S9.4b Terbium-Cu-Zn-Fe nanoparticles induced apoptosis and cell cycle arrest in multidrug-resistant Candida auris
S9.4c Free oral presentations (late breaking), September 23, 2022, 4:05-6:15 PM
Hammed Amin1, Ajark Ahmad1
1Clinical Microbiology and Infection Diseases, Faculty of Health Sciences, School of Pathology, University of the Witwatersrand, Johannesburg 2193, South Africa

Background: Candida species are opportunistic can cause serious infections, particularly in immunocompromised populations. The fungal burden has increased recently steadily with Candida species being responsible for 70% of these infections, particularly in hospitalized patients with significant underlying conditions. Phospholipase A2 in Candida species and the adhesion of Candida auris elevated candidiasis to a major public health concern. A growing number of infections in cats is an emerging and opportunistic fungal cause can cause hematological malignancies and high-fidelity race, particularly in hospitalized patients with major medical issues. Antifungal study of terbium nanoparticles (TNPs) of various types have been used as a therapy option for effective and safe control of candidias. These TNPs were highlighted for being environmentally friendly and suitable for nanosynthesis purposes.

Objective: To test the in vitro and in vivo antifungal and antiproliferative activity of terbium against Candida auris isolates.

Methods: The synthesis and characterization of Cu-Zn-Fe trinuclear nanoparticles was done by standard methods. The antifungal capabilities of these TNPs were determined by calculating minimal inhibitory concentrations (MIC) and minimal fungicidal concentrations (MFC) following CLSM recommended guidelines. Susceptibility on planktonic cells and biofilms was further confirmed by MTT cell count and viability assay and scanning electron microscopy (SEM) respectively. For in vivo antifungal and antiproliferative action against Candida auris isolates.

Results: Characteristics by Fourier-transform infrared spectroscopy (FTIR), differential scanning fluorimetry (DSF), electron microscopy (SEM) and transmission electron microscopy (TEM) determined the successful biosynthesis of Cu-Zn-Fe trinuclear. Susceptibility assay confirmed the functional activity of Cu-Zn-Fe NPs with MIC and MFC values of 32.1 and 25 μg/mL respectively. These results were further confirmed by viability assay reporting the cell viability of 45.1%, 13.5%, and 1.8% when C. auris cells were treated with 1/2 MIC, MFC, and MDC respectively.

Conclusion: Our results demonstrated that Cu-Zn-Fe NPs showed 91.3% of breakage developed uninfected cell wall was in GMS phase, whereas 3.7% and 3.0% of cells were in the S phase and G2/M phase, respectively. In contrast, N-P treated cells were observed to be arrested in S phase with 15.46% and 14.26% of total cell death caused by NPs, we investigated intracellular mitochondrial potential (ΔΨm), with cells having a stable (ΔΨm) when treated cells showed loss of (ΔΨm). Another important parameter of apoptosis in yeast cells is the release of cytochrome C from the mitochondria to the cytoplasm and NPs treated cells resulting in decreased FACS and increased Annexin V and Cytochrome C. Both conditions confirmed the role in NPs in catalyzing apoptosis and cell death in C. auris.

S9.4c Diverse environmental inputs mediate in -glucan exposure at the Candida albicans cell surface thereby modifying the host immune response to systemic infection
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Ammal Preethan1, Gino Mej1, Emer Healy2, Gabriela Avila3, Daniel Lancet1, Judith Bain1, Delma Childress1, Ivy Daniels1, Danir Ions1, Leonie A. Riggs1, Alastair J. P. Brown1
1IRC Centre for Medical Mycology, University of Exeter, Exeter, United Kingdom
2Department of Medical Science, University of Aberdeen, Aberdeen, United Kingdom
3University of Oxford, Oxford, United Kingdom

Background: Candida albicans is an opportunistic fungal pathogen that can cause skin and systemic infections. C. albicans expresses β-glucan on its cell wall, which plays a critical role in the immune response. Previous studies have shown that environmental inputs can mediate changes in β-glucan exposure at the Candida cell surface. Our objective was to investigate the nature and extent of environmental inputs that mediate β-glucan exposure at the Candida cell surface and to assess their potential role in modulating the host immune response.

Methods: We used a combination of live-cell imaging, flow cytometry, and cytokine assays to analyze the relative contributions of different environmental inputs to β-glucan exposure at the Candida cell surface. We exposed C. albicans to a range of environmental inputs, including temperature, pH, ROS, and antibiotics, and measured the impact on β-glucan expression using flow cytometry. We also assessed the effect of environmental inputs on the expression of additional cell-surface molecules, such as mannoproteins, using quantitative PCR.

Results: Our results showed that environmental inputs can modulate β-glucan exposure at the Candida cell surface. For example, exposure to high temperature resulted in increased β-glucan expression, whereas exposure to low pH or ROS led to decreased β-glucan expression. These changes in β-glucan exposure were associated with changes in the expression of additional cell-surface molecules.

Conclusion: Our findings suggest that environmental inputs can modulate β-glucan exposure at the Candida cell surface, which may have implications for the immune response to C. albicans infection. Future studies will be required to determine the mechanisms by which environmental inputs modulate β-glucan expression and how these changes impact the host immune response.