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The immune system possesses elegant strategies to cope with potential harmful invading microorganisms. Nevertheless, pathogens themselves have evolved mechanisms to deal with the threats imposed by the immune system. Yeasts like *Candida* species are common commensals of the human microbiota, yet also major opportunistic fungal pathogens that frequently cause superficial and even fatal infections. The commensal co-existence with the human host allows the co-evolution of fungal adaptation strategies in line with the threats imposed by the host.

While the competing microbiota is potentially the major challenge for host-associated *Candida* species during commensalism, the immune system is the major threat that can compromise the survival during infection. The fungus, therefore, employs strategies to evade immune recognition or even escape the immune cells after it has been attacked. A constant expression of these pathogenicity strategies is not efficient and may also jeopardize the commensal lifestyle of these pathogens. An adaptive regulation is essential to only engage these pathogenicity strategies when needed. We investigate the adaptations induced by host conditions (like temperature) and host molecules. In particular, we are interested in the underlying molecular mechanisms inducing these adaptations and the host proteins the fungus may sense to do so.

Further related topics dealing with immunotherapy and interaction with the microbiota are investigated in close collaboration with the Department of Microbial Pathogenicity Mechanisms.

[Microbial Pathogenicity Mechanisms](#)