Master's Defense Announcement

"Towards an understanding of the mechanism of action of the tumor-inhibitory triterpenoid saponin avicin G"

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Here I describe the use of *Schizosaccharomyces pombe (S. pombe)* as a model organism for determining the mechanism of action of the tumor-inhibitory triterpenoid saponin avicin G. The data here presented support a model in which avicin is internalized by endocytosis following an association with membrane ergosterol. After being taken up by endocytosis, avicin causes vacuole dysfunction, mitochondrial stress, and generation of reactive oxygen species. Additionally, I report an apparently novel HIP1-ortholog dependent survival mechanism in dividing *S. pombe* cells which results in asymmetric survival of daughter cells following avicin stress. Finally I present evidence suggesting that the HIP1-ortholog End4 is required for maintenance of normal mitochondrial morphology. Cumulatively the data here presented provide insight into the use of avicin as a therapeutic agent, highlight a novel *S. pombe* behavior, and suggest previously uncharacterized roles for End4.