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DELLA Proteins and GA Signalling in *Arabidopsis*

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Abstract

Gibberellin (GA) is a classical plant hormone involved in many aspects of plant growth and development. A family of five homologs called the DELLA proteins, comprised of GAI, RGA, RGL1, RGL2 and RGL3, were recently found to act as critical GA signal mediators in *Arabidopsis.* Reports have shown that GAI and RGA are coupled together to repress stem elongation growth whereas RGL2 is a major negative regulator of seed germination. GA down-regulates DELLA proteins through protein degradation likely via the proteasome pathway. The conserved and functionally important DELLA domain is responsible for protein stability in response to GA.

Key words: Gibberellin; DELLA proteins; GAI/RGA/RGL1/RGL2/RHT/d8; Internode elongation; Seed germination; Signal transduction; Proteasome

Introduction

Gibberellin is an essential plant hormone involved in regulating many aspects of plant growth and development including seed germination, hypocotyl and stem elongation, leaf expansion, pollen development and flower initiation (Harberd and others 1998; Richards and others 2001). Although the function of GA as a hormone in regulating plant growth was known as early as the 1950s (Brian and Hemming 1955; Vlitos and Meudt 1957), significant progress in understanding the biochemical and molecular genetic mechanism of GA function has been made very recently. Understanding of GA functions mainly comes from the investigation of mutant plants with aberrant GA responses. These plants can be divided into two groups. The first group comprises mutants defective in GA biosynthetic enzymes and are hence GA deficient. These mutants (for example, *ga1* alleles) are generally defective in seed germination and are severely dwarfed in the absence of exogenous GA. The other group of mutants, found in a number of plant species, are defective in the components of the GA signaling pathway but have normal or elevated levels of endogenous GA and are associated with variable phenotypes. This later group can further be divided into two classes: the semidominant semidwarf mutants with reduced GA sensitivity and the recessive null mutants, which are associated with GA over-dose phenotypes. Advances in molecular biological and genetic tools in recent years have lead to the identification of a number of genes involved in the GA signaling pathway. These genes include *GAI* (*GA-insensitive*), *RGA* (*Repressor of ga1-3*), *RGL1* (*RGA-like 1*), *RGL2*, *AGL20* (*AGAMOUS-LIKE 20*), *SUPERMAN*, *SPY* (*SPINDLEY*) and *PICKLED* in *Arabidopsis*, *GAMYB*, *PKABA1* and *SLN1* (*Slender 1*) in barley, *SLR1* (*Slender Rice 1*) in rice, *PHOR1*

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(*PHOTOPERIOD RESPONSIVE 1*) in potato, *KNOX* in tobacco, *rht* (*reduced height*) in wheat, *d8* (*dwarf 8*) in maize and *VvGAI1* (*Vitis Vinifera GA insensitive 1*) in grapevine (reviewed in Peng and Harberd 2002; Boss and Thomas 2002).

A number of review articles have recently been published on GA signaling reflecting the vigorous investigation and rapid progress in the understanding of GA function (Thornton and others 1999; Lovegrove and Hooley 2000; Sun 2000; Harberd and others 1998; Richards and others 2001; Peng and others 2002; Olszewski and others 2002). In this review, we summarize the roles of GAI and RGA in GA signaling and regulation of stem growth in *Arabidopsis* and the role of RGL2 in GA-dependent seed germination. In addition, we also discuss the importance of the DELLA domain for protein stability in response to GA.

GAI and *RGA*: Repressors of Plant Growth

Roles of GA in regulating growth of plants have been known for decades (Brian and Hemming 1955; Vlitos and Meudt 1957). Of particular interest is the reduced-height variety of wheat (*Rht-B1* or *Rht-D1*) which is relatively insensitive to GA and has a shorter stature with increased crop yield. Introduction of these varieties of wheat to farmers worldwide in the 1960s and 1970s dramatically increased world wheat production, a development popularly known as the 'Green Revolution' (Dyson 1996; Conway 1997; Evans 1993; Gale and Youssefian 1985). A similar dwarf allele, *gai* (*GA insensitive*), of *Arabidopsis*, having reduced GA sensitivity and shorter height, was reported in 1985 (Koornneef and others 1985). Cloning of the mutant *gai* gene demonstrated that it differed from the wild-type *GAI* gene by a 17-amino acid in-frame deletion near the N-terminus of the encoded protein (Peng and Harberd 1993; Peng and others 1997) and this deletion was responsible for the reduced GA sensitivity. Studies of a *Ds*-disrupted null-allele of *GAI*, named *gai-t6*, demonstrated that the growth of *gai-t6* required less GA than its wild-type counterpart, suggesting that *GAI* functions as a negative regulator of GA in plant growth (Figure 2) (Peng and others 1997).

Peng and colleagues, while cloning the *GAI* gene, also reported the cloning of a *GAI*-related sequence gene (*GRS*) having 83% identity in amino acid sequence of its encoded protein with that of *GAI*. The cloning of the *GRS* gene was independently reported as a repressor of the *ga1-3* phenotype and was named *RGA* (Silverstone and others 1998; Peng and others 1997). *RGA* was discovered as a suppressor of the phenotypes of the *ga1-3 Arabidopsis* mutant. The *ga1-3* plants are defective in GA biosynthetic enzyme with very little endogenous GA and are associated with severe dwarfism, dark green leaves, reduced apical dominance, male sterility and nongerminating seeds (Koornneef and van der Veen 1980). All of these phenotypes, however, can be completely rescued by exogenous GA application. Mutations in the *RGA* gene can partially suppress multiple aspects of the *ga1-3* phenotype and the double mutant *ga1-3 rga-24* plants are taller, paler green and more apically dominant compared to the *ga1-3* plants although they are relatively shorter, darker and less apically dominant than the wild-type plants (Silverstone and others 1998). However *ga1-3 rga-24 Arabidopsis* plants are still male sterile and their seeds cannot germinate, suggesting that *RGA* may not play a major role in regulating these functions.

A clear understanding of the growth-inhibitory function of *GAI* and *RGA* came from an investigation of the effect of the null-alleles of *GAI* (*gai-t6*) and *RGA* (*rga-24*) on the GA-deficient *ga1-3* plants (Dill and Sun 2001; King and others 2001). It was found that the *rga-24* allele substantially suppressed multiple aspects of *ga1-3* phenotypes (discussed above) whereas *gai-t6* allele had a weaker suppression. However, the combination of *gai-t6* and *rga-24* alleles with *ga1-3* resulted in a GA-overdose phenotype, with complete rescue of the retarded elongation growth and delayed flowering phenotypes of *ga1-3*. Apparently, both GAI and RGA act as repressors of plant growth and GA functions through removing the negative growth regulatory effect of GAI and RGA to promote growth (Figure 2), whereas in the *gai* mutant, the in-frame deletion of 17 amino acids in the DELLA domain (named after the first five residues) does not result in loss of function of GAI; instead it leads to a form that converts gai mutant protein into a constitutive repressor of plant growth (Figures 1, 2)

Soon after the cloning of *GAI* and *gai* (and *GRS*∼*RGA*) genes, Peng and others went on to clone the genes responsible for reduced GA sensitivity and shorter height varieties in wheat (*Rht-1*) and maize (*D8*). It was found that the *rht-1* and *d8* genes have significant homology in amino acid sequence with *GAI* and *RGA* in their encoded proteins while the reduced GA sensitivity and dwarfish phenotypes were conferred by mutation in a limited region near the N-terminus. Like the *gai* mutation, mutations in *Rht1* and *D8* converted rht1 and d8 into constitutive

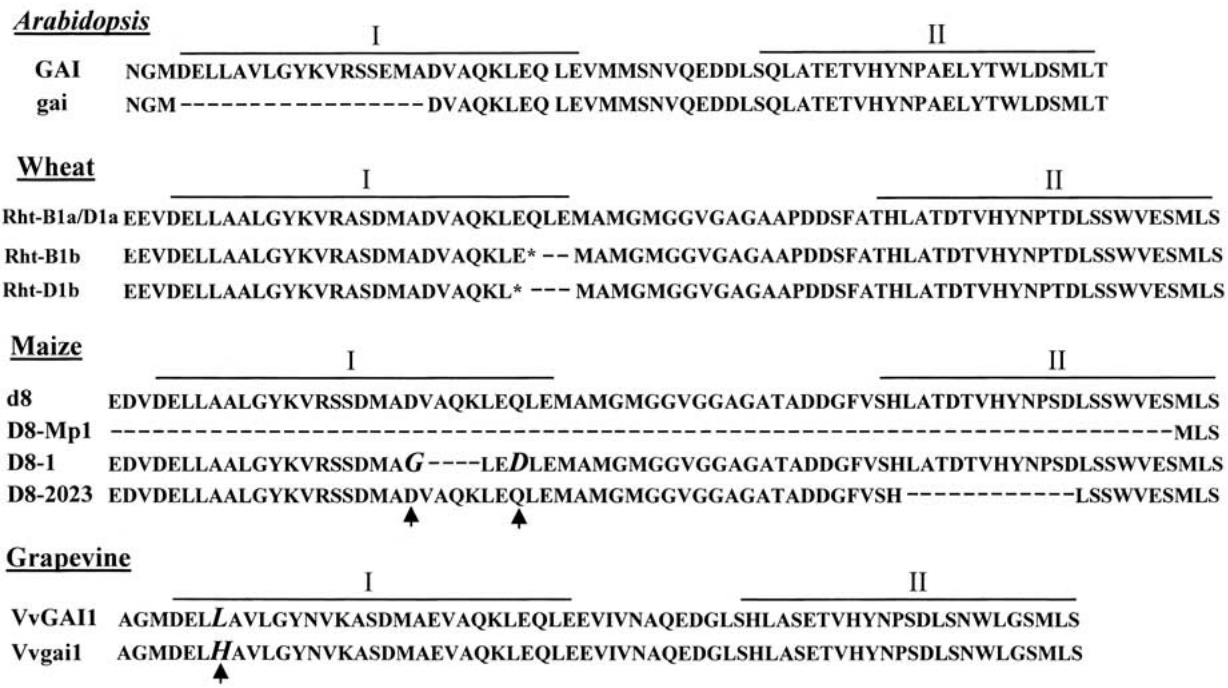

Figure 1. Comparison of mutations in DELLA proteins in various dominant mutant alleles of *Arabidopsis*, wheat, maize and grape. For each species, the wild-type sequence is shown above the mutant sequences. Amino acid deletions (−), mutational stop codons (*), and substitution of amino acid residues (larger fonts along with arrows) are shown. All mutations are confined to a highly conserved limited segment near the N-terminus, as indicated by regions I and II. *D8-2023* mutant allele also carries a 6-base deletion, which removes a GA dipeptide from 510GAGA513, plus a nucleotide substitution, which converts T519 to A519 near the C-terminus (not shown). Because these changes are poorly conserved, they may be of no phenotypic significance. In wheat, Q64 of *Rht-B1a* is equivalent to Q62 of *Rht-D1a* because of a difference of two amino acid residues in a poorly conserved N-terminal region (see text, data not highlighted).

repressors of plant growth. Thus, *GAI* and *RGA* (in *Arabidopsis*) and *rht-1* (in wheat) and *d8* (in maize) formed a family of orthologous genes in plants (Figure 1) (Peng and others 1999).

Genes, homologous to *GAI* and *RGA* have also been identified in other plant species. The null mutants, *sln1* (*slender 1*) in barley and *slr1* (*slender rice 1*) in rice are associated with tall and slender GA overdose phenotypes (Ikeda and others 2001; Chandler and Robertson 1999; Chandler and others 2002). Cloning of the *SLN1* and *SLR1* genes showed a strong homology with *GAI* and *RGA* of *Arabidopsis* including the conserved DELLA domain. Recently, a grapevine dwarf variant derived from the L1 cell layer of the champagne cultivar Pinot Meunier, which produces inflorescences along the length of the shoot where tendrils are normally formed, was found to be associated with a point mutation in the DELLA domain (DELLA in WT vs DELHA in mutant) gene homologous to wheat 'green revolution' genes *Rht-1* and *Arabidopsis* gene *GAI* and is named *VvGAI1* (Boss and Thomas 2002). All these proteins are now called DELLA proteins (Figures 1, 2).

Another GA response gene called *SPINDLY* (*SPY*), which is a *O*-linked N-acetylglucosamine transferase involved in *O*-GlcNAcylation of proteins, also functions as a negative regulator of GA action, similar to *GAI* and *RGA*, but has no homology with the latter (Jacobsen and others 1996; Hartweck and others 2002) and will be discussed in detail in a separate article in this issue.

RGL1 and *RGL2* in Seed Germination

Although the combination of *RGA* and *GAI* null mutations (*gai-t6/rga24*) resulted in restoration of retarded stem elongation and delayed flowering caused by GA deficiency of the *ga1-3* allele, the *ga1-3/rga-t2/gai-t6* plants were still severely defective in seed germination, leaf expansion and male sterility, suggesting that either GA may be essential for promoting these functions or other genes and possibly other DELLA proteins may be involved in regulating those functions in *Arabidopsis*. Indeed, *RGL1* has recently been shown to have inhibitory roles in

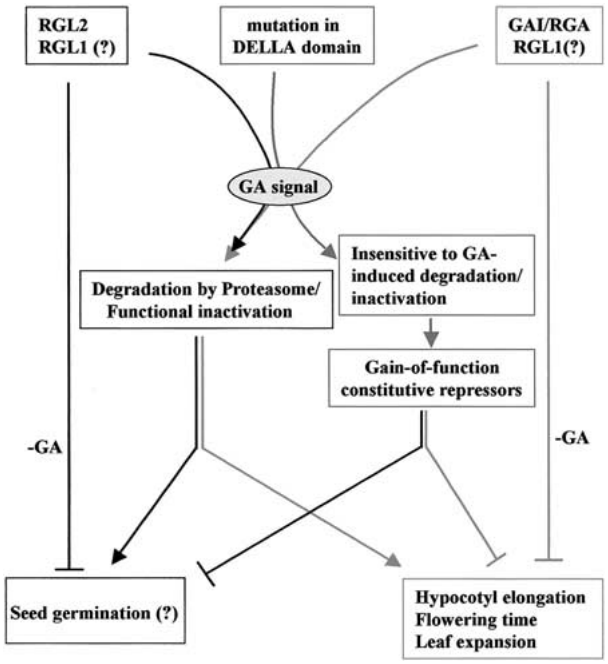

Figure 2. A model for the mechanism of DELLA protein functions in GA signaling in *Arabidopsis*. DELLA proteins, namely, GAI, RGA and RGL1, repress hypocotyl elongation, flowering time, leaf expansion and so on. Whereas RGL2 and possibly RGL1, repress seed germination. Under the normal condition (wild-type), endogenously synthesized GA induces degradation of RGA, RGL2 and GAI (?) by proteasome or functional inactivation of GAI (?) and RGL1 (?) via an unknown mechanism resulting in removal of the repressive effects of these proteins and thus allows normal growth and development. In the absence of GA, for example, in the GA biosynthesis mutant *ga1-3*, these repressors cannot be degraded which accounts for the severely retarded growth and development. Critical mutations in the DELLA domain render these proteins insensitive to GA-dependent degradation/inactivation while retaining their repressive functions, which make them constitutive repressors, resulting in similar phenotypes as those of GA-deficient mutants.

regulation of GA-dependent seed germination, leaf expansion, flowering, stem elongation and floral development (Wen and Chang 2002). These authors have demonstrated that a co-suppression line of *Arabidopsis*, which has reduced RGL1 expression, can withstand a PAC concentration (GA biosynthesis inhibitor) at which the wild-type seeds fail to germinate, suggesting that the RGL1 is a negative regulator of GA in promotion of germination. Our laboratory has shown that RGL2 plays a major role in seed germination because *RGL2*-null mutation can promote germination of non-germinating *ga1-3* seeds, which have an absolute requirement of exogenous GA for germination (Lee and others 2002). We have also demonstrated that *RGL2*-null mutant seeds of *Arabidopsis* can germinate at a concentration of PAC at which wild-type seeds cannot germinate, which again suggests that RGL2 inhibits the effect of GA on seed germination. *RGL2*-null mutation, however, does not affect other phenotypes of *ga1-3* plants. These results suggest that individual DELLA proteins in *Arabidopsis* may regulate different aspects of GA responses. It is possible that dicotyledonous plants such as *Arabidopsis* may have acquired multiple copies of the DELLA protein homologs during evolution and that an individual member of these genes is responsible for regulation of one or more aspects of GA functions whereas monocotyledonous plants like rice and barley possess only one copy of the DELLA protein gene in the genome but it regulates multiple aspects of GA response, including stem elongation, flowering and floral development, seed germination and so on. Assignment of one or more aspects of GA-regulated functions to individual DELLA proteins in dicots allows for temporal and tissue-specific expression whereas in monocots, the single copy of the DELLA protein genes are being expressed throughout their life-time and in all tissues. Indeed, the expression of *RGL2* is found to be more tissue-specific (highly expressed in inflorescence) and also changes dynamically during seed germination whereas GAI and RGA expression is more widespread (Lee and others 2002; Wen and Chang 2002).

The DELLA Proteins

The DELLA proteins in different species now include GAI, RGA, RGL1, RGL2 and RGL3 in *Arabidopsis*, rht-1 in wheat, d8 in maize, SLN1 in barley, SLR1 in rice and a recently identified VvGAI1 in grape. These genes actually fall into a bigger group of genes known as the *GRAS* family in *Arabidopsis* (Pysh and others 1999). The DELLA proteins share a high homology with other GRAS family proteins at the C-terminus but not at the N-terminus. The DELLA proteins from different plant species, however, share a significant homology in amino acid sequence at the N-terminus as well including the highly conserved DELLA domain (Figure 1). Apart from DELLA, these proteins also share multiple conserved sequence motifs, which include a putative nuclear localization signal (NLS), two LXXLL motifs, an src homology 2 (SH2)-like domain, similar to transcription factor, STATs (Peng and others 1997, 1999; Richards and others 2000).

GFP-fused RGA, RGL1 and SLR1 have been reported to translocate into the nuclei of cells

(Silverstone and others 2001; Wen and Chang 2002; Itoh and others 2002). Ongoing work in our laboratory demonstrates that RGL2, when expressed as a GFP fusion in the tobacco BY2 cell line, also localizes in the nuclei (Hussain and Peng unpublished). It would be interesting to prove if the proposed putative NLS in DELLA proteins alone could guide the nuclear localization. The LXXLL motifs are known to mediate binding of transcriptional co-activator to nuclear receptors, which suggests that DELLA proteins may function as a co-activator/regulator of transcription. The absence of a typical basic DNA binding domain suggests that DELLA proteins are more likely to function as transcriptional regulators instead of as transcription factors. SH2 domains are involved in interacting with phosphorylated tyrosine residues and are important components of many signaling pathways in metazoans and slime molds. However, this signaling mechanism seems to be absent in plants and yeast (Peng and others 1999; Richards and others 2000). The observation that DELLA proteins contain a conserved SH2-like domain raised the possibility that plants may also have SH2-phosphotyrosine signaling mechanisms. However, thus far there is no experimental evidence to support this idea.

How GA Overcomes DELLA Protein Functions

It is now clear that the DELLA proteins are negative regulators of GA function, and plants require GA to overcome the effects of these proteins on plant growth and development (Figure 2). The important question is how GA overcomes the inhibitory effects of these proteins. Exciting progress has been made in this area recently. The RGA, SLN1 and SLR1 proteins have recently been shown to disappear from the nucleus of cells when treated with GA, which suggests that GA promotes destabilization of these proteins (Silverstone and others 2001; Gubler and others 2002; Itoh and others 2002). Our ongoing work demonstrates that the RGL2 protein expressed in BY2 cells also quickly disappears upon treatment with GA (Hussain and Peng unpublished). These reports suggest that GA may overcome the inhibitory effect of DELLA proteins on plant growth and development by removing them from cells via degradation. It has also been shown that deletion of the DELLA domain from RGA and SLR1 rendered them insensitive to GA-dependent destabilization (Ito and others 2002; Dill and others 2001). Deletion of the DELLA and C-terminal 260-547 amino acid segment of RGL2 also makes the resultant proteins insensitive to GA-mediated degradation (Hussain and Peng unpublished) suggesting that both N- and C-terminal sequence elements are necessary for the responsiveness of the RGL2 protein to GA-dependent degradation.

Although the complete mechanism by which DELLA proteins are degraded by GA is not clear, involvement of the ubiquitin/proteasome-dependent mechanism has been predicted (Dill and others 2001; Gubler and others 2002; Itoh and others 2002). Indeed, the involvement of the ubiquitin/proteasome mechanism in GA-induced degradation of SLN1 has been demonstrated very recently (Fu and others 2002). Fu and colleagues also demonstrated a possible involvement of both serine-threonine and tyrosine kinases and phosphatases in this process.

Although the degradation of RGA, SLR1, SLN1 and RGL2 is GA-dependent, a GFP-fused form of GAI and RGL1 did not disappear upon treatment with GA (Fleck and Harberd 2002; Wen and Chang 2002). However, deletion of the DELLA domain of RGL1 repressed GA responses. Obviously, the DELLA domain is important for protein stability in response to GA, however, the biochemical mechanism behind this remains unknown (Figure 2).

Discussion and Future Perspectives

The major focus of recent research in the field of GA is to understand the signaling mechanism by which GA regulates plant growth and development. Indeed significant headway has been made in that direction in the last decade or so. It has now been demonstrated that the DELLA proteins in general have a repressive effect on various aspects of plant growth and development and GA functions simply by overcoming the repressive effect of these proteins. The major mechanism by which GA overcomes the effect of DELLA proteins is the destabilization/degradation of the latter. This degradation seems to be mediated via the ubiquitin/proteasome pathway for cellular protein degradation although detailed studies are needed to address the exact biochemical mechanism (Figure 2). There are several highly conserved domains in DELLA proteins. Apparently, it is also important to reveal the function of each individual domain.

Although the question of how GA overcomes the inhibitory effects of DELLA proteins is becoming increasingly clear, it is important to address the mechanism of how the presence of DELLA proteins arrests many aspects of plant growth and development. Because DELLA proteins are likely to be

regulators of gene transcription, it is time to focus attention on the target genes being regulated by DELLA proteins. In fact, a report has shown that the rice DELLA protein SLR1 affects gene transcription when expressed in spinach (Ogawa and others 2000). A very recent report, which employed a proteomics approach to compare protein profiles of wild-type and GA-deficient seeds during germination, has also identified a number of GA-regulated genes (Gallardo and others 2002). It is important to identify which sets of these genes are related to RGL2 and RGL1 and the mechanism by which RGL2 and RGL1 regulate these genes. Similarly, the target genes for regulation by RGA and GAI have to be identified and it must be determined how these genes arrest the proliferation and differentiation of cells at the growing tip of the shoot apical meristem.

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