Candida and Candidiasis

6–9 October 2025

The Estrel Congress Center (ECC)
Berlin, Germany

#Candida2025

POSTER ABSTRACT BOOK

MICROBIOLOGY SOCIETY

BLOCK B

Vaginal clinical isolates of *Candida albicans* differentially modulate type I interferon pathway in an *in vitro* infection model

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Abstract

Vulvovaginal candidiasis (VVC) affects up to 75% of women worldwide and is primarily caused by *Candida albicans* (*C. albicans*). Our studies demonstrated that two clinical isolates of *C. albicans*, a VVC-associated strain (Ca01887) and a Colonizing strain (Ca14314), elicit different epithelial responses. Ca01887 caused massive epithelial damage, elevated fungal shedding and exfoliation, without activating type I interferon (IFN-I) signaling. In contrast, Ca14314 induced limited damage and shedding and activated the IFN-I pathway.

Using an *in vitro* infection model with A-431 vaginal epithelial cells (VECs), we analyzed the IFN-I pathway's role. VECs were treated with a neutralizing IFNAR-2 antibody, the STING agonist 2'3'-cGAMP, and the JAK-STAT inhibitor Ruxolitinib, alone and in combination. After 24 hours, fungal shedding was quantified via colony-forming units (CFUs).

To assess inflammasome activation, IL-1β, IL-1α, and IL-1RA levels were measured in VECs supernatants using ELISA. Mitochondrial ROS (mtROS) production was monitored through MitoSOX Red staining and quantified by fluorometry and confocal microscopy. To correlate mtROS production with fungal shedding, cells were treated with the mtROS inhibitor Visomitin.

Our results showed that fungal shedding increased with Ruxolitinib and decreased with 2'3'-cGAMP in Ca14314-infected VECs, but not with Ca01887. Ca14314 induced significantly higher mtROS than Ca01887, while Ca01887 triggered a stronger inflammasome. Visomitin enhanced shedding only in Ca14314-infected VECs.

These findings reinforce the idea that IFN-I signaling and mtROS are key regulators in the vaginal epithelial cell defense to *C. albicans* infection, suggesting thus their possible employment as novel therapeutic targets in VVC/RVVC.