**Introduction**

*Rhodospirillum centenum* is a purple photosynthetic bacterium belonging to the class α-proteobacteria. This organism has the ability to transition into motile swim cells when growing in a liquid environment. Additionally, starvation induces the differentiation of vegetative cells into cysts. Previously, our lab has shown that a phosphorylated response regulator, CtrA, enhances motility while suppressing encystment. The current study is focused on characterizing the phosphotransferase protein, ChpT, to determine whether this protein affects CtrA activity in *R. centenum*. ChpT along with all contributing protein factors were identified as orthologs of a well-characterized phosphorelay that controls motility and cell division in another α-proteobacterium, *Caulobacter crescentus*.

Initial results partially suggest that the ortholog ChpT affects motility and encystment in *R. centenum*. ΔchpT displays consistent results with the model pathway however, ChpT overexpression plasmid failed to rescue the ChpT phenotype of wild type.

**Hypothesis**

![Diagram of DivK, Cyd2, ChpT, CtrA, and Encystment](Image)

**Methods**

A null chpT mutation was made along with a site directed mutation to the gene’s histidine residue protein product. A chpT expression plasmid and the chpT plasmid with a site directed mutation were conjugated into both ΔchpT strain and wild type cells. These eight strains were then grown on CENS to characterize cyst formation and CENMED to examine motility.

**Results**

![Figure 2: A: On CENMED soft agar medium, a wild type R. centenum colony exhibits rings of chemotactic cells that swim up a concentration gradient of their preferred carbon source, pyruvate. The ΔchpT strain exhibits a delayed chemotaxis response and the chemotaxis ring is much less dense suggesting that far fewer cells become motile in the absence of the phosphotransferase. B: On CENS medium wild type colonies wrinkle after 72 hours and many of the cells are cysts. Encystment appears to increase by 72 hours in ΔchpT. These results are consistent with the hypotheses.](Image)

**Discussion**

The data are partially consistent with the phosphorelay model (Fig 1). ΔchpT inhibits motility on soft agar media and exhibits a hypercyst phenotype. These observations are due to the inability of the phosphotransferase to directly phosphorylate the CtrA response regulator. Although these results are consistent with our hypothesized model, the reintroduction of chpT on a plasmid vector was not observed to rescue wild type motility in the ΔchpT strain. These results suggest further investigation of the effect of both the presence of the plasmid vector and the presence of gentamicin drugs in the media and how these factors effect motility. It is also important to recognize that the inability to rescue a wild type phenotype through the expression plasmid may suggest a more complex phosphorelay in that chpT might be acting in another pathway to phosphorylate two different proteins.

**References**
