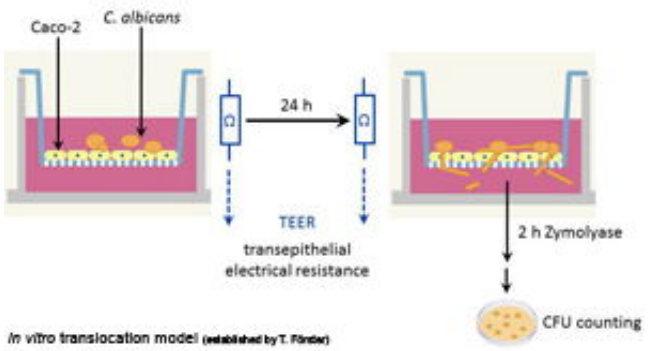
Pathogenicity mechanisms at the mucosal interphase



In vitro translocation model simulates the intestinal epithelial barrier

In the healthy host, certain *Candida* species live as harmless commensals on mucosal surfaces like the oral, vaginal or intestinal mucosa. Under predisposing conditions, these *Candida* species growing on the oral or vaginal mucosa can cause tissue damage associated with induction of inflammation, immunopathology, and disease that significantly impacts quality of life.

When immune defense and microbiota are compromised in hospitalized patients or when the intestinal barrier is disturbed, fungal populations of the intestinal tract can invade the intestinal epithelial barrier and translocate into the bloodstream. From here, the fungus can infect virtually all organs and cause systemic fungal infection. Our aim is to elucidate which fungal and host factors mechanistically allow fungal adhesion to, invasion into, and damage of epithelial cells as well as translocation through intestinal barriers and invasion of host tissues.

To unravel host-pathogen interactions at the epithelial interface for *C. albicans*, but also *C. glabrata* and the newly emerged, multidrug-resistant species *C. auris* we are using *in vitro* infection models, genome-wide dual-species transcription profiling techniques, and fungal gene deletion strains.