

Glucose sensing in yeast: site-directed and randomized mutagenesis of the Snf1 protein kinase regulatory subunit Snf4

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Abstract:

Many regulatory mechanisms have evolved to ensure proper cellular responses to environmental change. The *Saccharomyces cerevisiae* protein kinase Snf1 is required for cellular response to change of carbon source in the absence of glucose. Snf1 is the yeast homologue of the human enzyme AMP-activated protein kinase (AMPK), a major therapeutic target for Type II Diabetes. In humans, AMPK is activated when its ligand, AMP, binds to its regulatory subunit. Homologous residues involved in ligand binding were identified in the yeast Snf1 regulatory subunit Snf4. These residues were changed by site-directed mutagenesis and the functional consequences assessed. Preliminary results suggest that the homologous AMP binding residues are not important for Snf4 function. Therefore our results suggest that Snf4 may not bind AMP and the regulation of yeast Snf1 complex may be fundamentally different from the regulation of its mammalian homologues.