Jena School for Microbial Communication
Graduate School of Excellence

PhD project title

Elucidating different virulence strategies *in Candida albicans* clinical isolates with fluorescent reporters for host cell damage in intestinal epithelial cells

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Abstract: Despite its normally commensal nature, *Candida albicans* can cause severe disease in predisposed humans. The fungus is equipped with a set of virulence factors helping it to establish infections in the host. However, the vast majority of studies of *C. albicans* virulence factors and strategies so far have been based on SC5314, a clinical isolate commonly described as more virulent than most others. To better understand not only how the fungus can damage intestinal epithelial cells but also how the host cells respond to it, we screened over 60 isolates from different body sites and clades for their damage potential. Surprisingly, we found that almost one third of them is at least as damaging as SC5314. To better understand the interactions between the host and the fungus, in the first part of my project we established a new set of fluorescent reporter lines for host cell death and stress. With the use of these reporters, we discovered that the isolates all differ in their interactions with the host. Our interest was piqued by C227, a blood isolate, that causes as much damage, redox stress, apoptosis and inflammasome activation in intestinal epithelial cells as SC5314, but is lacking most of the typical virulence attributes of SC5314, such as adhesion, invasion, and efficient translocation. We believe that C227 could represent a group of clinical isolates that manage to establish infection in different ways than the reference strain SC5314, depending less on invasion and candidalysin expression. Our study is shedding light on alternative ways that C. albicans can damage and infect the host. This will help us to better understand the fungal pathogenicity mechanisms and their still unexplored natural variations.