The **GAOLAOZHUANGREN1** Gene Encodes a Putative Glycosyltransferase that is Critical for Normal Development and Carbohydrate Metabolism

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Glycosyltransferases are enzymes that catalyze the attachment of a sugar molecule to specific acceptor molecules. These enzymes have been shown to play important roles in a number of biological processes. Whereas a large number of putative glycosyltransferase genes have been identified by genomic sequencing, the functions of most of these genes are unknown. Here we report the characterization of an Arabidopsis mutant, designated **gaolaozhuangren1** (*glz1*), which is allelic to *parvus* characterized recently. The *glz1* mutant exhibited a reduced plant stature, reduced size of organs in the shoot and dark-green leaves, indicating an important role of **GLZ1** gene in normal development. The earliest **GLZ1** expression appears at the shoot apical region of 4-d-old seedlings, which coincides with the onset of the *glz1* morphological phenotypes. **GLZ1** is expressed in a tissue-specific and developmentally regulated manner, predominantly in the stem and siliques, and moderately in the flower. **GLZ1** expression is strong in the midrib of rosette and cauline leaves; however, its expression was not detectable in the midrib of the cotyledon. Further analyses revealed that carbohydrate composition and distribution were aberrant in the *glz1* mutant. These, together with the **GLZ1** expression pattern, suggest a requirement for the **GLZ1** function in normal sink–source transition during plant development.

Keywords: Arabidopsis — Assimilate distribution — Development — Glycosyltransferases.

Abbreviations: **GLZ1**, **GAOLAOZHUANGREN1**; Glc, glucose; Fru, fructose; Suc, sucrose.

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**Introduction**

Glycosyltransferases are involved in biochemical reactions that transfer sugar molecules to various acceptors in living cells. More than 7,200 glycosyltransferase genes have been identified thus far in prokaryotes and eukaryotes (Coutinho et al. 2003). On the basis of amino acid sequence, glycosyltransferases have been classified into 65 families (Campbell et al. 1997, Coutinho et al. 2003). Plant glycosyltransferases play divergent roles in different physiological processes including glycolipid (Jarvis et al. 2000, Jorasch et al. 2000) and polysaccharide synthesis (Myers et al. 2000), as well as those relating to cell wall synthesis (Reiter et al. 1997, Falk et al. 2000, Perrin et al. 2001, Bouton et al. 2002, Iwai et al. 2002). In addition, plant glycosyltransferases are capable of adding sugars to small molecules, such as plant hormones (Szerszen et al. 1994, Martin et al. 1999a, Martin et al. 1999b, Jackson et al. 2001), salicylic acid (Lee and Raskin 1999) and flavonoids (Hirotani et al. 2000).

In Arabidopsis, genomic sequencing and annotation have revealed more than 350 putative glycosyltransferase genes (Arabidopsis Genome Initiative 2000). However, the biochemical activities have only been determined for a handful of gene products. The difficulty in assessing the biochemical function of many glycosyltransferases may be in part due to the highly specific activities of these enzymes (Keegstra and Raikhel 2001). Genetic analysis of the *quasimodo1* (*qua1*) mutations in Arabidopsis revealed an important function for the **QUA1** gene, which encodes a putative membrane-bound glycosyltransferase (Bouton et al. 2002). Loss-of-function of **QUA1** resulted in a 25% reduction of galacturonic acid levels in mutant cell walls, compared with those in the wild-type plants. This defect severely affected the development of *qua1* mutant plants, resulting in a dwarf stature and numerous cell protrusions from leaves and hypocotyls (Bouton et al. 2002). These results indicate that **QUA1** is required for normal pectin synthesis and cell adhesion. The **QUA1** protein is a member of the glycosyltransferase family 8 (Bouton et al. 2002). This family comprises proteins of diverse organisms from bacteria to yeast, plant and human; their functions are largely unknown.

Sink to source transition is a defining feature during leaf development. For instance, young leaves are heterotrophic (sink); they depend in part on carbohydrate imported from other regions of the plant. In contrast, mature leaves are...
autotrophic (source); they produce excess amounts of assimilates, which are transported to sinks (Turgeon 1989). While it is known that glycosyltransferases are involved in complex carbohydrate metabolism, their effects on sink–source transition have not been revealed. Here we report the characterization of the Arabidopsis gaolaozhuangren1 (glz1) mutant and identification of the GLZ1 gene via an enhancer trap approach (Sundaresan et al. 1995). GLZ1 encodes a protein identical to PARVUS (Lao et al. 2003), which belongs to glycosyltransferase family 8. Because sugar was not translocated via a default pathway in the glz1 mutant, i.e. from source to sink, we propose that GLZ1 may play a role in sink–source transition by direct and indirect influence on carbohydrate metabolism, cell wall composition and transport function.

**Results**

**Isolation and characterization of the glz1 mutant**

To identify genes important for plant development, we screened Ds insertional lines for mutants with morphological defects. One Ds line was identified to segregate for mutant plants that showed a reduced plant stature and was named glz1. The name gaolaozhuangren1 (glz1) came from a group of small people in a modern Chinese novel. Segregation analyses indicated that the glz1 phenotype was recessive. F1 progeny of a cross between glz1 and Ler all showed wild-type phenotypes, and the F2 generation segregated normal to mutant plants in a 3 : 1 ratio (331 : 107), indicating a single nuclear loss-of-function mutation.

Compared with the wild type, the glz1 mutant was much smaller, with dark-green leaves (Fig. 1A). Although the glz1 mutant had nearly normal overall architecture and organ shapes, the size of all organs was reduced, including leaves (C), floral organs (D) and fruits (E). A top view of a flower shows the release of pollen from anthers in the wild type (F) but not in the glz1 mutant (G) grown at 28°C. Seedlings grown in soil for 7 d showing the size difference between wild type (H) and the glz1 mutant (I). Seedling morphology was nearly identical prior to the emergence of true leaves. In contrast to the wild type, glz1 cotyledon ceased to expand after the emergence of true leaves. WT, wild type Ler. Bars = 0.5 cm in (A), (C) and (D); bar = 1 cm in (B); and bars = 1 mm in (E), (F), (G), (H) and (I).
length of roots (Fig. 1B). The earliest difference in leaf size and leaf color was detected when the first pair of rosette leaves was expanding (Fig. 1I, H), at a time when photoassimilates are transported from the cotyledons to new leaves. These results suggest that GLZ1 function is not critical before the onset of true leaf development.

GLZ1 encodes a putative glycosyltransferase

The glz1 mutant contains a single Ds element (Southern results not shown) with a kanamycin resistance marker, which co-segregated with the glz1 mutant phenotypes (in total, 114 F2 lines with glz1 phenotypes were analyzed), indicating the link between the putative GLZ1 gene and the Ds insertion. The flanking sequence of the Ds insertion was obtained by using TAIL-PCR (Liu et al. 1995). A coding region (At1g19300) was identified by BLAST-searching of the Arabidopsis genome sequence using the Ds flanking sequence. Sequence analysis revealed that the Ds element was inserted into the transcribed region 36 bp upstream of the start codon of a gene (Fig. 2A), with a characteristic 8-bp duplication of genomic sequence flanking the Ds element. This gene encodes a protein in family 8 of the glycosyltransferase superfamily, identical to recently reported PARVUS (Lao et al. 2003).

To determine whether the glz1 mutation was caused by a Ds insertion, we screened for revertants and performed functional complementation by transforming glz1 mutant plants with the putative GLZ1 driven by CaMV 35S promoter (Fig. 2A). Revertants with complete wild-type phenotypes were identified from progeny of glz1 mutant plants carrying an Ac element, and the Ds insertion sites of three revertants were sequenced. Rev#1 has the wild-type sequence. The underlined bases are from the duplication due to Ds insertion; and the boldfaced ones are extra bases added during the repair following Ds excision. Fig. 2B shows a plant from line 1, with a copy of the CaMV35S::GLZ1 transgene in the glz1/glz1 background. Taken together, these results confirm that the Ds insertion into the GLZ1 gene is responsible for the glz1 phenotype.

GLZ1 expression pattern

To better understand GLZ1 function, we examined GLZ1 expression in different wild-type organs by RNA gel blot analyses. GLZ1 cDNA probe detected a 1.3-kb band predominantly in stem and young siliques, moderately in flower and
Role of GLZ1 in plant development

GLZ1 expression in roots was not detectable (Fig. 3). GLZ1 was not expressed in 4-week-old glz1 plants, indicating a block of transcription by Ds insertion (Fig. 3). The Ds insertion carried a GUS reporter gene when inserted into the 5′ untranslated region of the GLZ1 gene, providing a convenient system to monitor the spatial and temporal expression of GLZ1. To learn whether GLZ1 expression is developmentally regulated, we analyzed the patterns of GLZ1::GUS expression in plants heterozygous for the glz1 insertion.

GUS staining was not detectable in most organs in young seedlings (date not shown). Undetectable GUS expression was consistent with the fact that the glz1 mutant at the equivalent developmental stage does not show any phenotype (Fig. 1B). The earliest appearance of clear GUS staining was at the shoot apical region (Fig. 4A, arrowhead) 4 d after germination. GUS staining became more distinct after the onset of true leaf development. The GUS staining was intense in the class I vein (midrib) and trichomes, but weak in the class II vein (arising as single branches from the midrib) in nearly fully expanded leaves (Fig. 4B, C). Whereas GUS staining began to fade in other cell types in older leaves, midrib expression remained constant (Fig. 4D). The strongest GLZ1 expression was seen in inflorescence stems (Fig. 4E). Cross-sections showed that although GUS activity was detected in most cells, the highest expression appeared in the xylem, phloem and endodermis (Fig. 4F, G). In flower buds, GUS staining was visible in sepal (Fig. 4H); however, in mature flowers GUS expression only appeared in the stamen, filament and carpel (Fig. 4I). GUS activity was found in the septum in developing siliques (Fig. 4J). GUS staining was also detected in a region close to the shoot–root junction in 20-day-old plants (data not shown). This highly localized expression was not detectable in the root sample using RNA gel blot analysis.

Altered carbohydrate composition in glz1 mutant

Glycosyltransferases play pivotal roles in carbon metabolism in plants. To determine whether carbon metabolism in glz1 was affected, we conducted a simple comparison of carbohydrate composition between glz1 and wild-type leaves. In wild-type plants, starch content was markedly higher in the first pair of rosette leaves than in the second pair. However, there was no significant difference between the two pairs of rosette leaves in glz1. Furthermore, starch content of the second pair of leaves was slightly higher in glz1 than in the wild type (Fig. 5A). Soluble sugar content were also different between wild-type and mutant plants. Whereas glucose (Glc) and fructose (Fru) contents in the glz1 mutant were much higher in both the first and second pair of leaves than those in wild-type leaves, the

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**Fig. 3** Tissue-specific expression of GLZ1. RNAs were prepared from 4-week-old greenhouse soil-grown plants. The upper panel shows the expression using GLZ1 cDNA as a probe. ACTIN probe was used in the lower panel as control to adjust variation caused by RNA loading.

**Fig. 4** Histochemical analysis of GLZ1 expression in the GLZ1/glz1 plants where no developmental defect was observed. In a 4-day-old seedling (A), GUS staining only appeared at the shoot apex (arrowhead). Young leaves usually showed more extensive GUS activity in the class I and class II veins (B) and trichomes (C). Mature leaves showed more restricted GUS activity in the midrib (D). Strong GLZ1 expression was detected in stems (E, F) where GUS staining was more localized in phloem (ph), xylem (xy) and endodermis (en) (G). In flower buds, GUS staining was visible in sepal (H). However, in mature flowers, GUS activity only appeared in the anther, filament and carpel (I). In siliques, GUS activity was found in septum (arrow). Bars = 1 mm in (A), (B), (D), (E), (H) and (I); 0.5 mm in (C); 40 µm in (F) and (G); and 0.25 mm in (I).
sucrose (Suc) content was higher in the second pair of leaves but lower in the first pair of leaves in the mutant (Fig. 5B). These results suggest that glz1 has altered carbohydrate metabolism or transport.

Abnormal sucrose translocation in glz1 mutant

Sugar translocation is a means of providing energy and structural elements not only for plant growth and development but also for the defense response to biotic and abiotic stresses. To determine whether sugar translocation is defective in glz1, we fed 17-day-old plants with [14C]Suc and monitored its distribution. When [14C]Suc was fed via a single cotyledon, it could only be found in sink tissues including stem, root, developing inflorescence and the youngest rosette leaf of the glz1 mutant, respectively. Soluble sugar contents are higher in glz1 mutant than in the wild type. Mean ± SE is given for three independent samples.

Discussion

We have identified a GLZ1/PARVUS gene via the characterization of a dwarf mutant glz1. GLZ1/PARVUS encodes a putative glycosyltransferase containing 351 amino acids. The GLZ1/PARVUS protein belongs to family 8 of glycosyltransferases; members of this family share a conserved Prodom domain that includes a DXD motif, which is thought to be involved in metal ion binding (Unligil and Rini 2000). Several members of this family have been identified as being involved in the synthesis of lipopolysaccharides [lipopolysaccharide galactosyltransferase (EC 2.4.1.44), lipopolysaccharide glycosyltransferase (EC 2.4.1.58)], glycogen [glycogenin glycosyltransferase (EC 2.4.1.186)] and inositol 1-α-galactosyltransferase (EC 2.4.1.123, http://afmb.cnrs-mrs.fr/CAZY/GT_8.html). In Arabidopsis, the GLZ1/PARVUS protein belongs to a subfamily with 41 members. The C-terminus of these proteins is highly conserved in a region encompassing...
the Prodom domains, whereas the N-terminus is much less conserved (Tavares et al. 2000).

GLZ1 expression is under strict temporal and spatial control. In early seedling development, when cotyledons are expanding, nutrients needed for growth are provided mainly by the storage in the cotyledons, and long-distance sugar transport has not yet begun. At this stage, the mutant seedlings did not show an obvious phenotype and GLZ1 expression was undetectable. GLZ1 expression becomes detectable at the onset of emergence of the first pair of rosette leaves, which marks the beginning of assimilate transport from source (cotyledon) to sink (expanding rosette leaves). The timing of these events coincides with the appearance of the glz1 mutant phenotype. The glz1 phenotype becomes more severe after the first pair of rosette leaves have fully expanded and other rosette leaves begin to emerge. From this stage on, GLZ1 is strongly expressed in midribs of rosette leaves and cauline leaves, as well as inflorescence stems, especially in the vascular tissues. The GLZ1 expression pattern implicates its role in assimilate transport or sink–source transition.

In glz1, carbohydrate composition was abnormal. In the wild type, starch content was higher in the first (source) than in the second (sink) pair of rosette leaves, presumably due to higher photosynthetic competence. However, starch content was approximately the same in both pairs of rosette leaves (sink) in the glz1 mutant and the concentration was comparable to that in the second pair of wild-type leaves. By contrast, the soluble sugar content was much higher in both pairs of glz1 leaves, except that sucrose content was lower in the first pair of glz1 leaves. We cannot rule out the possibility that sucrose concentration was underestimated in the first pair of glz1 leaves. Together these results suggest that starch accumulation is somehow less active in the first pair of glz1 leaves. Intriguingly, this cannot be interpreted solely by the accumulation of glucose and fructose in glz1 because the difference in starch content (Fig. 5A, approximately 150 nmol mg$^{-1}$) was much greater than that in soluble sugar content (Fig. 5B, approximately 25 nmol mg$^{-1}$). As a result, total starch + soluble sugar content in the first pair of leaves was higher in the wild type than that in glz1 mutant. This implies that the first pair of glz1 leaves are not as photosynthetically competent as the wild type and thus suggests a delay/block of transition from sink to source. Starch content showed less difference in the second pair of leaves between glz1 and the wild type presumably because both remained as sink tissue. This notion is consistent with the results of the sugar transport assay (Fig. 6A) in which both pairs of glz1 leaves and the second pair of wild-type leaves were indicated as sugar-importing sink tissues. It is unclear whether accumulation of soluble sugar in glz1 leaves is a result of altered carbohydrate metabolism and/or transport from other organs. Possibilities include abnormal invertase and sugar transporter activities associated with a defective cell wall (Sherson et al. 2003) or decreased usage of glucose/fructose substrate due to defective glycosyltransferase.

The $[^{14}C]$Suc feeding experiment demonstrated that sucrose translocation was abnormal in glz1. In the wild-type plants, the fed $[^{14}C]$Suc was clearly targeted to the sink: young leaves or the apical region where new organs are initiated. Normal sugar transport from source to sink is important not only for the growth of new organs but also for the removal of excess assimilates to relieve feedback inhibition of photosynthesis by sugars in source leaves. In glz1, however, $[^{14}C]$Suc fed from either cotyledon or rosette leaf moved throughout the plant. This, together with low starch content in the first pair of rosette leaves, indicates a delay or block of transition from sink to source in the glz1 mutant. Growing leaves obtain carbon from two sources: import and photosynthesis. The carbon for protein synthesis in growing leaves is derived preferentially, but not exclusively, from photosynthesis, whereas imported sucrose is preferentially used in the synthesis of structural carbohydrates (Joy 1967, Dickson and Larson 1975, Ho and Shaw 1977). One possibility is that the GLZ1 gene was related to the synthesis of a certain carbohydrate structure, which may play an important role in leaf growth and expansion. Loss-of-function of the GLZ1 gene is likely to cause structural defects such as abnormal cell wall that directly or indirectly affect cell expansion and hence hinder sink–source transition. Bouton et al. (2002) proposed that all Arabidopsis members of the glycosyltransferase family 8 might be important for pectin biosynthesis. Pectins are a family of complex polysaccharides present in all plant primary cell walls and may have multiple functions in plant growth and development (Ridley et al. 2001). This is supported by recent characterization of parvus, a mutant allelic to glz1, in which mutation caused an altered cell wall carbohydrate composition and possibly abnormal pectin biosynthesis (Lao et al. 2003). In summary, we have shown that loss-of-function of GLZ1 causes abnormal carbohydrate composition and aberrant sugar translocation. While it is clear that glycosyltransferase is pivotal for plant growth and development, the precise biochemical and cellular functions of GLZ1/PARVUS remain to be elucidated.

Materials and Methods

Plant materials and growth conditions

Ds insertion lines in the Landsberg erecta (Ler) genetic background were generated as described previously (Sundaresan et al. 1995). Mutant and wild-type Ler plants were usually grown at about 22–24°C in a greenhouse with a photoperiod of 16 h light and 8 h dark. Plant growth on agar plates was according to our previous conditions under continuous illumination at 22°C (Li et al. 1999). Mutant plants were identified in the F3 generation following crosses between Ac and Ds plants. Heterozygous siblings of the glz1 mutant plants were allowed to mature and their seeds planted to determine segregation frequency. Normal and mutant plants segregated in an approximate 3 : 1 ratio, supporting the hypothesis that the mutant phenotype was caused by a single-gene recessive mutation.
Morphological observations and detection of GUS activity

Fresh tissues of wild-type and mutant plants were examined using a SZH10 dissecting microscope (Olympus, Japan), and photographed using a Nikon E995 digital camera (Nikon, Japan). Histochemical detection of GUS activity was carried out with 5-bromo-4-chloro-3-indolyl β-D-glucuronic acid (X-Gluc) as a substrate. Plant tissues were placed in X-Gluc solution [750 µg ml⁻¹ X-Gluc, 100 mM NaPO₄ (pH 7), 3 mM K₃PO₄, 10 mM EDTA and 0.1% Nonidet-P40] under vacuum for 10 min at room temperature, then incubated overnight at 37°C for 1–3 d. Tissues were cleared with 95% ethanol, and viewed and photographed in 50% glycerol, using the SZH10 dissecting microscope.

Molecular cloning and revertant analysis

Genomic DNA was isolated as described previously (Huang et al. 1994). To isolate the GLZ1 gene, we obtained a 0.6 kb genomic fragment from the 3’ end of the Ds element, using the TAIL-PCR procedure (Liu et al. 1995). The TAIL-PCR was performed with the Ds-specific primers (Ds3-1, Ds3-2 and Ds3-3; Grossniklaus et al. 1998) and arbitrary degenerate primers (Liu et al. 1995). The GLZ1-specific primers were designed based on the sequence information of the TAIL-PCR product: 2247–3, 5’-AGAGCTTAAGAATCCAACGCGA-3’; and 2247-5, 5’-AGGATCCATGT CCCAACATCTT-3’ (Fig. 2A).

To determine whether the glz1 mutation was caused by a Ds insertion, we screened for revertants. No obvious somatic revertant sectors in the F2 progeny from a cross between glz1 mutant plants and an Ac line (Ac–1, Sundaresan et al. 1995). We then analyzed F2 progeny from individual F2 lines with phenotypes, and identified F3 plants with wild-type-like plants. We PCR-amplified and sequenced the genomic region flanking the Ds insertion from these wild-type-like plants, and found that the Ds element had been excised with footprints left in two of those lines (Fig. 2B), indicating that Ds insertion resulted in glz1 phenotypes.

Complementation of the glz1 mutation

DNA BLAST analysis revealed that there is no intron in the GLZ1 gene, so that the genomic DNA and 2247-3 and 2247-5 primers were used to amplify the entire GLZ1 coding region. To complement the glz1 mutations, the amplified DNA fragment containing the entire putative GLZ1 coding region from the wild-type Columbia ecotype was cloned into the vector pGEM7Z(+)/+, between BamHI and SacI sites to yield the plasmid H451. After sequence verification, a HindIII–BamHI fragment containing a 35S promoter from the pBI121 vector was inserted into the H451, upstream of the GLZ1I fragment, resulting in the plasmid H452. The 35S:GLZ1 fragment was then released from H452 by HindIII and NsiI digestion and inserted into the HindIII and PstI sites of a binary T-DNA vector pCAMBIA1301 to yield the construct H453. This construct was then introduced into plants heterozygous for glz1 mutant. We also analyzed T2 segregating progeny of line 1, 3 and 5 transformants. Our results demonstrated that plants with the glz1 phenotype in T2 all lost the 35S:GLZ1I transgene in the glz1/glz1 genotype (data not shown).

RNA gel blot hybridization analysis

Total RNA isolation, gel separation and RNA blotting were according to our previous protocols (Huang et al. 1995). Blots were made using Amersham Nylon N° membranes and hybridization was performed as recommended by the manufacturer using 32P-labeled probes (Pharmacia, Sunnyvale, CA, U.S.A.). Hybridization and washing were conducted at a stringent temperature (65°C).

Carbohydrate analysis

Seventeen-day-old seedlings of wild type and glz1 mutant were used to detect the carbohydrate content. The first and second pairs of rosette leaves were collected in liquid nitrogen separately and lyophilized when used. The lyophilized leaves (2.5 mg each) were heated in boiling water in Eppendorf tubes for 20 min to destroy the endogenous enzymes. The leaves were then ground and mixed with 200 µl double distilled water in a vortex. The resulting samples were incubated at room temperature for 25 min with occasional vortexes, followed by centrifugation at 10,000g for 10 min. The supernatants were used for soluble sugar assays with an enzymic method (Hampp et al. 1994). The absorbance was recorded by a microplate reader system (Sunrise, Phenix, Candler, NC, U.S.A.). Starch in pellets was determined following enzymic digestion as described previously (Ooblen and Brown 1994). Three independent samples were measured to get the mean ± SE.

[14C]Feeding

The [14C]Suc feeding experiment was performed according to the method described previously (Gottwald et al. 2000), with modifications. Briefly, one cotyledon (from 17-day-old seedlings) or rosette leaf (from 28-day-old seedlings) from each plant to be fed was crimped with forceps, and 0.3 µl of [14C]Suc (0.2 mM, m⁻¹, Sigma, St. Louis, MO, U.S.A.) was placed at the crimped point on the cotyledon or rosette leaf. The drop was left for 2 h before the seedlings were dissected. Each leaf from a fed seedling was wrapped with the Saran Wrap and exposed to a phosphor screen (Molecular Dynamics, Boston, MA, U.S.A.) for 24–48 h before the screen was scanned.

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References


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