Mitochondria are essential organelles in eukaryotes. Mitochondria synthesize ATP, supplying the cell with energy necessary for metabolic processes, hence its nickname of the cell’s “powerhouse”. Mitochondria have individual genomes, separate from the nuclear DNA, that encode proteins vital for respiration. In humans, mutations in the mitochondrial DNA (mtDNA) lead to several neuromuscular and neurodegenerative disorders due to the compromised stability of mtDNA. This particular study focuses on a nuclear gene, *SGS1*, and its significance in mtDNA stability in the budding yeast, *Saccharomyces cerevisiae*. *SGS1* is a member of the recQ family of helicases and therefore aids in the unwinding of chromatin at the duplex as it prepares for replication. Similar mutations in homologs of *SGS1* helicase lead to specifically, Bloom Werner and Rothmund-Thomson syndromes in humans. Yeast lacking functional Sgs1p protein display hypersensitivity to DNA-damaging agents and hyper-recombination and exhibit signs of premature aging. The quantitative impacts of *SGS1* mutations on mtDNA stability in budding yeast was studied via genetic assays that measured spontaneous respiration loss and direct repeat mediated deletion. Budding yeast *sgs1Δ* display an ~2.2 fold increase in respiration loss. From two independent isolates, *sgs1Δ* mutants have also shown an ~1.7 and ~1.5 decrease in mitochondrial homologous recombination, but ~2.4 and ~2.8 increase in nuclear homologous recombination. The nuclear data supports existing conclusions. Our data shows that the presence of Sgs1p protein plays a role in mitochondrial genome stability.