Fungal pathogens are estimated to infect over 1 billion people and kill over 1.5 million people a year, establishing themselves as a global emerging threat. *Candida albicans* is a prominent opportunistic fungal pathogen that is able to undergo morphogenesis and cause severe, invasive bloodstream infections, with mortality rates ranging from ~30-70%. In the clinic, these infections have been treated with antifungals that target *C. albicans* and disrupt its cell integrity to induce lysis. However, a predictable consequence of the extensive use of antifungals is the subsequent rise in antifungal drug resistance. These new drug resistant isolates proliferate unchecked and cause further harm to the infected host. The fundamental problem of antimicrobials is the high selection pressure they impose on microbial pathogens to evolve resistance. A promising alternative, and the focus of my research, is to find compounds that target and inhibit microbial virulence factors (the pathogen components that damage the host) rather than attempting to eliminate the pathogen altogether. For my research, *C. albicans*’ morphogenetic switching presents itself as a potent virulence target. I hypothesize that screening the supernatant of diverse microbes, will yield antivirulence compounds that will selectively inhibit *C. albicans*’ ability to undergo morphogenesis and form hyphae. Once compounds are found, I will quantify their ability to prevent hyphal formation in drug-resistant isolates in vitro and then attempt to induce and quantify resistance to the compounds by serial passaging. This research will help uncover microbial factors that can directly influence the pathogenesis of *C. albicans* without the development of resistance.