Public Abstract

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Title:KINETIC STUDY OF HISTIDINE KINASE CHEA IN BACTERIAL CHEMOTAXIS

Autophosphorylating histidine kinase CheA, the enzyme of bacterial chemotactic signaling, is conserved across bacterial diversity. It is activated approximately 1000-fold upon interaction with transmembrane chemoreceptors to form signaling complexes. In these complexes, its activity is inhibited approximately 100-fold by chemoreceptor saturation with attractant and modulated over 200-fold by chemoreceptor adaptational modification. We characterized these allosteric effects by Michaelis-Menten kinetic analysis and found that all three altered apparent k_{cat} with little effect on K_M for either substrate, ATP or the phosphoryl-accepting kinase domain. Our results indicate that the allosteric effectors target the kinase catalytic mechanism not substrate affinity or availability.