The Role of the Nuclear Gene *RAD1* in the Stability of the Mitochondrial Genome in *Saccharomyces cerevisiae*

Amber Altrieth aaltr1@brockport.edu (585) 857-4195

Mentor: Dr. Rey Sia rsia@brockport.edu

The College at Brockport, State University of New York, Department of Biology

The mitochondria or the powerhouse of the cell is an essential because it produces much of the cells energy in the form of ATP. This energy is used to successfully complete many different cellular processes. Mitochondria have their own genome, separate from the nuclear genome, which encodes for proteins specifically for respiration. In humans, mutations in the mitochondrial DNA (mtDNA) results in the loss of mitochondrial function which leads to neuromuscular and neurodegenerative disorders. To help prevent mutations in the nuclear genome, there are genes that express proteins involved in DNA repair mechanisms. For example, the *RAD1* gene is involved in the nucleotide excision repair (NER) pathway. NER is a repair mechanism that utilizes double-strand breaks and cuts out damaged portions of DNA and replaces it by copying the template strand. Research so far has found that the Rad1p and Rad10p protein complex target lesion sites on nuclear DNA. This laboratory focuses on determining the role of the nuclear gene *RAD1* in maintaining the stability of the mitochondrial genome in *Saccharomyces cerevisiae*. To do this, the lab observes how the loss of the *RAD1* gene plays a role in the stability of the mtDNA. An assay was done to measure the percent of spontaneous respiration loss in *rad1*Δ mutants. It was observed that *rad1*Δ mutants did not show a significant increase in spontaneous respiration loss compared to that of wild type. In addition to respiration loss assays, a direct repeat-mediated deletion (DRMD) assay was also used to determine if there was a change in the stability of the mtDNA. It was found that the *rad1*Δ mutants did not show a significant increase in mtDNA mutations. However, the *rad1*Δ mutants did show a 2-fold decrease in nuclear mutations.

Keywords: *Saccharomyces cerevisiae,* Rad1, Nucleotide Excision Repair (NER), mitochondrial genome

Type of presentation/time request: poster presentation, morning time preferred