Candida albicans

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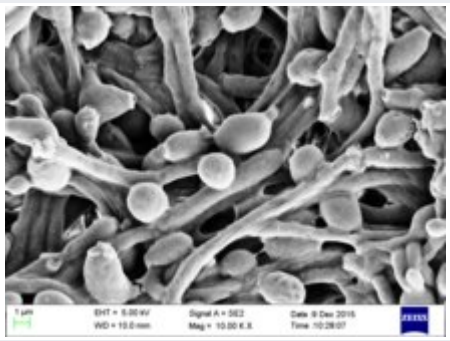
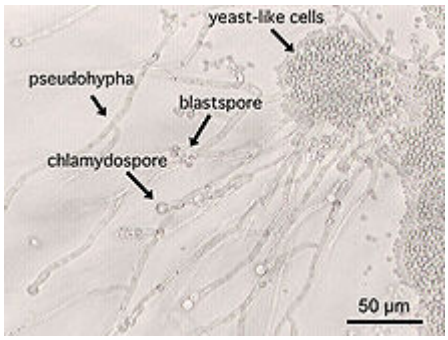
Candida albicans is a type of yeast that is a common member of the human gut flora. It does not proliferate outside the human body.[4] It is detected in the gastrointestinal tract and mouth in 40-60% of healthy adults.[5][6] It is usually a commensal organism, but can become pathogenic in immunocompromised individuals under a variety of conditions.[6][7] It is one of the few species of the *Candida* genus that causes the human infection candidiasis, which results from an overgrowth of the fungus.[6][7] Candidiasis is for example often observed in HIV-infected patients.[8]

C. albicans is the most common fungal species isolated from biofilms either formed on (permanent) implanted medical devices or on human tissue.[9][10] *C. albicans*, together with *C. tropicalis*, *C. parapsilosis* and *C. glabrata*, is responsible for 50–90% of all cases of candidiasis in humans.[7][11][12] A mortality rate of 40% has been reported for patients with systemic candidiasis due to *C. albicans*.[13] Estimates range from 2800 to 11200 deaths caused annually in the USA due to *C. albicans* causes candidiasis.[14]

C. albicans is commonly used as a model organism for biology. It is generally referred to as a dimorphic fungus since it grows both as yeast and filamentous cells. However it has several different morphological phenotypes. *C. albicans* was for a long time considered an obligate diploid organism without a haploid stage. This is however not the case. Next to a haploid stage *C. albicans* can also exist in a tetraploid stage. The latter is formed when diploid *C. albicans* cells mate when they are in the opaque form.[15] The diploid genome size is approximately 29Mb, and up to 70% of the protein coding genes have not yet been characterized.[16] *C. albicans* is easily cultured in the lab and can be studied both *in vivo* as *in vitro*. Depending on the media different studies can be done as the media influences the morphological state of *C. albicans*. A special type of medium is CHROMagar™ Candida which can be used to identify different species of candida.[17][18]

Candida albicans

Candida albicans visualised using scanning electron microscopy. Note the abundant hypal mass.
Scientific classification
Kingdom: Fungi
Division: Ascomycota
Class: Saccharomycetes
Order: Saccharomycetales
Family: Saccharomycetaceae
Genus: *Candida*
Species: ***C. albicans***
Binomial name
Candida albicans (C.P.Robin) Berkhout (1923)
Synonyms
Candida stellatoidea[1] *Monilia albicans* [2] *Oidium albicans*[3]

Contents



Different morphological forms of *Candida albicans*: the scale bar is 50 µm.

Etymology

Candida albicans can be seen as a tautology. Candida comes from the Latin word candidus, meaning white. Albicans itself is the present participle of the Latin word albicō, meaning becoming white. This leads to white becoming white, making it a tautology.

It is often shortly referred to as thrush, candidiasis or candida. More than hundred synonyms have been used to describe *C. albicans*.[2][19] Over 200 species have been described within the candida genus. The oldest reference to thrush, most likely caused by *C. albicans*, dates back to 400 B.C. in Hippocrates' work Of the Epidemics describing oral candidiasis.[20][2]

Genome

Candida albicans growing on Sabouraud agar

The genome of *C. albicans* is almost 16Mb large, 8 chromosomes (28Mb for the diploid stage) and contains 6198 Open Reading Frames (ORFs). 70% of these ORFs have not yet been characterized. The whole genome has been sequenced making it one of the first fungi to be completely sequenced (next to *Saccharomyces cerevisiae* and *Schizosaccharomyces pombe*).[16][8] All open reading frames (ORFs) are also available in gateway adapted vectors. Next to this ORFeome there is also the availability of a GRACE (gene replacement and conditional expression) library to study essential genes in the genome of *C. albicans*.[21][22] The most commonly used strains to study *C. albicans* are the WO-1 and SC5314 strains. The WO-1 strain is known to switch between white-opaque form with higher frequency while the SC5314 strain is the strain used for gene sequence reference.[23]

One of the most important features of the *C. albicans* genome is the high heterozygosity. At the base of this heterozygosity lies the occurrence of numeric and structural chromosomal rearrangements and changes as means of generating genetic diversity by chromosome length polymorphisms (contraction/expansion of repeats), reciprocal translocations, chromosome deletions, Nonsynonymous single-base polymorphisms and trisomy of individual chromosomes. These karyotypic alterations lead to changes in the phenotype, which is an adaptation strategy of this fungus. These mechanisms are further being explored with the availability of the complete analysis of the *C. albicans* genome.[24][25][26]

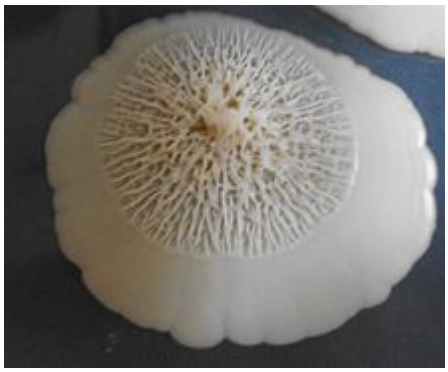
An unusual feature of the *Candida* genus is that in many of its species (including *C. albicans* and *C. tropicalis*, but not, for instance, *C. glabrata*) the CUG codon, which normally specifies leucine, specifies serine in these species. This is an unusual example of a departure from the standard genetic code, and most such departures are in start codons or, for eukaryotes, mitochondrial genetic codes.[27][28][29] This alteration may, in some environments, help these *Candida* species by inducing a permanent stress response, a more generalized form of the heat shock response.[30] However this different codon usage makes it more difficult to study *C. albicans* protein-protein interactions in the model organism *S. cerevisiae*. To overcome this problem a *C. albicans* specific two-hybrid system was developed.[31]

The genome of *C. albicans* is highly dynamic, contributed by the different CUG translation, and this variability has been used advantageously for molecular epidemiological studies and population studies in this species. The genome sequence has allowed for identifying the presence of a parasexual cycle (no detected meiotic division) in *C. albicans*.[32] This study of the evolution of sexual reproduction in six *Candida* species found recent losses in components of the major meiotic crossover-formation pathway, but retention of a minor pathway.[32] The authors suggested that if *Candida* species undergo meiosis it is with reduced machinery, or different machinery, and indicated that unrecognized meiotic cycles may exist in many species. In another evolutionary study, introduction of partial CUG identity redefinition (from *Candida* species) into *Saccharomyces cerevisiae* clones caused a stress response that negatively affected sexual reproduction. This CUG identity redefinition, occurring in ancestors of *Candida* species, was thought to lock these species into a diploid or polyploid state with possible blockage of sexual reproduction.[33]

Morphology

C. albicans exhibits a wide range of different morphological phenotypes due to phenotypic switching and bud to hypha transition. The yeast to hyphae transition is a rapid process and induced by environmental factors. Phenotypic switching is spontaneous, happens at lower rates and in certain strains up to seven different phenotypes are known. The best studied switching mechanism is the white to opaque switching (an epigenetic process). Other systems have been described as well. Two systems (the high frequency switching system and white to opaque switching) were discover by David R. Soll and colleagues.[34][35] Switching in *C. albicans* is often, but not always, influenced by environmental conditions such as the level of CO_2, anaerobic conditions, medium used and temperature.[36]

Yeast to hyphae switching

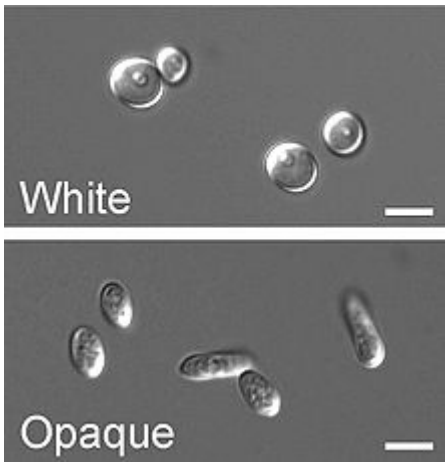


An opaque colony of *C. albicans* growing as yeast like cells with on top filamentous like *C. albicans* cells

Although often referred to as **dimorphic**, C. albicans is in fact polyphenic (often also referred to as pleomorphic).[37] When cultured in standard yeast laboratory medium, *C. albicans* grows as ovoid "yeast" cells. However, mild environmental changes in temperature, CO_2, nutrients and pH can result in a morphological shift to filamentous growth.[38][39] Filamentous cells share many similarities with yeast cells. Both cell types seem to play a specific, distinctive role in the survival and pathogenicity of *C. albicans*. Yeast cells seem to be better suited for the dissemination in the bloodstream while hyphal cells have been proposed as a virulence factor. Hyphal cells are invasive and speculated to be important for tissue penetration, colonization of organs and surviving plus escaping macrophages.[40][41][42] The transition from yeast to hyphal cells is termed to be one of the key factors in the virulence of *C. albicans*, however it is not deemed necessary.[43] When *C. albicans* cells are grown in a medium that mimics the physiological environment of a human host, they grow as filamentous cells (both true hyphae and pseudohyphae). *Candida albicans* can also form Chlamydospores, the function of which remains unknown, but it is speculated they play a role in surviving harsh environments as they are most often formed under unfavorable conditions.[44]

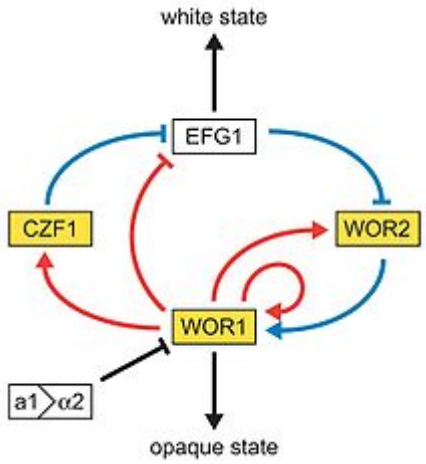
The cAMP-PKA signaling cascade is crucial for the morphogenesis and an important transcriptional regulator for the switch from yeast like cells to filamentous cells is EFG1.[45][46]

High frequency switching



Round, white-phase and elongated, opaque-phase *Candida albicans* cells: the scale bar is 5 μm.

Besides the well studied yeast to hyphae transition other switching systems have been described.[47] One such system is the "high frequency switching" system. During this switching different cellular morphologies (phenotypes) are generated spontaneously. This type of switching does not occur en masse, represents a variability system and it happens independently from environmental conditions.[48] The strain 3153A produces at least seven different colony morphologies.[49][50][51] In many strains the different phases convert spontaneously to the other(s) at a low frequency. The switching is reversible, and colony type can be inherited from one generation to another. While several genes that are expressed differently in different colony morphologies have been identified, some recent efforts focus on what might control these changes. Further, whether a potential molecular link between dimorphism and phenotypic switching occurs is a tantalizing question.[52] Being able to switch through so many different (morphological) phenotypes makes *C. albicans* able to grow in different environments and this both as a commensal and as a pathogen.[53]



In this model of the genetic network regulating the white-opaque switch, the white and gold boxes represent genes enriched in the white and opaque states, respectively. The blue lines represent relationships based on genetic epistasis. Red lines represent Wor1 control of each gene, based on Wor1 enrichment in chromatin immunoprecipitation experiments. Activation (arrowhead) and repression (bar) are inferred based on white- and opaque-state expression of each gene.

In the 3153A strain, a gene called *SIR2* (for silent information regulator), which seems to be important for phenotypic switching, has been found. *SIR2* was originally found in *Saccharomyces cerevisiae* (brewer's yeast), where it is involved in chromosomal silencing—a form of transcriptional regulation, in which regions of the genome are reversibly inactivated by changes in chromatin structure (chromatin is the complex of DNA and proteins that make chromosomes). In yeast, genes involved in the control of mating type are found in these silent regions, and *SIR2* represses their expression by maintaining a silent-competent chromatin structure in this region. The discovery of a *C. albicans SIR2* implicated in phenotypic switching suggests it, too, has silent regions controlled by *SIR2*, in which the phenotype-specific genes may reside. How *SIR2* itself is regulated in *S. cerevisiae* may yet provide more clues as to the switching mechanisms of *C. albicans*.

White to opaque switching

Next to the dimorphism and the first described high frequency switching system *C. albicans* undergoes another high frequency switching process called white to opaque switching, which is another phenotypic switching process in *C. albicans*. It was the second high-frequency switching system discovered in *C. albicans*.[54] The white to opaque switching is an epigenetic switching system.[55] Phenotypic switching is often used to refer to white-opaque switching, which consists of two phases: one that grows as round cells in smooth, white colonies (referred to as white form) and one that is rod-like and grows as flat, gray colonies (called opaque form). This switch from white cells to opaque cells is important for the virulence and the mating process of *C. albicans* as the opaque form is the mating competent form, being a million times more efficient in mating compared to the white type.[56][55][57] This switching between white and opaque form is regulated by the WOR1 regulator (White to Opaque Regulator 1) which is controlled by the mating type locus (MTL) repressor (a1-α2) that inhibits the expression of WOR1.[58] Besides the white and opaque phase there is also a third one: the gray phenotype. This phenotype

shows the highest ability to cause cutaneous infections. The white, opaque and gray phenotypes form a tristable phenotypic switching system. Since it is often difficult to differentiate between white, opaque and gray cells phloxine B, a dye, can be added to the medium.[53]

A potential regulatory molecule in the white to opaque switching is *Efg1p*, a transcription factor found in the WO-1 strain that regulates dimorphism, and more recently has been suggested to help regulate phenotypic switching. *Efg1p* is expressed only in the white and not in the gray cell-type, and overexpression of *Efg1p* in the gray form causes a rapid conversion to the white form.[59][60]

White-GUT switch

A very special type of phenotypic switch is the white-GUT switch (Gastrointestinally-IndUced Transition). GUT cells are extremely adapted to survival in the digestive tract by metabolic adaptations to available nutrients in the digestive tract. The GUT cells live as commensal organisms and outcompete other phenotypes. The transition from white to GUT cells is driven by passage through the gut where environmental parameters trigger this transition by increasing the WOR1 expression.[61][62]

Role in disease

Candida is found worldwide but most commonly compromises immunocompromised individuals diagnosed with serious diseases such as HIV and cancer. *Candida* are ranked as one of the most common groups of organisms that cause nosocomial infections. Especially high risk individuals are patients that have recently undergone surgery, a transplant or are in the Intensive Care Units (ICU),[63] *Candida albicans* infections is the top source of fungal infections in critically ill or otherwise immuncompromised patients.[64] These patients predominantly develop oropharyngeal or thrush candidiasis, which can lead to malnutrition and interfere with the absorption of medication.[65] Methods of transmission include mother to infant through childbirth, people-to-people acquired infections that most commonly occur in hospital settings where immunocompromised patients acquire the yeast from healthcare workers and has a 40% incident rate. Men can become infected after having sex with a woman that has an existing vaginal yeast infection.[63] Parts of the body that are commonly infected include the skin, genitals, throat, mouth, and blood.[66] Distinguishing features of vaginal infection include discharge, and dry and red appearance of vaginal mucosa or skin. *Candida* continues to be the fourth most commonly isolated organism in bloodstream infections.[67]

Superficial and local infections

It commonly occurs, as a superficial infection, on mucous membranes in the mouth or vagina. Once in their life around 75% of women will suffer from vulvovaginal candidiasis (VVC) and about 90% of these infections are caused by *C. albicans*. It however may also affect a number of other regions. For example, higher prevalence of colonization of *C. albicans* was reported in young individuals with tongue piercing, in comparison to unpierced matched individuals.[68] To infect host tissue, the usual unicellular yeast-like form of *C. albicans* reacts to environmental cues and switches into an invasive, multicellular filamentous form, a phenomenon called dimorphism.[69] In addition, an overgrowth infection is considered superinfection, usually applied when an infection become opportunistic and very resistant to antifungals. It then becomes suppressed by antibiotics. The infection is prolonged when the original sensitive strain is replaced by the antibiotic-resistant strain.[70]

Candidiasis is known to cause GI symptoms particularly in immunocompromised patients or those receiving steroids (e.g. to treat asthma) or antibiotics. Recently, there is emerging literature that an overgrowth of fungus in the small intestine of non-immunocompromised subjects may cause unexplained GI symptoms. Small intestinal fungal overgrowth (SIFO) is characterized by the presence of excessive number of fungal organisms in the small intestine associated with gastrointestinal (GI) symptoms. The most common symptoms observed in these patients were belching, bloating, indigestion, nausea, diarrhea, and gas. The underlying mechanism(s) that predisposes to SIFO is unclear. Further studies are needed; both to confirm these observations and to examine the clinical relevance of fungal overgrowth.[6][7][71]

Systemic infections

Systemic fungal infections (fungemias) including those by *C. albicans* have emerged as important causes of morbidity and mortality in immunocompromised patients (e.g., AIDS, cancer chemotherapy, organ or bone marrow transplantation). *C. albicans* often forms biofilms inside the body. Such *C. albicans* biofilms may form on the surface of implantable medical devices or organs. In these biofilms it is often found together with *Staphylococcus aureus*.[72][73][9][10] Such multispecies infections lead to higher mortalities.[74] In addition hospital-acquired infections by *C. albicans* have become a cause of major health concerns.[75][8] Especially once candida cells are introduced in the bloodstream a high mortality, up to 40-60% can occur.[8][76]

Although *Candida albicans* is the most common cause of candidemia, there has been a decrease in the incidence and an increases isolation of non-albicans species of *Candida* in recent years.[77] Preventive measures include keeping a healthy lifestyle including good nutrition, proper nutrition, and careful antibiotic use.

Economic implications

Given the fact that candidiasis is the fourth (to third) most frequent hospital acquired infection worldwide it leads to immense financial implications. Approximately 60000 cases of systemic candidiasis each year in the USA alone lead up to a cost to be between $2–4 billion.[78] The total costs for candidiasis are among the highest compared to other fungal infections due to the high prevalence.[79] The immense costs are partly explained by a longer stay in the intensive care unit or hospital in general. An extended stay for up to 21 more days compared to non infected patients is not uncommon.[80]

Proteins important for pathogenesis

Hwp1

Hwp1 stands for Hyphal wall protein 1. Hwp1 is a mannoprotein located on the surface of the hyphae in the hyphal form of *Candida albicans*. Hwp1 is a mammalian transglutaminase substrate. This host enzyme allows *Candida albicans* to attach stably to host epithelial cells.[81] Adhesion of *Candida albicans* to host cells is an essential first step in the infection process for colonization and subsequent induction of mucosal infection.

Slr1

RNA-binding protein Slr1 was recently discovered to play a role in instigating the hyphal formation and virulence in *C. albicans*.[82]

Candidalysin

Candidalysin is a cytolytic 31-amino acid α-helical peptide toxin that is released during hyphal formation. It contributes to virulence during mucosal infections.[83]

Genetic and genomic tools

Due to his nature as a model organism, being an important human pathogen and the alternative codon usage (CUG translated into serine rather than leucine), several specific projects and tools have been created to study *C. albicans*.[8]

Full sequence genome

The full genome of *C. albicans* has been sequenced and made publicly available in a candida database. The heterozygous diploid strain used for this full genome sequence project is the laboratory strain SC5314. The sequencing was done using a whole-genome shotgun approach.[84][85]

ORFeome project

Every predicted ORF has been created in a gateway adapted vector (pDONR207) and made publicly available. The vectors (plasmids) can be propagated in *E.coli* and grown on LB+gentamicin medium. This way every ORF is readily available in an easy to use vector. Using the gateway system it is possible to transfer the ORF of interest to any other gateway adapted vector for further studies of the specific ORF.[86][22]

CIp10 integrative plasmid

Contrary to the yeast *S. cerevisiae* episomal plasmids do not stay stable in *C. albicans*. In order to work with plasmids in *C. albicans* an integrative approach (plasmid integration into the genome) thus has to be used. A second problem is that most plasmid transformations are rather inefficient in *C. albicans*, however the CIp10 plasmid overcomes these problems and can be used with ease to transform *C. albicans* in a very efficient way. The plasmid integrates inside the RP10 locus as disruption of one RP10 allele does not seem to affect the viability and growth of *C. albicans*. Several adaptations of this plasmid have been made after the original became available.[87][88]

Candida two-hybrid (C2H) system

Due to the aberrant codon usage of *C. albicans* it is less feasible to use the common host organism (*Saccharomyces cerevisiae*) for two-hybrid studies. To overcome this problem a *Candida albicans* two-hybrid (C2H) system was created. The strain SN152 that is auxotrophic for leucine, arginine and histidine was used to create this C2H system. It was adapted by integrating a HIS1 reporter gene preceded by five LexAOp sequences. In the C2H system the bait plasmid (pC2HB) contains the staphylococcus aureus LexA BD, while the prey plasmid (pC2HP) harbors the viral AD VP16. Both plasmids are integrative plasmids since episomal plasmids do not stay stable in *C. albicans*. The reporter gene used in the system is the HIS1 gene. When proteins interact, the cells will be able to grow on medium lacking histidine due to the activation of the HIS1 reporter gene.[31][8]

Microarrays

Both DNA and protein microarrays were designed to study DNA expression profiles and antibody production in patients against *C. albicans* cell wall proteins.[89][90]

GRACE library

Using a tetracycline-regulatable promoter system a gene replacement and conditional expression (GRACE) library was created for 1152 genes. By using the regulatable promoter and having deleted 1 of the alleles of the specific gene it was possible to discriminate between non-essential and essential genes. Of the tested 1152 genes 567 showed to be essential. The knowledge on essential genes can be used to discover novel antifungals.[91]

Application in engineering

Candida albicans has been used in combination with carbon nanotubes (CNT) to produce stable electrically conductive bio-nano-composite tissue materials that have been used as temperature sensing elements[92]

Treatment

Treatment commonly includes:[93]

- amphotericin B, echinocandin, or fluconazole for systemic infections
- Nystatin for oral and esophageal infections
- Clotrimazole for skin and genital yeast infections[94]

Important to note is that similar to antibiotic resistance, resistance to many anti-fungals is becoming a big problem. New anti-fungals have to be developed to cope with this problem since only a limited number of anti-fungals are available.[95][96]

Notable *C. albicans* researchers

- David R. Soll
- Neil A. R. Gow
- Fred Sherman

See also

- Intestinal permeability
- Torula yeast (*Candida utilis*)
- Neonatal infection
- codon usage

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External links

- *Candida* Genome Database
- U.S. National Institutes of Health on the *Candida albicans* genome
- Mycobank data on *Candida albicans*
- Labs working on Candida