Virulence of *Aspergillus fumigatus* and Host-Pathogen Interactions

During the past decades, the incidence of systemic fungal infections in humans has risen considerably. More than 90% of all cases of invasive mycoses caused by aspergilli are due to an infection with *Aspergillus fumigatus*. The Department focuses on two aspects: (1) the identification of virulence determinants of the fungus and (2) the elucidation of the interaction between *A. fumigatus* and the immune system, in particular neutrophilic granulocytes, macrophages, epithelia and T cells. We are in particular interested in mechanisms of immune evasion such as the ability of the fungus to avoid the recognition immune effector cells due to its hydrophobin layer or the manipulation of the intracellular processing of conidia by their dihydroxy naphthalene (DHN) melanin layer. DHN melanin inhibits the acidification of phagolysosomes and inhibits apoptosis of macrophages, epithelia cells and neutrophils. In addition, we are interested in further secondary metabolites produced by the fungus like gliotoxin, to elucidate their biological activity also with respect to virulence.

Research Projects

- Interaction of *A. fumigatus* with neutrophilic granulocytes
- Interaction of *A. fumigatus* with macrophages: Interference of conidial DHN melanin with the host endocytosis pathway
- Biosynthesis and regulation of secondary metabolites related to virulence, e.g. gliotoxin and fumipyrryl
- Function and impact of fungal signal transduction pathways, including G protein-coupled receptors, on virulence
- Virulence assays of *A. fumigatus* mutants and testing of new antifungal compounds in a murine infection model of invasive pulmonary aspergillosis

Electron micrograph of an *A. fumigatus* conidiophor
Interaction of *A. fumigatus* with the human innate immune system