

Our laboratory looks at the mechanisms used by *Candida albicans* to generate phenotypic diversity and to adapt to different niches in the human host. Genetic mechanisms of diversity include an unusual parasexual mating cycle in which a non-meiotic program of concerted chromosome loss reduces the ploidy of mating products. We also examine epigenetic mechanisms of diversity including those responsible for heritable ‘phenotypic switching’ between alternative cell states in pathogenic *Candida* species. We use both commensal models of GI tract colonization and systemic models of disease to determine how genetic and epigenetic diversity impact infection of the mammalian host.