Mechanisms of Cross Talk between Gibberellin and Other Hormones[1]

David Weiss* and Naomi Ori

Robert H. Smith Institute of Plant Sciences and Genetics in Agriculture, Faculty of Agricultural, Food and Environmental Quality Sciences, Hebrew University of Jerusalem, Rehovot 76100, Israel

It has always been clear that different plant hormones affect overlapping processes, such that the output of plant hormone action depends on specific hormone combinations rather than on the independent activities of each. In the last two decades, numerous components of the signal transduction pathways of various plant hormones have been identified, leading to the elucidation of partial or entire signaling cascades. These findings have provided the tools to begin addressing the mechanisms underlying the cross talk among different hormone signal transduction pathways. Such cross talk involves diverse mechanisms, which act at both the hormone response and biosynthesis levels, creating a delicate response network. In this review, we describe how gibberellin (GA) interacts with other plant hormones, concentrating on its interactions with abscisic acid (ABA), auxin, ethylene, and cytokinin. Although evidence for interactions of GA with brassinosteroids (Bouquin et al., 2001) and jasmonate (Traw and Bergelson, 2003) also exists, we focus on studies addressing the mechanisms governing the cross talk.

GA BIOSYNTHETIC AND RESPONSE PATHWAYS

GAs regulate various developmental processes throughout the life cycle of the plant, from seed germination through leaf expansion, stem elongation, flower induction, and development to seed development (Sun and Gubler, 2004). As the interactions between GA and other hormones involve components from the GA biosynthetic and response pathways, we first briefly introduce a few relevant players in these pathways. For a more comprehensive description of these pathways, see recent reviews (Hedden and Phillips, 2000; Sun and Gubler, 2004; Hartweck and Olszewski, 2006; Lange and Lange, 2006; Razem et al., 2006). The GA biosynthetic pathway has been elucidated by a combination of biochemical and genetic approaches. The first few steps of the pathway, from trans-geranylgeranyl diphosphate to GA12-aldehyde, are common to all species. The final steps to produce active GAs are species specific but in most cases require activity of the GA 20-oxidase (GA20ox) and GA3ox enzymes. In contrast, the enzyme GA2ox antagonizes GA activity by deactivating GAs. The level of endogenous active GA is governed by feedback regulation, where active GAs suppress the expression of the *GA20ox* and *GA3ox* genes and promote the expression of the *GA2ox* gene.

Studies of GA signal transduction, using genetic approaches, have led to the identification of positive and negative signaling components (Sun and Gubler, 2004). The most extensively characterized among these are the DELLA proteins, a class of nuclear proteins that belong to the GRAS family of transcriptional regulators and act as suppressors of GA signaling. The molecular mechanism by which DELLA proteins suppress GA responses is not yet clear. The Arabidopsis (*Arabidopsis thaliana*) genome contains five *DELLA* genes (*REPRESSOR OF ga1-3* [*RGA*], *GA INSENSITIVE* [*GAI*], *RGA LIKE1* [*RGL1*], *RGL2*, and *RGL3*), whereas in the rice (*Oryza sativa*) genome, only one family member has been identified (*SLENDER1* [*SLR1*]; Itoh et al., 2002).

A major breakthrough in our understanding of the GA-signaling cascade has been the recent discovery of the soluble GA receptor GA INSENSITIVE DWARF1 (GID1) in rice and Arabidopsis (Ueguchi-Tanaka et al., 2005; Nakajima et al., 2006; Griffiths et al., 2007). GA binding to GID1 triggers its interaction with the DELLA domain of the DELLA proteins (Griffiths et al., 2007). This interaction stimulates binding of the DELLA proteins to an SCF E3 ubiquitin ligase via specific F-box proteins (GID2/SLY), leading to polyubiquitination and degradation of the DELLA protein by the 26S proteosome (Sasaki et al., 2003; Dill et al., 2004; Griffiths et al., 2007).

While this relatively simple GA-signaling cascade involves three major players, a receptor, a DELLA protein, and an F-box protein, other studies have identified additional factors that affect GA responses (Hartweck and Olszewski, 2006). One of these is the GA response inhibitor SPINDLY (SPY; Jacobsen and

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* Corresponding author; e-mail weiss@agri.huji.ac.il; fax 972–8–9468263.

The author responsible for distribution of materials integral to the findings presented in this article in accordance with the policy described in the Instructions for Authors (www.plantphysiol.org) is: David Weiss (weiss@agri.huji.ac.il).

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Olszewski, 1993; Filardo and Swain, 2003). The SPY protein resembles mammalian enzymes that modify proteins posttranslationally by a specific type of glycosylation, termed tetratricopeptide repeat-containing Ser and Thr-*O*-linked GlcNAc transferases (OGT). OGT transfers the single sugar moiety GlcNAc from UDP-GlcNAc to specific Ser/Thr residues via an *O*-linkage. This posttranslational modification can affect protein localization, phosphorylation, interaction with other proteins, and/or stability (Wells et al., 2001). While there is no direct biochemical evidence for the interaction of SPY with the DELLA proteins, genetic evidence suggests that SPY is required for DELLA's GA response suppression activity (Hartweck and Olszewski, 2006; Silverstone et al., 2007).

INTERACTION OF GA WITH OTHER HORMONES

The mode of GA action in planta is still far from being understood, as numerous positive and negative functional interactions with other endogenous and environmental cues affect GA responses (Fig. 1). Nemhauser et al. (2006) have identified robust target genes that are affected specifically by a single hormone. However, in the case of GA-induced genes, no specific robust targets were identified. This may suggest that interactions with other hormones play major roles in GA action, which necessitates the existence of efficient and sensitive cross talk mechanisms among the corresponding signaling pathways. Recently, several studies have focused on the molecular machinery behind the interactions between GAs and other hormones, uncovering a complex network.

MECHANISM OF THE ANTAGONISTIC INTERACTION BETWEEN GA AND ABA

GA and ABA play antagonistic roles in the regulation of numerous developmental processes. Whereas GA is associated with the promotion of germination, growth, and flowering, ABA inhibits these processes. Moreover, the antagonistic relationship and the ratio between these two hormones regulate the transition from embryogenesis to seed germination (Razem et al., 2006). Several different mechanisms have been shown to underlie this antagonistic interaction in different developmental processes (Fig. 2). During cereal seed germination, the developing embryo releases GAs to the aleurone cells where they induce the transcription of several genes encoding hydrolytic enzymes, including α-amylase. These enzymes are then secreted to the endosperm and hydrolyze starch and proteins, supplying nutrients to the developing embryo. In contrast, ABA suppresses *α-amylase* expression. The GA-induced, ABA-suppressed transcription of *α-amylase* in the aleurone layer of cereal seeds was classically used as an experimental system to study the interaction between GA and ABA. The *α-amylase* promoter contains a GA response element, required for both its activation by GA and suppression by ABA (Rogers

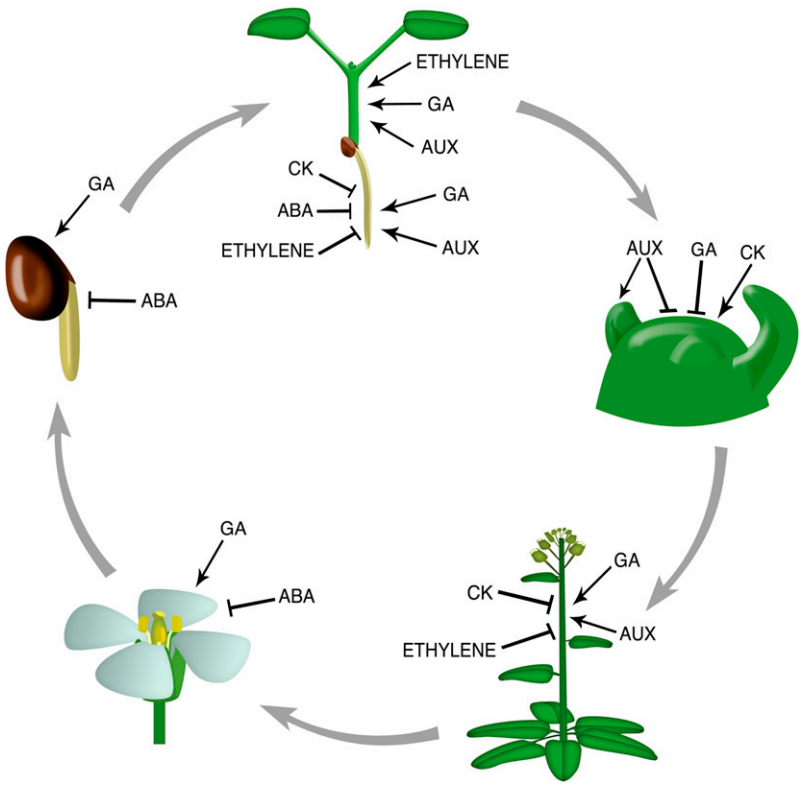

Figure 1. GA interacts positively and negatively with other plant hormones throughout the plant's life cycle. Some of the effects are shown. CK, Cytokinin.

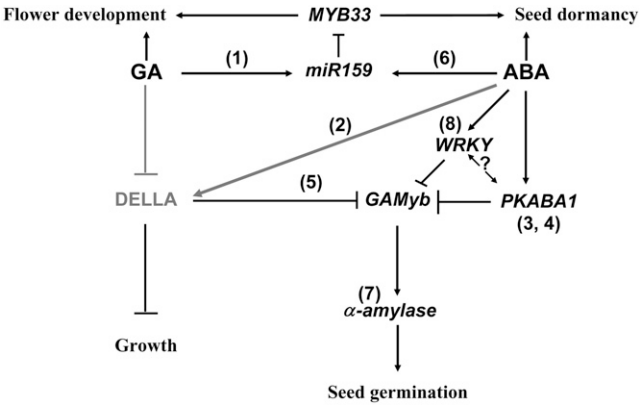

Figure 2. Network of interactions between GA and ABA. ABA suppresses GA responses through DELLA-dependent and -independent pathways. Interactions mediated by changes in protein activity or stability are in gray and those mediated by gene expression are in black. Numbers in parentheses indicate the respective reference as follows: 1, Achard et al., 2004; 2, Achard et al., 2006; 3, Gomez-Cadenas et al., 1999; 4, Gomez-Cadenas et al., 2001; 5, Gubler et al., 1995; 6, Reyes and Chua, 2007; 7, Rogers and Rogers, 1992; 8, Xie et al., 2006.

and Rogers, 1992). Gubler et al. (1995) identified a GA-induced Myb-like protein (GAMyb) that binds to the GA response element box of the *α-amylase* promoter. Induction of *GAMyb* and *α-amylase* transcription was shown to be mediated by the DELLA protein SLR1, as both are up-regulated in *slr1* mutants, even in the absence of GA. How does ABA affect this pathway? The induction of *GAMyb* and *α-amylase* by GA is suppressed by an ABA-induced Ser/Thr protein kinase, PKABA1. ABA and PKABA1 inhibited the up-regulation of *GAMyb* and *α-amylase* in *slr1* mutants as well, suggesting that the inhibition of *GAMyb* and *α-amylase* by PKABA1 occurs downstream of DELLA (Gómez-Cadenas et al., 1999, 2001). However, a more recent study has shown that when *PKABA1* is suppressed by RNAi, ABA still inhibits the GA-induced *α-amylase* expression. This finding indicates that ABA affects this process through an additional, PKABA1-independent pathway (Zentella et al., 2002). A candidate alternative ABA-signaling pathway to suppress GA responses in rice may involve two ABA-induced WRKY transcriptional regulators (Xie et al., 2006). These proteins suppress GA-induced *α-amylase* transcription, but their interaction with PKABA1 is not yet clear.

A different mechanism of interaction between GA and ABA in the regulation of root growth was described by Achard et al. (2006). In Arabidopsis, GA promotes and ABA suppresses root growth, and both effects seem to be mediated by the DELLA proteins. ABA application increased the stability of RGA and blocked its GA-induced degradation. Moreover, the quadruple-DELLA mutant (loss of *GAI*, *RGA*, *RGL1*, and *RGL2*) is relatively resistant to the growth-inhibitory effects of ABA. Therefore, while during cereal seed germination ABA seems to act downstream of DELLA, it affects Arabidopsis growth via DELLA. It is thus possible that distinct mechanisms of interaction between GA and ABA are utilized for different developmental decisions.

The complexity of the interaction between GA and ABA and its possible organ-specific mechanism were recently demonstrated in Arabidopsis. Both ABA and GA induce the accumulation of *microRNA159* (*miR159*), which targets the *MYB33* mRNA. Interestingly, MYB33 promotes ABA responses in seeds and GA responses in flowers. Thus, these two antagonistic hormones exert their action through a common mediator, MYB33, and desensitize their signaling through the same homeostatic mechanism, *miR159*, at different developmental stages (Achard et al., 2004; Reyes and Chua, 2007). Whether *miR159* and MYB33 are part of the mechanism of interaction between the two hormones or are just used by both in a development-specific manner is not yet clear.

AUXIN INTERACTS POSITIVELY WITH GA

The activities of GA and auxin overlap with respect to the regulation of cell expansion and tissue differentiation. Auxin affects GA signaling as well as GA biosynthesis (Fig. 3). In Arabidopsis, GA stimulation of root elongation has been shown to require auxin. GA-induced root elongation was inhibited by the removal of the shoot apex that is a major auxin source, and this effect was reversed by auxin application. Moreover, application of the auxin-transport inhibitor 1-*N*-naphthylphthalamic acid or mutation in the auxin-efflux regulator *AtPIN1* suppressed the effect of GA on root elongation and on RGA degradation in the

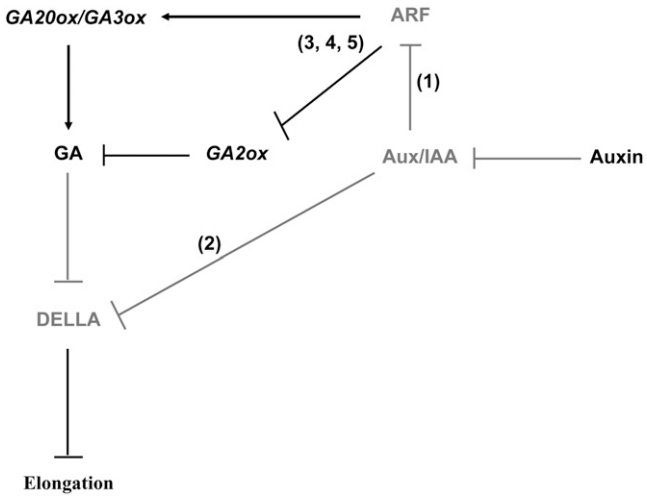

Figure 3. Network of interactions between GA and auxin. Auxin promotes GA responses by destabilizing DELLA and by promoting the expression of GA biosynthetic genes. Interactions mediated by changes in protein activity or stability are in gray and those mediated by gene expression are in black. Numbers in parentheses indicate the respective reference as follows: 1, Frigerio et al., 2006; 2, Fu and Harberd, 2003; 3, O'Neill and Ross, 2002; 4, Ross et al., 2000; 5, Wolbang and Ross, 2001.

root cells. GA-induced RGA degradation was also inhibited in the mutant *axr1* in which auxin signaling is compromised. These results suggest that auxin promotes the degradation of DELLA in root cells in response to GA, which is a prerequisite for GA-induced root elongation (Fu and Harberd, 2003). Thus, the DELLA protein RGA seems to act as an integrator of GA and auxin signals in the root.

In addition to its requirement for GA signaling in the root, auxin also affects GA production in the stem by positively regulating the expression of GA biosynthetic genes (Nemhauser et al., 2006). Decapitation of pea (*Pisum sativum*) and tobacco (*Nicotiana tabacum*) shoot apices reduced the level of active GAs in the stems, and this effect was reversed by auxin application (Ross et al., 2000; Wolbang and Ross, 2001). Auxin was shown to induce the expression of the GA biosynthetic gene *GA20ox* in tobacco and Arabidopsis, whereas in pea, the hormone induced the expression of *GA3ox* and suppressed the expression of *GA2ox*, which is involved in GA deactivation (O'Neill and Ross, 2002; Frigerio et al., 2006). Loss of the DELLA genes *GAI* and *RGA* had no effect on the induction of *GA20ox* by auxin. Thus, auxin induces GA biosynthesis through a DELLA-independent pathway or via other DELLA proteins. Addressing this problem in rice, which contains only a single DELLA, or in Arabidopsis plants completely lacking DELLA activity may help distinguish between these possibilities. The effect of auxin on GA biosynthesis was shown to transduce via the degradation of auxin signaling suppressors Aux/IAA proteins (for review, see Teale et al., 2006) and the resulting activation of the transcription factor *AUXIN RESPONSE FACTOR7* (*ARF7*). Moreover, loss of the auxin receptor TIR1, an F-box protein that mediates Aux/IAA degradation and the consequent ARF activation, suppressed auxin regulation of GA biosynthetic gene expression (Frigerio et al., 2006). Therefore, auxin positively interacts with GA either at the biosynthesis level or by promoting DELLA degradation.

DEVELOPMENTAL AND ENVIRONMENTAL CIRCUMSTANCES DETERMINE WHETHER INTERACTIONS BETWEEN GA AND ETHYLENE ARE POSITIVE OR NEGATIVE

The interaction between GA and the stress-related gaseous hormone ethylene is rather complex, as both negative and positive reciprocal effects have been demonstrated (Fig. 4). Ethylene inhibits growth in a GA-antagonistic manner. Achard et al. (2003) have shown that at least part of the inhibitory effect of ethylene on growth and its interaction with GA in this regard is mediated by the DELLA proteins. GA promotes seedling root elongation in Arabidopsis, and this effect is inhibited by ethylene. However, in *gai rga* double mutants, GA stimulated root elongation also in the presence of ethylene, suggesting that ethylene acts through these DELLA proteins in this process. In

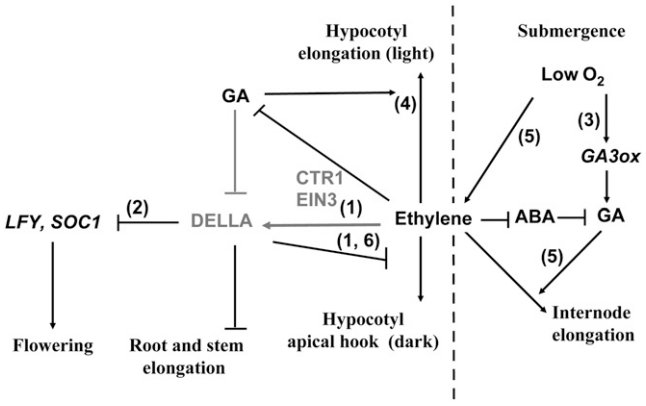

Figure 4. Network of positive and negative interactions between GA and ethylene. Ethylene represses GA biosynthesis or suppresses GA responses via DELLA stabilization. GA promotes ethylene responses in dark- and light-grown seedling (apical hook formation in the dark and hypocotyl elongation in the light). Submergence promotes ethylene and GA synthesis in deepwater rice and *R. palustris*, and GA promotes ethylene-induced internode elongation. Interactions mediated by changes in protein activity or stability are in gray and those mediated by gene expression are in black. Numbers in parentheses indicate the respective reference as follows: 1, Achard et al., 2003; 2, Achard et al., 2007; 3, Benschop et al., 2006; 4, Sabio et al., 2003; 5, Sauter et al., 1995; 6, Vriezen et al., 2004. EIN3, ETHYLENE INSENSITIVE3; *LFY*, *LEAFY*; *SOC1*, *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1*.

agreement with this, ethylene inhibited RGA degradation in root-cell nuclei in response to GA. The effect of ethylene on RGA stability was mimicked by the loss of its signaling suppressor CONSTITUTIVE TRIPLE RESPONSE1 (CTR1), suggesting that ethylene's RGA-stabilizing signal is transduced via a CTR1-dependent pathway (for review, see Guo and Ecker, 2004).

Negative interaction between ethylene and GA was also shown in mature plants. The induction of several GA-responsive genes by GA was enhanced in the Arabidopsis ethylene-resistant mutant *etr1* or when plants were pretreated with the ethylene perception inhibitor 1-methylcyclopropene. Thus, ethylene inhibits GA response in mature Arabidopsis plants (De Grauwe et al., 2007). Furthermore, ethylene delayed the transition to flowering in Arabidopsis under short days, and this effect was suppressed by GA treatment and in *gai rga* double mutant (Achard et al., 2007). While in seedlings, ethylene was shown to affect DELLA stability directly, during the transition to flowering, it affected GA biosynthesis. The resulting reduction in the level of biologically active GAs repressed the expression of two central flowering genes, *LEAFY* and *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* and the consequent transition to flowering. This effect of ethylene on the accumulation of active GA is transduced via CTR1 and the downstream transcriptional regulator ETHYLENE INSENSITIVE3. This study suggests that the antagonistic interaction between ethylene and GA mediates the timing of the decision to flower in response to changing environmental conditions.

While ethylene is classically considered to be a growth inhibitor that antagonizes GA responses, positive interactions have also been described. In etiolated seedlings, ethylene induces a triple response that includes the formation of an apical hook and the inhibition of hypocotyl and root growth. The apical hook is formed by asymmetric elongation of the inner and outer sides of the hypocotyl and helps the young seedling grow through the soil. Induction of the apical hook by ethylene requires GA activity (Achard et al., 2003; Vriezen et al., 2004). Ethylene-treated hypocotyls of the GA-deficient mutant *ga1-3* did not form an apical hook, but the response to ethylene was restored by GA treatment or when *ga1-3* was combined with the loss of both *RGA* and *GAI*. A synergistic promotive effect of GA and ethylene was also shown in light-grown Arabidopsis seedlings (Saibo et al., 2003); while GA is the major factor controlling cell elongation in light-grown hypocotyls, ethylene enhances this effect. Whether this positive interaction is mediated by the DELLA proteins, i.e. ethylene reduces DELLA stability, is not yet known. These studies indicate that GA and ethylene stimulate each other's activities reciprocally under specific circumstances.

Ethylene plays a central role in regulating the plant's developmental reaction to stress. GA has been shown to be positively involved in ethylene activity under conditions of oxygen deficiency. The elongation of deepwater-rice internodes requires the activity of GA and ethylene (Sauter et al., 1995). Reduced O_2 level during rice submergence was suggested to induce ethylene production, which in turn inhibited ABA synthesis. This changed the balance between ABA and GA, resulting in GA-induced stem elongation. In *Rumex palustris*, submergence induces the expression of *GA3ox*, suggesting that ethylene induces GA synthesis (Benschop et al., 2006). Thus, during submergence, ethylene affects the GA-to-ABA ratio by inhibiting ABA production and inducing GA synthesis. Ethylene also induced adventitious root growth in submerged rice. While GA had no effect on its own on this process, it acted synergistically with ethylene to promote root growth (Steffens et al., 2006).

Depending on the developmental process and environmental conditions, ethylene thus interacts both positively and negatively with GA. Furthermore, the interaction between these two hormones operates at both the biosynthesis and signal transduction levels, exhibits reciprocal effects of these hormones on one another, and involves both additive and synergistic effects.

RECIPROCAL INTERACTION BETWEEN GA AND CYTOKININ

GA and cytokinin exert antagonistic effects on numerous developmental processes, including shoot and root elongation, cell differentiation, shoot regeneration in culture, and meristem activity (Greenboim-Wainberg et al., 2005; Jasinski et al., 2005). Several recent studies have shown development-dependent reciprocal interactions between the two hormones, where cytokinin inhibits the production of GA and promotes its deactivation and GA inhibits cytokinin responses (Fig. 5).

High cytokinin and low GA signals are required for normal shoot apical meristem (SAM) function (Sakamoto et al., 2001; Jasinski et al., 2005; Yanai et al., 2005). SAM regulators from the KNOTTED1-like homeobox (KNOXI) protein family were shown to induce expression of the cytokinin-biosynthesis gene *ISOPENTENYL TRANSFERASE7* and accumulation of the hormone in the meristem (Jasinski et al., 2005; Yanai et al., 2005). Proteins from this family were also shown to negatively control GA level in the SAM by binding to the *GA20ox* promoter and directly repressing its transcription (Sakamoto et al., 2001; Hay et al., 2002; Chen et al., 2004). KNOXI and cytokinin both induced the expression of the gene encoding the GA-deactivating enzyme GA2ox at the base of the SAM, perhaps to block biologically active GAs transported to the SAM from flanking tissues (Jasinski et al., 2005). Thus, KNOXI proteins control the balance between cytokinin and GA in the SAM by inducing cytokinin production, directly inhibiting GA synthesis, and indirectly promoting GA deactivation. Genome-wide expression profiling of cytokinin-treated Arabidopsis seedlings revealed that cytokinin inhibits the expression of *GA20ox* and *GA3ox* and promotes that of *RGA* and *GAI*, further expanding upon the negative interactions between these hormones (Brenner et al., 2005).

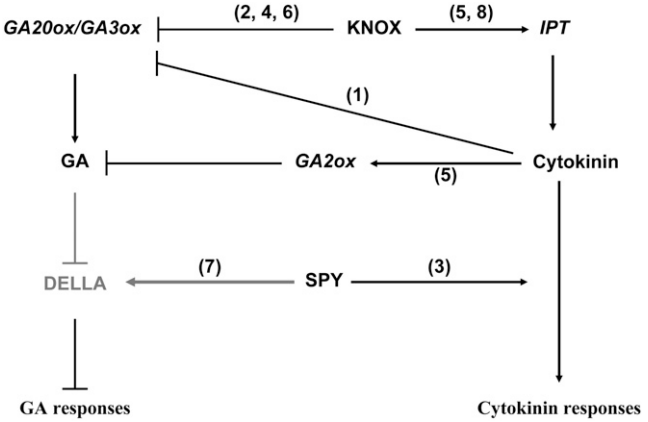

Figure 5. Network of reciprocal interactions between GA and cytokinin. Two major players control the balance between GA and cytokinin. KNOXI proteins control the balance between the two hormones in the SAM by inducing cytokinin production, directly inhibiting GA synthesis, and indirectly promoting GA deactivation. SPY regulates the balance between the response pathways of these two hormones via suppression of GA signal and promotion of cytokinin responses. Interactions mediated by changes in protein activity or stability are in gray and those mediated by gene expression are in black. Numbers in parentheses indicate the respective reference as follows: 1, Brenner et al., 2005; 2, Chen et al., 2004; 3, Greenboim-Wainberg et al., 2005; 4, Hay et al., 2002; 5, Jasinski et al., 2005; 6, Sakamoto et al., 2001; 7, Silverstone et al., 2007; 8, Yanai et al., 2005. *IPT*, *ISOPENTENYL TRANSFERASE*.

Whereas SAM activities require high cytokinin and low GA signals, later stages of cell maturation and elongation require the opposite: low cytokinin and high GA signals. A reverse antagonistic interaction, in which GA inhibits cytokinin, has also been demonstrated. Greenboim-Wainberg et al. (2005) have shown that GA, or a mutation in the GA-signaling suppressor SPY, inhibit cytokinin responses in Arabidopsis. Several lines of evidence suggest SPY acts directly to promote cytokinin responses and that GA suppresses cytokinin responses via SPY. This suggests that in the absence of GA, SPY represses GA signaling and promotes cytokinin responses, but when cellular GA levels increase, the hormone suppresses SPY activity and, therefore, cytokinin responses. How GA suppresses the cytokinin response via SPY is not yet clear. Recent results have suggested that GA has no effect on SPY activity toward the inhibition of GA responses (Silverstone et al., 2007). It is possible, however, that GA inhibits a component that interacts with SPY to specifically promote cytokinin responses. How might SPY promote cytokinin responses? Because GA and *spy* inhibited the induction of the cytokinin primary-response genes *TYPE-A RESPONSE REGULATOR*s, it was suggested that SPY interacts with, and perhaps modifies (via *O*-GlcNAc modification), elements of the cytokinin phosphorelay cascade (for review of the cytokinin-signaling pathway, see Hutchison and Kieber, 2002; Kakimoto, 2003; Ferreira and Kieber, 2005). However, elucidation of the mechanism by which SPY affects the cytokinin pathway still requires intensive study.

Despite the pronounced effect of *spy* on cytokinin responses, the *spy* phenotype is much less severe than that of the triple mutant of the cytokinin receptors (Higuchi et al., 2004). This could result from functional redundancy with either GA-related or nonrelated components. The Arabidopsis genome contains one additional OGT gene, *SECRET AGENT* (*SEC*; Hartweck et al., 2002). While *sec* mutations do not show any obvious phenotypic alteration, the *sec spy* double mutant is lethal (Hartweck et al., 2002). As high GA levels or signal do not cause lethality, this lethality could result from an effect of the double mutant on both GA and cytokinin pathways.

Interestingly, GA and *spy* suppress phenotypes caused by *KNOXI* overexpression (Hay et al., 2002). Although GA and *spy* may simply restore the inhibition effect of KNOXI on GA biosynthesis, it is also possible that they inhibit KNOXI-induced cytokinin responses or that SPY is required directly for KNOXI activity, the latter representing another possible level of interaction between GA and cytokinin.

Hence, cytokinin and GA act mostly in an antagonistic manner. The reciprocal interaction is regulated at both the biosynthesis and signal transduction levels.

CONCLUSION AND PERSPECTIVES

GA interacts with all other plant hormones, in some cases reciprocally, whereby GA affects but is also being affected by the other hormone. The direction and type (positive or negative) of the interaction depends on the biological process, tissue, developmental stage, and/or environmental conditions. The network likely features further levels of complexity, as interactions between more than two hormones to regulate specific developmental processes have been documented. For example, GA, auxin, and ethylene interact to promote elongation of light-grown seedlings (Saibo et al., 2003), and GA, cytokinin, and auxin are all involved in SAM development (Shani et al., 2006). This naturally results in a seemingly infinite number of possible combinations for regulation, which may contribute to the plant's ability to cope with a constantly changing environment with high flexibility (Brady and McCourt, 2003). It is clear, however, that the current knowledge is just the tip of the iceberg in a complex network of interactions between the various plant hormones.

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LITERATURE CITED

Achard P, Baghour M, Chapple A, Hedden P, Van Der Straeten D, Genschik P, Moritz T, Harberd NP (2007) The plant stress hormone ethylene controls floral transition via DELLA-dependent regulation of floral meristem-identity genes. Proc Natl Acad Sci USA **104:** 6484–6489

Achard P, Cheng H, De Grauwe L, Decat J, Schoutteten H, Moritz T, Van Der Straeten D, Peng J, Harberd NP (2006) Integration of plant responses to environmentally activated phytohormonal signals. Science **311:** 91–93

Achard P, Herr A, Baulcombe DC, Harberd NP (2004) Modulation of floral development by a gibberellin-regulated microRNA. Development **131:** 3357–3365

Achard P, Vriezen WH, Van Der Straeten D, Harberd NP (2003) Ethylene regulates Arabidopsis development via the modulation of della protein growth repressor function. Plant Cell **15:** 2816–2825

Benschop JJ, Bou J, Peeters AJM, Wagemaker N, Gühl K, Ward D, Hedden P, Moritz T, Voesenek LACJ (2006) Long-term submergence-induced elongation in *Rumex palustris* requires abscisic acid-dependent biosynthesis of gibberellin. Plant Physiol **141:** 1644–1652

Bouquin T, Meier C, Foster R, Nielsen ME, Mundy J (2001) Control of specific gene expression by gibberellin and brassinosteroid. Plant Physiol **127:** 450–458

Brady SM, McCourt P (2003) Hormone cross-talk in seed dormancy. J Plant Growth Regul **22:** 25–31

Brenner WG, Romanov GA, Kollmer I, Burkle L, Schmulling T (2005) Immediate-early and delayed cytokinin response genes of *Arabidopsis thaliana* identified by genome-wide expression profiling reveal novel cytokinin-sensitive processes and suggest cytokinin action through transcriptional cascades. Plant J **44:** 314–333

Chen H, Banerjee AK, Hannapel DJ (2004) The tandem complex of BEL and KNOX partners is required for transcriptional repression of *ga20ox1*. Plant J **38:** 276–284

De Grauwe L, Vriezen WH, Bertrand S, Phillips A, Vidal AM, Hedden P, Van Der Straeten D (March 10, 2007) Reciprocal influence of ethylene and gibberellins on response-gene expression in Arabidopsis thaliana. Planta doi/10.1007/s00425-007-0501-7

Dill A, Thomas SG, Hu J, Steber CM, Sun T-p (2004) The Arabidopsis F-box protein SLEEPY1 targets GA signaling repressors for GA-induced degradation. Plant Cell **16:** 1392–1405

Ferreira FJ, Kieber JJ (2005) Cytokinin signaling. Curr Opin Plant Biol **8:** 518–525

Filardo FF, Swain SM (2003) SPYing on GA signaling and plant development. J Plant Growth Regul **22:** 163–175

Frigerio M, Alabadi D, Perez-Gomez J, Gracia-Cacel L, Phillips AL, Hedden P, Blazquez MA (2006) Transcriptional regulation of gibberellin metabolism genes by auxin signaling in Arabidopsis. Plant Physiol **142:** 553–563

Fu XD, Harberd N (2003) Auxin promotes Arabidopsis root growth by modulating gibberellin response. Nature **421:** 740–743

Gómez-Cadenas A, Verhey SD, Holappa LD, Shen Q, Ho THD, Walker-Simmons MK (1999) An abscisic acid-induced protein kinase, PKABA1, mediates abscisic acid-suppressed gene expression in barley aleurone layers. Proc Natl Acad Sci USA **96:** 1767–1772

Gómez-Cadenas A, Zentalla R, Walker-Simmons M, Ho THD (2001) Gibberellin/abscisic acid antagonism in barley aleurone cells: site of action of the protein kinase PKABA1 in relation to gibberellin signaling molecules. Plant Cell **13:** 667–679

Greenboim-Wainberg Y, Maymon I, Borochov R, Alvarez J, Olszewski N, Ori N, Eshed Y, Weiss D (2005) Cross talk between gibberellin and cytokinin: the Arabidopsis GA-response inhibitor SPINDLY plays a positive role in cytokinin signaling. Plant Cell **17:** 92–102

Griffiths J, Murase K, Rieu I, Zentella R, Zhang ZL, Powers SJ, Gong F, Phillips AL, Hedden P, Sun TP, et al (2007) Genetic characterization and functional analysis of the GID1 gibberellin receptors in Arabidopsis. Plant Cell **18:** 3399–3414

Gubler F, Kalla R, Roberts JK, Jacobsen JV (1995) Gibberellin-regulated expression of a myb gene in barley aleurone cells: evidence for Myb transactivation of a high-pI α-amylase gene promoter. Plant Cell **7:** 1879–1891

Guo H, Ecker JR (2004) The ethylene signalling pathway: new insights. Curr Opin Plant Biol **7:** 40–49

Hartweck LM, Olszewski NE (2006) GIBBERELLIN INSENSITIVE DWARF1 is a gibberellin receptor that illuminates and raises questions about GA signaling. Plant Cell **18:** 278–282

Hartweck LM, Scott CL, Olszewski NE (2002) Two O-linked N-acetylglucosamine transferase genes of *Arabidopsis thaliana* L. Heynh. have overlapping functions necessary for gamete and seed development. Genetics **161:** 1279–1291

Hay A, Kaur H, Phillips AS, Hedden P, Hake S, Tsiantis M (2002) The gibberellin pathway mediates knotted1-type homeobox function in plants with different body plans. Curr Biol **12:** 1557–1565

Hedden P, Phillips AL (2000) Manipulation of hormone biosynthetic genes in transgenic plants. Curr Opin Biotechnol **11:** 130–137

Higuchi M, Pischke MS, Mahonen AP, Miyawaki K, Hashimoto Y, Seki M, Kobayashi M, Shinozaki K, Kato T, Tabata S, et al (2004) In planta functions of the Arabidopsis cytokinin receptor family. Proc Natl Acad Sci USA **101:** 8821–8826

Hutchison CE, Kieber JJ (2002) Cytokinin signaling in Arabidopsis. Plant Cell **14:** S47–S59

Itoh H, Ueguchi-Tanaka M, Sato Y, Ashikari M, Matsuoka M (2002) The gibberellin signaling pathway is regulated by the appearance and disappearance of SLENDER RICE1 in nuclei. Plant Cell **14:** 57–70

Jacobsen SE, Olszewski NE (1993) Mutations at the *SPINDLY* locus of Arabidopsis alter gibberellin signal transduction. Plant Cell **5:** 887–896

Jasinski S, Piazza P, Craft J, Hay A, Woolley L, Rieu I, Phillips A, Hedden P, Tsiantis M (2005) KNOX action in *Arabidopsis* is mediated by coordinate regulation of cytokinin and gibberellin activities. Curr Biol **15:** 1560–1565

Kakimoto T (2003) Perception and signal transduction of cytokinins. Annu Rev Plant Biol **54:** 605–627

Lange MJP, Lange T (2006) Gibberellin biosynthesis and the regulation of plant development. Plant Biol **3:** 281–290

Nakajima M, Shimada A, Takashi Y, Kim YC, Park SH, Ueguchi-Tanaka M, Suzuki H, Katoh E, Iuchi S, Kobayashi M, et al (2006) Identification and characterization of Arabidopsis gibberellin receptors. Plant J **46:** 880–889

Nemhauser JL, Hong FX, Chory J (2006) Different plant hormones regulate similar processes through largely nonoverlapping transcriptional responses. Cell **126:** 467–475

O'Neill DP, Ross JJ (2002) Auxin regulation of the gibberellin pathway in Arabidopsis. Plant Physiol **130:** 1974–1982

Razem FA, Baron K, Hill RD (2006) Turning on gibberellin and abscisic acid signaling. Curr Opin Plant Biol **9:** 454–459

Reyes JL, Chua NH (2007) ABA induction of miR159 controls transcript levels of two MYB factors during Arabidopsis seed germination. Plant J **49:** 592–606

Rogers JC, Rogers SW (1992) Definition and functional implications of gibberellin and abscisic acid *cis*-acting hormone response complexes. Plant Cell **4:** 1443–1451

Ross JJ, O'Neill DP, Smith JJ, Kerckhoffs LHJ, Elliott RC (2000) Evidence that auxin promotes gibberellin A1 biosynthesis in pea. Plant J **21:** 547–552

Saibo NJM, Vriezen WH, Beemster GTS, Van der Straeten D (2003) Growth and stomata development of Arabidopsis hypocotyls are controlled by gibberellins and modulated by ethylene and auxins. Plant J **33:** 989–1000

Sakamoto T, Kamiya N, Ueguchi-Tanaka M, Iwahori S, Matsuoka M (2001) KNOX homeodomain protein directly suppresses the expression of a gibberellin biosynthetic gene in the tobacco shoot apical meristem. Genes Dev **15:** 581–590

Sasaki A, Itoh H, Gomi K, Ueguchi-Tanaka M, Ishiyama K, Kobayashi M, Jeong DH, An G, Kitano J, Ashikari M, et al (2003) Accumulation of phosphorylated repressor for gibberellin signaling in an F-box mutant. Science **299:** 1896–1898

Sauter M, Mekhedov SL, Kende H (1995) Gibberellin promotes histone H1 kinase-activity and the expression of *CDC2* and cyclin genes during the induction of rapid growth in deep-water rice internodes. Plant J **7:** 623–632

Shani E, Yanai O, Ori N (2006) The role of hormones in shoot apical meristem function. Curr Opin Plant Biol **9:** 484–489

Silverstone AL, Tseng TS, Swain SM, Dill A, Jeong SY, Olszewski NE, Sun TP (2007) Functional analysis of SPINDLY in gibberellin signaling in Arabidopsis. Plant Physiol **143:** 987–1000

Steffens B, Wang JX, Sauter M (2006) Interactions between ethylene, gibberellin and abscisic acid regulate emergence and growth rate of adventitious roots in deepwater rice. Planta **223:** 604–612

Sun TP, Gubler F (2004) Molecular mechanism of gibberellin signaling in plants. Annu Rev Plant Biol **55:** 197–223

Teale WD, Paponov IA, Palme K (2006) Auxin in action: signalling, transport and the control of plant growth and development. Nat Rev Mol Cell Biol **7:** 847–859

Traw MB, Bergelson J (2003) Interactive effects of jasmonic acid, salicylic acid, and gibberellin on induction of trichomes in Arabidopsis. Plant Physiol **133:** 1367–1375

Ueguchi-Tanaka M, Ashikari M, Nakajima M, Itoh H, Katoh E, Kobayashi M, Chow TY, Hsing YI, Kitano H, Yamaguchi I, et al (2005) GIBBERELLIN INSENSITIVE DWARF1 encodes a soluble receptor for gibberellin. Nature **437:** 693–698

Vriezen WH, Achard P, Harberd NP, Van Der Straeten D (2004) Ethylene-mediated enhancement of apical hook formation in etiolated *Arabidopsis thaliana* seedlings is gibberellin dependent. Plant J **37:** 505–516

Wells L, Vosseller K, Hart GW (2001) Glycosylation of nucleocytoplasmic proteins: signal transduction and O-GlcNac. Science **291:** 2376–2378

Wolbang CM, Ross JJ (2001) Auxin promotes gibberellin biosynthesis in decapitated tobacco plants. Planta **214:** 153–157

Xie Z, Zhang ZL, Zou XL, Yang GX, Komatsu S, Shen QXJ (2006) Interactions of two abscisic-acid induced WRKY genes in repressing gibberellin signaling in aleurone cells. Plant J **46:** 231–242

Yanai O, Shani E, Dolezal K, Tarkowski P, Sablowski R, Sandberg G, Samach A, Ori N (2005) *Arabidopsis* KNOXI proteins activate cytokinin biosynthesis. Curr Biol **15:** 1566–1571

Zentella R, Yamauchi D, Ho THD (2002) Molecular dissection of the gibberellin/abscisic acid signaling pathways by transiently expressed RNA interference in barley aleurone cells. Plant Cell **14:** 2289–2301

CORRECTIONS

Vol. 144: 682–694, 2007

Pislariu C. and Dickstein R. An *IRE*-Like AGC Kinase Gene, *MtIRE*, Has Unique Expression in the Invasion Zone of Developing Root Nodules in *Medicago truncatula.*

On Page 687, the first full sentence in the right column should read as follows: ''Plants were inoculated with rhizobia carrying a constitutive *lacZ* gene, and nodulated roots were harvested at 15 dpi.''

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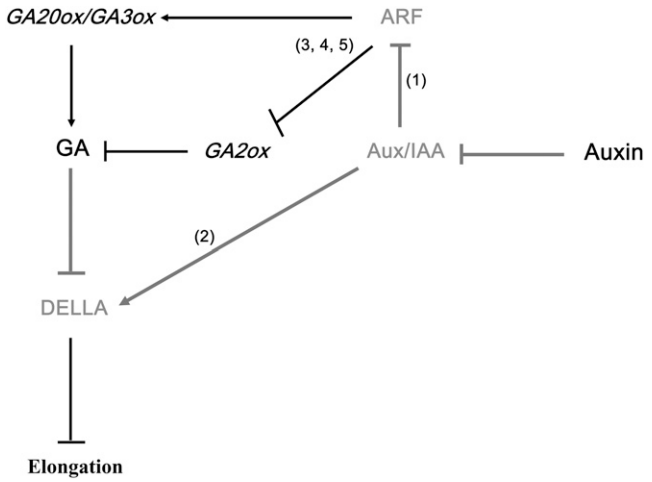
Weiss D. and Ori N. Mechanisms of Cross Talk between Gibberellin and Other Hormones.

In Figure 3 of this *Update*, the inhibition bar from Aux/IAA to DELLA should be an arrow. The corrected figure is below.



www.plantphysiol.org/cgi/doi/10.1104/pp.104.900232