Plant Morphology of Heterotrimeric G Protein Mutants

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The heterotrimeric G protein complex, comprising Gα, Gγ and Gβ subunits, is an evolutionarily conserved signaling molecular machine that transmits signals from transmembrane receptors to downstream target proteins. Plants conserved the core G protein elements, while developing their own regulatory systems differently from animals. Genetic evidence supports the conclusion that the heterotrimeric G proteins regulate shoot, root and epidermis development, as well as sugar sensing, hormone responsiveness and abiotic and biotic stress tolerance. This review is a compendium of the known morphological changes conferred by loss- and gain-of-function mutations of the G protein subunit genes across three higher land plant models, namely Arabidopsis, rice and maize.

Keywords: AGB1, GPA1, CT2, d1, DEP1, GS3.

Abbreviations: AGB1, Arabidopsis G protein β subunit 1; AGG, Arabidopsis G protein γ subunit; CT2, Compact Plant2; DEP1, Dense and Erect Panicle 1; d1, dwarf1; GCR1, G protein-coupled receptor 1; GPA1, G protein α subunit 1; GPCR, G protein-coupled receptor; GS3, Grain Size 3; IM, inflorescence meristem; QTL, quantitative trait locus; RGS, regulator of G protein signaling; SAM, shoot apical meristem; 7TM, seven transmembrane; XLG, extra-large G protein.

Introduction to G Protein Signaling

Animal heterotrimeric G proteins serve as physical couplers between seven transmembrane (7TM) G protein-coupled receptors (GPCRs) and downstream components designated as effectors (Kaziro et al. 1991). G proteins have three subunits: Gα, Gβ and Gγ, among which the Gα subunit binds a guanine nucleotide: GDP or GTP. A ligand-bound GPCR induces exchange of GDP for GTP on Gα leading to its conformational change and G protein complex dissociation. The active Gα or Gβγ subunits then interact with downstream effectors and modulate their activities. Intrinsc GTP hydrolysis by Gα returns it to the GDP-bound, basal state. Regulator of G protein signaling (RGS) proteins accelerate GTP hydrolysis by Gα, thereby suppressing G protein activity. Plants lack the conventional G protein regulation by GPCRs, because their G proteins spontaneously activate themselves without GPCRs (Johnston et al. 2007, Urano et al. 2012). Plants have G protein-coupled receptor 1 (GCR1), a 7TM protein weakly homologous to the Dictyostelium CAMP receptor (Colucci et al. 2002); however, its action on G proteins remains equivocal (Chen et al. 2004, Pandey et al. 2006). Most vascular plants, except cereals, utilize a 7TM RGS protein to modulate their G protein activity (Chen et al. 2003, Urano et al. 2012), although the entire regulatory system still remains unclear (Urano et al. 2013). The Arabidopsis genome encodes four Gα genes, one canonical Gα (AtGPA1) and three non-canonical extra-large Gα (XLG1, XLG2 and XLG3), a single Gβ gene (AGB1), three Gγ genes, i.e. two typical Gγ (AGG1 and AGG2) and an atypical Gγ (AGG3), and one 7TM RGS (AtRGS1). The Gγ gene duplications and evolution led to functional specialization in the plant G protein network (Chakravorty et al. 2011, Li et al. 2012, Thung et al. 2012, Trusov et al. 2008). The non-canonical Gγ proteins, XLG1, XLG2 and XLG3, have an N-terminal cysteine-rich domain and a C-terminal Gα-like domain, although the Gα-like domain lacks several Gα signatures required for GTP hydrolysis and Gβγ and RGS interactions. Fig. 1 summarizes the domain structures and the nomenclature of G protein components along with mutations discovered by forward genetics in rice.

Shoot Morphologies of Gα, RGS1 and GCR1 Mutants

Arabidopsis, rice and maize Gα mutants, gpa1, ‘daikoku’ dwarf1 (d1) and compact plant2 (ct2), respectively, produce shorter but wider shoot tissues (Fujisawa et al. 1999, Ullah et al. 2001, Bommert et al. 2013). The Arabidopsis gpa1 mutation confers a shortening and a widening of hypocotyls, flowers, siliques and seeds to different degrees. Fig. 2A–C presents some obvious phenotypes (e.g. leaf shape), while others (e.g. silique length) are mildly affected (Ullah et al. 2001, Ullah et al. 2003, J.G. Chen et al. 2006, Chakravorty et al. 2011). Rice and maize Gα null alleles exhibit more severe defects; nearly all mutant shoot tissues are approximately 25–50% shorter than those of the wild-type siblings (Fujisawa et al. 1999, Bommert et al. 2013). Fig. 2D–K presents side-by-side views of the morphologies of the wild type and Gα mutants of rice and maize. Gα null rice DK22, one of five original rice d1 alleles (Fujisawa et al. 1999), shortens plant height by 52%, the floral bract by 25%, the seeds...
Fig. 1 Domain structures of plant G protein components. (A) Two types of Gα subunits, namely canonical Gα and non-canonical XLG. The Gα proteins have a single Gα domain comprising two subdomains, i.e. the Ras-homology domain and the Helical domain. Canonical Gα has a well-conserved myristoylation site at the second glycine, and guanine nucleotide-binding motifs. Non-canonical XLG proteins have an N-terminal cysteine-rich domain, a nuclear localization signal and an unusual Gα-like domain, which lacks some residues essential in nucleotide hydrolysis. (B) The Gβ subunit has N-terminal coiled-coil helices and a tryptophan–aspartic acid 40 (WD40) repeat domain. (C) Three types of Gγ proteins: type-A, -B and -C Gγ subunits. An N-terminal Gγ domain forms a coiled-coil with the Gβ subunit. Type-A Gγ has a well-conserved prenylation motif (CaaX motif) and a potential palmitoylation site near the C-terminus. While type-B Gγ proteins lack the prenylation motif, the rice type-B Gγ protein (RGG2) is membrane associated by an unknown interaction. Type-C Gγ has a transmembrane (tm) helix and a C-terminal extracellular cysteine-rich domain. Some type-C Gγ proteins have a CaaX motif. Rice forward genetics identified point mutations, frameshifts and truncations in canonical Gα (RGA1, not shown) and type-C Gγ genes (DEP1 and GS3, shown in C) that confer developmental anomalies. Note that rice DEP1 and GS3 proteins vary in size (426 and 232 residues, respectively), due to a highly divergent extracellular domain. (D) Two seven transmembrane (7TM) proteins, RGS1 and GCR1. RGS1 has a 7TM region, a cytoplasmic RGS domain and C-terminal phosphorylation sites. The 7TM region has no homology to any reported GPCRs or to GCR1. GCR1 has a 7TM region, presumably having a protein fold similar to GPCRs. GCR1 is genetically uncoupled with the G protein complex in Arabidopsis development and any role for GCR1 in G protein-dependent signaling is not clear. (E) A regulatory model of the G protein complex. GDP-bound Gα forms an inactive heterotrimer with GβGγ in the resting state. Gα spontaneously exchanges GDP for GTP, releases GβGγ and then modulates downstream target proteins, also known as effectors. Freed GβGγ also modulates its own effectors. 7TM RGS1 promotes GTP hydrolysis by Gα, returning to an inactive state. An action of GCR1 on the G protein complex remains equivocal. A XLG pathway is largely unknown, except the physical and genetic association with GβGγ. The illustrations were modified from Urano et al. (2013).
modulates shoot morphologies. Arabidopsis rgs1 null alleles, in which Gx signal is presumably hyperactive, enhance leaf and hypocotyl outgrowths similar to the ectopic Gx-Q222L expression (J.G. Chen et al. 2006, Chen et al. 2003), while RGS1 overexpression confers shorter hypocotyls, smaller rosettes and delayed flowering (Y. Chen et al. 2006, Johnston et al. 2007). The gpa1 rgs1 double mutant shows an epistatic interaction with the archetypical gpa1 shoot phenotypes, indicating that these two components work in the same genetic pathway (Y. Chen et al. 2006). In contrast, knockout of a putative 7TM receptor, GCR1, in the Col-0 ecotype or in the G protein mutants causes no developmental abnormality except an early-flowering phenotype observed in an overexpression line of GCR1, suggesting no connection with the G protein complex in shoot development (Colucci et al. 2002, Chen et al. 2004, Chakraborty et al. 2015). Arabidopsis xlg3 mutants, like gpa1, displayed a shorter and wider hypocotyl (Pandey et al. 2008); however, epistasis analysis that would reveal its interaction with other G protein subunits has not been reported.

**Shoot Morphologies of Gβ and Gγ Mutants**

Compared with gpa1 null alleles, Arabidopsis Gβ null mutants, agg1, have more severe shortening of the hypocotyls, leaves, petioles, flowers, siliques and seeds (Fig. 2A–C), while their widths are increased to a similar level (Lease et al. 2001, Ullah et al. 2003, J.G. Chen et al. 2006, Chakraborty et al. 2011). The agg1 null mutants produce more flowers (Trusov et al. 2008). The gpa1 agg1 double knockout mutants indicate an apparent epistasis of the agg1 null allele to the gpa1 null allele (J.G. Chen et al. 2006), implying that AGB1 acts downstream of GPA1, that the intact G2βγ complex is essential for the function or that atypical XLGs function redundantly in the same pathway. No Gβ knockout line has been isolated in rice, probably due to its embryonic lethality (Utsunomiya et al. 2012). A reduced expression of the rice Gβ gene by RNA interference shortens and narrows leaf sheaths and blades (Utsunomiya et al. 2011), while the ectopic expression of Gβ increases tillers and reduces leaf length (Sun et al. 2014). None of these Gβ or Gβ mutations decrease cell size in shoot tissues (Ullah et al. 2001, Ullah et al. 2003, Oki et al. 2009b, Utsunomiya et al. 2011, Bommer et al. 2013); therefore, the shortened organs, caused by the Gx or Gβ mutations, are due to reduced cell proliferation (Fig. 3A, B).

Seed plants possess three types of Gγ subunits classified by their domain structures and lipid modification sites (Trusov et al. 2012). Type-A Gγ has a prenylation site (CaaX motif) at the C-terminus, while type-B Gγ lacks this motif (Fig. 1C). Type-C Gγ has a transmembrane region and an extracellular cysteine-rich domain (Wolfenstetter et al. 2015). Gβ primarily co-operates with the atypical type-C Gγ (e.g. Arabidopsis agg3) in shoot development. Null mutations of Arabidopsis agg3 lead to abnormal shoot morphologies, including shorter hypocotyls, siliques and seeds (Chakravorty et al. 2011, Li et al. 2012), whereas overexpression of AAG3 enlarges leaves, flowers, seeds and siliques (Li et al. 2012). Mutations in the two type-A Gγ subunits agg1 and agg2 did not lead to abnormal shoot development (Fig. 2A–C); however, AGG1 and AGG2 may still support longitudinal shoot growth, as the agg1 agg2 agg3 triple mutant shows more severe shortening of leaves, flowers and siliques than the agg3 single allele (Trusov et al. 2008, Thung et al. 2012). The agg1 agg2 agg3 triple mutant shares all the agg1 mutant shoot morphologies (Thung et al. 2012, Chakravorty et al. 2015), probably because Gβ is degraded in planta without Gγ (Wolfenstetter et al. 2015), indicating that Gγ is an
The Arabidopsis G protein network also regulates stomata formation, most probably through control of cell proliferation. G protein network in rice development involves different G\textgamma\gamma subtypes to sort G protein pathways.

Forward genetics studies using rice substantiate the type-specific G\textgamma\gamma function. Rice has five G\textgamma\gamma homologs, a type-A G\textgamma\gamma 1 (RGG1), a type-B G\textgamma\gamma 2 (RGG2) and three type-C G\textgamma\gamma genes, Dense and Erect Panicle 1 (DEP1)/qPE9-1/DN1, Grain Size 3 (GS3) and G\textgamma\gamma type-C 2 (OsGGC2) (Kato et al. 2004, Fan et al. 2006, Huang et al. 2009, Zhou et al. 2009, Taguchi-Shiobara et al. 2011, Trusov et al. 2012). Two rice quantitative trait loci (QTLs), which are associated with grain density per panicle or grain size, arise from point mutation, frameshifts or deletions of the DEP1 and GS3 genes (Fig. 1C). Similar to the type-C G\textgamma\gamma AGG3, DEP1 and GS3 proteins have an N-terminal G\textgamma\gamma domain, a transmembrane region and a predicted extracellular cysteine-rich domain. A premature stop codon of GS3 in the middle of the G\textgamma\gamma domain (c165a, TGC\textgamma\gamma\gamma) confers increased grain length by approximately 10%, whereas several premature terminations or frameshifts in the cysteine-rich domain (e.g. a 1 bp deletion at c357, Fig. 1C) decrease grain length (Fan et al. 2006, Takano-Kai et al. 2009, Mao et al. 2010, Takano-Kai et al. 2013). The c165a allele (gs3-3, also known as Minghui 63) is a recessive loss-of-function mutation. Suppression of the short-grain gs3 gene (the c57– allele, gs3-4) by RNA interference expands grain length (Mao et al. 2010), suggesting that the GS3 protein gains a function by the elimination of the cysteine-rich domain. Another G\textgamma\gamma gene, DEP1/qPE9-1/DN1, regulates plant height, panicle erectness, and grain density and yield (Huang et al. 2009) (Fig. 2D, G). The dep1-1 allele, whose protein product lacks the entire cysteine-rich domain, increases grain quantity and primary and secondary branches per panicle, and enlarges shoot apical meristems while decreasing plant height, panicle length and grain weight (Huang et al. 2009). Other DEP1 mutations, which similarly truncate the protein, demonstrate comparable phenotypes (Zhou et al. 2009, Taguchi-Shiobara et al. 2011, Sun et al. 2014). The dep1-1 allele is partially dominant, as ectopic expression of the truncated DEP1 protein recapitulates all the phenotypes in the near isogenic line (Sun et al. 2014), whereas dep1-32 (g277t, GGA\textgamma\gamma\gamma\gamma>TGA) that expresses a G\textgamma\gamma domain and a few residues of the transmembrane region is a recessive loss-of-function allele. These observations lead to the proposition of a model whereby the cysteine-rich domain inhibits the G\textgamma\gamma/DEP1 or G\textgamma\gamma/GS3 signals, and that eliminating part of or the entire cysteine-rich domain releases the G\textgamma\gamma\gamma dimers from this autoinhibition (Botella 2012). Rice plants overexpressing RGG1, RGG2 or GS3 are shorter compared with the parental line, although this effect has not been quantified (Mao et al. 2010, Sun et al. 2014). Further mutant analyses, including loss-of-function alleles for RGG1, RGG2 or OsGGC2 genes, are necessary for understanding of the G protein network in rice development.

**Stomatal Development in G Protein Mutants**

G proteins are firmly established as being involved in the control mechanism for cell proliferation. The increased shoot branches of rice are related to enhanced cell proliferation or reduced determinacy of meristems. The rice G\textgamma\gamma dep1 mutant has an enlarged inflorescence meristem (Huang et al. 2009), with increased panicle branches. Maize G\textgamma\gamma also regulates both the shoot apical meristem (SAM) and inflorescence meristem (IM). The maize G\textgamma\gamma mutant ct2 has enlarged SAMs; however, their identity and organization are normal, as determined by KNOTTED1 expression analyses (Bommert et al. 2013). ct2 ear primordia have enlarged IMs, starting very early in development, leading to the initiation of extra rows of spikelet pair meristems. The tassel IMs of ct2 are also larger (Bommert et al. 2013). Abnormal meristems are similarly produced in Arabidopsis G protein mutants. While Arabidopsis gpa1 mutants display no obvious change in SAM height, the gbg1 and gbg1 agg2 double null alleles have approximately 40% taller meristems (Ishida et al. 2014). Both maize CT2 and Arabidopsis AGB1 function in the CLAVATA pathway, and transmit CLAVATA3 ligand-dependent signals to control meristem size, through leucine-rich repeat receptors for CLAVATA3, maize FASCIATED EAR2 or Arabidopsis Receptor-like kinase2 (Bommert et al. 2013, Ishida et al. 2014). Although these studies suggest that the G protein network co-operates with CLAVATA receptors to regulate stem cell fate, further studies are needed to understand fully the roles of G proteins in meristem regulation.
et al. 2001) and approximately 10% more in cotyledons (Zhang et al. 2008). The rgs1 null allele similarly enhances stomatal density (Zhang et al. 2008), probably due to increasing the steady-state GPA1 activity. In contrast to the gpa1 null allele, the Arabidopsis agb1 null mutant shows slight stomatal clustering, and increased stomatal density by 25% (Zhang et al. 2008). The Gα and Gβ pathways seem to control stomatal production in cotyledons antagonistically, because the gpa1 and agb1 mutations display an additive effect on stomatal formation (Zhang et al. 2008).

The role of Gβ in stomatal development is coupled primarily with the typical Gγ gene, AGG1. Loss-of-function alleles of agg1, but not agg2 or agg3, promoted stomatal proliferation to a level similar to agb1 (Chakravorty et al. 2015). Interestingly, the agg1 agg2 double mutant exhibited the highest stomatal density, even greater than the agb1 or agg1 agg2 agg3 triple mutant (Chakravorty et al. 2015), implying that the typical Gγ subunit suppresses while the atypical Gγ subunit partially promotes stomatal development. The xlg1 xlg2 xlg3 triple knockout, but none of the xlg single null alleles, also enhances stomatal formation (Chakravorty et al. 2015). Epistasis analysis with the Gβ or Gγ null alleles has not been tested. Insights into the underlying cellular mechanisms have come from findings that the gpa1 null mutations delay and agb1 null mutations promote asymmetric cell divisions during stomatal lineage progression (Zhang et al. 2008). Further research over successive developmental stages should elucidate how these G protein mutants alter stomatal proliferation at a molecular level.

**Root Morphologies of G Protein Mutants**

Arabidopsis, rice and maize Gα null alleles decrease root growth similarly, despite their different root architectures, namely taproots in Arabidopsis vs. fibrous roots in rice and maize (Ullah et al. 2003, Izawa et al. 2010, Urano et al. 2015b). The Arabidopsis gpa1 mutant has a normal primary root length but fewer lateral roots, leading to a more compact root architecture (Ullah et al. 2003, J.G. Chen et al. 2006) (Fig. 4A), although the gpa1 effect is subtle and therefore often is overlooked with agar plate-based assays. The null alleles in rice (d1) and maize (ct2; Fig. 4B) also exhibit a slight reduction in root growth, approximately 10% shorter roots and 15% fewer seminal or crown roots compared with their wild-type sibs (Izawa et al. 2010, Urano et al. 2015b). The Gx-null mutations probably lead to a decrease in cell proliferation at the root apical meristem, because Gx function does not affect root cell elongation (Izawa et al. 2010). The ectopic Gx-Q222L mutation promotes primary root elongation in the opposite way due to increased cell proliferation (Chen et al. 2003).

The Arabidopsis agb1 null mutant shows a more expanded root architecture, presumably due to increased cell proliferation and lateral root formation (Ullah et al. 2003) (Fig. 4A). The agb1 phenotype is epistatic to gpa1, because the root architecture of the gpa1 agb1 double mutant resembles that of the agb1 mutant (J.G. Chen et al. 2006). AGB1 overexpression decreases lateral root formation, opposite to the loss-of-function phenotype (J.G. Chen et al. 2006). The rgs1 null allele accelerates primary root elongation but does not affect lateral root formation (J.G. Chen et al. 2006), whereas the gcr1 null alleles show no defect in root development or in shoot development (Pandey et al. 2006, Pandey et al. 2008), again questioning its potential involvement in G protein signaling. The xlg1 xlg2 xlg3 triple null mutant, like agb1, has a longer primary root and more lateral roots (Ding et al. 2008), although the two genotypes should be compared under the same growing conditions. However, xlg1, xlg2 or xlg3 single or double knockouts show barely changed root growth, presumably due to redundancy (Ding et al. 2008).

The Arabidopsis G protein complex uses Gγ subunits spatially in shoot and root development. While the atypical AGG3 gene plays a main role in shoot development (see above), the two typical AGG1 and AGG2 genes mainly contribute to root development (Trusov et al. 2007), particularly to lateral root formation. The agg1 or agg2 mutants produce more lateral roots than Col-0, and the double mutants additively increase lateral roots to a level comparable with the agb1 allele (Trusov et al. 2007). It remains untested if this functional selectivity for Gγ subunits occurs similarly in rice and other plants.

**Summary**

Arabidopsis, rice and maize G protein mutants display comparable morphological anomalies, despite their distinct plant architectures. Consistent defects observed in G protein mutants are
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<th>Mutant</th>
<th>Shoot morphology</th>
<th>Root morphology</th>
<th>Others</th>
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<tr>
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<td>gpa1 agb1</td>
<td>Phenocopies agb1 (Chen et al. 2006a)</td>
<td>Phenocopies agb1 (Chen et al. 2006, Pandey et al. 2008)</td>
<td>Phenocopies agb1 (Zhang et al. 2008)</td>
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<tr>
<td>agg1</td>
<td>Wild-type-like hypocotyls, leaves, petioles, flowers and siliques (Trusov et al. 2008)</td>
<td>More lateral roots (Trusov et al. 2007)</td>
<td>Higher stomatal density (Chakravorty et al. 2015)</td>
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<tr>
<td>agg2</td>
<td>Wild-type-like hypocotyls, leaves, petioles, flowers and siliques (Trusov et al. 2008)</td>
<td>More lateral roots (Trusov et al. 2007)</td>
<td>Wild-type-like stomatal density (Chakravorty et al. 2015)</td>
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<tr>
<td>agg3</td>
<td>Shorter and wider leaves, shorter hypocotyls (Chakaravorty et al. 2011, Thung et al. 2012),</td>
<td>Wild-type-like roots (Chakravorty et al. 2011)</td>
<td>Wild-type-like stomatal density (Chakravorty et al. 2011, Chakravorty et al. 2015)</td>
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<tr>
<td>agg1 agg2</td>
<td>Wild-type-like hypocotyls, leaves, petioles, flowers and siliques (Trusov et al. 2008)</td>
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<td>agg1 agg2 agg3</td>
<td>Shorter and wider leaves, shorter siliques and flowers (Thung et al. 2012)</td>
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<td>Higher stomatal density (Zhang et al. 2008)</td>
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<td>rgs1</td>
<td>Longer etiolated hypocotyls, leaves and seeds (Chen et al. 2003, Chen et al. 2006a)</td>
<td>Longer primary roots (Chen et al. 2003)</td>
<td>Higher stomatal density (Zhang et al. 2008)</td>
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<td>gcr1</td>
<td>Wild-type-like hypocotyls, plant height, leaves and siliques (Chen et al. 2004, Chakraborty et al. 2015)</td>
<td>Wild-type-like roots (Pandey et al. 2008)</td>
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<td>35S::GPA1</td>
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<td>Shorter primary roots (Chen et al. 2006a)</td>
<td>Higher stomatal density (Zhang et al. 2008)</td>
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<td>Rice</td>
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more compact shoot architectures and altered branching patterns during the reproductive stages. The reduced organ sizes are due to lower cell proliferation activity along the longitudinal axis (Ullah et al. 2001, Ullah et al. 2003, Oki et al. 2009b, Utsunomiya et al. 2011), while changes in branching patterns are associated with enlarged meristems (Huang et al. 2009, Bommert et al. 2013, Ishida et al. 2014) and could in part be explained by control of Gz by a master regulator of branching, as evident in the case of the regulation of maize CT2 expression by the RAMOSA1 gene (Eveland et al. 2014). The G protein complex modulates longitudinal growth potential in response to environmental factors such as light, temperature, nutrients and ions (Utsunomiya et al. 2011). This idea is supported by evidence that the maize Gz null ct2 mutant shows effects resembling the inhibitory effect of sodium chloride on cell proliferation (Uranov et al. 2014), and the rice Gγ mutant dep1 also phenocopies the growth inhibition caused by nitrogen deficiency (Sun et al. 2014). Classical plant hormone pathways including auxin, abscisic acid and gibberellin also co-ordinate with the G protein complex in various developmental processes (Uranov et al. 2013). Future research should elucidate: (i) the cell type-specific function of the G protein network in cell proliferation; (ii) their co-ordination with environmental factors with regard to cell proliferation; and (iii) the regulatory systems of the G protein network in greater depth.

There are also important differences between species. For example, maize Gz mutants have larger shoot meristems, but similar phenotypes are not seen in Arabidopsis (Bommert et al. 2013, Ishida et al. 2014). Some of these differences could be due to redundancy, as plants increased the repertoire of G protein genes during evolution, while deleting some genes in specific lineages, resulting in diversity in this signaling system. For example, Arabidopsis and its close relatives lack the type-B Gγ gene (Trusov et al. 2012), and most cereals lack the RGS gene (Urano et al. 2015a). The observed natural variation in primary structures presumably makes G protein interactions selective and signaling outputs specific. The lack of a 7TM RGS gene in cereals makes research with rice and maize of paramount importance, because no regulatory element has been identified. Experimental evidence with multiple models will lead to unexpected discoveries as well as strengthening of our current knowledge of G protein function during plant development. These hopefully will translate into improvements in crop architecture for increased harvest index.

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<td>rgb1-RNAi</td>
<td>Longer, wider and heavier seeds (Utsunomiya et al. 2011)</td>
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<td>slower, shorter, and narrower seeds. Brown lamina joint regions and nodes (Utsunomiya et al. 2011)</td>
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<td>Wild-type-like plant height (Sun et al. 2014)</td>
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<td>35S::RGA1-Q223L</td>
<td>Longer and heavier seeds, longer internodes (35S::RGA1-Q223L in d1 background) (Oki et al. 2005)</td>
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<td>pActin::RGG1</td>
<td>Shorter mature plants (Sun et al. 2014)</td>
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Disclosures

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References


