Abstract

*Candida albicans* is the most prevalent human fungal pathogen. Unfortunately, there still remains much that is unknown about it, including the reason for maintaining a diploid genome and the function of many genes. In this thesis I describe the phenotypic consequences of up and downregulating Pseudouridine Synthase 4 (*PUS4*) of *C. albicans*, as well as an examination of the differences in the expression level of the entire transcriptome. These experiments and resulting bioinformatic analysis have lead to our conclusion that that *PUS4A* is more lowly transcribed than *PUS4B*. This data suggests *PUS4A* may be nonfunctional or play a different role in *C. albicans* biology.

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