



SINCOTRÓN

Aplicaciones en las investigaciones biomédicas.

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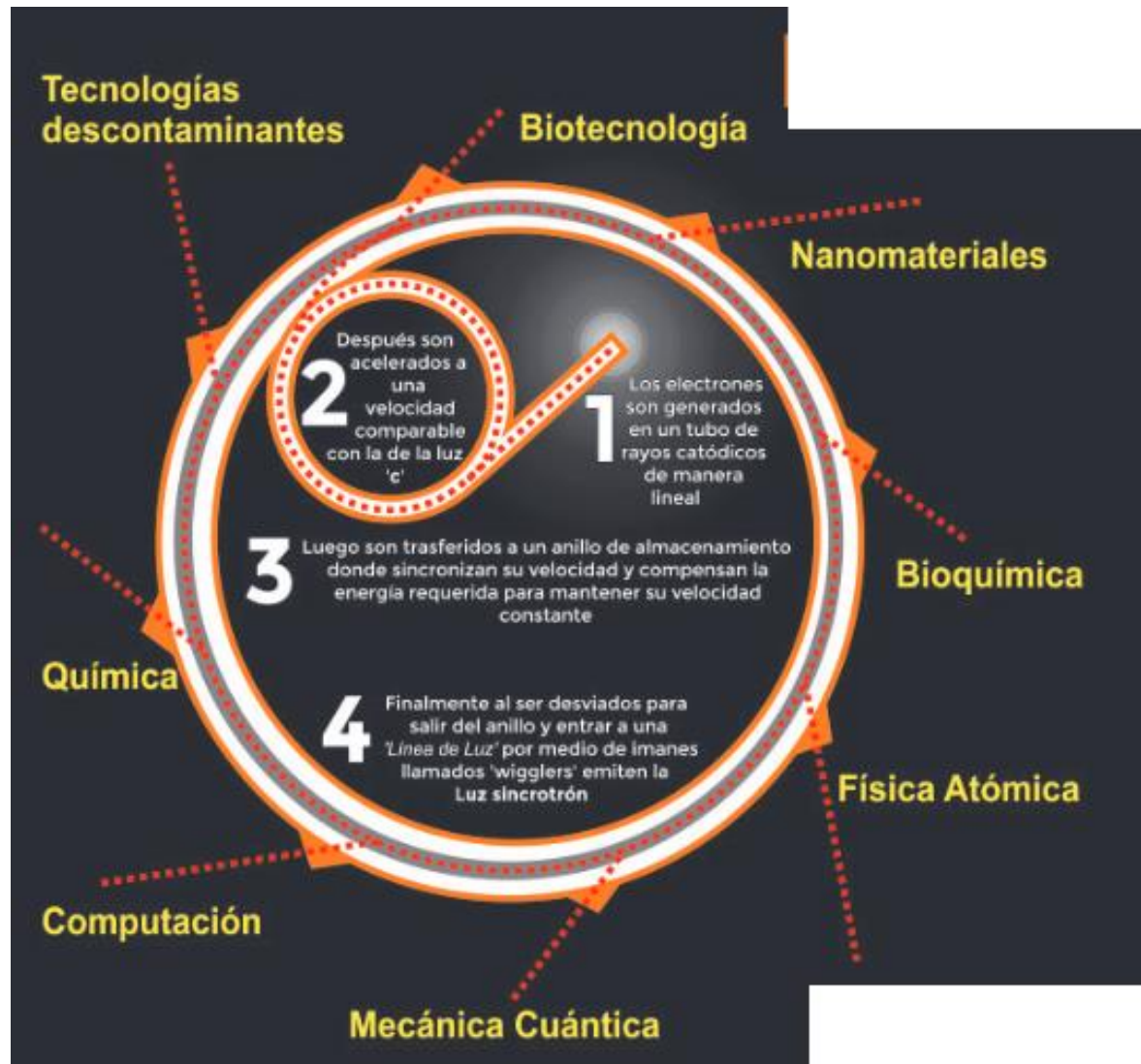


SINCOTRÓN

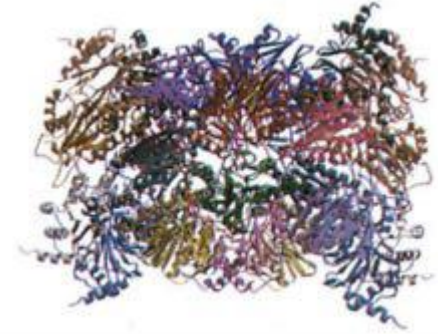
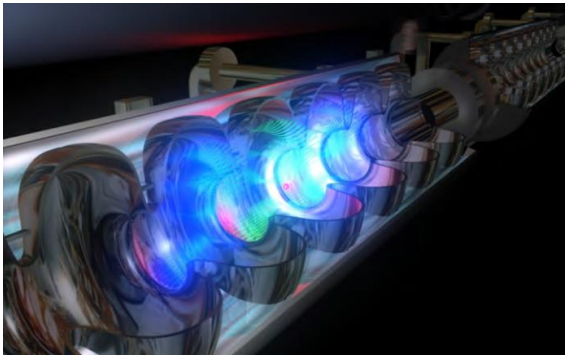
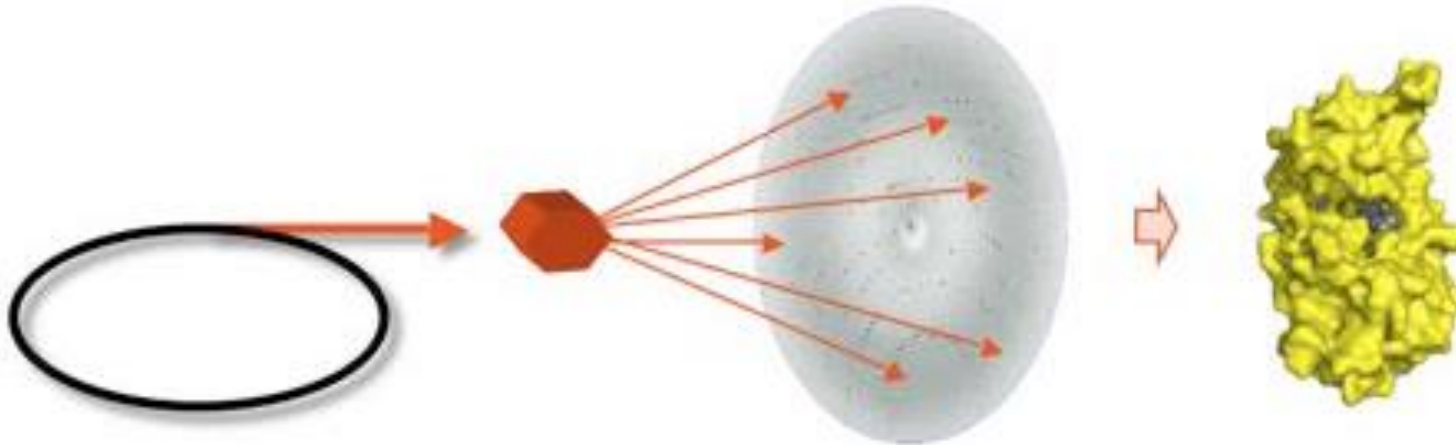


- ✓ Es un tipo de **acelerador de partículas**.
- ✓ Generan **energía extremadamente altas**, creando un haz de electrones que viaja casi a la velocidad de la luz
- ✓ Estas instalaciones aceleran partículas a gran velocidad y las guían dentro de una trayectoria utilizando **campos electromagnéticos**.
- ✓ Permiten **estudiar la materia** (viva o inerte) y sus propiedades del orden del radio atómico.

PARTES DEL SINCOTRÓN



FUNCIONAMIENTO DEL SINCOTRON



APLICACIONES DEL SINCOTRÓN

La luz de sincrotrón se utiliza en múltiples industrias. Destacan los siguientes:

Investigación médica: microbiología, estudio de enfermedades, generación de imágenes de alta resolución y radioterapia contra el cáncer.

Contaminación: detección de partículas contaminantes de agua y polvos cósmicos.

Exploración de minerales/fósiles: análisis rápido de minerales para facilitar el procesamiento de minerales.

Ingeniería: imágenes de procesos industriales en tiempo real y imágenes de alta resolución de grietas y defectos en estructuras.

APLICACIONES DEL SINCOTRÓN

Estudia cristales a nivel macromolecular

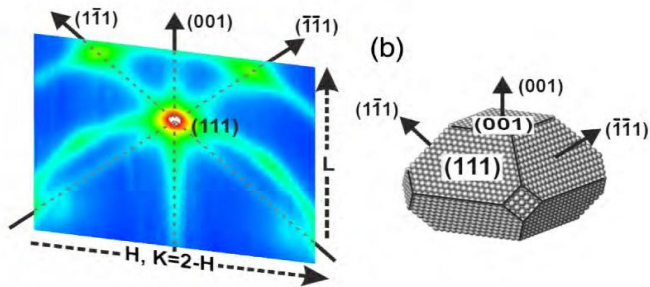
MATERIALES

- ✓ Preciosos.
- ✓ Que no pueden ser reproducidos.
- ✓ Contiene diferentes estructuras mezcladas.
- ✓ Frágiles.

APLICACIONES DEL SINCOTRÓN

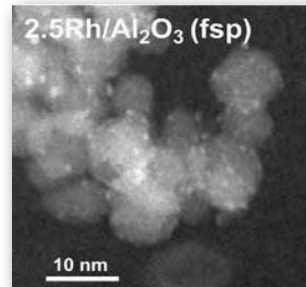
Física

- ✓ Disposición de electrones
- ✓ Películas magnéticas
- ✓ Fotones



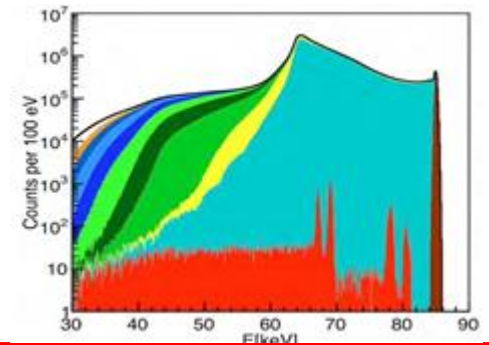
Química

- ✓ Catálisis
- ✓ Reacciones superficiales



Materia blanda

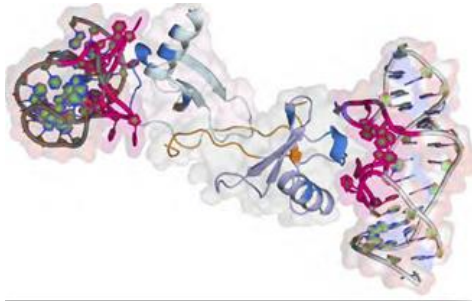
- ✓ Coloides
- ✓ Fase de transición



APLICACIONES DEL SINCOTRÓN

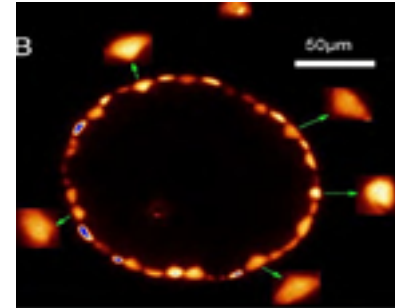
Ciencias de la Vida

- ✓ Estructura de proteínas
 - ✓ Fisiología celular
 - ✓ Diseño de fármacos



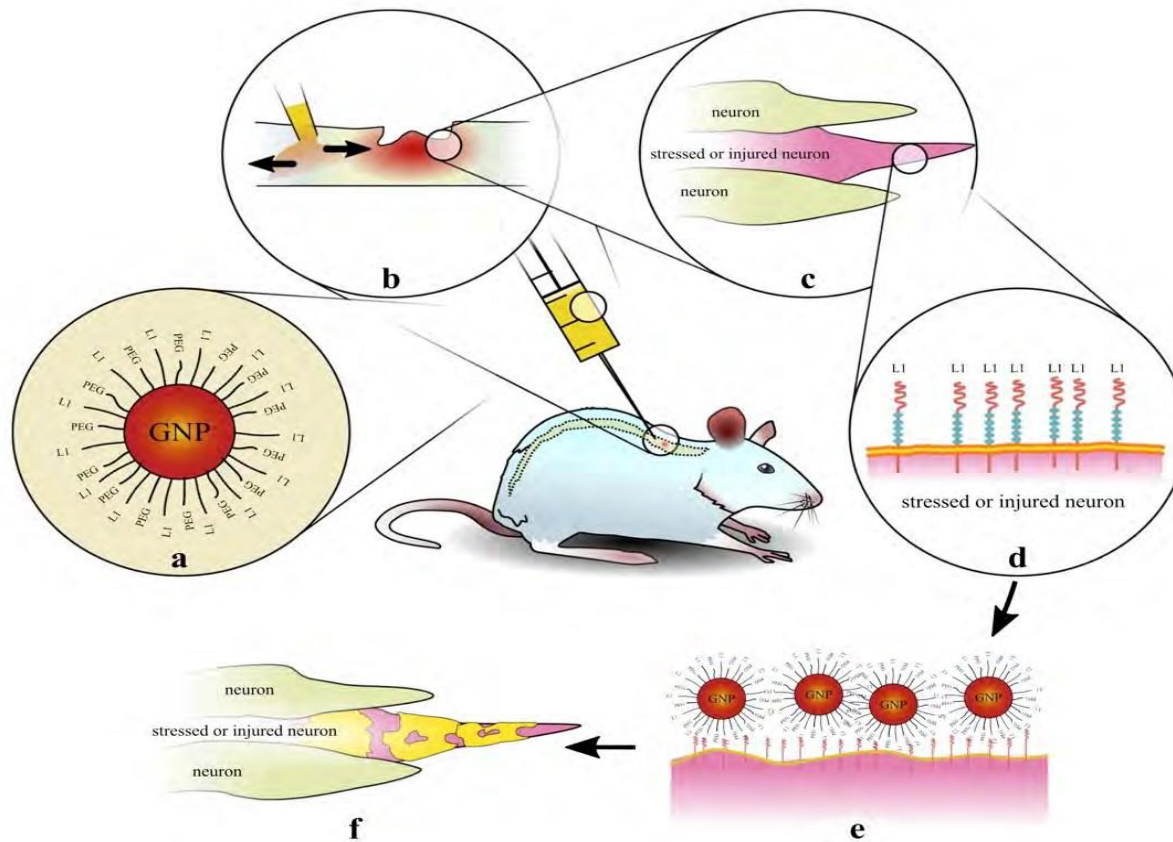
Ciencias medioambientales

- ✓ Contaminación
- ✓ Reacciones



APLICACIONES DEL SINCOTRÓN

DIAGNÓSTICO TEMPRANO DE TUMOR

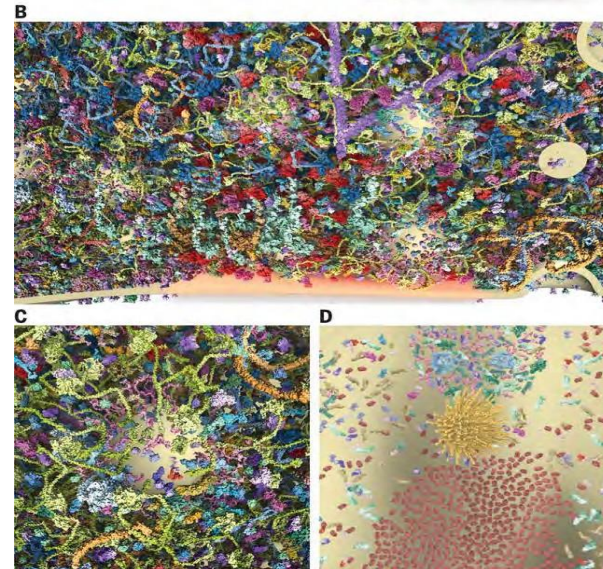
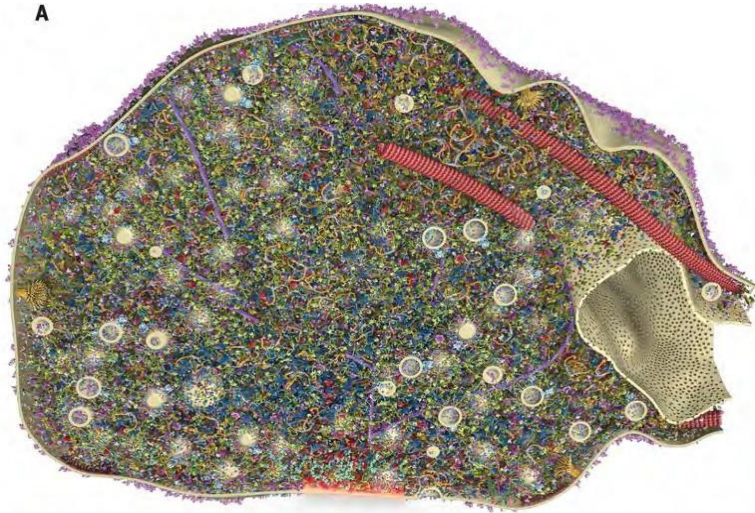


APLICACIONES DEL SINCOTRÓN

ENFERMEDADES INFECCIOSAS

Unión de virus a la superficie de una célula

- ✓ Puede resultar segundos
- ✓ Reacción química que ocurre a nivel cuántico



SINCOTRONES EN EL MUNDO

HAY MÁS DE 60 SINCROTRONES



SINCOTRÓN EN BRASIL

SIRIUS Ciudad de Campinas



SINCOTRÓN EN BRASIL

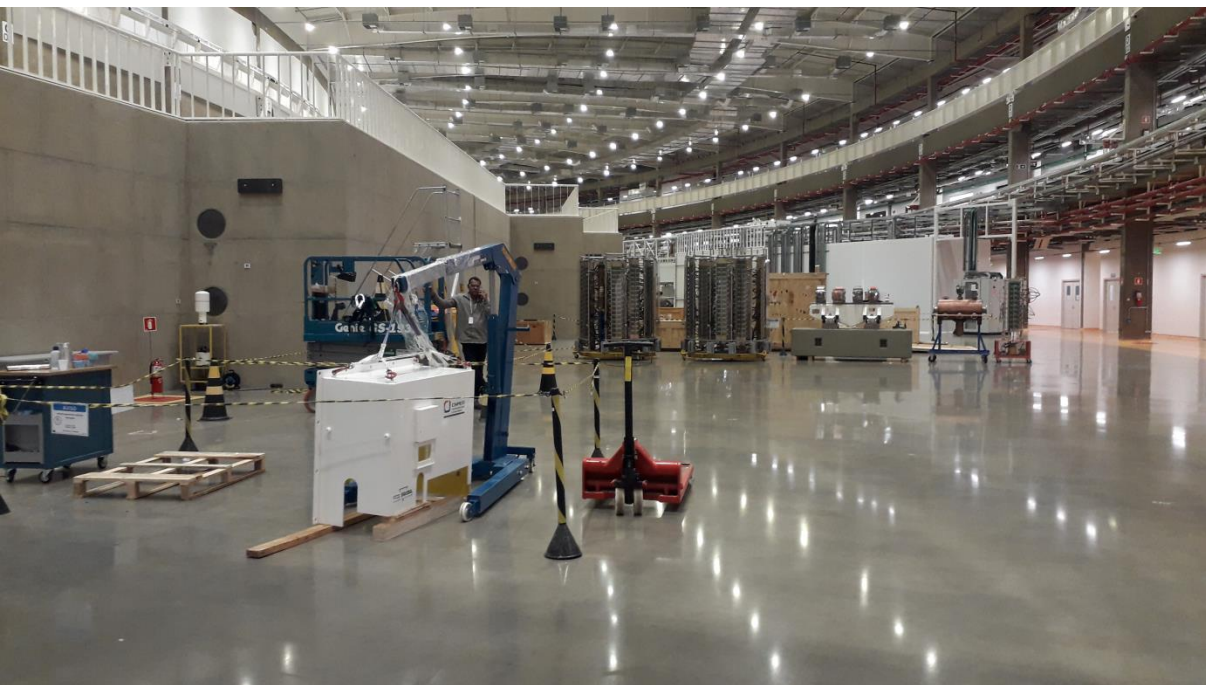
SIRIUS

Ciudad de Campinas



- ✓ Empezó a contruirse en 2015.
- ✓ Se terminó 2020.
- ✓ Circunferencia de 518 metros y emitancia de 0,27 nm-radianes.
- ✓ Está blindado por 1 kilómetro de muros de hormigón de 1,5 metros de espesor y 3 metros de altura.
- ✓ La inversión fue de 1.800 millones de reales.
- ✓ El proyecto científico más ambicioso jamás realizado en Brasil.
- ✓ Todo es tecnología brasileña.
- ✓ Primeros estudios dirigidos al Sar-CoV-2.





SINCOTRÓN EN BRASIL

SIRIUS

Ciudad de Campinas



- ✓ Estudiar estructuras tridimensionales.
- ✓ Posición de los átomos en el espacio y soluciones.
- ✓ Interacciones entre moléculas
- ✓ Creación de materiales superconductores.

**Aprueban proyectos 2 veces
al año libre de costo**

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Abstract: Viruses are obligate parasites that do consequently, to fully understand the viral process fundamentally to study them in the cellular context in the subcellular organization of the host cell by cytoelectronology and/or budding of newly formed virus particles in infected cells can thus provide antiviral drug development. Among the X-ray tomography stands out for its large depth of field to be imaged without further thinning; plus to detect small particles (the minimal size fraction), unfixed and unstained whole cells) as fractal, unified and unstrained whole cells) as fractal, unified and unstrained whole cells). In (i.e., correlative fluorescence microscopy). In X-ray tomography, its simple requirements potential of this technique, examples of viruses also presented.

Keywords: cryo-soft X-ray tomography (cr
Zika virus (ZIKV); direct-acting antiviral (D

1. Introduction

1. Introduction

Despite their vast diversity, viruses share one thing: that all viruses are obligate intracellular parasites. In other words, they cannot generate energy or to synthesize proteins in a cellular environment, any proliferation or smaller degree, to alterations in the favorable environment for viral multiplication.

In eukaryotic viruses, one of the viral factories or viral replication compartments or inclusions that are and membrane compartments and morphogenesis. Viral factories concentrating the required cellular defenses [4-7].

de ferres [4-7].

Viruses **2021**, *13*, 2109; <https://doi.org/10.3390/v13112109>

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Structural Genomics and Drug Discovery for Infectious D

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Abstract

The application of structural genomics methods and approaches to proteins from organisms causing infectious diseases is making available the three dimensional structures of many previously unknown drug targets and laying the groundwork for structure aided drug discovery. The Center for Structural Genomics of Infectious Diseases (CSGID) is a multidisciplinary center established to apply state-of-the-art high throughput structural biology technology to the characterization of proteins from the National Institutes for Allergy and Infectious Diseases category A-C pathogens and organisms causing emerging, or re-emerging infections. The center's research program emphasizes potential biomedical benefits. Selected protein targets and their homologs, essential enzymes, virulence factors and vaccine antigens are being characterized. The CSGID also provides a structure determination service for the infectious disease community. The ultimate goal is to generate a library of structures that are available to the scientific community and can serve as a starting point for further research and structure aided drug discovery. To achieve this goal, the CSGID will determine protein crystal structures of selected drug targets and protein-ligand complexes using proven, rapid, highly integrated, and cost-effective methods, primarily by X-ray crystallography. High throughput structure determination is greatly aided by frequent, convenient access to high-performance synchrotron X-ray sources.

Keywords

CSGID; structural genomics; infectious diseases; drug discovery; structure

ANTI-MICROBIAL DRUG DISCOVERY AND STRUCTURAL DISCOVERY

It is widely recognized that anti-microbial drug discovery [1]. Coupled with the rise in drug resistance is a need for new targets and new approaches to anti-screening for broad spectrum antibiotics in particular are a number of factors that have limited recent *in vitro* spectrum antibacterials is lagging behind society's explored is to focus on new drug targets and approach therapeutics, may be useful for treating

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Blotitis as a virulence factor of *Candida* species

infections due to *Candida* species are the leading cause of hospital morbidity and mortality [2-4], and *Candida albicans* is the organism most frequently isolated from patients with systemic candidiasis [5-7]. However, because of the indiscriminate use of broad-spectrum antibiotics and antifungals [8-10], combined with immunosuppressive therapies, the non-*C. albicans* *Candida* (NCAC) species such as *Candida parapsilosis*, *Candida glabrata* and *Candida lusitana*, which were previously considered to be nonpathogenic, have emerged as the second or third most common species causing candidemia and systemic candidiasis in immunocompromised patients, organ transplant or hematopoietic stem cell recipients, with neoplasms [11,42-45]. The infections caused by these species are difficult to treat, since *C. glabrata* and *C. cryptococcus* possess an innate resistance to azole-based compounds [30,12,46]. The second most frequently isolated species of *Candida* in the USA and Canada is presently *C. glabrata* [30, 31], whereas *C. parapsilosis* is the second most frequent in Europe and some regions of Latin America [30,47,48]. Even though *C. parapsilosis* and *C. glabrata* are important nosocomial pathogens, little is known about their virulence. One virulence factor involved in the establishment of

Candida infection is the adherence of the yeast to epithelial and endothelial cells, mobile factors in the extracellular matrices and inert material implants in the body of the host [24]. Once *Candida* has adhered and evaded the immune system of the patient [29-31], it can form biofilms that colonize the internal organs and medical implants such as prostheses, contact or intraocular lenses, breast implants, vascular inserts, endotracheal tubes and pacemakers [32-36]. In the last two decades, important virulence factors for the establishment of recurring candidiasis [37], as their sterile living cells show high resistance to antifungal treatment and host defense mechanisms [38-40]. This resistance greatly impacts the patient's health, since the infected medical implant must be removed when the infection does not respond adequately to treatment. This can damage surrounding tissues, as the resistance to antifungal treatment required by the resistance of the micro-organisms [40,41]. It has been reported that the NCAC species, *C. albicans*, *C. parapsilosis*, *C. glabrata* and *C. cryptococcus* that were isolated during a fungal infection, capable of forming biofilms, cause high levels of mortality (25-50%) as compared with clinical isolates unable to form biofilms [34,42]. In other studies have shown that *C. glabrata* is phylogenetically closer to *Saccharomyces cerevisiae* than to *C. albicans*, as supported by evolutionary divergence between the

Keywords

- adhesive. AU
- *Candida* species
- EPR. CDR protein
- morphological device. synchrony

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Candida infection is the adherence of the yeast to epithelial and endothelial cells, notable factors, extracellular matrices and inert material implants in the body of the host [24]. Once *Candida* has adhered and evaded the immune system of the host, it can form biofilms that colonize the internal organs and medical implants such as catheters, contact or intraocular lenses, breast prostheses, vascular inserts, endotracheal tubes, pacemakers [25–30]. In the last two decades, there have come to be considered as very important virulence factors for the establishment of recurring candidiasis [30], as their resident cells show high resistance to antifungal treatment and host defense mechanisms [31–34]. Once the infectious medical implant must be removed when the infection does not respond to treatment. This can damage tissues, as can the prolonged antifungal treatment by the resistance of the microorganism. It has been reported that *C. albicans*, *C. parapsilosis*, *C. glabrata* and *C. guilliermondii* are the most responsible for isolated during a fungal infection of forming biofilms, cause mortality (23–50%) as compared with those unable to form biofilms [35]. Several studies have shown that *C. glabrata* is more virulent than *S. aureus* in murine models, as suggested by the following

Keywords

- adhesins - ALS, biofilm
- *Candida* species - cell wall
- EPA - GPI proteins
- mannoproteins - medical device - synchrotron

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Biofilms of *Candida albicans*, *Candida parapsilosis*, *Candida glabrata* and *Candida tropicalis* are associated with high indices of hospital morbidity and mortality. Major factors involved in the formation and growth of *Candida* biofilms are the chemical composition of the medical implant and the cell wall adhesins responsible for mediating *Candida*-*Candida*, *Candida*-human host cell and *Candida*-medical device adhesion. Strategies for elucidating the mechanisms that regulate the formation of *Candida* biofilms combine tools from biology, chemistry, nanoscience, material science and physics. This review proposes the design of new technologies, such as synchrotron radiation, to study the mechanisms of biofilm formation. In the future, this information is expected to facilitate the origin of new materials and antifungal compounds that can eradicate *Candida* biofilm infections, thus reducing the dissemination of candidiasis and hopefully improve the quality of patients.

as a virulence factor of *Candida*

***Candida* infections**

infections due to *Candida* species are the cause of hospital morbidity and mortality [1]. *Candida albicans* is the organism most isolated from patients with systemic mycoses. However, because of the widespread use of broad-spectrum antibiotics and immunosuppressive drugs combined with immunoprecipitation, the non-*C. albicans Candida* species, such as *C. albicans parapsilosis*, *C. glabrata*, *C. tropicalis*, and *Candida krusei*, which were considered to be nonpathogenic, have now been found to be responsible for first, second or third most common candidemia and systemic candidiasis in compromised persons, organ transplant recipients, elderly adults and patients undergoing treatment. Since *C. glabrata* has an innate resistance to fluconazole [2], the second most frequent species of *Candida* in the community *C. glabrata* [3]. In *C. tropicalis* [4] and *C. parapsilosis* [5], the second most frequent of Latin America, *C. glabrata* [6] and *C. parapsilosis* [7] are also nosocomial pathogens.

The virulence of *C. albicans* is well established. The establishment of

Candida infection is the adherence of the yeast to epithelial and endothelial cells, soluble factors, extracellular matrices and inert material implanted in the body of the host [8]. Once *Candida* has adhered and evaded the immune system of the patient [9–11], it can form biofilms that colonize the internal organs and medical implants such as prostheses, contact or intraocular lenses, breast implants, vascular intakes, endotracheal tubes and pacemakers [12–16]. In the last two decades, biofilms have come to be considered as very important virulence factors for the establishment of recurring candidiasis [9], as their sessile treatment and host defence mechanisms [17–19]. This resistance negatively impacts the patient's health, since the infected medical implant must be removed when the infection does not respond adequately to treatment. It can damage surrounding tissue, as can the prolonged antifungal treatment required by the resistance of the microorganism [10, 20]. It has been reported that the strains of *C. albicans*, *C. parapsilosis*, *C. glabrata* and *C. tropicalis* that were isolated during a fungal infection, capable of forming biofilms, cause high levels of mortality (23–50%) as compared with clinical isolates unable to form biofilms [21, 22]. *In vitro* studies have shown that *C. glabrata* is phylogenetically closer to *Saccharomyces cerevisiae* than to *C. albicans*, as suggested by evolutionary divergence between

Keywords:

- adhesins - AL
- *Candida* species - CRI profile
- microorganisms - device - synthesis

Review

Future Microbiology