

We study systemic fungal infections

Fungal infections triggering sepsis have increased dramatically in recent years. There is a common characteristic that is apparent in all septic fungal infections: they are difficult to diagnose and they have an above-average mortality rate. For this reason, Fungal Septomics focuses specifically on infection biology of systemic fungal infections.

The research group Fungal Septomics aims at analysing both the variability of the pathogen and the regulation of the human immune system during sepsis. The most important model organism is the polymorphic yeast *Candida albicans*. We investigate how *C. albicans* can transform from a harmless 'resident' into an invasive pathogen. The ability of *C. albicans* to switch from its yeast form to filamentous forms, enabling tissue-invasive growth, is essential for pathogenesis. *Fungal Septomics* analyses the mechanisms of this morphotypic switch in parallel with the associated changes in the epithelial barrier. However, invasive fungal infections caused by *C. albicans* are almost always also associated with a dysfunction of the host's immune response. Here, neutrophilic granulocytes are of particular importance. The action of neutrophilic granulocytes in concert with other components of the immune system as well as the functions of other immune cells are yet to be fully understood. The work of *Fungal Septomics* focuses on these questions, using established infection models for primary human immune cells, as well as a whole-blood infection model, as a simple but meaningful *in vitro* infection model.

Numerous studies show that the risk for invasive fungal infections of immunosuppressed patients is also dependent on genetic predispositions. The AsplRS (*Aspergillosis intrinsic risk stratification*) study coordinated by *Fungal Septomics* is the first systematic genome wide approach to identify genetic polymorphisms which are associated with invasive aspergillosis.