Position by institution 6

ESR No. 9
Host Institution: Hans Knöll Institut Jena, Germany
ESR enrolled at: Friedrich Schiller University Jena

Institute	Hans Knöll Institut Jena, Germany
Lab	Microbial Pathogenicity Mechanisms
Responsible person	Bernhard Hube, PhD
Job title	Early Stage Researcher: PhD thesis on molecular microbiology of *C. albicans* infections
Job description	Short description: - Required degree: MSc in microbiology, biology, biochemistry or equivalent - Preferred qualification and expertise: microbiology, infection biology, molecular biology, functional genomics - Duration: 36 months - Language: English (essential), - Contact: Prof. Bernhard Hube, Tel.: +49 3641 532-1401; Mail: bernhard.hube@leibniz-hki.de **The Department of Microbial Pathogenicity Mechanisms :** The Department of Microbial Pathogenicity Mechanisms (MPM) investigates infections caused by human pathogenic fungi. Research is focused on the pathogenesis of mycoses caused by yeasts such as Candida albicans or C. glabrata. Using cellular, microbial, molecular and biochemical methods and C. albicans or C. glabrata as model organisms, the goal of our research is to identify factors which fungal pathogens need to cause diseases. In addition to these efforts to increase our understanding of the basics of pathogenesis of fungal infections, we also seek to identify new biomarkers for diagnostic approaches and potential targets for antimycotic drug development. **PhD project** Objectives: To (i) extend and refine existing in vitro and ex vivo commensal and infection models for C. albicans and C. glabrata; (ii) to dissect the different stages of infection; (iii) to characterize the fungal and host transcriptional profiles during infection; (iv) to identify stage-specific marker genes of C. albicans and C. glabrata infection; (v) to identify and characterize genes that are required for pathogenicity of C. albicans and C. glabrata. Methodology: In collaboration with ESR5 and 6 (P3), ESR9 will monitor the interaction of C. albicans and C. glabrata with epithelial and endothelial cells, and will dissect the infection process. RNA-seq will be used to determine the stage-specific transcriptional profile of the pathogen and the host. Genes associated with the different stages of infection will be identified using RNA-seq and compared to data obtained during infections with other species (P3) to identify common and species-specific signatures. QRT-PCR will be used to validate results. Candidate marker genes will be developed into prototype diagnostic tools. Candidate genes will be disrupted and their roles in infection analysed. Furthermore, to identify genes crucial for pathogenicity, mutant strains from a collection of C. albicans and C. glabrata knock out strains will be monitored in the established infection models. Expected Results: Identification of markers and transcriptional profiles common to fungal infection, and specific to C. albicans and C. glabrata infection. **Planned secondment(s):** P3 UCD (1 month; Y1; to establish standard operation procedures together with ESR5 and 6 for infection models and RNA isolation); P9 UGENT (1 month; Y2; to obtain exposure to clinical environment and to establish standard operation procedures together with ESR12 for infection RNA isolation and microarray analysis).