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CHEMORECEPTOR SIGNALING ROLE OF TSR RESIDUE G393

Tyler Frederick Crum (John S. Parkinson)
Department of Biology

Escherichia coli cells contain chemoreceptor signaling complexes that detect external chemical gradients and transmit internal signals that control the direction of flagellar motor rotation. These signaling complexes enable the cells to carry out chemotactic behaviors: movements toward metabolizable chemicals (attractants) and away from harmful compounds (repellents).

Chemoreceptor teams form signaling complexes with a kinase, CheA, and modulate its activity in response to stimuli. Results from previous functional studies done in the receptor-kinase interaction domain suggest that packing and dynamic shifts within the receptor protein in the vicinity of its hairpin turn play a role in kinase regulation. We explored this idea by performing mutagenesis on the serine receptor, Tsr, targeting a glycine residue near the hairpin turn, and characterizing the signaling properties of the mutant receptors (Figure 1).

Replacing this glycine with any other residue abolished chemotactic ability in host cells expressing mutant proteins. Several mutants were incapable of assembling into larger signaling arrays, and kinase activity varied between receptors with different amino acid replacements.

The data suggest that changing the structure near the hairpin turn prevents signaling by disrupting stimulus-induced conformations or dynamic behavior of the receptor. Further assays of Tsr-G393* mutants and of receptors subjected to targeted mutagenesis at different sites in this region are underway to further understand the mechanisms behind receptor-kinase control.

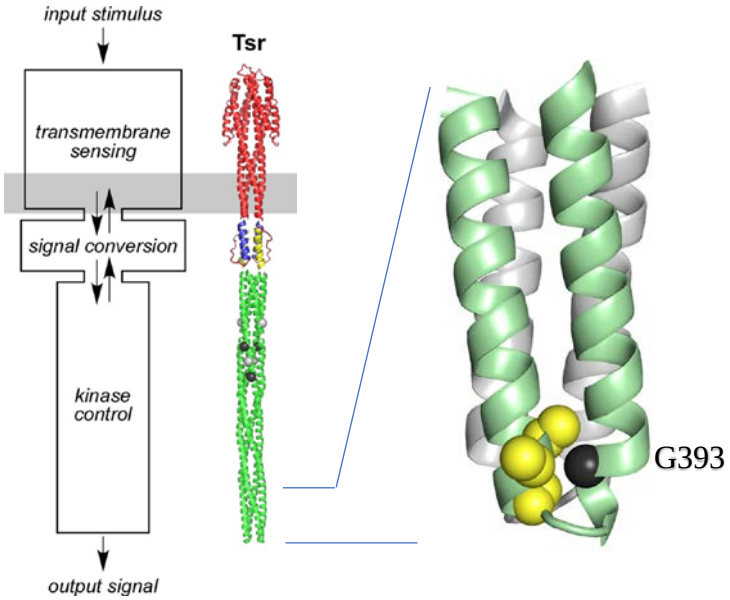

Figure 1. Functional architecture of Tsr.