

CALIFORNIA STATE SCIENCE FAIR 2009 PROJECT SUMMARY

Name(s)

David K. Tang-Quan

Project Number

S1799

Project Title

Isolation of Kinase Mutant Genes Governing Stress Response of Candida albicans

Abstract

Objectives/Goals In immuno-compromised patients, the fungus Candida albicans can enter the bloodstream, infecting most organs of the body and resulting in disseminated candidiasis, which has a 50% mortality rate, even with treatment. In healthy individuals, white blood cells protect against candidiasis by secreting antimicrobial peptides. For C. albicans to colonize patients and cause disease, it must be able to withstand these antimicrobial peptides.

Methods/Materials

Approximately 100 strains were screened for hyper-susceptibility to antimicrobial peptides. Hyper-susceptible strains were retested alongside a second independent clone. Additionally, gene deletion mutants and complemented strains were acquired and tested. Finally, the kinase insertion mutants were transformed with the HIS1 gene and retested in the absence of histadine.

Results

Hyper-susceptible strains such as SSK2, PBS2, and HOG1 were discovered, proving their necessity for C. albicans stress response. Hypo-susceptible strains were also discovered, suggesting that C. albicans has an adaptive response when certain genes are removed.

Conclusions/Discussion

Kinases are indeed required for C. albicans to grow in the presence of antimicrobial peptides. Most significantly, three members of the HOG1 kinase pathway, Ssk2, Pbs2, and Hog1, are required for antimicrobial peptide resistance in C. albicans. Pharmacologists can then develop medication that can inhibit the HOG1 kinase pathway and thereby prevent Candida infections.

Summary Statement

This study discovered that the HOG1 kinase pathway controls the resistance of the fungus Candida albicans to antimicrobial peptides.

Help Received

Mentored by Dr. Scott Filler, overseen by Ms. Norma Solis, at Los Angeles Biomedical Research Institute