

P006 Novel Glc7-Reg1 phosphatase – Yck1,2 kinase glucose signalling pathway in *Saccharomyces*

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In *Saccharomyces* glucose causes a rapid decrease in maltose transport activity and degradation of maltose permease, called glucose-induced inactivation of maltose permease. Michels and coworkers reported significant defects in inactivation upon loss of the Glc7 phosphatase type-1 targeting subunit Reg1p. Surprisingly, the extent of maltose permease phosphorylation decreases in a *reg1Δ* strain, suggesting that Reg1p-Glc7p phosphatase acts indirectly on maltose permease via a downstream kinase. We report that casein kinase 1 encoded by *YCK1,2* is involved in maltose permease phosphorylation and glucose-induced inactivation of maltose permease is blocked in a *yck^{ts}* strain. We show that *reg1Δ* and *yck^{ts}* mutants exhibit very similar phenotypes regarding maltose permease localization, phosphorylation, and resistance to inactivation. Additionally, the phenotype of the *reg1Δ yck^{ts}* double mutant is not significantly enhanced compared to the single mutants. Glucose repression-insensitive *glc7* mutations that alter Reg1p-binding also cause insensitivity to maltose permease inactivation, demonstrating that Glc7p – Reg1p interaction, and thus likely phosphatase activity, is required. Overexpression of *YCK1* suppresses *reg1Δ* whereas overexpression of *REG1* does not suppress *yck^{ts}*. These results establish that *REG1* acts upstream of *YCK1,2* in a novel Glc7-Reg1 phosphatase – Yck1,2 kinase signaling pathway controlling glucose-induced inactivation of maltose permease.