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Green Revolution DELLA Proteins: Functional Analysis and Regulatory Mechanisms

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DELLA proteins, transcription regulators, gibberellin signaling

Abstract

The *DELLA* genes, also referred to as Green Revolution genes, encode conserved master growth regulators in plants. The nuclear-localized DELLA proteins are transcription regulators that interact with hundreds of transcription factors and other transcription regulators. They not only function as gibberellin signaling repressors in vascular plants but also play a central role in coordinating diverse signaling pathways in response to both internal hormonal signals and external cues (e.g., light and nutrient conditions, biotic and abiotic stresses). Through a combination of genetic, genomic, biochemical, and structural studies, significant advances have been made in understanding both the functional domains and motifs within DELLAs and the molecular mechanisms underlying their function. Here, we highlight new insights into the molecular workings of DELLA proteins, including an evolutionary perspective.

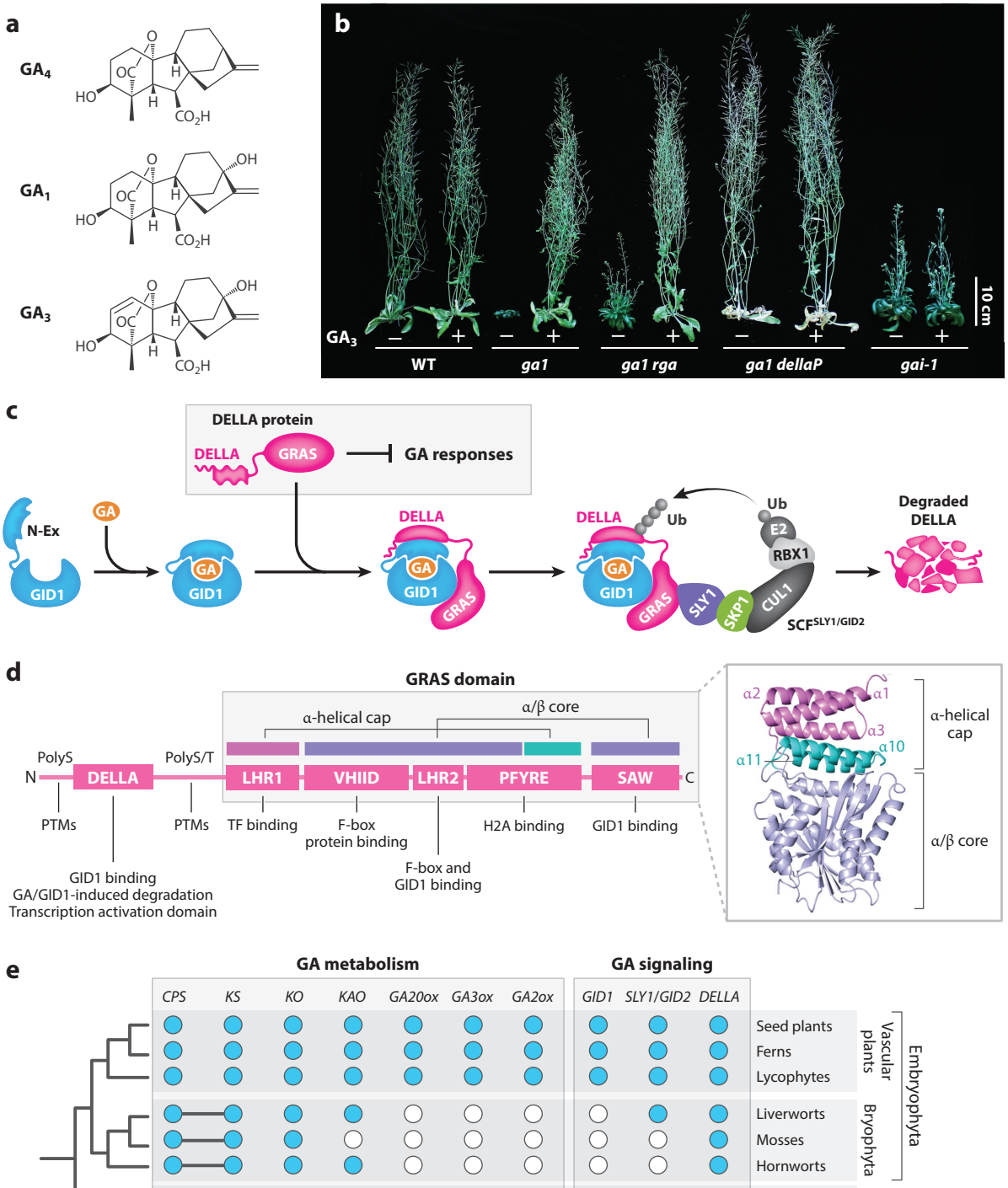
Contents

1. INTRODUCTION

Bioactive gibberellins (GAs) are diterpenoid phytohormones (**Figure 1*a***), which play a key role in regulating diverse processes throughout the plant life cycle. These functions range from promoting seed germination to modulating vegetative and reproductive development (117). GA research has had profound impacts on agriculture and horticulture, exemplified by the development of the high-yielding semidwarf rice and wheat varieties that catalyzed the Green Revolution in the 1960s (93). The dwarfing traits observed in these crops were later shown to be the result of reduced GA biosynthesis (106) or GA response (92). Furthermore, GA biosynthesis inhibitors are employed to reduce the stature of nursery plants, while applications of GAs are routine practices in agriculture. For instance, they are used to increase the fruit size of seedless grapes, to improve skin finish in apples, and to increase malting of barley during beer production (98).

Green Revolution: a period of technological advances in agriculture leading to a dramatic increase in crop yields

Over the last three decades, the GA biosynthesis and catabolism pathways in plants have been elucidated at the molecular level (reviewed in 40, 108). Significant strides have also been achieved in understanding GA perception and early signaling pathways through genetic, biochemical, and structural analyses (108). GA binds to and activates its nuclear receptor, GIBBERELLIN-INSENSITIVE DWARF1 (GID1), which in turn triggers polyubiquitylation and rapid degradation of downstream repressors known as DELLA proteins via the 26S proteasome. This GA-induced ubiquitylation of DELLAs is mediated by specific F-box proteins, GID2 in rice (*Oryza sativa*) and SLEEPY1 (SLY1) in *Arabidopsis*, which are an integral component of



(Caption appears on following page)

Figure 1 (*Figure appears on preceding page*)

Structure and function of DELLAs as GA signaling repressors. (*a*) The major bioactive GAs. Although many GAs have been identified, most are biosynthetic intermediates or catabolites of bioactive GAs, and only very few are active growth regulators in plants. GA_4 and GA_1 are the major bioactive GAs produced in plants, whereas GA_3 (also known as gibberellic acid) is mainly made in fungi. (*b*) Phenotypes of GA-related *Arabidopsis* mutants. Shown are 50-day-old WT and mutant lines in the Col-0 background. All plants were untreated (−) or treated (+) weekly with 100 μM GA_3. We thank Jianhong Hu for help with the photo. (*c*) A model for GA-induced GID1–DELLA interaction followed by $SCF^{SLY1/GID2}$ binding and DELLA degradation. GA binding to GID1 induces a conformational switch in the N-Ex of GID1, facilitating its binding to the DELLA domain. This interaction causes the DELLA domain to transition from a disordered state to a stable structure, illustrated by the change from a wavy to a smooth shape. This interaction then promotes the GRAS domain of the DELLA protein binding to GID1. This stable complex formation enables recognition of DELLA by $SCF^{SLY1/GID2}$, leading to its polyubiquitylation and degradation by the proteasome. (*d*) Schematic of functional domains/subdomains within the DELLA protein and the predicted 3D structure of the GRAS domain. The 3D model of the RGA GRAS domain was generated using the SCARECROW protein (PDB ID 5B3G) (47) as a scaffold. The GRAS domain contains an α-helical cap (α1–α3 in magenta and α10 and α11 in cyan) and an α/β core (*purple*). Panel adapted from Reference 52. (*e*) Evolution of GA metabolism and signaling genes. Panel adapted from Reference 41 with permission from Elsevier. Abbreviations: 3D, three-dimensional; *dellaP*, *della pentuple*; GA, gibberellin; GAI, GIBBERELLIN INSENSITIVE; GID, GIBBERELLIN-INSENSITIVE DWARF; LHR, Leu heptad repeat; N-Ex, amino-terminal extension; PDB ID, Protein Data Bank identifier; PTM, posttranslational modification; RGA, REPRESSOR OF *ga1-3*; SCF, Skp1-Cullin-F-box protein; SLY1, SLEEPY1; TF, transcription factor; Ub, ubiquitin; WT, wild type.

the SCF (Skp1–Cullin–F-box protein) E3 ubiquitin ligase complex. DELLAs function as nuclear transcription regulators lacking a canonical DNA-binding domain. Extensive studies in the past 15 years revealed that DELLAs not only repress GA signaling but also are master growth regulators, integrating the activities of various signaling pathways through direct protein–protein interaction with a myriad of regulatory proteins (23, 108, 124, 128). Phylogenetic and functional studies indicate that the GA-GID1-DELLA module is highly conserved among vascular plants but not in the bryophytes (41). Intriguingly, DELLAs are conserved in all land plants. This review aims to highlight new insights into the molecular mechanisms of DELLA function and the evolution of GA and DELLA signaling in plants.

2. DELLA PROTEINS AND THE GA-GID1-DELLA REGULATORY MODULE

2.1. The Historical Discovery of DELLAs as Gibberellin Signaling Repressors

Forward genetic screens for mutants with either impaired or elevated GA response were pivotal in identifying GA signaling components. Recessive mutants that do not respond to GA show a characteristic dark green, dwarf phenotype similar to that of GA biosynthesis mutants, but their growth cannot be restored by GA treatment. This mutant class includes defects in genes encoding the GA receptor GID1 in rice or the downstream activator F-box protein GID2/SLY1 in rice and *Arabidopsis* (81, 107, 126). Conversely, gain-of-function DELLA mutants are semidominant GA-unresponsive dwarves, exemplified by *gibberellin insensitive-1* (*gai-1*; **Figure 1*b***) in *Arabidopsis* (65) and *Reduced height* (*Rht*) varieties in wheat (*Triticum aestivum*), which significantly contributed to the Green Revolution by augmenting grain yield in the 1960s–1970s (92, 93). By contrast, loss-of-function DELLA mutants, for example, *la cry* in pea (*Pisum sativum*) and *slender* (*sln*) in barley (*Hordeum vulgare*), are recessive and display a slender, tall phenotype mimicking wild-type plants overdosed with GA (8, 32). With the emergence of *Arabidopsis* as a model system for plant research, loss-of-function DELLA mutants were isolated by screening for suppressors of the extreme dwarfism of the GA biosynthesis mutant *ga1-3* and were named *repressor of ga1-3* (*rga*) (114) (**Figure 1*b***). Ultimately, cloning of *GAI* and *RGA* genes was achieved, aided by the isolation of intragenic suppressors of *gai-1* containing transposon *Ds* insertions (90, 91) and through genomic subtraction using *rga* alleles containing large deletions (112). The *Arabidopsis* genome contains

SCF E3 ubiquitin ligase: Skp1, Cullin, and F-box protein complex that catalyzes the covalent linkage of a ubiquitin chain to a specific target protein

five *DELLA* genes: *RGA*, *GAI*, *RGA-LIKE1* (*RGL1*), *RGL2*, and *RGL3*. RGA and GAI serve as the major repressors for vegetative growth and floral induction (26, 63). RGL2 plays a predominant role in seed germination, although the four other DELLAs also regulate this process (67, 125). Floral development is modulated by RGA, RGL1, and RGL2. Due to the functional redundancy among DELLAs, the absence of RGA function only partially alleviates the *ga1* phenotype (114) (**Figure 1*b***). However, knocking out all five *DELLA* genes in the quintuple *della* mutant [named *global-DELLA* or *della pentuple* (*dellaP*)] fully restores all GA-deficient phenotypes of *ga1* (87) (**Figure 1*b***). Further examination of orthologs in various species reveals the conservation of *DELLA* genes in plants. These include *Rht* in wheat (92), *SLENDER RICE1* (*SLR1*) in rice (55), *SLN1* in barley (15), *D8* and *D9* in maize (*Zea mays*) (92), and *LA* and *CRY* in pea (132).

GRAS transcription regulators: a family of plant-specific transcription regulators, named after the founding members GAI, RGA, and SCARECROW

Yeast two-hybrid (Y2H): an experimental test for protein–protein interaction based on the modularity of the yeast transcription factor Gal4

2.2. GA-GID1-Dependent DELLA Degradation Mediated by Ubiquitylation via SCF$^{SLY1/GID2}$ E3 Ligase

Following the identification of DELLAs as conserved GA signaling repressors, further research revealed an exciting GA-mediated regulatory mechanism controlling their stability. In addition, DELLAs have emerged as master growth regulators that integrate multiple signaling pathways in response to internal and external cues (23, 108, 124, 128).

2.2.1. The GA-GID1 complex promotes DELLA degradation via SCF$^{SLY1/GID2}$ E3 ligase.

DELLAs comprise a subfamily of the plant-specific GRAS (for GAI, RGA, and SCARECROW) transcription regulators, which all contain a conserved C-terminal GRAS domain (92, 97, 112) (**Figure 1*c*,*d***). The N-terminal DELLA domain, named after the conserved DELLA sequence motif, is unique to the DELLA subfamily (97, 122). The levels of DELLA proteins in vascular plants are tightly regulated by the bioactive GAs. GA-induced DELLA degradation was first revealed by observing dramatic reductions in the levels of both endogenous RGA and green fluorescent protein (GFP)-RGA proteins in *Arabidopsis* seedlings just minutes after GA treatment (113). Characterization of the *gai-1* mutant predicts that this semidominant allele encodes a mutant protein with a 17-amino-acid in-frame deletion spanning its DELLA motif, suggesting that this lesion converts the GAI protein into a constitutive GA signaling repressor (90). Moreover, deletion of the identical DELLA motif in RGA (rga-Δ17) blocks GA-induced degradation and confers a GA-unresponsive dwarf phenotype, confirming that the DELLA motif is required for GA-dependent proteolysis (25). Similarly, the semidwarf Green Revolution varieties of wheat and maize were later found to be caused by mutations within the DELLA domain of Rht and D8, respectively (92). The *D8* alleles have either short internal in-frame deletions, like *gai-1*, or truncations of N-terminal coding sequences. However, the *Rht-B1b* and *Rht-D1b* alleles harbor nucleotide substitutions that create an early stop codon within the DELLA domain. This leads to the production of an N-terminal truncated protein through translational reinitiation at a downstream AUG site. Interestingly, this reinitiation process is tissue-specific, resulting in a dwarf phenotype without affecting seed dormancy (129).

Mutant analysis and molecular cloning of GID1 and SLY1/GID2 further showed that they are required for GA-induced DELLA degradation. GID1 is a nucleocytoplasmic protein belonging to the hormone-sensitive lipase family, although it lacks lipase activity, likely due to the absence of a conserved catalytic residue (126). SLY1 and GID2 are nuclear-localized F-box proteins in *Arabidopsis* and rice, respectively (81, 107). The molecular mechanism of GA-GID1-dependent DELLA degradation was elucidated by biochemical and structural studies. Yeast two-hybrid (Y2H) and in vitro pull-down assays indicated that GA promotes binding of GID1 to DELLAs (126) and that the DELLA domain is essential for the interaction (38, 127, 133). X-ray crystallography further unveiled the intricate molecular details of the three-dimensional (3D) structures of

the GA-AtGID1A-DELLA domain (GAI) and the GA-OsGID1 complexes (84, 110). The amino-terminal extension (N-Ex) domain of GID1 acts as a lid, while its carboxy-terminal domain forms a GA-binding pocket. Bioactive GA acts as an allosteric inducer of GID1. Upon GA binding, a conformational switch of its N-Ex domain occurs, covering the GA-bound pocket and creating hydrophobic surfaces for DELLA domain binding (**Figure 1*c***). The DELLA domain is intrinsically disordered in the absence of its binding partner, and binding of GID1 induces a conformational change in DELLA to achieve a stable conformation. Within the DELLA domain, three conserved motifs—DELLA, LExLE, and VHYNP—directly contact with GID1, further stabilizing the GA-GID1-DELLA complex. GA-activated GID1 also serves as a ubiquitylation chaperon, promoting the recruitment of $SCF^{SLY1/GID2}$ for DELLA ubiquitylation. In vitro pull-down, Y2H, and Y3H experiments showed that DELLAs bind to SLY1/GID2 via their GRAS domain (27, 45), with GA-bound GID1 enhancing DELLA-SLY1/GID2 interaction (38, 45). These findings suggest that the interaction of the GA-GID1-DELLA domain induces conformational changes in the GRAS domain for F-box protein recognition (38). Additional mutant and biochemical analyses showed that the GRAS domain also interacts with GID1 to further stabilize the GA-GID1-DELLA complex, thereby enhancing F-box protein binding (45) (**Figure 1*c***).

Inducible promoter: a synthetic DNA fragment that enables the expression of the downstream gene in response to an exogenous stimulus (environmental or chemical)

Chromatin immunoprecipitation–sequencing/quantitative polymerase chain reaction (ChIP-seq/qPCR): a technique for identifying where in the genome a protein binds (ChIP-seq) or whether it binds to a specific site (ChIP-qPCR)

2.2.2. Crosstalk between the GA-GID1-DELLA module and other pathways. The GA-GID1-DELLA regulatory module plays a vital role in governing GA responses in vascular plants. Extensive evidence indicates that this signaling module interacts with numerous pathways in response to internal developmental programs (e.g., other phytohormones) and environmental stimuli (e.g., light, nutrient availability, and biotic and abiotic stresses). One mechanism of interaction involves the modulation of GA metabolic gene expression, thereby influencing levels of bioactive GAs and DELLAs (reviewed in 18, 108, 121). Another mechanism of cross-regulation occurs through direct protein interactions of DELLAs with a wide array of transcription factors (TFs) and regulators involved in diverse cellular processes (108, 124, 128). These interactions often lead to alterations in the activities of DELLAs and/or their interacting partners, consequently influencing the transcription of target genes, as discussed in detail in Sections 2.3 and 3. Notably, the blue light receptor CRYPTOCHROME 1 interacts with both DELLA and GID1, inhibiting the binding of GID1 and SLY1 to DELLA and thereby stabilizing DELLA proteins (137, 150). Consequently, blue light–mediated photomorphogenesis, such as reduced hypocotyl growth, is achieved, at least in part, by impeding GA-induced DELLA degradation.

2.3. Early Evidence of DELLA Proteins as Transcription Regulators

The presence of protein motifs typical of transcription regulators in GAI and RGA, as well as in other members of the GRAS family, suggests that they may act in transcription regulation (97, 101). Indeed, both DELLA proteins can rapidly modulate gene transcription, as shown with transgenic lines expressing them under inducible promoters (35, 145). This ability of DELLA proteins is consistent with the rapid changes in gene expression elicited by GAs that can be observed as quickly as 15 min after treatment (145). In addition, SLR1 behaves as a transcription activator in both yeast and rice (46). Importantly, chromatin immunoprecipitation–sequencing/quantitative polymerase chain reaction (ChIP-seq/qPCR) performed in *Arabidopsis* showed that RGA can exert this role by associating with the promoter region of target genes (145). This view has been supported by ChIP-seq in *Arabidopsis* showing that DELLAs are indeed bound to hundreds of loci (108), suggesting that they have an extensive role as transcription regulators.

Inspection of the protein sequence of DELLAs and other members of the GRAS family reveals that these proteins have no known DNA-binding domain, and it was suggested that the LPVVV/II and VHIID motifs may function as such (97, 101). Although the GRAS proteins of *Medicago*

truncatula NODULATION SIGNALING PATHWAY1 and rice SCARECROW-LIKE7 (SCL7) are able to bind DNA in vitro (48, 73), DELLAs and other GRAS proteins are not thought to bind DNA directly in vivo. Mechanistic insights into how DELLAs regulate transcription emerged from two pioneering studies in *Arabidopsis*, which showed that they interact with TFs to orchestrate gene expression (24, 31). DELLAs prevent the binding of the light-regulated basic helix-loop-helix (bHLH) TFs PHYTOCHROME-INTERACTING FACTOR3 (PIF3) and PIF4 to DNA by interacting with their DNA-binding domains. It was therefore assumed that the TFs are kept away from the target chromatin by DELLAs. Thus, in addition to describing the first mechanism of action of DELLA proteins, a complete set of components linking the hormone GA to target genes and a molecular mechanism for the cross-regulation between GAs and light signaling pathways were identified for the first time. These two articles represent only the tip of the iceberg, as subsequent studies showed that DELLAs are promiscuous proteins capable of interacting with hundreds of TFs and transcription regulators (66, 80), as well as with chromatin remodelers and modifiers (68, 105, 146, 147) and histones (52). The identification of DELLA interacting partners was critical not only to determine how DELLA proteins regulate transcription but also to understand, from a mechanistic perspective, how these proteins regulate a variety of physiological processes and, in vascular plants, serve to integrate GAs and other signaling pathways. Currently, DELLAs are thought to act as transcriptional hubs that connect environmental and developmental cues to transcriptional networks (9, 17). This is due to the sensitivity of DELLAs to these cues, which is mediated by GA/GID1 or other signaling pathways (see Section 4), as well as their ability to interact with many TFs and transcription regulators. Notably, DELLAs can activate or repress target gene expression, depending on their interacting partners, as we discuss in Section 3.

Chromatin: form in which DNA is organized in the cell nucleus; contains proteins such as histones

Chromatin remodeler: a molecular machine that changes the conformation or composition of nucleosomes or moves them along the DNA

Transcriptional hub: protein that interacts with many transcription factors and transcription regulators and regulates their activity in response to a specific signal

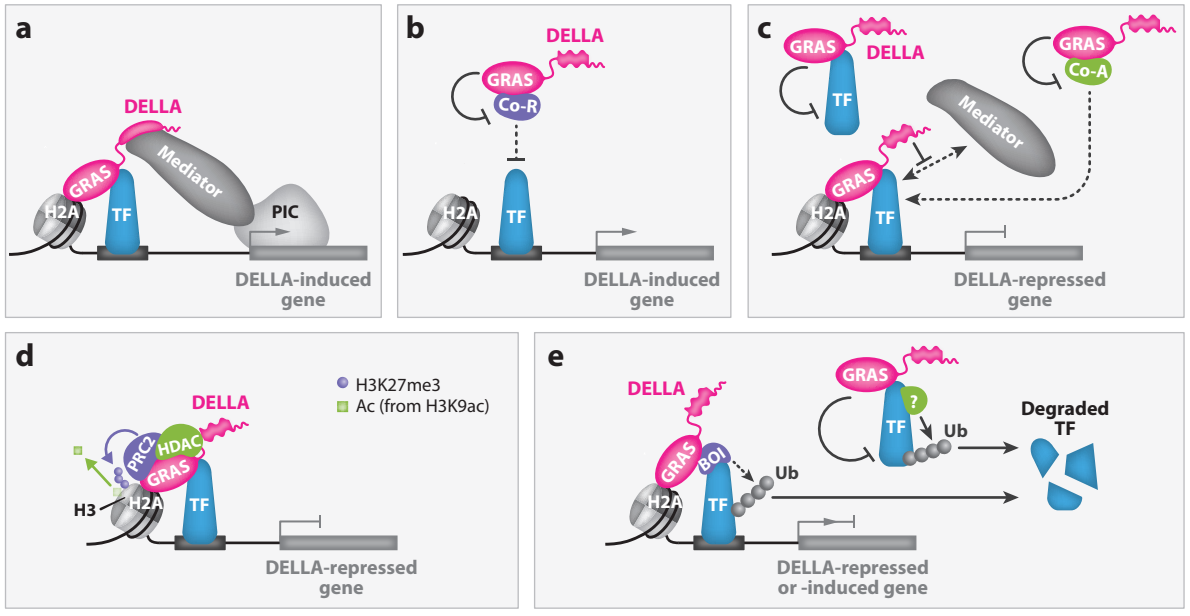
Nonvascular plants: lineage of land plants without vascular tissue, consisting of mosses, liverworts, and hornworts

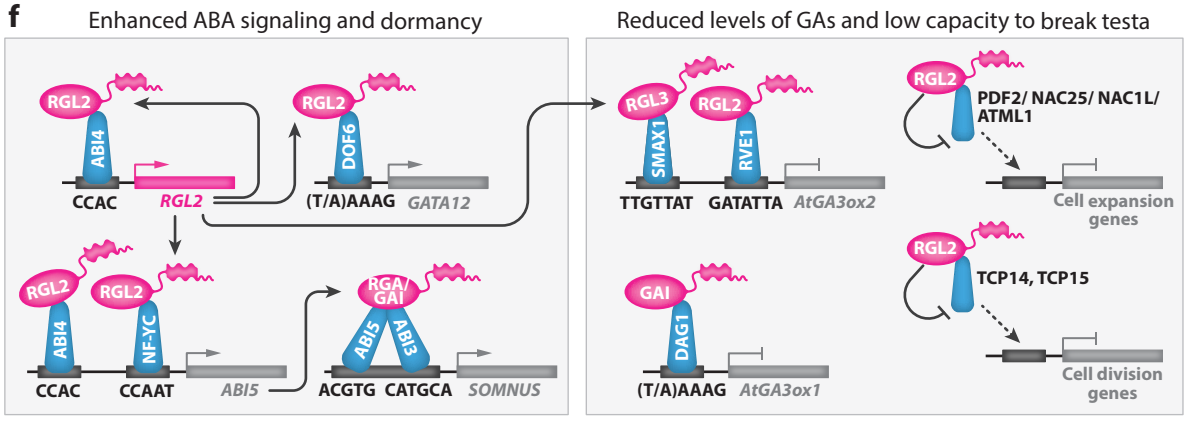
2.4. Identification of DELLAs' Functional Motifs Through Genetic, Biochemical, and Structure Analyses

Extensive studies have helped to dissect the roles of various functional domains and motifs in DELLAs as summarized below.

2.4.1. DELLA domain: GID1 binding and transcription activation. As described above, the DELLA domain serves as a regulatory domain, which is essential for GID1 binding and GA-GID1-dependent degradation. Intriguingly, this domain also exhibits transactivation activity, which enhances the growth suppression activity in planta (46). Initial transcriptomics and ChIP-qPCR analyses identified early RGA-induced direct target genes, encoding either positive components for GA biosynthesis and GA responses [e.g., *GA20ox2*, *GID1*s, and *SCARECROW-LIKE3* (*SCL3*)] or downstream repressors [e.g., *BOTRYTIS SUSCEPTIBLE1 INTERACTORs* (*BOIs*)] (87, 145, 149). These results reveal that DELLAs not only function as GA signaling repressors but also modulate GA homeostasis. Transient expression assays in *Nicotiana benthamiana* also demonstrated RGA-induced transcription of the *SCL3* promoter (149). Y1H assays further showed that the DELLA domain, including both DELLA and VHYNP motifs, is required for the transactivation activity of SLR1 (46). The important role of this activity in growth suppression in planta was elegantly illustrated by expressing wild-type *SLR1* and DELLA/VHYNP-deleted *slr1* transgenes separately in the *gid1 slr1* mutant background (46). The slr1 mutant protein lacking the DELLA domain displayed reduced growth suppression activity compared to SLR1.

An analysis of the DELLA protein sequence from an evolutionary perspective provided clues to the molecular mechanism by which DELLAs activate transcription (42) (**Figure 2*a***). Hernández-García et al. (42) found that the N-terminal DELLA domain for GID1 binding is conserved in nonvascular plants, even though they lack *GID1* genes (**Figure 1*e***). Notably, they found that the DELLA domain of nonvascular plants can also activate transcription. Additionally, the αD helix,





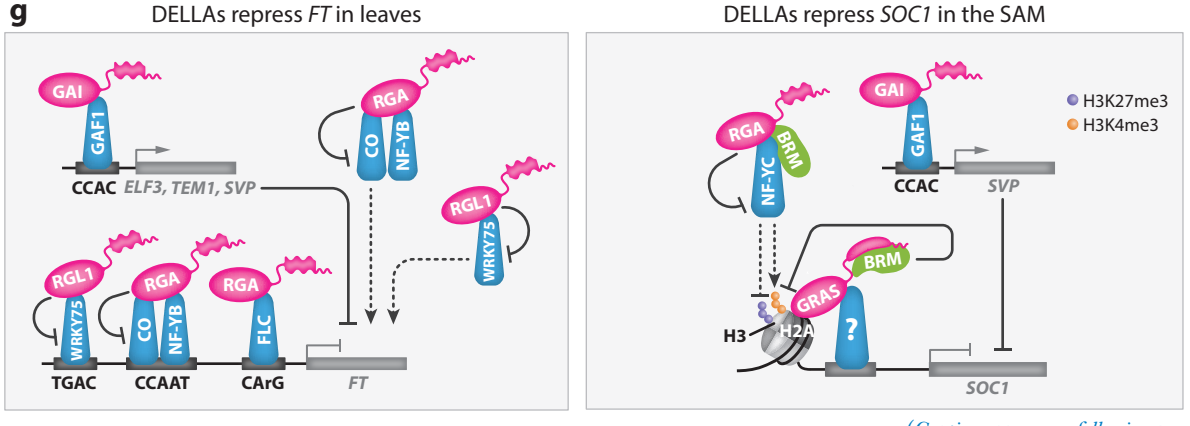


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Figure 2 (*Figure appears on preceding page*)

Mechanisms of DELLA-mediated transcription regulation. (*a*) DELLAs bind to the promoter of DELLA-activated genes via interaction with a TF and then recruit Mediator. The Mediator complex recruits the PIC, which contains general transcription factors and RNA polymerase II, to initiate transcription. The DELLA protein stabilizes the DELLA-TF complex at chromatin via interaction with H2A in a nearby nucleosome. The smooth shape of the DELLA domain indicates that it is likely to form a stable conformation upon binding to Mediator or another partner, while the wavy DELLA domain in panels *b*–*g* indicates that it is disordered when not interacting with a partner. (*b*) DELLAs can activate transcription indirectly by antagonizing the function of a transcription Co-R. (*c*) DELLAs can act as Co-Rs by antagonizing the function of transcription activators through sequestration and at chromatin. In the latter case, DELLAs can prevent the recruitment of Mediator to DELLA-repressed genes by the transcription activator. In addition, DELLAs can antagonize the function of transcription Co-As. (*d*) DELLAs recruit the PRC2 and HDAC complexes to genes repressed by DELLA. PRC2 promotes trimethylation of H3K27, a repressive mark, and HDAC removes H3K9ac, an active histone mark. (*e*) DELLAs can destabilize certain TFs. The question mark indicates that the ubiquitin ligases that mediate TF ubiquitylation are unknown. We propose that DELLAs may also promote the destabilization of TFs on the chromatin through their interaction with BOIs, which are ubiquitin E3 ligases. However, whether BOIs mediate the ubiquitylation of TFs on chromatin requires further investigation, indicated by the dashed arrow. The effect of TF destabilization on the expression of the target gene depends on whether the TF is an activator or a repressor. (*f*) Illustration of the different modes of action with which DELLAs regulate seed germination. The left panel shows how DELLAs, mainly RGL2, promote seed dormancy. The right panel shows how DELLAs repress the expression of genes necessary for seed germination, such as genes encoding GA biosynthetic enzymes and proteins involved in cell elongation and cell division. The mechanisms for the antagonistic effect of RGL2 on these TFs have not been determined. The *cis*-element sequence below the TF indicates its binding site. (*g*) Illustration depicting the different modes of action with which DELLAs regulate flowering. The left panel shows the direct and indirect repression of the flowering integrator gene *FT* by DELLAs, which takes place in the leaves. The right panel shows the direct and indirect repression of the flowering integrator gene *SOC1* by DELLAs, which takes place in the SAM. The *cis*-element sequence below the TF indicates its binding site. The question mark indicates that the TF that mediates the binding of DELLA and BRM to the *SOC1* promoter is unknown. In all panels, the solid arrows indicate positive effects, and solid T-bars indicate inhibitory effects. Arrows at the 5′ end of genes indicate active transcription, and T-bars indicate reduced or no transcription. In panels *b*, *c*, *f*, and *g*, the dashed arrows/T-bars indicate the action the TFs would take if they were not antagonized by DELLAs. Abbreviations: ABA, abscisic acid; ABI, ABA-INSENSITIVE; ATML1, *Arabidopsis thaliana* MERISTEM LAYER1; BOI, BOTRYTIS-SUSCEPTIBLE1 INTERACTOR; BRM, BRAHMA; Co-A, coactivator; Co-R, corepressor; DAG1, DOF AFFECTING GERMINATION1; DOF6, DNA BINDING WITH ONE FINGER6; *ELF3*, *EARLY FLOWERING3;* FLC, FLOWERING LOCUS C; *FT*, *FLOWERING LOCUS T*; GAF1, GAI-ASSOCIATED FACTOR1; GAI, GIBBERELLIN-INSENSITIVE; HDAC, HISTONE DEACETYLASE; NF-Y, NUCLEAR FACTOR Y; PDF2, PROTODERMAL FACTOR2; PIC, preinitiation complex; PRC2, POLYCOMB-REPRESSIVE COMPLEX2; RGL2, RGA-LIKE2; RVE1, REVEILLE1; SAM, shoot apical meristem; SMAX1, SUPPRESSOR OF MAX2 1; *SOC1*, *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1*; *SVP*, *SHORT VEGETATIVE PHASE*; *TEM1*, *TEMPRANILLO1*; TF, transcription factor; Ub, ubiquitin.

the most conserved region within the DELLA domain, is required for this activity. Interestingly, inspection of the αD sequence revealed striking similarity to the 9-amino-acid transactivation domain found in transcription activators responsible for recruiting the coactivator MEDIATOR (MED) complex (42). This notion was recently substantiated by findings that the DELLA domain recruits MED15 to DELLA-activated promoters in *Arabidopsis* (43).

2.4.2. GRAS domain: a hub for protein–protein interactions. The GRAS domain comprises five conserved subdomains: Leu Heptad Repeat 1 (LHR1), VHIID, LHR2, PFYRE, and SAW (**Figure 1*d***). Expression of the GRAS domain of DELLA proteins in both rice and *Arabidopsis* confers a GA-unresponsive semidwarf phenotype (46, 52). Mutant studies further confirmed the essential role of the GRAS domain in the growth suppression activity of DELLA proteins. Missense mutations that reduce the activity of DELLAs across various plant species are all located within the GRAS domain, leading to slender phenotypes (14, 45, 52). Moreover, the GRAS domain has been shown to interact with numerous TFs and transcription regulators (124, 128), indicating its important role in transcription regulation. Serial deletion studies via Y2H assays and in vitro pull-down experiments have suggested that LHR1 is crucial for binding to DELLA-interacting TFs and transcription regulators. However, truncations that include the C terminus of the GRAS domain often disrupt these interactions as well. Nonetheless, this approach alone cannot discern whether these deletions directly impact the interaction motif or lead to general

changes in protein conformation and structural stability. Further dissection of the distinct roles of various GRAS subdomains has been facilitated by extensive collections of *della* mutants, primarily in *Arabidopsis*, rice, barley, and wheat (14, 45, 52).

Nucleosome: an octamer consisting of pairs of histones H2A, H2B, H3, and H4, surrounded by 147 DNA base pairs

The essential role of LHR1 in TF binding is accentuated by comparing the effects of missense *rga* mutations within this subdomain on TF binding, transcription regulatory activity, and growth suppression in planta. Most notably, rga^{A268V}, corresponding to the *rht-7* allele in wheat (14), with a single A268V substitution completely abolishes all these activities (52). The importance of the VHIID subdomain for F-box protein binding is emphasized by the observation that deletion of VHIID in GAI eliminates the SLY1 interaction in the Y2H assay (27). Additionally, a novel semidominant GA-unresponsive dwarf in *Brassica rapa* (*Brrga1-d*) was found to contain a Q328R substitution in the VHIID subdomain, leading to the abolition of interaction with SLY1 and thereby stabilization of the mutant rga1-d protein (83). Furthermore, alanine scanning mutagenesis followed by Y2H/Y3H and surface plasmon resonance (SPR) assays identified the residues in SLR1 involved in the interactions with GID2 (an F-box protein) and/or GID1 (45). Multiple triple-alanine substitutions in the VHIID subdomain disrupt binding of GID2, while mutations in LHR2 reduce interactions with both GID2 and GID1. These observations support the role of VHIID and LHR2 in the binding of the F-box protein and the involvement of LHR2 in GID1 interaction.

A novel role of the PFYRE subdomain in binding histone H2A was discovered recently by functional analysis of missense *rga* alleles (52). Specifically, the *rga-2* and *rga-11* alleles, harboring D478N or S474L substitutions within PFYRE, respectively, completely abolish RGA's function in transcription regulation of target genes and in growth suppression in planta. ChIP-seq analysis further revealed that these mutations led to a dramatic reduction in the association of RGA with target chromatin globally. Surprisingly, rga-2 and rga-11 mutant proteins do not disrupt the interaction with several TFs, although both LHR1 and PFYRE subdomains are crucial for target chromatin binding. Instead, the PFYRE subdomain was found to enhance histone H2A binding. Consequently, a model was proposed: DELLAs are recruited to target promoters by binding to TFs via their LHR1 subdomain. Subsequent interaction between DELLAs and the histone H2A of nearby nucleosomes through the PFYRE subdomain is necessary for stabilizing the TF-DELLA-H2A complex at the target chromatin.

Characterization of a novel semidominant dwarf allele in rice, *slr1-d4*, has unveiled the role of the SAW subdomain in GID1 binding (45). This allele carries a G576V substitution in the SAW subdomain, resulting in increased stability of the slr1-d4 protein. Notably, assays including Y2H, Y3H, and SPR demonstrate that slr1-d4 not only fails to bind GID2 but also exhibits diminished interaction with GID1. Additionally, C-terminal truncations of the GRAS domain caused by nonsense mutations in RGA also produce mutant proteins that are unresponsive to GA-induced degradation (27). These findings suggest that sequential binding of the DELLA domain followed by the GRAS domain to GID1 leads to a stable DELLA protein–GID1 complex, facilitating efficient recognition by the F-box protein (45) (**Figure 1*c***).

To understand the structure–function relationship of the DELLA GRAS domain, a 3D-structure model of the RGA GRAS domain was generated based on the crystal structure of another *Arabidopsis* GRAS protein, SCARECROW (47, 52) (**Figure 1*d***). The predicted RGA GRAS domain contains an α/β core subdomain with an α-helical cap. The α-helical cap consists of five α-helixes: α1–α3 (representing LHR1) and α10 and α11 (a segment of the PFYRE subdomain). This α-helical cap is required for DELLA's function as a transcription regulator and growth repressor, although the LHR1 and PFYRE subdomains play distinct roles in binding TFs or H2A, respectively. The remaining GRAS sequence including VHIID, LHR2, a segment of PFYRE, and SAW forms the α/β core, which is involved in GID1 and F-box protein binding to stabilize the

GA–GID1–DELLA–F-box protein complex for GA-dependent degradation. Besides playing a role in regulating DELLA protein stability, the α/β core also contributes to DELLA protein activity. Most of the missense mutations within the α/β core and C-terminal truncations of SAW lead to reduced growth suppression activity in planta (14, 45, 52). Y2H assays also showed that deletions of the SAW subdomain decrease interaction with several TFs (128). These loss-of-function mutations in the C-terminal α/β core may affect TF binding directly or indirectly by destabilizing the overall protein conformation. Structural studies will help to identify key residues in the DELLA protein directly involved in binding to various interacting partners.

Eudicots: flowering plants with embryos with two embryonic leaves

Homologous genes: genes that are present in two species and originate from a common ancestor

2.5. DELLAs Are Conserved in Land Plants and Predate GAs

DELLAs and other GRAS subfamilies arose in the most recent common ancestor of land plants (42). Phylogenetic analyses have provided a high-resolution evolutionary history of DELLAs. They show that DELLAs in extant plants are grouped into three clades, with two major duplications occurring after the divergence of nonvascular and vascular plants and in the common ancestor of eudicots (42). Further losses and duplications occurred in vascular plants. The presence of more than one DELLA in many species has not led to the acquisition of new functions. On the contrary, the biochemical properties of the different DELLAs in each species appear to be conserved, with the different expression patterns of their genes providing specificity, as shown in *Arabidopsis* (37).

It is intriguing that although DELLAs are present in all land plants, nonvascular plants lack the complete set of GA signaling components that are conserved in vascular plants (41, 42). The presence of GA signaling elements other than DELLAs in the clades of nonvascular plants is quite variable (41) (**Figure 1*e***). Homologous genes of *SLY1/GID2* exist, for example, only in liverworts, where SLY1/GID2 has a broad affinity for DELLAs, which has narrowed during evolution, reaching high specificity in species where GAs regulate DELLA stability (59). The consequences of the SLY1/GID2 interaction for DELLA in liverworts are currently unknown. Genes encoding putative GID1 receptors are not present in nonvascular plants. Accordingly, the N-terminal domain of DELLAs from liverworts and mosses is highly divergent compared to those of vascular plants (42). However, the N-terminal domain of hornwort DELLAs exhibits all the motifs required for DELLA–GID1 interaction (42). Indeed, the DELLA from the hornwort *Nothoceros vicentianus* can interact with the three *Arabidopsis* GID1s in Y2H experiments in a GA-dependent manner (42). Therefore, researchers have suggested that GA signaling may have originated in the common ancestor of vascular plants (42). In accordance with this idea, phylogenetic analyses show that nonvascular plants may only be able to synthesize the GA precursors *ent*-kaurenoic acid (KA) and GA_{12} (12, 41, 60) (**Figure 1*e***). Indeed, different KA derivatives regulate very specific processes independent of DELLAs in the moss *Physcomitrium patens* and in the liverwort *Marchantia polymorpha*, consistent with the absence of *GID1* genes in these species (82, 94, 120).

3. FUNCTIONS AND MECHANISMS OF ACTION OF DELLA PROTEINS

3.1. Mechanisms of DELLA-Mediated Transcription Regulation

Recent studies have uncovered complex mechanisms by which DELLAs regulate transcription of their target genes as summarized in this section.

3.1.1. DELLAs can activate or repress transcription on chromatin. Shortly after observing that DELLA proteins interact with PIF3 and PIF4 and hinder their binding to target promoters, numerous studies—mainly in *Arabidopsis* and rice—demonstrated that this mode of antagonizing

the TF function extends to many other interacting TFs (128). However, the predominance of this mechanism has been challenged by a study showing that DELLAs act primarily at chromatin (52). The current model is that DELLAs interact with TFs on target chromatin and cooperate with or antagonize the activity of the TF to regulate gene expression. In this scenario, DELLAs can act as either a transcription coactivator or corepressor of the target gene (**Figure 2*a*,*c*–*e***). This model does not preclude the role of DELLAs in sequestering a fraction of the TFs, such as PIFs, in the nucleoplasm and preventing their binding to chromatin (**Figure 2*c***). Therefore, DELLAs can inactivate TFs on two levels: by antagonizing the activity of chromatin-bound TFs and by preventing the binding of the TF to chromatin.

Nucleoplasm: the content of the cell nucleus with the exception of the chromatin

3.1.2. Mechanisms for transcription activation by DELLAs on chromatin. One of the mechanisms by which DELLAs activate transcription is by acting as transcription coactivators. The 9-amino-acid transactivation domain in the DELLA domain interacts with MED15 to recruit the MED complex to chromatin and activate gene expression in *Arabidopsis* and *M. polymorpha*, providing a conserved molecular mechanism for DELLA-mediated transcriptional activation (43) (**Figure 2*a***). Most DELLA-induced genes in *Arabidopsis* depend on MED15, and their promoters are enriched in binding sites for TFs of families that include those previously shown to require DELLA to activate gene expression. This is the case of the INDETERMINATE DOMAIN (IDD) TFs (34, 142). GAI-ASSOCIATED FACTOR1 (GAF1)/IDD2 recruits DELLA to activate the expression of *AtGA20ox2* and *AtGA3ox1* (34), and, accordingly, upregulation of these two genes by DELLAs requires MED15 (43). Reducing DELLA levels alters the transcriptional fate of IDD target genes by allowing IDDs to recruit the transcriptional repressor TOPLESS-RELATED4 (TPR4) (34). The alternative recruitment of DELLA or TPR4 to IDD2/GAF1 bound to the *AtGA20ox2* gene contributes to control GA content (34). DELLAs are also coactivators of type-B ARABIDOPSIS RESPONSE REGULATORs (ARRs), which are TFs involved in cytokinin-induced gene expression (79). Consistent with MED activity being required for the activation of DELLA-ARR targets, *med15*-defective plants are impaired in DELLA-induced cotyledon opening and reduction in root apical meristem size (43), and both processes are dependent on DELLA and ARR (79).

The spectrum of DELLAs' action in activating transcription includes the sequestration of transcriptional corepressors. DELLAs compete with the regulated TF for binding to the corepressor, allowing the TF to activate transcription (**Figure 2*b***). This is exemplified by the JA ZIM-DOMAIN (JAZ) proteins, which are negative regulators in jasmonate (JA) signaling that undergo degradation in response to the hormone (16). JAZ proteins recruit the transcription repressors TOPLESS and TPR to MYC2-regulated genes (89). The interaction between RGA and JAZ1 prevents JAZ1 from interacting with MYC2 and also with the MYB-bHLH-WD40 complex, thereby allowing the transcription of JA-induced genes (49) and of anthocyanin biosynthesis (135), respectively.

3.1.3. Mechanisms for transcription repression by DELLAs on chromatin. DELLAs also function in transcription repression by antagonizing transcription activators (**Figure 2*c***). One of the mechanisms involves the MED complex, although this may seem paradoxical. RGA and GAI repress the expression of target genes of the flowering time regulator SQUAMOSA PROMOTER BINDING PROTEIN-LIKE15 (SPL15) through direct interaction with this TF at the chromatin, where DELLAs prevent the recruitment of MED18 and the subsequent transcription activation (54) (**Figure 2*c***). The hypothesis is that the interaction between DELLA and SPL15 might cause structural changes in SPL15, preventing its interaction with MED18. DELLA-SPL15 can also cause structural changes in DELLA and hinder the recruitment of MED15.

A second mechanism has been described in which SLR1 antagonizes the transcription activator by recruiting POLYCOMB-REPRESSIVE COMPLEX2 (PRC2) and HISTONE DEACETYLASE702 (HDA702) to GA-induced loci in rice (68) (**Figure 2*d***). PRC2 deposits the repressive mark trimethylation of lysine 27 of histone H3 (H3K27me3) (95), while histone deacetylases (HDACs) remove histone acetylation, a hallmark of active genes (21). ChIP-seq analysis showed that H3K27me3 levels were reduced in a large number of gene bodies in *slr1* compared to those in wild type. SLR1 forms a tripartite complex with PRC2 and HDA702, and their recruitment to GA-induced genes is SLR1-dependent, via an interaction with at least the zinc-finger TF YABBY4 (68). Consistent with this, GA-induced gene expression is associated with a decrease in H3K27me3 and an increase in the acetylation of histone H3K9 (H3K9ac). It is likely that a similar mechanism is at work in other plants, given the conservation of PRC2 and HDAC complexes.

DELLAs can also prevent the activation of target genes by sequestering transcription coactivators (**Figure 2*c***). In the context of growth–defense trade-off, salicylic acid (SA) production in response to *Pseudomonas* attack requires the transcription coactivator ENHANCED DISEASE SUSCEPTIBILITY1 (EDS1). SA triggers the stabilization of RGA and RGL3 in an EDS1-dependent manner to restrain growth upon pathogen attack (74). DELLAs, in turn, sequester EDS1 and prevent its recruitment to chromatin via an unknown TF, thereby preventing the overactivation of SA biosynthesis genes (74).

3.1.4. DELLAs can destabilize transcription factors. DELLAs not only regulate the activity of TFs but can also destabilize them via the 26S proteasome (**Figure 2*e***). Li et al. (69) showed that PIF3 protein levels were reduced in lines that overaccumulate DELLAs, while they increased in response to treatments with GAs or in the *dellaP* mutant. The reduction in PIF3 levels was posttranscriptional and counteracted by the proteasome inhibitor MG132. Simultaneous treatment with a GA biosynthesis inhibitor (paclobutrazol) and MG132, which uncouples PIF3 degradation from PIF3 sequestration, demonstrated that both mechanisms contribute to inhibit this TF (69). This effect extends to other PIFs and to ETHYLENE-INSENSITIVE3 (69). Similarly, the protein content of the DELLA interactor HAT1 (for *Arabidopsis thaliana* HOMEODOMAIN-LEUCINE ZIPPER PROTEIN1) was reduced by DELLAs posttranscriptionally, an effect abolished by MG132 (123). This suggests the possibility that DELLAs antagonize HAT1 and promote its degradation to control its activity. By contrast, protein levels of other TFs antagonized by DELLAs, such as BRASSINAZOLE-RESISTANT1 (BZR1) and LEAFY COTYLEDON1, are not affected by the interaction (4, 36, 51, 71). Overall, these results suggest that DELLAs may exert dual control over certain TFs, that is, antagonizing their activity by both interaction and degradation, while in other cases only antagonizing their activity without affecting their stability.

The view that DELLAs potentially act on all TFs on chromatin (52) suggests that DELLA-induced degradation can occur while the TF is bound to chromatin. In this context, it should be noted that DELLAs interact with BOIs, which are RING-type E3 ubiquitin ligases, and both are targeted to the promoters of a subset of GA-induced genes, which are subsequently repressed (87). Because the RING domain is required for the role of BOIs in gene repression, it is possible that DELLAs recruit BOIs to target promoters, where they ubiquitylate and destabilize the TF that brings them to chromatin and that would otherwise activate transcription (**Figure 2*e***). Therefore, DELLAs may recruit E3 ubiquitin ligases to trigger the degradation of specific TFs on chromatin, serving as a mechanism to antagonize their function.

3.2. Nongenomic Roles of DELLAs

The ability of DELLAs to regulate certain physiological processes without requiring a change in gene expression demonstrates their functional versatility. The first example of this type was the

discovery that DELLAs can inhibit cell elongation by interfering with the assembly of cortical microtubules (77). The prefoldin complex is a cochaperone assisting in the folding of tubulins in the cytoplasm, and *Arabidopsis* mutants defective in its activity show disorganized microtubules and reduced growth (5). Interestingly, DELLAs interact with the prefoldin complex and sequester it in the nucleus, preventing its role as a cochaperone in the cytoplasm, resulting in reduced α-/β-tubulin heterodimers and shorter and disorganized microtubules. The negative nongenomic effect of DELLAs on the arrangement of cortical microtubules also reduces protein trafficking to the plasma membrane, in particular, the auxin transporter PIN-FORMED2 (103).

DELLAs also prevent chloroplast development before germination by sequestering the chloroplast import receptor TRANSLOCON AT THE OUTER ENVELOPE MEMBRANE OF CHLOROPLAST159 (TOC159) in the nucleus (109). RGL2 interacts with TOC159 in the nucleus and promotes its degradation by proteasome 26S, thereby preventing chloroplast development until conditions are favorable for germination and GA levels increase.

3.3. DELLAs at Work in Biological Contexts: Regulation of Seed Germination and Flowering Time

The promiscuity of DELLAs for protein–protein interactions is the molecular basis for their ability to act as transcriptional hubs, particularly in response to changes in the environment (9, 17). In this section, we illustrate how DELLAs regulate germination and flowering, two developmental transitions that are strongly influenced by environmental variables, by deploying their distinct modes of action.

3.3.1. DELLAs' block of germination involves various mechanisms. Seed germination is triggered when environmental conditions become favorable, for example, increased soil moisture. The decision to germinate is controlled by two antagonistic hormones, GAs and abscisic acid (ABA) (111). ABA accumulates during seed development and promotes primary dormancy, preventing premature germination. Upon seed imbibition, the GA levels increase, opposing the effects of ABA to induce germination. In *Arabidopsis*, the major DELLA protein that blocks seed germination is RGL2 (67), although the other DELLAs also regulate this process. RGL2 controls two aspects of germination via different mechanisms: It enhances primary seed dormancy by promoting ABA action, and it suppresses germination by downregulating the expression of GA biosynthetic genes and genes involved in endosperm and embryo cell expansion and division (**Figure 2*f***).

RGL2 enhances the effect of ABA by acting as a transcription coactivator to promote the expression of an ABA signaling activator, *ABA-INSENSITIVE5* (*ABI5*), via interaction with the bZIP TF ABI4 (134) and subunits of the NUCLEAR FACTOR Y (NF-Y) TF complex NF-YC3, NF-YC4, and NF-YC9 (76). *RGL2* is induced by RGL2-ABI4 and thus acts in a feedforward mechanism to enhance the response (134). RGL2 and the NF-YCs bind to the *ABI5* promoter synergistically. RGL2 also interacts with DNA BINDING WITH ONE FINGER6 (DOF6) to promote dormancy by activating *GATA12* expression (99). High temperatures suppress *Arabidopsis* seed germination, at least in part by the action of RGA and GAI. These proteins form a complex with ABI3 and ABI5, which are TFs of the B3 and bZIP families, respectively, to activate the transcription of *SOMNUS* (75), a CCCH-type zinc finger that negatively regulates germination by both promoting ABA biosynthesis and repressing GA biosynthesis (30). It is tempting to speculate that RGL2 activates transcription via recruitment of the MED complex.

Additionally, RGL2 inhibits processes that would otherwise trigger germination. RGL2, together with its interacting Myb-like TF, REVEILLE1 (RVE1), represses the expression of *AtGA3ox2* (141). Interestingly, the RVE1–RGL2 interaction stabilizes RGL2 by interfering with

SLY1 binding. *AtGA3ox2* is also repressed through interaction between RGL3 and the strigolactone and karrikin signaling TF SUPPRESSOR OF MAX2 1 (SMAX1), which is important for preventing germination in low light (138). The effect of DELLAs also continues after germination, when the interaction of GAI with DOF AFFECTING GERMINATION1 (DAG1) is required to repress *AtGA3ox1* expression to slow down the germination rate (7).

The negative effect of RGL2 on germination also includes the downregulation of genes required for cell elongation or cell cycle progression. In this case, RGL2 antagonizes several transcription activators (**Figure 2*f***). RGL2 inhibits NAC25 and NAC1-LIKE (NAC1L) in the endosperm (104) and the homeodomain-leucine zipper TFs *Arabidopsis thaliana* MERISTEM LAYER1 (ATML1) and PROTODERMAL FACTOR2 (PDF2) in the epidermis (102) to prevent the expression of cell expansion genes. Similarly, RGL2 prevents TEOSINTE BRANCHED1/CYCLOIDEA/PROLIFERATING CELL FACTOR14 (TCP14) and TCP15 from activating the cell cycle in the embryonic root meristem (100). Both cell expansion and cell division are necessary for the embryo to emerge out of the seed coat.

3.3.2. DELLAs deploy various mechanisms to delay flowering. The transition to flowering is an important decision in the life of a plant and is regulated by several pathways, including the GA pathway (64). In *Arabidopsis*, these pathways converge to control the expression of integrator genes such as *FLOWERING LOCUS T* (*FT*), a protein belonging to the phosphatidylethanolamine-binding protein family, in the leaves and the MADS-box TF *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*) in the shoot apex. Under inductive conditions, for example, long days in *Arabidopsis*, *FT* expression is induced in the leaves, and the FT protein migrates to the shoot apical meristem, where it induces the expression of *SOC1* and the flower identity gene *APETALA1* (*AP1*). Under noninductive conditions, for example, short days, flowering of *Arabidopsis* plants is delayed and depends on GAs (64). Under these conditions, DELLAs prevent flowering by acting on the expression of the integrator genes in different ways, both in the leaves and in the shoot apex (**Figure 2*g***).

In leaves, DELLAs antagonize various TFs that otherwise would bind to and activate *FT* (**Figure 2*g***). One target is the B-box TF CONSTANS (CO), which is one of the main transcription activators of *FT*. The DELLA–CO interaction suppresses *FT* expression, likely via sequestration of CO (130) and by preventing the interaction of CO with NF-YB2 (13, 50, 136). DELLAs also antagonize the activities of other activators of *FT*, such as WRKY75 (148). Moreover, DELLAs together with the MADS-box TF FLOWERING LOCUS C (FLC), a major repressor of flowering time, inhibit *FT* expression (70). In addition, DELLAs indirectly suppress *FT* by acting as transcription coactivators of GAF1/IDD2 to promote the expression of *EARLY FLOWERING3* (*ELF3*), *TEMPRANILLO1* (*TEM1*), and *SHORT VEGETATIVE PHASE* (*SVP*), which encode negative regulators of *FT* (33).

In the shoot apex, DELLAs prevent the activation of floral integrator genes, such as *SOC1* (**Figure 2*g***). DELLAs prevent the expression of *SOC1* by sequestering the NF-YC TFs through the formation of a protein complex with the chromatin remodeler BRAHMA (BRM) (146). This results in an increase of the repressive mark H3K27me3 over the *SOC1* locus, as NF-Ys are needed to recruit H3K27 demethylase (49, 147). In addition, DELLA and BRM are recruited to the *SOC1* promoter via interaction with an unknown TF, thereby reducing the active mark H3K4me3, contributing to inhibition of *SOC1* (146). Another DELLA-GAF1/IDD2 coactivator complex also operates at the shoot apex to indirectly repress *SOC1* expression via the activation of *SVP* (33). The activity of DELLAs as coactivators extends after the plant is committed to flowering, as they mediate the activation of the floral identity gene *AP1* via interaction with SPL9 (139).

3.4. The Evolutionary Perspective on the Action of DELLAs

Coexpression network: a network formed by genes that have similar expression patterns; the more similar, the higher the compaction of the network

Gene ontology: attributes defining the protein encoded by the gene, such as molecular function, biological process, and cellular component

The current view of DELLAs as transcriptional hubs is supported by studies conducted in angiosperms, particularly *Arabidopsis* and rice. However, the recent adoption of various nonvascular plant species as experimental models, such as the liverwort *M. polymorpha* and the moss *P. patens*, has not only enabled investigation into the role of DELLAs in these species but also facilitated inferences about their potential ancestral role.

3.4.1. The ancestral function of DELLAs as hubs in transcriptional networks. The first indication that the function of DELLAs as transcriptional hubs may be a conserved property during evolution comes from an in silico analysis of transcriptional networks (10). In this study (10), the coexpression network formed by DELLA-interacting TFs and their coexpressing genes in *Arabidopsis* was compared with the networks constructed for tomato, *P. patens*, and the green algae *Chlamydomonas reinhardtii*, which lacks DELLAs, based on orthology. The comparison showed a higher compaction and connectivity of the transcriptional network in organisms with DELLAs, especially in *Arabidopsis* and tomato, where DELLAs are regulated by GAs, suggesting that DELLAs may have evolved as transcriptional hubs (10). Interactomic analyses further indicated that DELLAs from representative species of major plant lineages, including nonvascular plants, exhibit promiscuity in interaction with TFs, which is the molecular basis for their role as transcriptional hubs. Importantly, a large portion of the transcriptome in each representative species is dependent on DELLA, supporting the idea that their function as transcriptional hubs is ancient (9).

3.4.2. The conserved role of DELLAs as coordinators of growth and defense. Studies in *Arabidopsis* have shown that DELLA proteins play an important role in coordinating growth inhibition and defense when the plant is exposed to abiotic stress (1, 2). Does this physiological coordination represent an ancestral role of DELLAs? Gene ontology analyses have shown that gene ontology terms associated with stress responses are overrepresented in the DELLA-dependent transcriptomes and transcriptional networks of representative species from diverse lineages, including at least one nonvascular plant (9, 10). The evidence provided by the transcriptomic analyses was confirmed experimentally in *M. polymorpha*. Genetic manipulation of the levels of the sole DELLA in this species showed that the effects of oxidative stress can be counteracted by overaccumulation of MpDELLA, probably due to the increased production of protective compounds, for example, flavonoids (44). Importantly, overaccumulation of MpDELLA also suppresses growth via impairment of cell proliferation, consistent with the accumulation of MpDELLA in the apical notches where cell divisions occur (44). The function of DELLAs in coordinating growth rate and defense against stress therefore most likely represents an ancestral role of these proteins. Interestingly, the negative regulation of MpPIF is conserved via interaction with MpDELLA, and MpPIF can promote growth when it is expressed in vascular plants. However, MpPIF is not involved in growth regulation in *M. polymorpha*. Rather, it promotes gemma dormancy and gametangiophore formation, which MpDELLA inhibits (44). The role of DELLAs in coordinating growth and defense against abiotic stress has been lost, however, in *P. patens* (94). Mutant plants lacking the two *PpDELLA* genes do not show growth defects or an impaired response to abiotic stress. This is probably a consequence of the growth conditions of this species (94). Instead, PpDELLAs promote spore germination and sporophyte formation (94).

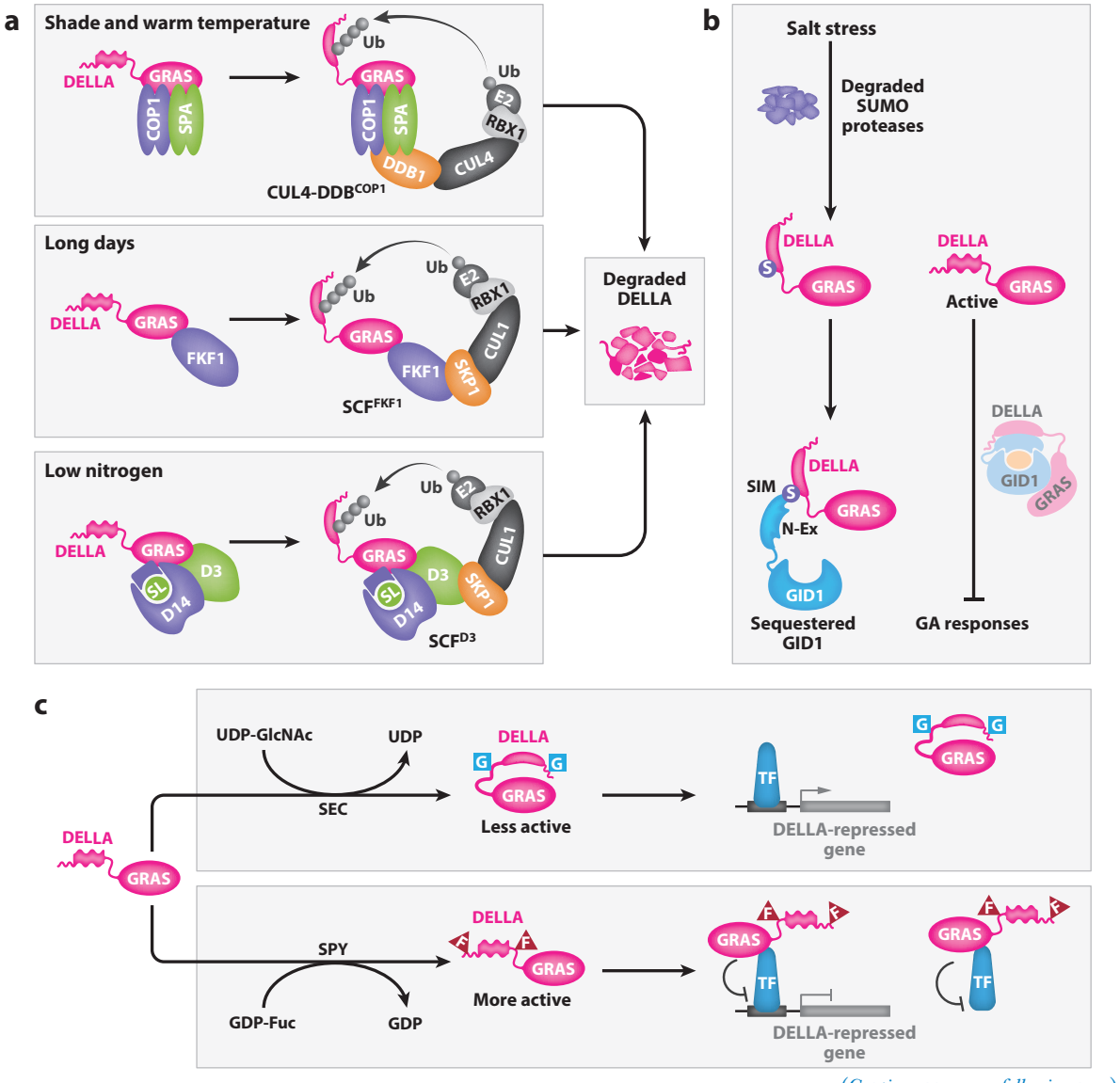
4. GA-GID1-INDEPENDENT POSTTRANSLATIONAL MECHANISMS REGULATING DELLA ACTIVITY AND LEVELS

Posttranslational destabilization of DELLAs by the GA-GID1 pathway is arguably the most important mechanism for regulating DELLA levels in vascular plants. However, recent studies have

unveiled a more intricate scenario for regulating DELLAs, wherein their stability is governed by pathways distinct from GAs, and their activity and stability are modulated by posttranslational modifications.

4.1. GA-Independent Ubiquitylation of DELLAs

The importance of tight regulation of DELLA levels is shown by the fact that plants use several other pathways besides GA-GID1 for this purpose (**Figure 3*a***). This additional control of DELLA levels works together with GA-GID1 to provide a faster response or additional temporal control under certain environmental conditions.



(Caption appears on following page)

Figure 3 (*Figure appears on preceding page*)

Posttranslational regulation of DELLAs. (*a*) Illustration of three GA-GID1-independent mechanisms to mediate ubiquitylation and degradation of DELLA via the 26S proteasome. Each mechanism functions under the environmental conditions indicated in each panel. The K residues that are polyubiquitylated in DELLAs by each pathway are not known. For simplicity, we have drawn the polyubiquitin chain bound to the DELLA domain in all cases. COP1 and SPA proteins are active as a tetramer. (*b*) Salt stress–induced stabilization of DELLAs by SUMOylation (denoted as S). After the SUMO proteases OTS1 and OTS2 are degraded under salt stress, SUMOylated DELLAs accumulate and sequester GID1 by interacting via the SIM motif in the N-Ex of the receptor. The sequestered GID1 proteins are unable to mediate DELLA degradation. The amount of active GID1 proteins is reduced (represented by the light-colored GA-GID1-DELLA complex), allowing DELLAs to suppress GA responses. (*c*) The model proposes contrasting roles for *O*-GlcNAcylation and *O*-fucosylation in regulating DELLA activity. *O*-GlcNAcylation (denoted as G) mediated by SEC may trigger the DELLA protein to adopt a closed conformation, thereby reducing its activity in binding TFs (such as BZR1 and PIFs). Conversely, *O*-fucosylation (denoted as F) catalyzed by SPY may induce the DELLA protein to assume an open conformation, enhancing its ability to bind TFs to inhibit the expression of target genes and restrict plant growth. Two DELLA-mediated inhibitory mechanisms are shown: inhibition of TF activity at the target chromatin or by sequestration of the TFs. This diagram only depicts DELLA-mediated transcription repression. In all panels, the solid arrows indicate positive effects, and solid T-bars indicate inhibitory effects. In panel *c*, an arrow at the 5′ end of a gene indicates active transcription, and a T-bar indicates reduced or no transcription. Abbreviations: COP1, CONSTITUTIVELY PHOTOMORPHOGENIC1; F, *O*-fucosylation; FKF1, FLAVIN-BINDING, KELCH REPEAT, F-BOX1; Fuc, fucose; G, *O*-GlcNAcylation; GA, gibberellin; GID1, GIBBERELLIN-INSENSITIVE DWARF1; GlcNAc, *N*-acetylglucosamine; N-Ex, amino-terminal extension; OTS, OVERLY TOLERANT TO SALT; S, SUMOylation; SCF, Skp1-Cullin-F-box protein; SEC, SECRET AGENT; SIM, SUMO-interacting motif; SL, strigolactone; SPA, SUPPRESSOR OF *phyA-105*; SPY, SPINDLY; SUMO, small ubiquitin-related modifier; S, SUMOylation; TF, transcription factor; Ub, ubiquitin.

The *Arabidopsis* DELLAs GAI and RGA are directly targeted for proteasomal degradation by the E3 ubiquitin ligase CONSTITUTIVELY PHOTOMORPHOGENIC1 (COP1) to promote hypocotyl elongation in shade and under warm temperature in a GA-independent manner (6) (**Figure 3*a***). COP1 forms a complex with SUPPRESSOR OF *phyA-105* proteins (SPA1–SPA4 in *Arabidopsis*) that, among other processes, promotes hypocotyl elongation by targeting TFs such as LONG HYPOCOTYL5 for degradation and is inactivated by light and low/moderate temperatures (96). The hypocotyl elongates when the seedling is shaded or when the ambient temperature rises moderately, and GA-induced DELLA degradation contributes in both cases (28, 118). In response to either environmental stimulus, phytochrome B is rapidly inactivated, thus lifting the inhibition it imposes on COP1 (78, 88), which in turn triggers the degradation of DELLA (6). Importantly, the action of COP1 precedes the increase in GA levels and the activation of GA signaling. Although the effect of COP1 on DELLAs is direct and does not depend on GAs, the rapid effect on growth rate is not observed when the basal concentration of DELLAs is high, suggesting that GA signaling determines the range of DELLA concentrations sensitive to COP1 (6). The concerted action of both pathways thus provides the plant with a mechanism to adjust DELLA levels in response to fluctuating environmental conditions on an organismal scale via GAs and to respond rapidly to these conditions via COP1.

Phytochrome B: red and far-red photoreceptor and thermoreceptor, which is inactivated by far-red light and warm temperature

The F-box protein FLAVIN-BINDING, KELCH REPEAT, F-BOX1 (FKF1) promotes the degradation of GAI and RGA by the 26S proteasome to modify their diurnal levels under long days and trigger the transition to flowering in *Arabidopsis* (140) (**Figure 3*a***). FKF1 interacts with DELLAs to promote their ubiquitylation and subsequent degradation. FKF1 is a flowering time regulator that promotes flowering under inductive photoperiods by stabilizing CO and triggering the degradation of CYCLING DOF FACTOR1, a negative regulator of *CO* expression (116). DELLAs function as important negative regulators of flowering in *Arabidopsis*, and part of their action is exerted via inactivation of CO (**Figure 2*g***). Thus, the action of FKF1 on DELLAs adds an additional layer of regulation to this developmental transition by temporally restricting the action of DELLAs, allowing CO and other flowering time regulators to act at the right time of the day. The effect of FKF1 on DELLAs is not limited to the regulation of flowering time, as the expression of DELLA-repressed genes involved in cell elongation, *EXPANSIN A8* and *PACLOBUTRAZOL*

RESISTANCE1, is reduced in *fkf1* mutants, indicating that FKF1 also contributes to the regulation of this process by DELLAs (140).

A recent study in rice shows that low nitrogen in soil triggers strigolactone-dependent degradation of SLR1 to increase nitrogen use efficiency (NUE) (119) (**Figure 3*a***). SLR1 inactivates GROWTH-REGULATING FACTOR4, a TF that promotes nitrogen uptake, assimilation, and translocation. This is the cause of the low NUE of Green Revolution rice varieties, which contain elevated SLR1 due to reduced GA levels (72). Similar to GA, strigolactone enhances NUE, partly through strigolactone-induced SLR1 degradation (119). The mechanism by which strigolactone promotes SLR1 degradation mirrors the mechanism that triggers degradation of the negative regulator of strigolactone responses, D53. Specifically, the strigolactone-loaded and catalytically active D14 receptor interacts with SLR1 (85), leading to ubiquitylation of SLR1 and subsequent degradation by the 26S proteasome via the F-box protein D3 (119).

4.2. DELLAs Are Stabilized by SUMOylation

Small Ubiquitin-Related Modifier (SUMO) is a protein conserved in eukaryotes that can be attached to certain K residues of target proteins via a mechanism similar to ubiquitylation. SUMOylation can reversibly alter target protein activity, localization, or stability thanks to the action of SUMO proteases (3). DELLAs can be stabilized by SUMOylation, a process reverted by the SUMO proteases OVERLY TOLERANT TO SALT1 (OTS1) and OTS2 (19) (**Figure 3*b***). In *Arabidopsis*, GAI is SUMOylated at the conserved K49, and the mutation K49R, which can no longer be modified, results in a DELLA protein with reduced stability. The stabilization of DELLA involves the recognition of SUMOylated DELLA by the SUMO-interacting motif (SIM) present in the N-terminal domain of GID1 (19). Interestingly, SUMOylated DELLAs compete with non-SUMOylated ones for binding to GID1. Thus, the more SUMOylated DELLAs are present, the less GID1 protein is available to trigger GA-dependent DELLA degradation. This mechanism is important to modulate DELLA levels quickly in response to salt stress in a GA-independent manner. Under salt stress, OTS1 and OTS2 are rapidly degraded (20), leading to an increase in SUMOylated DELLAs, which in turn interact with and sequester GID1 receptors. This allows the accumulation of DELLAs that limit growth and prepare the plant to withstand stress, while the subsequent reduction in GAs consolidates the response for the long term (19). Expression of OTSs, on the other hand, increases during stamen development to promote filament elongation by deSUMOylation of DELLA in *Arabidopsis* (11). The regulation of DELLA levels by SUMOylation is more intricate than expected, as SLY1 is also stabilized by this posttranslational modification, and SUMOylation thus indirectly contributes to the degradation of DELLA (61, 62). SLY1 is SUMOylated by the E3 ligase AtSIZ1, and as with DELLAs, SLY1 levels appear to be determined by the activity of the SUMO protease EARLY IN SHORT DAYS4 (62).

4.3. *O*-GlcNAcylation and *O*-Fucosylation via SEC and SPY

The discovery of *O*-glycosylation's impact on DELLA activity originated from the characterization of hypomorphic *Arabidopsis spindly* (*spy*) mutants, which partially reversed the dwarf phenotype caused by GA deficiency (57, 58, 114, 115). These genetic studies indicate that SPY functions as a GA signaling repressor. The molecular cloning of *SPY* and its paralog *SECRET AGENT* (*SEC*) unveiled that their protein products contain a tetratricopeptide repeat (TPR) domain and a putative *O*-linked N-acetylglucosamine (*O*-GlcNAc) transferase (OGT) catalytic domain (86). Indeed, SEC was confirmed as an OGT (39), and it *O*-GlcNAcylates DELLAs by transferring GlcNAc from UDP-GlcNAc to Ser and Thr residues within the polyS and polyS/T regions flanking the

DELLA domain (143) (**Figures 1*d*** and **3*c***). However, subsequent investigations by mass spectrometry (MS) analysis and in vitro enzymatic assays revealed surprising results. Unlike SEC, SPY is a protein *O*-fucosyltransferase, which transfers mono-fucose from GDP-fucose to specific Ser/Thr residues in DELLAs (144). Genetic analysis and pull-down assays demonstrated that SPY-mediated *O*-fucosylation enhances DELLA binding to TFs such as BZR1 and PIFs involved in brassinosteroid and light responses. By contrast, SEC-catalyzed *O*-GlcNAcylation reduces DELLA activity. Consequently, these two distinct *O*-glycosyl modifications of DELLAs by SPY and SEC oppositely modulate GA, brassinosteroid, and light signaling pathways to regulate plant growth and development. The identified *O*-GlcNAcylation and *O*-fucosylation sites in DELLA partially overlap within the two disordered polyS and polyS/T regions (143, 144). A model was proposed wherein highly *O*-GlcNAcylated DELLA may assume a closed form impeding the binding of target proteins. Conversely, increasing *O*-fucosylation may change the DELLA conformation to an open form, promoting interaction with target proteins (**Figure 3*c***).

4.4. Phosphorylation and Dephosphorylation

The role of phosphorylation in regulating DELLA function is still unclear due to conflicting and fragmented findings. Initially, studies indicated that phosphorylation of SLR1 promoted its degradation by facilitating binding to GID2 (107). However, subsequent research found that SLR1 phosphorylation has no effect on binding to GID2, suggesting that SLR1 degradation might not require phosphorylation (56). Conversely, treatments with phosphatase inhibitors or silencing of *TOPP4* encoding a type-one phosphatase in *Arabidopsis* reduced GA-induced DELLA degradation, suggesting that dephosphorylation may promote its proteolysis. Additionally, phosphorylation of SLR1 by a casein kinase I, EARLIER FLOWERING1 (EL1), was suggested to stabilize SLR1 (22), although EL1-mediated SLR1 phosphorylation was only shown in vitro. Substitutions of conserved Ser and Thr residues to Ala within the GRAS domain of *Arabidopsis* RGA decreased protein stability, also suggesting a potential role of phosphorylation in enhancing DELLA stability (131). However, phosphorylation of these Ser and Thr residues in planta has not been shown, and these Ser/Thr-to-Ala substitutions may affect DELLA activity directly or impair SLY1 binding (and thereby increase stability) independent of phosphorylation. Recently, MS analysis of RGA purified from *Arabidopsis* extracts has identified several phosphorylation sites within the polyS and polyS/T regions (53). The substitution of multiple Ser and Thr residues with Ala within the polyS and polyS/T regions additively decreased RGA activity in planta but did not affect its stability. Furthermore, co-immunoprecipitation experiments revealed that phosphorylation of the polyS/T region in RGA promotes its binding to histone H2A at target chromatin in *Arabidopsis* (53). A recent study of Rht-B1b in wheat also suggested that phosphorylation of the polyS/T region enhances DELLA function. Notably, a GSK3/SHAGGY-like kinase was reported to phosphorylate DELLA (Rht-B1b) in wheat (29), although there is no evidence for phosphorylation of Rht-B1b by GSK3 in vivo.

5. CONCLUDING REMARKS

Investigations of GA signaling in vascular plants have uncovered the GA-GID1-DELLA regulatory module, which controls plant growth and development in response to internal and environmental signals. Moreover, DELLAs are now considered master growth regulators in land plants, integrating diverse signaling pathways through protein–protein interactions with hundreds of TFs and transcription regulators to exert transcription reprogramming. Over the past 15 years, studies have significantly advanced our understanding of the molecular mechanisms

underlying DELLA function. However, several important questions persist: (*a*) What factor(s) determine the specificity of DELLAs as transcription coactivators or corepressors for specific target genes? (*b*) How do DELLAs interact with so many different proteins? (*c*) What roles do DELLAs play in different tissues/cell types to coordinate plant development? Utilizing combined approaches of structural/functional analyses and single-cell omics holds promise in addressing these fundamental inquiries.

SUMMARY POINTS

1. DELLAs function as master growth regulators that are present in all land plants, where they coordinate growth and defense by integrating different signaling pathways in response to internal and external cues.
2. DELLAs are recruited to target promoters by specific transcription factors and then form stable protein complexes at the chromatin by binding to histone H2A of nearby nucleosomes.
3. DELLAs recruit the MEDIATOR (MED) complex for transcription activation and the POLYCOMB-REPRESSIVE COMPLEX2 (PRC2) and histone deacetylase complexes for transcription repression.
4. Although DELLAs are found in all land plants, the recruitment of the gibberellin (GA)-GIBBERELLIN-INSENSITIVE DWARF1 (GID1) module probably occurred in the common ancestor of vascular plants.
5. In vascular plants, DELLA protein levels are strictly regulated by GA-GID1-dependent ubiquitylation and degradation, although GA-independent ubiquitylation mechanisms can also modulate DELLA stability under particular environmental conditions.
6. DELLAs are stabilized under salt stress by SUMOylation, and DELLA activity is increased or decreased by *O*-fucosylation or *O*-GlcNAcylation, respectively.

FUTURE ISSUES

1. To understand how DELLAs function, it is important to determine the molecular basis of DELLA promiscuity in protein–protein interactions. Structural studies will shed light on how the well-defined structure of the GRAS domain interacts with diverse partners.
2. How do DELLAs activate transcription of selected target genes but repress others?
3. The advent of single-cell omics makes it possible to determine the function of proteins at unprecedented spatial resolution. It is imperative to investigate the contribution of DELLA in various processes at the single-cell level.
4. What is the role of phosphorylation in regulating DELLA stability and/or activity? Are the conflicting findings due to complex regulation by more than one protein kinase?
5. The presence of DELLAs in bryophytes, which lack *GID1* genes, suggests that DELLAs in these species are regulated by GA-GID1-independent mechanisms. Future studies are needed to determine how DELLAs are regulated in species lacking GA-GID1, providing insights into ancestral DELLA regulatory mechanisms.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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