact with epithelial or abiotic surfaces, yeast cells switch to filamentous hyphal growth. On mucosal and other solid surfaces, the developing hyphae can penetrate the underlying substratum and also contribute to biofilm formation. [4,10]

Another important characteristic of hyphal cells is thigmotropism, which refers to directional hyphal growth in response to physical surface cues such as anatomical ridges or grooves. This property enables hyphae to actively navigate along intercellular junctions and invade host tissues more effectively. [4,10,11]

Compared to yeast cells, hyphal forms adhere more strongly to epithelial surfaces because of the expression of hypha-associated adhesins and virulence

factors. Thus, morphological switching enhances the pathogenic potential of *Candida albicans* by promoting adhesion, invasion, and biofilm formation. [3,4,11]

Mechanisms Involved in Hyphal Formation

Environmental Triggers of Hyphal Formation

Hyphal formation in *Candida albicans* is initiated in response to various environmental and host-related stimuli such as elevated temperature, alkaline pH, increased carbon dioxide levels, nutrient deprivation, hypoxia, and contact with solid surfaces. These environmental cues activate multiple intracellular signaling pathways that promote the transition of *Candida* from yeast form to the invasive filamentous hyphal form. [3,6]

Symmetry Breaking and Polarity Establishment

Hyphal growth in *Candida albicans* is characterized by anisotropic growth, a process in which cellular expansion occurs in a non-uniform manner along a defined polarized axis. [12,13]

Hyphal development begins with symmetry breaking and the establishment of a polarity axis, which determines the site of directional growth. Unlike yeast cells, which generally exhibit isotropic or uniform growth, hyphal cells undergo highly polarized growth restricted to a single axis. [12-15]

Germ Tube Formation

Once polarity is established, a small projection, known as a germ tube, emerges from the selected growth site. Germ tube formation represents the earliest stage of hyphal development and marks the initiation of filamentous growth. As the germ tube extends, growth becomes restricted to the apical region, leading to the development of elongated, parallel-walled hyphae. [12,16]

Polarized Tip Growth and Hyphal Elongation

Hyphal elongation occurs through continuous expansion at the apical tip. The hyphal tip possesses a plastic and expandable cell wall, whereas the lateral cell walls remain rigid and elastic. Internal hydrostatic pressure drives extension at the growing tip, thereby facilitating directional filamentous growth and tissue penetration. [12]

Role of Cytoskeleton in Hyphal Growth

The cytoskeleton plays a crucial role in maintaining polarized hyphal growth by directing vesicle transport toward the growing apex. During hyphal elongation, the Golgi apparatus becomes polarized toward the hyphal tip, facilitating the production of secretory vesicles required for apical expansion. [12,17,18]

These vesicles carry membrane components and cell wall-synthesizing enzymes and are transported with actin cables and myosin proteins to the growing tip

critical for continuous tip expansion and maintenance of polarized growth. [12,17]

The transported vesicles accumulate at a specialized apical structure known as the Spitzenkorper, which functions as a dynamic vesicle-organizing center and coordinates vesicle trafficking during hyphal extension. [12,19,20]

These vesicles are subsequently tethered to the plasma membrane through the exocyst complex, enabling localized delivery of membrane and cell wall-synthesizing enzymes at the growing tip. [12,19,20]

Another important regulator of polarized growth is the polarisome complex, a group of proteins localized near the hyphal apex. The polarisome organizes the actin cytoskeleton and maintains cell polarity by directing vesicle transport toward the growing tip. [18-21]

Through these functions, the polarisome maintains the directionality of polarized hyphal growth by coordinating the organization of the actin cytoskeleton and targeted vesicle delivery, thereby preventing random or isotropic expansion of the fungal cell. [18-21]

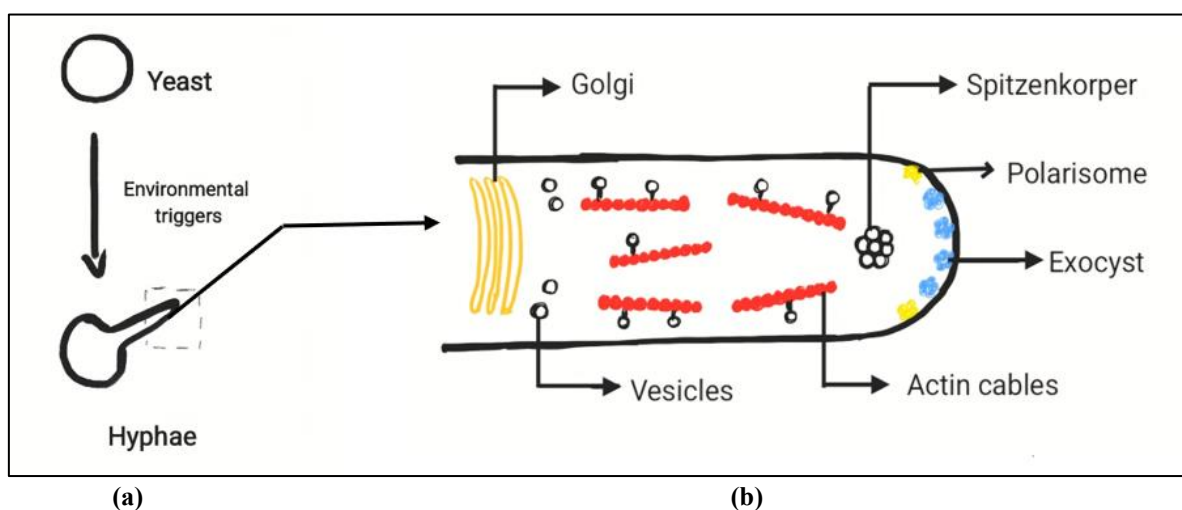


Figure 2: Mechanism of hyphal formation and polarized hyphal growth in *Candida albicans*

(a) Environmental stimuli induce the transition of *Candida albicans* from the yeast form to the filamentous hyphal form. (b) Schematic representation of polarized hyphal growth. Secretory vesicles produced by the Golgi apparatus are transported along actin cables toward the hyphal apex, where they accumulate at the Spitzenkorper. Vesicles are subsequently tethered to the plasma membrane by the exocyst complex, enabling localized delivery of membrane components and cell wall-synthesizing enzymes required for apical extension. The polarisome maintains cell polarity by organizing the actin cytoskeleton and directing vesicle transport, thereby ensuring continuous directional hyphal elongation.

BIOFILM FORMATION

Biofilm formation is considered one of the major virulence attributes of *Candida albicans* and plays a crucial role in the persistence and pathogenicity of candidal infections. [4,22]

Biofilms are structured microbial communities composed of yeast cells, pseudohyphae, and hyphae embedded within a self-produced extracellular matrix. These biofilms can develop on both biotic surfaces such as oral mucosa as well as abiotic surfaces including dentures. Once established, biofilms exhibit increased expression of virulence factors along with marked resistance to antifungal agents, host immune responses, and phagocytosis, thereby contributing to chronic and recurrent infections. [23,24]

Stages of Biofilm Formation

Biofilm formation in *Candida albicans* is a highly regulated sequential process. The development of candidal biofilm can be divided into four major stages:

adherence, initiation, maturation, and dispersal. [24,25] (Figure 3)

Adherence Stage

The initial stage involves attachment of planktonic yeast cells to biotic or abiotic surfaces, resulting in formation of a basal yeast cell layer. [24,25]

Initiation Stage

Following adhesion, the attached yeast cells proliferate and undergo morphological transition from the yeast form to filamentous hyphal forms. Hyphal formation is considered a hallmark of biofilm initiation and contributes significantly to biofilm architecture and stability. [23,24,25]

Maturation Stage

As biofilm development progresses, the fungal cells secrete extracellular polymeric substances (EPS), which accumulate around the microbial community, leading to the formation of a mature three-dimensional

biofilm structure. The mature biofilm consists of polymorphic fungal cells embedded within an extracellular matrix composed predominantly of β -1,3-glucan, β -1,6-glucan, mannans, proteins, and lipids. This matrix provides structural stability and acts as a protective barrier against antifungal agents and host immune mechanisms. [23-25]

Dispersal Stage

During the dispersal stage, round daughter yeast cells are released from the mature biofilm into the

surrounding environment. These dispersed cells can subsequently adhere to new biotic or abiotic surfaces and initiate the formation of secondary biofilms. [26]

Clinically, biofilm dispersal is highly significant because the released cells can colonize new host niches, invade surrounding tissues, and contribute to the development of disseminated candidiasis and systemic infections. [26]

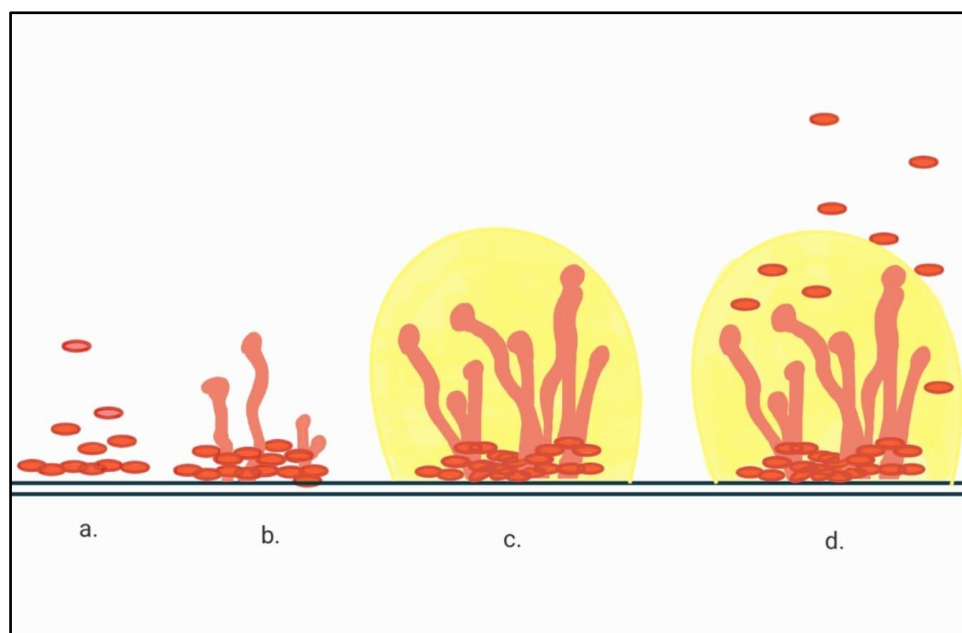


Figure 3: Stages of *Candida albicans* biofilm formation. (a) Adherence of planktonic yeast cells to the surface. (b) Initiation characterized by yeast proliferation and hyphal development. (c) Maturation with accumulation of extracellular polymeric matrix surrounding polymorphic fungal cells. (d) Dispersal of daughter yeast cells from the mature biofilm facilitating colonization of new host niches and biofilm formation at distant sites

Invasion of Epithelial Cells

Candida albicans utilizes two different mechanisms to invade host cells: induced endocytosis and active penetration.[11]

Induced endocytosis

It is a host cell-driven process in which fungal cells stimulate their own uptake by epithelial or endothelial cells. During this process, hyphal cells express specialized surface proteins known as invasins, primarily Als3 and Ssa1, which interact with host cell receptors including E-cadherin and the epidermal growth factor receptor (EGFR)-HER2 complex on epithelial cells, and N-cadherin on endothelial cells. [6,27,28]

These interactions activate host cell signaling pathways (clathrin dependent pathway) and trigger cytoskeletal rearrangements, leading to the formation of pseudopod-like membrane extensions that engulf the fungal hyphae. [28,29]

Active Penetration

Unlike induced endocytosis, active penetration is primarily driven by the fungus itself. This process involves the extension of hyphae, which exert mechanical force to penetrate epithelial barriers. Although the precise molecular mechanisms underlying active penetration remain incompletely understood, fungal adhesion, hyphal elongation, and the generation of physical force and secretion of hydrolytic enzymes including proteases, phospholipases and lipases are considered as contributors.[11,30,31]

Role of Secreted Aspartyl Proteinases (SAPs) in Active Penetration

Candida albicans secretes hydrolytic enzymes, particularly secreted aspartyl proteinases (SAPs), which facilitate tissue invasion by degrading epithelial junctional proteins such as E-cadherin. Among the SAP family, eight members (Sap1–Sap8) are secreted extracellularly, whereas Sap9 and Sap10 remain attached to the fungal cell surface through glycosylphosphatidylinositol (GPI) anchors. [31,32]

This disruption of epithelial adhesion creates intercellular spaces that enable hyphae to penetrate between adjacent epithelial cells. SAPs are capable of hydrolyzing various host extracellular matrix components, including collagen, laminin, fibronectin, and mucin, thereby facilitating deeper tissue invasion. [31,32]

***Candida albicans*-induced epithelial damage**

Once *Candida albicans* hyphae have gained access to ECs or to sub-mucosal layers, the final feature of the invasion process is induction of tissue damage. This might occur via a combination of two distinct mechanisms: necrosis and apoptosis. [3,4]

Necrosis is characterized by mitochondrial swelling and increased plasma membrane permeability and is induced by external factors, including hyphal-associated and secreted virulence factors of *Candida albicans*. In contrast, apoptosis is a regulated form of programmed cell death characterized by an orderly biochemical breakdown of the cell into membrane-bound apoptotic bodies. [3,4]

Role of Candidalysin

Candidalysin is a pore-forming cytolytic peptide toxin produced exclusively by the hyphal form of *Candida albicans* and is considered the principal mediator of epithelial cell damage.[33]

It is derived from the hypha-specific ECE1 (Extent of Cell Elongation 1) gene, whose protein product is proteolytically cleaved by the Kex2 protease into eight peptides, of which only candidalysin possesses cytolytic activity. [33]

Upon secretion, candidalysin inserts into epithelial cell membranes, causing membrane permeabilization, calcium influx, and cellular damage. In addition to its direct cytotoxic effects, candidalysin activates epithelial signaling pathways, resulting in the release of pro-inflammatory cytokines, chemokines, and antimicrobial peptides that initiate the host immune response. Thus, candidalysin plays a dual role by acting as both a virulence factor responsible for epithelial injury and a trigger of mucosal innate immunity. [33]

Interestingly, a study in mice found that regulated candidalysin expression also helps *Candida albicans* establish and maintain oral colonization, suggesting that its role is not limited to epithelial damage during infection [34].

The transition of *Candida albicans* from oral commensal to pathogen is determined by the interaction between fungal activity and host susceptibility, rather than by the presence of a single virulence factor. Adhesion and filamentation can support colonization, whereas altered local conditions or impaired mucosal defenses may permit greater fungal growth, epithelial

invasion, and tissue damage [3,4]. Thus, the expression, timing, and host context of these mechanisms help determine whether *Candida albicans* persists without symptoms or contributes to oral candidiasis.

CONCLUSION

Candida albicans possesses an intricate network of virulence factors that enable its transition from a commensal colonizer to an opportunistic pathogen. The coordinated action of adhesion molecules, morphological switching, biofilm formation, tissue invasion, hydrolytic enzymes, and candidalysin promotes successful colonization, epithelial invasion, immune evasion, and host tissue damage. These virulence mechanisms are tightly regulated in response to environmental and host-derived cues, allowing *Candida albicans* to adapt to diverse host niches and establish persistent infection. Elucidating the molecular basis of these pathogenic processes enhances our understanding of host-fungal interactions and may facilitate the identification of novel diagnostic biomarkers and therapeutic targets for the effective prevention and management of oral candidiasis.

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