# Three Genes That Affect Sugar Sensing (*Abscisic Acid Insensitive 4*, *Abscisic Acid Insensitive 5*, and *Constitutive Triple Response 1*) Are Differentially Regulated by Glucose in Arabidopsis[1]

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Mutant characterization has demonstrated that *ABI4* (*Abscisic Acid* [*ABA*] *Insensitive 4*), *ABI5* (*ABA Insensitive 5*), and *CTR1* (*Constitutive Triple Response 1*) genes play an important role in the sugar signaling response in plants. The present study shows that the transcripts of these three genes are modulated by glucose (Glc) independently of the developmental arrest caused by high Glc concentrations. *ABI4* and *ABI5* transcripts accumulate in response to sugars, whereas the *CTR1* transcript is transiently reduced followed by a rapid recovery. The results of our kinetic studies on gene expression indicate that *ABI4*, *ABI5*, and *CTR1* are regulated by multiple signals including Glc, osmotic stress, and ABA. However, the differential expression profiles caused by these treatments suggest that distinct signaling pathways are used for each signal. *ABI4* and *ABI5* response to the Glc analog 2-deoxy-Glc supports this conclusion. Glc regulation of *ABI4* and *CTR1* transcripts is dependent on the developmental stage. Finally, the Glc-mediated regulation of *ABI4* and *ABI5* is affected in mutants displaying Glc-insensitive phenotypes such as *gins*, *abas*, *abi4*, *abi5*, and *ctr1* but not in *abi1-1*, *abi2-1*, and *abi3-1*, which do not show a Glc-insensitive phenotype. The capacity of transcription factors, like the ones analyzed in this work, to be regulated by a variety of signals might contribute to the ability of plants to respond in a flexible and integral way to continuous changes in the internal and external environment.

Sugars play a central role as signaling molecules that modulate the metabolism, development, and physiology of plants (Koch, 1996; Sheen et al., 1999; Smeekens, 2000; Coruzzi and Zhou, 2001; Rolland et al., 2002). Despite their central function, the molecular mechanisms underlying sugar signaling are still poorly understood. Evidence based on the use of sugar analogs such as 6-deoxy-Glc, 2-deoxy-Glc (2DG), and 3-*O*-methyl-Glc, shows that sugar-mediated regulation occurs through distinct signaling pathways (Sheen et al., 1999; Smeekens, 2000). One of these pathways responds exclusively to Suc (Chiou and Bush, 1998; Loreti et al., 2000); others respond to hexoses, such as Glc and Fru. Some signaling systems rely only on hexose sensing without any catabolism (hexokinase [HXK] independent), whereas others require hexose phosphorylation or even further hexose metabolism (Sheen et al., 1999; Fujiki et al., 2000; Smeekens, 2000; Xiao et al., 2000; Rolland et al., 2002).

At present, few components of the sugar signaling network are known. Evidence exists that, similar to yeast (*Saccharomyces cerevisiae*), the plant enzyme involved in hexose phosphorylation, HXK, functions as a primary sugar sensor (Jang and Sheen, 1997; Rolland et al., 2002; Moore et al., 2003). Protein phosphorylation and dephosphorylation also have been implicated in sugar regulation (Takeda et al., 1994; Ehness et al., 1997; Halford and Hardie, 1998; Fujiki et al., 2000; Smeekens, 2000; Halford et al., 2003).

Genetic approaches also have been used to identify components of the plant sugar signaling cascade (Smeekens, 2000; Rolland et al., 2002). Sugar response mutants have been isolated based on the effects caused by high or low sugar levels during germination or early seedling development. Others have been selected by screening transgenic plants with altered expression of sugar-regulated promoters. Interestingly, several sugar-insensitive mutants affect known genes involved in hormone biosynthesis or signaling (Coruzzi and Zhou, 2001; Gazzarrini and McCourt, 2001; Finkelstein and Gibson, 2002; Leon and Sheen, 2003). Mutants such as *gin1* and *gin5* (Glc-insensitive), *isi4* (impaired Suc induction), and *sis4* (Suc insensitive) affect genes involved in the biosyn-

[1] This work was supported by Consejo Nacional de Ciencia y Tecnológia (grant no. 31791–N and fellowship to A.A.), by the Dirección General de Asuntos para el Personal Académico (grant no. IN210200), by Howard Hughes (grants), and by DGEP (fellowship to A.A.).

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Article, publication date, and citation information can be found at http://www.plantphysiol.org/cgi/doi/10.1104/pp.103.021089.