Among ~5,000,000 fungal species on this planet, Candida albicans is unique in its lifelong association with humans, either as a stable component of the gastrointestinal microbiome or as the most common fungal pathogen. We hypothesize that this close relationship with the host has driven the evolution of specialized fungal programs for occupying a diversity of host niches. The core research objective of our laboratory is to define the molecular basis of host-microbe interactions that foster both commensalism and pathogenesis.

To approach these questions, we exploit genetic resources, animal models of virulence and commensalism, and techniques such as whole-genome chromatin immunoprecipitation and high throughput sequencing. To date we have identified a series of novel virulence factors that act independently of the yeast-to-hyphal transition, which was previously thought to be this species’ major virulence specialization (Noble et al., Nature Genetics 2010). Our detailed characterization of the Sef1 virulence factor uncovered a C. albicans-specific transcriptional network whose activity has opposite roles in commensalism and disseminated infections (Chen et al., Cell Host and Microbe 2011; Chen and Noble, PLoS Pathogens 2012).

More recently, we discovered that passage of C. albicans through the mammalian gastrointestinal milieu triggers a developmental switch to a commensalism-specific cell type (Pande et al., Nature Genetics 2013). The ability to switch between specialized cell types that are optimized for specific host niches helps to explain the ubiquity and versatility of this microorganism and offers opportunities for treatment and prevention of clinical infectious diseases.