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Hormone Cross-Talk in Seed Dormancy

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Abstract

The choice to become dormant versus continuing to grow is observed in a variety of organisms in response to specific developmental and environmental signals. In higher plants this is most obvious during both the establishment and breaking of seed dormancy. With the advent of molecular genetic analysis, particularly in *Arabidopsis*, genes involved in the establishment and breaking of seed dormancy have been identified. Genetic analysis suggests a web of hormone-derived information is required in the regulation of these processes. In this review we focus on examples of where hormones, and in particular cross-talk between hormones, is used to regulate both the establishment and release of seed dormancy. The use of multiple hormones that overlap in their control of specific developmental programs allows seeds to be flexible in making decisions in response to specific developmental and environmental cues.

Key words: Hormone mutants; *Arabidopsis*; Gibberellins; Abscisic acid; Ethylene; Brassinosteroid; Seed development; Signal transduction; Hormone interaction

Dormancy: Making Up and Breaking Up

The usual botanical definition of seed dormancy is the failure of an intact viable seed to complete germination under favorable conditions (Koornneef and others 2002). There are many situations that can be envisioned where seed dormancy could be adaptive. For example, dormant seeds ensure persistence of a species in risky or randomly varied environments or can prevent seedlings from competing with the mother plant or siblings (Baskin and Baskin 1998). A recurring theme that arises from these scenarios, however, is that the seed needs a way to interpret information, whether it is developmental or environmental, in a dynamic way so as to respond with flexibility. In looking for regulatory molecules that link developmental and environmental signals, plant growth regulators or hormones are often found to be important. In seed dormancy, for example, mutations that decrease either abscisic acid (ABA) or gibberellins (GA) have profound effects on the establishment and breaking of seed dormancy (McCourt 1999). However, the construction of double mutants deficient in the synthesis of both of these hormones indicates it is a balance between these two regulators that results in the level of overall dormancy or ability to germinate (Debeaujon and Koornneef 2000; Steber and others 1998). Hence, it is the interaction of ABA and GA or cross-talk between the two hormones that is required for an understanding of how seed dormancy is regulated. In this review we have focused on evidence of how hormones may cross-talk to

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control seed dormancy programs with an emphasis on studies using *Arabidopsis thaliana* L. (*Arabidopsis*). For obvious reasons, seeds of many laboratory-friendly *Arabidopsis* ecotypes are not highly dormant. Thus, many of the mutants that are identified that have altered responses to hormones such as ABA or GA are actually scored by their germination profiles rather than by their change in dormancy. Many of these mutants, therefore, do not strictly follow the botanical definitions of producing a more dormant or less dormant seed. However, the concentration on *Arabidopsis* is because this model genetic system has been instrumental in the identification of components that define how and where hormones are synthesized and how sensitivity to a particular hormone is established. With the identification of these genes, future goals will be to take these components and study them in a more traditional seed dormancy system such as wild oats.

Hormone Cross-Talk: What's all the Gossip About?

Cross-talk means different things to different people and therefore has many definitions. In this review we have defined cross-talk in a number of ways. First, it can occur not only within a cell but also between cells and tissues. Hence, the action of one hormone on another hormone's synthesis within or between cells can be considered as cross-talk. Cross-talk can also occur via shared signalling components or by control of common downstream targets. In these cases we are most likely discussing intracellular interactions. Whatever definitions or mechanisms are used, a central conclusion that has arisen from hormone cross-talk studies is the concept of a linear genetic pathway giving way to the acceptance that hormone signalling forms a complex web of overlapping information (Gazzarini and McCourt 2003).

Examples of hormones regulating each other's synthesis are becoming very common (Grsic and others 1999; Grossmann and others 1996; Hansen and others 2000; O'Neill and Ross 2002; Ross and others 2001; Wolbang and others 2001; Yi and others 1999). An isoform of a key regulatory enzyme in the ethylene biosynthetic pathway, 1-aminocyclopropane-1-carboxylic acid synthase 4 (*ACS4*) is a primary auxin response gene, and this regulation is dependent upon the auxin signalling genes, *AXR1* and *AXR2* and on the auxin transporter, *AUX1* (Abel and others 1995). The ability of ethylene to rescue the germination defect of an *Arabidopsis* GA auxotroph (Karssen and others 1989; Koornneef and Karssen 1994) suggests that control of ethylene synthesis could play a role in the breaking of seed dormancy. On this note, any hormone that encourages cell expansion of the hypocotyl or radicle cell elongation, or degradation of the surrounding seed tissues would contribute to breaking dormancy. As a result, it might be expected that the action of GA, auxin, and ethylene, which are all known to be involved in cell elongation, could result in the same outcome and hence appear as if they cross-talk in a single process such as germination.

However, auxin is unable to rescue the *ga1* mutant, and while ethylene is able to rescue the germination of *ga1*, this most likely is due to a nonspecific stimulation of hypocotyl elongation. Although this result demonstrates that auxin does not influence these GA and ethylene interactions, molecular mechanisms of auxin action do give hints as to how hormones may cross-talk using similar downstream components. For example, the *AXR1* gene encodes the ubiquitin-activating enzyme E1 and was initially found in screens for genes required for the auxin response (Leyser and others 1993). However, *AXR1* is also a signalling component of the jasmonic acid-signalling pathway. The *axr1-24* allele has decreased sensitivity to IAA, jasmonate, ACC, cytokinin, brassinosteroid (BR) and ABA. IAA was also shown to induce jasmonic acid (JA)-responsive genes, and this induction is dependent on the presence of AXR1 (Tiryaki and others 2002).

ABA and GA: Who's on Top?

As mentioned, ABA and GA have opposing actions in control of seed dormancy. ABA auxotrophic mutants possess decreased seed dormancy, whereas GA auxotrophs have increased seed dormancy, and need application of exogenous GA to germinate (Karssen and others 1989; Koornneef and Karssen 1994; Leon-Kloosterziel and others 1996). GA is proposed to have two roles in the process of dormancy. The first is the induction of expression of genes encoding enzymes that hydrolyze the endosperm and the second is the direct stimulating effect on the growth potential of the embryo. However, the ability of double mutants auxotrophic for both hormones to germinate suggests that the effect of GA in the embryo appears to be restricted in counteracting ABA-regulated events such as seed dormancy (Debeaujon and Koornneef 2000).

The role of GA in the endosperm has been demonstrated by several experiments. First, removal of the seed coat of a GA biosynthetic mutant relieves its requirement for exogenous GA to germinate. Second, the introduction of a testa mutation or an

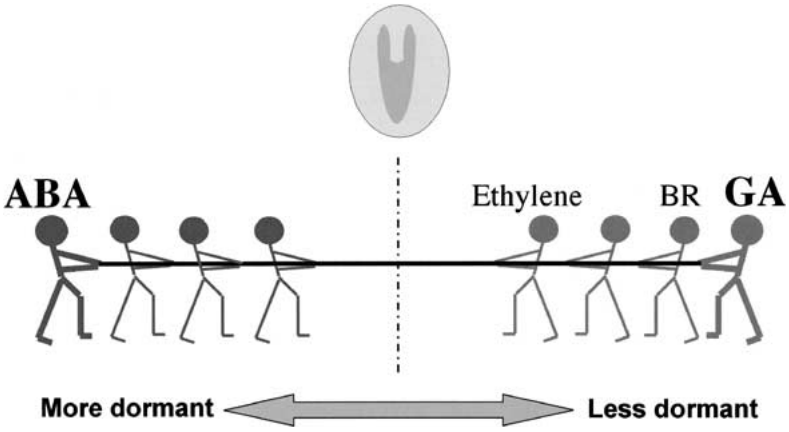

Figure 1. Balance model of hormone action in the establishment of seed dormancy. Genetic and physiological analysis has determined that the overall level of seed dormancy is often established during embryogenesis by a tug-of-war among various hormones. Mutations that increase or decrease a particular hormone's sensitivity or synthesis will pull the seed towards or away from a more dormant state. This can often make the classification of mutants based on preliminary phenotypes difficult. For example, GA auxotrophs in *Arabidopsis* do not germinate because of the ABA they synthesize during embryogenesis. Suppressor mutations often are defective in ABA synthesis or sensitivity.

ABA auxotrophic mutation functions in the same way (Debeaujon and Koornneef 2000). The role of ABA in this process is less clear. In *Arabidopsis*, an extensin-like gene, *AtEPR1* is expressed in the endosperm at the micropylar end of the germinating seed, and this expression is under control of GA, but not of ABA (Dubreucq and others 2000). However, in tobacco and rice, ABA does appear to be involved in this seed-coat regulation by GA. In tobacco, GA is correlated with testa and endosperm rupture resulting in dormancy release (Leubner-Metzger 2002). In this system GA is also associated with the expression of the βGLU1gene, which has been used as a reporter gene for dormancy release. ABA is able to delay this endosperm rupture and inhibits induction of βGLU1. In rice, the *slr1* mutant behaves as if it has a constitutive GA response, with GA-independent expression of α-amylase. This GA-independent expression is inhibited by ABA application (Ikeda and others 2002), suggesting that the effect of GA in the tissues surrounding the embryo is not exclusive to ABA signalling (Ikeda and others 2002).

Signalling between ABA and GA in the embryo is very common, and studies in *Arabidopsis* have revealed many interactions between these two signalling pathways. Using suppression of ABA insensitivity of the *abi1-1* mutant, it was possible to identify mutations with decreased GA biosynthesis (*ga1*) or sensitivity (*sly1*) (Steber and others 1998). Because *sly1* alleles cannot respond to GA they require the nondormancy conferred by the *abi1* lesion to germinate (Steber and others 1998). On the other hand, ABA auxotrophic and ABA-insensitive mutants suppress the requirement for GA in germination of *Arabidopsis* (Koornneef and others 1982; Nambara and others 1992; Leon-Kloosterziel and others 1996). Furthermore, a mutation in the *Arabidopsis SPY1* gene that confers increased GA signalling results in reduced ABA sensitivity (Steber and others 1998). These experiments suggest a genetic framework in which there is a push and pull of hormone balance between ABA and GA action that determines the overall seed dormancy and germination capacity (Figure 1). High ABA or low GA levels of sensitivity results in more dormant seeds, and conversely, low ABA or high GA levels of sensitivity results in less dormant seeds. The mutations identified in these suppressor screens, however, only partially suppress germination to wild type displaying more of an additive rescue, suggesting parallel pathways (Steber and others 1998). The relationship between ABA and GA in seed dormancy is therefore more complex. Added to this complexity are other hormone biosyntheses and signal transduction pathways, including those of ethylene and brassinosteroid which also appear to influence ABA and GA action in the seed (Figure 1).

Ethylene, ABA and GA: A Menage Á Trois

The plant hormone, ethylene, is usually associated with action in fruit ripening and leaf and flower senescence. Ethylene has also been implicated in the release of seed dormancy, root hair formation and in wounding and pathogen responses (Reid 1995; Bleecker and others 1998). Ethylene is able to break the dormancy of peanut, apple, redroot pigweed, cocklebur, lamb's quarters, *Amaranthus retroflexus* and sunflower (Kepczynski and Kepczynska 1997; Kepczynski and others 1997). Ethylene gas is able to fully rescue the germination defect of the *Arabidopsis* ga-*1* mutant in the light, however, the resulting seedlings display the triple response (Karssen and others 1989; Koornneef and Karssen

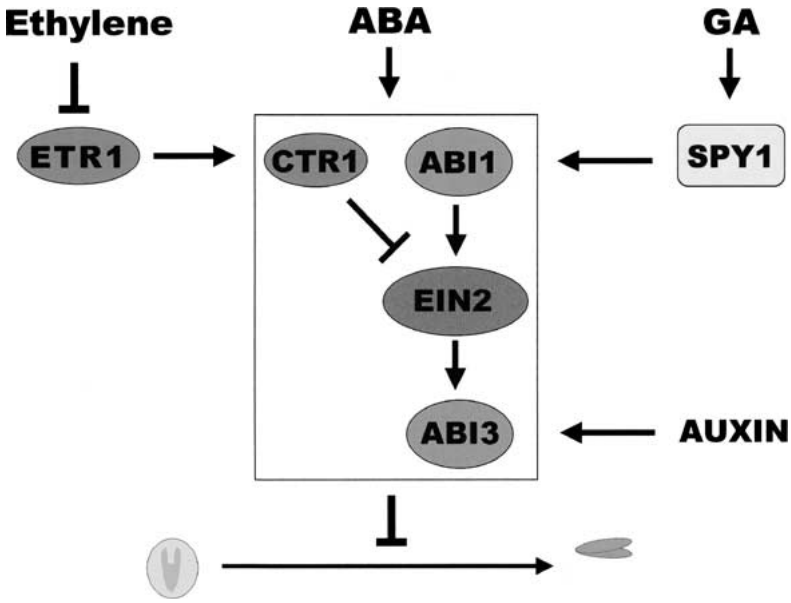

Figure 2. Genetic interactions in hormone signalling in *Arabidopsis* seeds. Mutations in ethylene, ABA and GA pathways have all been shown to increase or decrease *Arabidopsis* seed germination. Furthermore, suppressor and double-mutant analysis among various pathways has uncovered an extensive genetic cross-talk between these pathways. For example, all the genes shown in the box show simple epistatic interactions with each other at the level of seed sensitivity to ABA. *ETR1*, *CTR1* and *EIN2* are ethylene-response genes, *ABI1* and *ABI3* are ABA-response genes, and *SPY1* is a GA-response gene. Arrows represent a positive genetic interaction and bars represent a negative genetic interaction. This representation is a genetic relationship and not necessarily a mechanistic one.

1994), suggesting that some components of ethylene signalling are involved. The genetic mechanisms of ethylene action on seed germination have not been elucidated. Analysis of ABA and GA biosynthesis and signalling would be useful to fully determine if ethylene stimulates germination by altering ABA or GA biosynthesis or sensitivity, or if it acts through its own signalling components in these cases. Ethylene could also interact with other signalling components not previously identified through traditional ethylene response mutant screens. Furthermore, the detailed knowledge of the molecular mechanisms of early ethylene action should be extremely useful in understanding how ethylene stimulates germination.

In ethylene signalling, the hormone binds to the 2-component receptor ETR1, which stops the ETR1 receptor from activating CTR1, a downstream Raf-like serine/threonine kinase. Lack of CTR1 activation releases positive regulators such as EIN2 and EIN3 from the negative regulation of CTR1 allowing an ethylene response (McCourt 1999). These signalling components have been shown to interact with genes involved in ABA signalling, or their mutants have altered ABA responses (Figure 2). Mutations in *CTR1* are able to enhance the ABA insensitivity of *abi1-1* whereas ethylene-insensitive mutants like *ein2* are able to suppress ABA insensitivity in *abi1* seed (Beaudoin and others 2000). When tested for general dormancy defects, the ethylene-insensitive mutants *ein2* and *etr1* have enhanced dormancy (Ghassemian and others 2000). Furthermore, loss-of-function *abi3* alleles, which have reduced seed dormancy, are epistatic to *ein2*. Together, these results further strengthen the genetic relationship between ABA and ethylene signalling components and it appears that ethylene negatively regulates ABA signalling in the seed.

Although exogenous ABA does not induce ethylene biosynthesis and exogenous ethylene does not induce the ABA-response gene, *RAB 18*, loss-of-function mutations in *EIN2* plants have higher amounts of leaf ABA compared to wild type (Ghassemian and others 2000). Thus, it appears that decreasing ethylene signalling can translate into increases in ABA synthesis (Ghassemian and others 2000). Surprisingly, decreases in ethylene signalling can also decrease ABA sensitivity in the root. Although the mechanism of this root sensitivity change is unclear, it may mean that the ethylene signalling pathway shares signalling components with ABA signal transduction. Further evidence for shared roles of ethylene signalling components has been uncovered in studies of sugar sensing and mutations in *EIN2* affect a wide range of signalling pathways including those involved in auxin transport, senescence, and in the action of cytokinin, ABA and jasmonic acid (for review, Gazzarini and McCourt 2001), suggesting that EIN2 is not entirely specific to ethylene signalling.

Brassinosteroid, ABA and GA: Size Matters

Brassinosteroids (BRs) are involved in a variety of physiological processes including cell elongation, cell division, reproductive and vascular development, leaf morphogenesis and epinasty, and induction of ethylene biosynthesis (for review, Clouse and Sasse

1998; Schumacher and Chory 2000). Although not generally accepted as a primary regulator of seed dormancy, BR has been reported to enhance the germination of rice, *Orobranche minor* and *Striga asiatica* seeds (Takeuchi and others 1991, 1995; Yamaguchi and others 1987). However, BR is unable to enhance germination in wild type *Arabidopsis* seeds, and can only induce endosperm and not testa rupture in light and dark-imbibed photo-dormant tobacco seeds (Steber and McCourt 2001; Leubner-Metzger 2001).

Although BR is unable to stimulate germination in wild type *Arabidopsis* seeds, it can rescue the germination of GA biosynthetic mutants (*ga1, ga2,* and *ga3*) and the GA-insensitive mutant *sly1.* BR is also able to partially rescue the hypocotyl elongation of both *ga1* and *sly1* in the dark. Stimulation of hypocotyl elongation may be the mechanism for the rescue of the germination defects of these two mutants by BR as both BR and GA have been shown to additively enhance cell elongation in other plants (Clouse and Sasse 1998).

In addition to an interaction with GA, the BR biosynthetic mutant *det2* and the *bri1* BR receptor mutant show increased sensitivity to ABA in germination (Steber and McCourt 2001). This phenotype may be due to increased sensitivity to ABA or increased ABA biosynthesis. Alternatively, BR mutants may have decreased GA biosynthesis or sensitivity. Measuring ABA and GA content in the seeds may further elucidate this mechanism. Aside from germination, hormone cross-talk has already been demonstrated among BR, GA and ABA at the whole plant level. There is ectopic accumulation of the GA-responsive γ-*TIP* gene in BR-deficient and BR-signalling mutants and the *Arabidopsis MERI-5* gene's expression is regulated by either BR or GA (Kauschmann and others 1996; Medford and others 1991). Finally, in *Arabidopsis,* BR is reported to up-regulate *GA5,* a gene involved in GA biosynthesis, and this up-regulation is dependent upon the *BRI1* receptor (Bouqin and others 2001).

Conclusion: Surfing the Web

The regulation of seed dormancy by hormones is certainly complex and as more genetic analysis is applied to this process, the complexity will increase. Some information has sent up warning flags that a mutant originally identified as specific for a particular hormone response may be involved in other hormone signalling pathways (Brady and others 2003). Thus, classifying genes as ABA- or GA-sensitivity genes, for example, will most likely become a relic of the past. Nevertheless, mutant isolation also has allowed the testing of hormone interactions in a more precise way than simply adding the hormone exogenously. The use of mutations that decrease the biosynthesis of a specific hormone has been instrumental in clearing up the relationship between hormones in the establishment of seed dormancy in *Arabidopsis* for example.

With the molecular information listed in this limited review it still seems that the question of why hormones should cross-talk in the first place to establish and break dormancy is unanswered. Why should a plant want to use a spider web of regulation rather than a linear control and command system to decide the level of seed dormancy or when to break dormancy and germinate? Perhaps the analogy to a spider web is appropriate since what is important about a web is not the individual threads and nodes but the overall oscillation of the web in response to a stimulus. For example, a spider wants to know when an insect is caught, which in turn causes an oscillation of the web that is very different than a trivial movement such as wind blowing through the web. This oscillation is dependent on the overall web topology that has been built through placement of nodes, numbers of nodes and distance between the nodes. Moreover, another important aspect of a web structure is the redundancies of threads connecting nodes that give it strength or flexibility. Perhaps the decision to germinate, which is a critical event in the plant's life cycle, uses a variety of hormone levels and responses that interact to give the response the strength and flexibility required to relay complex information to the embryo. The redundancy of hormone action would also be enhanced if its actions are instead overlapping, allowing a more robust response to a variety of environmental cues.

To be understood such a model does not really require the identification of all the threads and nodes that make up a signalling web. However, with actual signalling components in hand we can now begin to manipulate the web in meaningful ways to test its topology. With genomics programs it is now possible to use a computer to identify candidate genes that may be involved in a particular problem, and the advent of complete knockout lines in *Arabidopsis* and rice will allow direct testing of any gene in a particular process. The untangling of the hormone web is most likely in hand.

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REFERENCES

Abel S, Nguyen MD, Chow W, Theologis A. 1995. ACS4, a primary indoleacetic acid-responsive gene encoding 1-aminocyclopropane-l-carboxylate synthase in *Arabidopsis thaliana*. Structural characterization, expression in *Escherichia coli*, and expression characteristics in response to auxin [corrected]. J Biol Chem 270:19093–9, 26020.

Baskin CC, Baskin JM. 1998. Seeds: ecology, biogeography, and evolution of dormancy and germination. San Diego: Academic Press.

Beaudoin N, Serizet C, Gosti F, Giraudat J. 2000. Interactions between abscisic acid and ethylene signalling cascades. Plant Cell 12:1103–1105.

Bleecker AB, Esch JJ, Hall AE, Rodriguez FI, Binder BM. 1998. The ethylene-receptor family from *Arabidopsis*: structure and function. Philos Trans R Soc Lond B Biol Sci 353:1405–1412.

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

Brady SM, Sarkar SF, Bonetta D, McCourt P. 2003. The *ABSCISIC ACID INSENSITIVE 3* (*ABI3*) gene is modulated by farnesylation and is involved in auxin signalling and lateral root development in *Arabidopsis*. Plant J (in press).

Clouse SD, Sasse JM. 1998. Brassinosteroids: essential regulators of plant growth and development. Annu Rev Plant Physiol Plant Mol Biol 49:427–451.

Debeaujon I, Koornneef M. 2000. Gibberellin requirement for *Arabidopsis* seed germination is determined both by testa characteristics and embryonic abscisic acid. Plant Physiol 122:415–424.

Dubreucq B, Berger N, Vincent E, Boisson M, Pelletier G, Caboche M, Lepiniec L. 2000. The *Arabidopsis* AtEPR1 extensin-like gene is specifically expressed in endosperm during seed germination. Plant J 23:643–652.

Gazzarini S, McCourt P. 2003. Cross talk in plant hormone signalling: What *Arabidopsis* mutants are telling us. Ann Botany (in Press).

Ghassemian M, Nambara E, Cutler S, Kawaide H, Kamiya Y, McCourt P. 2000. Regulation of abscisic acid signalling by the ethylene response pathway in *Arabidopsis*. Plant Cell 12:1117–1126.

Grossmann K, Scheltrup F, Kwiatkowski J, Caspar G. 1996. Induction of abscisic acid is a common effect of auxin herbicides in susceptible plants. J Plant Physiol 149:475–478.

Grsic S, Kircheim B, Pieper K, Fritsch M, Hilgenberg W, Ludwig-Muller J. 1999. Induction of auxin biosynthetic enzymes by jasmonic acid and in clubroot diseased Chinese cabbage plants. Physiol Plant l05:521–531.

Hansen H, Grossmann K. 2000. Auxin-induced ethylene triggers abscisic acid biosynthesis and growth inhibition. Plant Physiol 1241437–1241438.

Ikeda A, Sonoda Y, Vernieri P, Perata P, Hirochika H, Yamaguchi J. 2002. The slender rice mutant with constitutively activated gibberellin signal transduction, has enhanced capacity for abscisic acid level. Plant Cell Physiol 43:974–979.

Karssen CM, Zagorski S, Kepczynski J, Groot SPC. 1989. Key role for endogenous gibberellins in the control of seed germination. Ann Botany 63:71–80.

Kauschmann A, Jessop A, Koncz C, Szekeres M, Willmitzer L, Altmann T. 1996. Genetic evidence for an essential role of brassinosteroids in plant development. Plant J 9:701–713.

Kepczynski J, Bihun M, Kepczynska E. 1997. In: Kanellis AK, Chang C, Kende H, Grierson H, editors. Biology and biotechnology of the plant hormone ethylene. pp 113–122.

Kepczynski J, Kepczynska E. 1997. Ethylene in seed dormancy and germination. Physiol Plant 101:720–726.

Koornneef M, Bentsink L, Hillhorst H. 2002. Seed dormancy and germination. Curr Opin Plant Biol 5:33–36.

Koornneef M, Jorna ML, Brinkhorst-van der Swan DLC, Karssen CM. 1982. The isolation and analysis of abscisic acid (ABA)-deficient mutants by selection of induced revertants in non-germinating gibberellin-sensitive lines of *Arabidopsis thaliana* (L.) Heynh Theor Appl Genl 61:385–393.

Koorneef M, Karssen CM. 1994. Seed dormancy and germination. In: Somerville CR, Meyerowitz EM, editors. Arabidopsis. New York: Cold spring Harbor Laboratory Press, pp 313–334.

Leon-Kloosterziel KM, Gil MA, Ruijs GJ, Jacobsen SE, Olszewski SH, Schwartz SH, Zeevart JAD, Koornneef M. 1996. Isolation and characterization of abscisic acid-deficient *Arabidopsis* mutants at two new loci. Plant J 10:655–661.

Leubner-Metzger G. 2001. Brassinosteroids and gibberellins promote tobacco seed germination by distinct pathways. Planta 213:758–763.

Leubner-Metzger G. 2002. Seed after-ripening and over-expression of class I B-1,3-glucanase confer maternal effects on tobacco testa rupture and dormancy release. Planta Med 215:959–968.

Leyser HM, Lincoln CA, Timpte C, Lammer D, Turner J, Estelle M. 1993. *Arabidopsis* auxin-resistance gene *AXR1* encodes a protein related to ubiquitin-activating enzyme E1. Nature 364:161–164.

Medford JI, Elmer JS, Klee HJ. 1991. Molecular cloning and characterization of genes expressed in shoot apical meristems. Plant Cell 3:359–370.

McCourt P. 1999. Genetic analysis of hormone signalling. Annu Rev Plant Physiol Plant Mol Biol 50:219–243.

Nambara E, Akazawa T, McCourt P. 1991. Effects of the gibberellin biosynthetic inhibitor uniconazol on mutants of *Arabidopsis*. Plant Physiol 97:736–738.

Nambara E, Naito S, McCourt P. 1992. A mutant of *Arabidopsis* which is defective in seed development and storage protein accumulation is a new *abi3* allele. Plant J 2:435–441.

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

Reid MS. 1995. Ethylene in plant growth, development and senescence. In: Davies PJ, editor. Plant hormones.. Dordrecht: Kluwer Academic Publishers, .

Ross JJ, O'Neill DP, Wolbang CM, Symons GM, Reid JB. 2001. Auxin-gibberellin interactions and their role in plant growth. J Plant Growth Regul 20:336–353.

Schumacher K, Chory J. 2000. Brassinosteroid signal transduction: still casting the actors. Curr Opin Plant Biol 3:79–84.

Steber CM, Cooney SE, McCourt P. 1998. Isolation of the GA-response mutant *sly1* as a suppressor of *abi1-1* in *Arabidopsis thaliana*. Genetics 149:509–521.

Steber CM, McCourt P. 2001. A role for brassinosteroids in germination in *Arabidopsis*. Plant Physiol 125:763–769.

Srivastava LM. 2002. Plant growth and development: hormones and environment. San Diego: Academic Press, pp 431–471.

Takeuchi Y, Worsham AD, Awad AE. 1991. Effects of brassinolide on conditioning and germination of witchweed (*Striga asiatica*) seeds. In: Cuttler HG, Yokota T, Adam G, editors. Brassinosteroids: chemistry, bioactivity and application. Washington DC: ACS Symposium Series 474, pp 298–305.

Takeuchi Y, Omigawa Y, Ogasawara M, Yoneyama K, Konnai M, Worsham D. 1995. Effects of brassinosteroids on conditioning and germination of clover broomrape (*Orobranche minor*) seeds. J Plant Growth Regul 16:153–160.

Tiryaki I, Staswick PE. 2002. An *Arabidopsis* mutant defective in jasmonate response is allelic to the auxin-signalling mutant *axr1*. Plant Physiol 130:887–894.

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

Yamaguchi T, Wakizuka T, Hirai K, Fujii S, Fujita A. 1987. Stimulation of germination in aged rice seeds by pretreatment with brassinolide. Proc Plant Growth Regul Soc Am 14:26–27.

Yi HC, Joo S, Nam KH, Lee JS, Kang BG, Kim WT. 1999. Auxin and brassinosteroid differentially regulate the expression of three members of the 1-aminocyclopropane-1-carboxylate synthase gene family in mung bean (*Vigna radiata* L.). Plant Mol Biol 41:443–454.