The Hickman lab investigates the genome plasticity of the yeast *Candida albicans*, a commensal that primarily resides in human gastrointestinal tracts and causes superficial infection in healthy individuals and serious infection in immunocompromised individuals. Such plasticity can lead to the acquisition of antifungal drug resistance. *C. albicans,* was considered to be an 'obligate' diploid organism for nearly a century until recently when a viable haploid stage was discovered (Hickman et al 2013). Furthermore, tetraploid cells have been recovered after mating between diploid partners as well as isolated from clinical samples. Unlike other budding yeasts, *C. albicans* does not go through meiosis to reduce ploidy, but rather utilizes stochastic and imprecise concerted chromosome loss processes that frequently result in a heterogeneous population as some cells will have chromosomal aneuploidy and/or homozygosis (Hickman et al 2015). We examine how shifts in ploidy, mediated by sexual cycles in addition to asexual mechanisms promote genetic diversity within a population of cells. These ploidy transitions facilitate large-scale mutations including recombination, aneuploidy and homozygosis of whole chromosomes within a single cell division and fuel rapid adaptation.