For mitosis to be successful, sister chromatids must be properly separated into two forming daughter cells during anaphase. This process is mediated by the kinetochore, a multi-protein structure that connects centromeric nucleosomes to microtubules during chromosome segregation. Abnormalities in chromosome number can occur when there are defects in this connection, leading to aneuploidy and an increased risk of cellular death. In the kinetochores of *Saccharomyces cerevisiae* (budding yeast), the Dsn1 protein (Dsn1p) acts as a bridge between proteins contacting centromeres and proteins contacting microtubules. Dsn1p's function is known to be affected by phosphorylation, so we wanted to explore how phosphorylation regulates Dsn1p's interactions within the kinetochore, due to its unique placement in the middle of this protein structure. Amino acid residues T380 and T386 are known to be phosphorylated, but the functional consequences of these modifications are unknown. We studied the effect of phosphorylation at these residues by using the CRISPR-Cas9 system to induce phosphomimetic mutations to the *DSN1* gene. A repair template was designed that changes the uncharged threonine residues to aspartic acid, mimicking a state of constant phosphorylation through the presence of a negative charge. We successfully made the T380D mutation, but have yet to introduce the mutation at site T386. We saw no significant changes in growth between the wild type and mutant strains. Since phosphorylation sites often function together, it is imperative to create the mutation at the proximal T386 residue and conduct further phenotypic testing to understand the function of phosphorylation at these sites.