PRODUCTION OF ISOGENIC PRION STRAINS FOR TRANSCRIPTIONAL PROFILING STUDIES. ***Seth Zimmerman*****,** *and *Irene Evans*, *Department of Biological Sciences*, *COS, RIT*, spz8913@mail.rit.edu, imesbi@rit.edu.

In the model organism *Saccharomyces cerevisiae* (yeast), the gene SUP35 codes for a translational release factor protein eRF3. This protein has been the subject of intense study as a model for prion proteins in eukaryotic organisms. A prion is a protein that is capable of assuming an alternate conformation distinct from that of its known functional conformation; this often gives the protein a different functional activity. Prions are also infectious particles, meaning that they have the ability to convert other functionally conformed proteins into prion conformations. In mammals this can cause neurodegenerative disease called transmissible spongiform encephalopathies. However, in yeast this prion system causes no known pathology and is actually thought to give an evolutionary advantage. This is because the prion conformation of eRF3 (the gene product of Sup35) is inefficient at translational release causing stop codon read through and alternate protein production. The variability in proteins produced may have a positive effect allowing the yeast to survive in diverse environments. Previously our lab has asked the question whether the presence of the prion protein in isogenic strains, [*PSI*+] and [*psi*-], causes changes in gene transcription. [*PSI*+] contains the prion form of eRF3 while [*psi*-] contains the functional form. Our previous results suggested there were over a hundred gene expression changes in the [*PSI*+] prion containing strain. In order to easily distinguish between the two strains, they each contain a nonsense mutation in the ADE3 gene which places a stop codon in the middle of the gene and in the [*psi*-] strain causes a disruption in adenine production. Due to this stop codon mutation, the [*psi*-] strain produces a byproduct red pigment. In contrast the *[PSI+]* strain reads through the stop codon and is able to produce adenine normally leaving *[PSI+]* colonies a cream color. In order to ensure that our *[PSI+]* and [*psi*-] strains are isogenic, our lab has attempted to cure or revert our [*PSI*+] strain back to [*psi*-] using millimolar concentrations of guanidine hydrochloride. In theory this should change our cream colored [*PSI*+] to a red [*psi*-] strain. However, upon attempting this in a variety of ways, the strains failed to show the appropriate color change. We theorized that the [*PSI*+] had acquired a mutation earlier in the adenine production pathway that kept the color change from occurring, meaning that the strains were indeed cured, but no longer isogenic. To test this theory our lab attempted to cure the original stocks of our [*PSI*+] strain and achieved a successful color reversion. We now plan on running further microarray studies on the truly isogenic strains to be certain of our earlier findings.