# Introduction

## Cellular aging and ROS, TOR 1 pathway, PCP1 function and its connection to CR and aging.

Cellular aging in Saccharomyces cerevisiae is an invasive aspect of research that can potentially lead to several implications related to human aging diseases. There are many factors that may influence cellular aging in yeast such as reactive oxidative species (ROS), osmotic pressure, vacuole acidification and calorie restriction (CR). It has been proposed that the reduction in calorie intake slows aging and increases lifespan by suppressing the TOR signaling pathway. **(DIAGRAM)** Suppressing the TOR pathway consequently reduces endogenous ROS levels which we know is the causative agent of cellular fragmentation as well as protein, lipid, and DNA damage. The research under study focuses on how CR alters oxidative stress levels in the ∆PCP1 mitochondrial gene and in turn how it affects the longevity of yeast cells. Mitochondria play a critical role in life span extension effects of CR because it houses the electron transport chain which has been found to be the center of superoxide production. PCP1, a rhomboid serine protease, participates in mitochondrial dynamics and the processing of cytochrome c peroxidase (CCP1) which is involved in degradation of hydrogen peroxide species. Additionally PCP1 partakes in the processing of MGM1 a dynamin- like GTPase involved in mitochondrial fusion, fission and cristae formation. **(DIAGRAM)**

\*Corrlelation btw TOR and CR and PCP1. Then hypothesis.

How h2o2 activates sod and converts o2-

Notes

\*The length of a cell's telomeres can be used to determine the cell's age and how many more times is will replicate. When a cell stops replicating, it enters into a period of decline known as "cell senescence," which is the cellular equivalent of aging. Replicative lifespan in budding yeast is assessed by determining the number of times cells divide in the presence of nutrients before they senesce and die via an apoptotic-like mechanism.

\*In efforts to determine how calorie restriction would affect a ∆PCP1 gene we grew ∆Pcp1 mutant cells as well as BY4743 cells which served as the wild type control. A single colony from both were taken and grown at 30° degrees in separate tubes containing 20% glucose YPD liquid. Using two test tubes the ∆Pcp1 culture was exposed to 2% glucose which is normal nutrient conditions and .05% glucose which is calorie restricted conditions. The same was done for the wild type BY4743. Using a technique known as DHE

# Materials and Methods

# Results

## CR suppress DHE signals

## Pcp1D has low DHE signals reardless of glucose concentration

# Disucssion

## If CR works through H2O2 and endogenous H2O2 level are extremely low in pcp1D, then CR should not extend life span of pcp1D. A hypthoseiss that can be tested.

## 

# References

# Tables

## List of strains used

# Figures

## Diagram of cellular aging, ROS, and PCP1 funciton (with hypothesis highlighted) (tell people what and why we are doing this project)

## DHE stain FACS data, BY47473 and pcp1D

## Todo: Tor1D