Abstract Submitted
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Insights in connecting phenotypes in bacteria to coevolutionary information.[1] RYAN CHENG, Rice University, FARUCK MORCOS, University of Texas at Dallas, RYAN HAYES, University of Michigan, RODNEY HELM, University of Houston, HERBERT LEVINE, JOSE ONUCHIC, Rice University — It has long been known that protein sequences are far from random. These sequences have been evolutionarily selected to maintain their ability to fold into stable, three-dimensional folded structures as well as their ability to form macromolecular assemblies, perform catalytic functions, etc. For these reasons, there exist quantifiable mutational patterns in the collection of sequence data for a protein family arising from the need to maintain favorable residue-residue interactions to facilitate folding as well as cellular function. Here, we focus on studying the correlated mutational patterns that give rise to interaction specificity in bacterial two-component signaling (TCS) systems. TCS proteins have evolved to be able to preferentially bind and transfer a phosphate group to their signaling partner while avoiding phosphotransfer with non-partners. We infer a Potts model Hamiltonian governing the correlated mutational patterns that are observed in the sequence data of TCS partners and apply this model to recently published in vivo mutational data. Our findings further support the notion that statistical models built from sequence data can be used to predict bacterial phenotypes as well as engineer interaction specificity between non-partner TCS proteins.

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Ryan Cheng
Rice University

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