

A Unique Short-Chain Dehydrogenase/Reductase in Arabidopsis Glucose Signaling and Abscisic Acid Biosynthesis and Functions

Wan-Hsing Cheng,^{a,b,1} Akira Endo,^{c,1} Li Zhou,^a Jessica Penney,^a Huei-Chi Chen,^a Analilia Arroyo,^d Patricia Leon,^d Eiji Nambara,^e Tadao Asami,^f Mitsunori Seo,^{c,e} Tomokazu Koshiba,^c and Jen Sheen^{a,2}

^a Department of Genetics, Harvard Medical School, and Department of Molecular Biology, Massachusetts General Hospital, Boston, Massachusetts 02114

^b Institute of Botany, Academia Sinica, Taipei, Taiwan, Republic of China

^c Department of Biological Sciences, Tokyo Metropolitan University, Hachioji-shi, Tokyo 192-0397, Japan

^d Departamento de Biología Molecular de Plantas, Instituto de Biotecnología, Universidad Nacional Autónoma de México, Cuernavaca, Morelos 62271, México

^e Plant Science Center, RIKEN (The Institute of Physical and Chemical Research), Wako, Saitama 351-0198, Japan

^f Plant Functions Laboratory, RIKEN (The Institute of Physical and Chemical Research), Wako, Saitama 351-0198, Japan

Glc has hormone-like functions and controls many vital processes through mostly unknown mechanisms in plants. We report here on the molecular cloning of *GLUCOSE INSENSITIVE1 (GIN1)* and *ABSCISIC ACID DEFICIENT2 (ABA2)* which encodes a unique Arabidopsis short-chain dehydrogenase/reductase (SDR1) that functions as a molecular link between nutrient signaling and plant hormone biosynthesis. SDR1 is related to SDR superfamily members involved in retinoid and steroid hormone biosynthesis in mammals and sex determination in maize. Glc antagonizes ethylene signaling by activating *ABA2/GIN1* and other abscisic acid (ABA) biosynthesis and signaling genes, which requires Glc and ABA synergistically. Analyses of *aba2/gin1* null mutants define dual functions of endogenous ABA in inhibiting the post-germination developmental switch modulated by distinct Glc and osmotic signals and in promoting organ and body size and fertility in the absence of severe stress. SDR1 is sufficient for the multistep conversion of plastid- and carotenoid-derived xanthoxin to abscisic aldehyde in the cytosol. The surprisingly restricted spatial and temporal expression of *SDR1* suggests the dynamic mobilization of ABA precursors and/or ABA.

INTRODUCTION

Plant growth and development is governed by signaling networks that connect inputs from environmental cues, hormone signals, and nutrient status. Recent studies have suggested a pivotal role of sugars as signaling molecules in plants that integrate external environmental conditions and other nutrients with intrinsic developmental programs modulated by multiple plant hormones (Smeekens, 2000; Coruzzi and Zhou, 2001; Finkelstein et al., 2002; Rolland et al., 2002). How sugar signals influence vital processes from germination, seedling development, root and leaf differentiation, and senescence to stress tolerance remains mostly unknown. Cellular and transgenic studies have indicated the

involvement of hexokinase (HXK) as a sugar sensor with both signaling and metabolic functions in plants (Jang et al., 1997; Smeekens, 2000; Rolland et al., 2002). To further elucidate the molecular mechanisms underlying the plant sugar signaling network, a genetic approach was used to isolate sugar response mutants in Arabidopsis. Based on a bioassay in which a high level of Glc blocks the switch to postgermination development in Arabidopsis, both Glc-insensitive (*gin*) and Glc-oversensitive (*glo*) mutants were isolated. Phenotypic, genetic, and molecular characterization of these mutants has provided new insights into the regulatory mechanisms that link nutrient status to plant hormone synthesis and signaling (Zhou et al., 1998; Arenas-Huertero et al., 2000; Rolland et al., 2002).

Phenotypic analysis of the *gin1* mutant has suggested an interaction between the signaling pathways mediated by Glc and the plant stress hormone ethylene. Further characterization of the ethylene overproduction mutant *eto1*, the constitutive ethylene signaling mutant *ctr1*, and the ethylene-insensitive mutant *etr1* has led to the discovery that the Glc

¹ These authors contributed equally to this work.

² To whom correspondence should be addressed. E-mail sheen@molbio.mgh.harvard.edu; fax 617-726-6893.

Article, publication date, and citation information can be found at www.plantcell.org/cgi/doi/10.1105/tpc.006494.