P010 G-Protein-Coupled Sugar Sensing in *Arabidopsis* Involving a Novel Plastid Protein

Jirong Huang[1], Jin-Gui Chen[1], Joachim F. Uhrig[2], KwangChul Oh[1], Tsuyoshi Nakagawa[3], Ken L. Korth[4], Daniel Osuna[5], Wolf-Rüdiger Scheible[5], Mark Stitt[5] , Alan M. Jones[1]

[1]University of North Carolina, [2]Max Planck Institute for Plant Breeding Research, [3]Shimane University, [4]University of Arkansas, [5]Max Planck Institute of Molecular Plant Physiology

Regulators of G-protein Signaling (RGS) desensitize G-protein-coupled receptor- (GPCR) mediated signal transduction by accelerating the GTPase activity of the GPCR-activated alpha subunit of the heterotrimeric G protein complex. In yeast and metazoans, GPCR and RGS proteins are separate and antagonistic, however, the single RGS protein in Arabidopsis (AtRGS1) is unusual in that it contains both a 7 transmembrane domain in its amino-terminal half and a functional RGS domain in its carboxy-terminal half. *atrgs1* mutants are insensitive to high concentrations of D-glucose and have altered glucose-induced gene expression. D-glucose causes AtRGS1 to internalizes rapidly, reminiscent of ligand-regulated internalization of GPCRs. AtGIP1, a plastid protein, represents a new AtGPA1 interaction partner in the AtRGS1 glucose sensing pathway. The a*tgip1-1* null mutant is hypersensitive to high D-glucose concentrations and AtGIP1 is rapidly degraded by D-glucose addition. AtRGS1 trafficking requires functional AtGIP1 protein. In summary, a new signal pathway for extracellular D-glucose is defined here by a cell-surface GPCR-like protein coupled via a heterotrimeric G protein complex to an intracellular compartment through interaction with a plastid protein.