Molecular Aspects of Seed Dormancy*

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Abstract

Seed dormancy provides a mechanism for plants to delay germination until conditions are optimal for survival of the next generation. Dormancy release is regulated by a combination of environmental and endogenous signals with both synergistic and competing effects. Molecular studies of dormancy have correlated changes in transcriptomes, proteomes, and hormone levels with dormancy states ranging from deep primary or secondary dormancy to varying degrees of release. The balance of abscisic acid (ABA):gibberellin (GA) levels and sensitivity is a major, but not the sole, regulator of dormancy status. ABA promotes dormancy induction and maintenance, whereas GA promotes progression from release through germination; environmental signals regulate this balance by modifying the expression of biosynthetic and catabolic enzymes. Mediators of environmental and hormonal response include both positive and negative regulators, many of which are feedback-regulated to enhance or attenuate the response. The net result is a slightly heterogeneous response, thereby providing more temporal options for successful germination.

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INTRODUCTION

Seed dormancy has been described as "one of the least understood phenomena in seed biology" (50) and remains confusing despite much recent progress. This confusion reflects the likelihood that dormancy is not a single phenomenon but a condition with many contributing causes. Traditionally this condition has been primarily negatively defined as a developmental state in which a viable seed fails to germinate under superficially favorable environmental conditions (e.g., adequate moisture). Seed dormancy has been further negatively categorized in terms of the requirements for release from this block, such as disruption of the seed coat (scarification), a period of dry storage (after-ripening) or moist chilling (stratification), or exposure to light. The situation is further complicated by the fact that, although germination is an all-or-nothing event for each seed, populations display variable degrees of dormancy that are reflected in the rate or percentage of germination under specific conditions. Presumably, each seed is in a state somewhere along the continuum from deeply dormant to nondormant, but it remains unclear how the tipping point between nonpermissive and permissive for germination is sensed for each seed. However, this point is of critical agronomic and ecological significance because it determines both the degree of synchronous germination in a given season and the reservoir of ungerminated viable seeds remaining in the soil until a later season, i.e., the seed bank (**Figure 1**).

Recently, genetic approaches have led to the identification of specific loci correlated with altered dormancy, and -omics approaches in many species have identified numerous transcripts and proteins correlated with dormant versus nondormant seeds. Such studies should lead toward a positive definition of dormancy. Comparative analyses should identify characteristics common to many forms of dormancy, and possibly other features unique to certain classes of dormancy. Until then, dormancy remains in the realm of

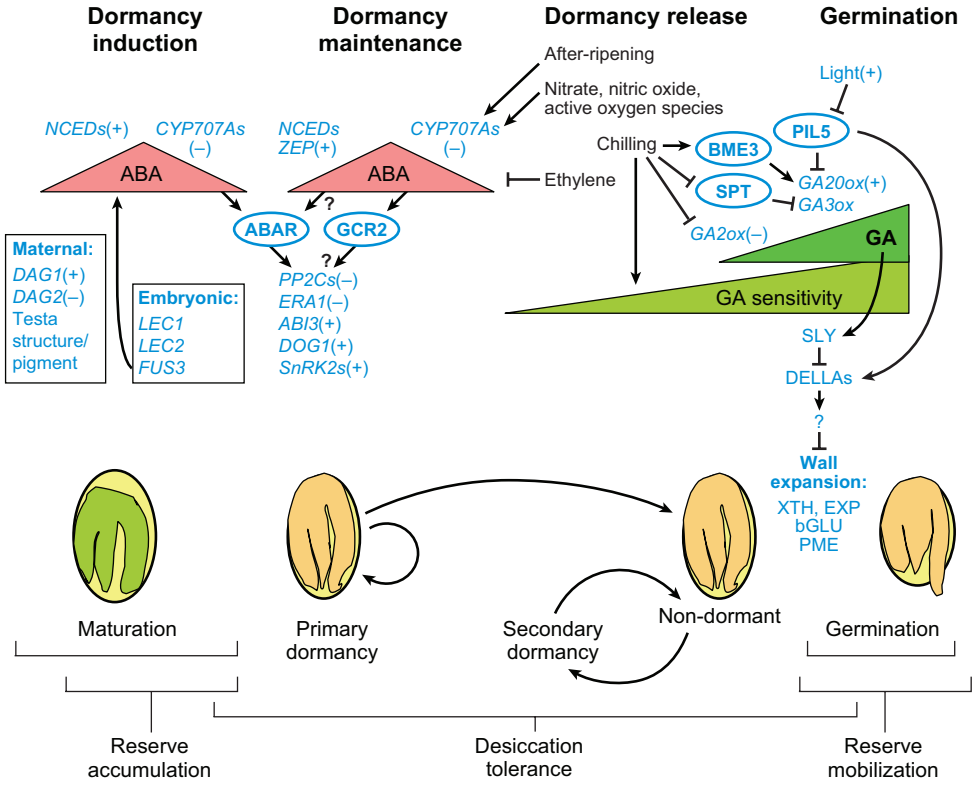

Figure 1

Time course of dormancy induction and release, including partial summary of regulatory factors. Width of abscisic acid (ABA) and gibberellin (GA) symbols represents relative hormone levels due to action of indicated biosynthetic and catabolic loci. Induction depends on combination of ABA-independent maternal and embryonic factors and ABA-dependent signaling. Release is promoted by many environmental factors, largely integrated through changes in ABA:GA signaling balance, eventually resulting in wall expansion to permit radicle emergence. Positive regulation is indicated by (+) and arrows, negative regulation by (–) and bars. NCED, 9-*cis*-epoxycarotenoid dioxygenase; CYP707A, Cytochrome P450 707A; ZEP, zeaxanthin epoxidase; NO, nitric oxide; AOS, active oxygen species; BME3, BLUE MICROPYLAR END 3; PIL5, PHYTOCHROME-INTERACTING FACTOR 3-LIKE 5; SPT, SPATULA; GA20ox, GIBBERELLIN 20 OXIDASE; GA3ox, GIBBERELLIN 3 OXIDASE; GA2ox, GIBBERELLIIN 2 OXIDASE; SLY, SLEEPY; XTH, xyloglucan endotransferase/hydrolase; EXP, expansins; bGLU, β-1,3 glucanase; PME, pectin methylesterase; DAG, DOF (DNA-binding with one zinc finger) AFFECTING GERMINATION; LEC, LEAFY COTYLEDON; FUS3, FUSCA 3; PP2C, protein phosphatase 2C; ERA, ENHANCED RESPONSE TO ABA; ABI, ABA INSENSITIVE; DOG, DELAY OF GERMINATION; SnRK, SNF1-related protein kinase; ABAR, ABA receptor; GCR, G protein–coupled receptor.

Supreme Court Justice Stewart's definition of obscenity in that it's notoriously hard to define but "[we] know it when [we] see it."

WHAT IS DORMANCY?

Seed dormancy is common in wild plants, where it may ensure the ability of a species to survive natural catastrophes, decrease competition between individuals of the same species, or prevent germination out of season. In contrast, domesticated species have been selected for fast and uniform germination, followed by rapid seedling establishment (termed stand establishment), to achieve good crop yield.

After-ripening: period of dry storage during which dormancy progressively decreases

Stratification: incubation of seeds in moist conditions to break dormancy, usually in cold to simulate overwintering

-omics: global studies of functional aspects of entire genomes, transcriptomes (all expressed transcripts in a tissue), proteomes (all expressed proteins in a tissue), etc.

Seed longevity: duration of seed viability during prolonged storage

ABA: abscisic acid

However, lack of seed dormancy is not desirable because it may cause preharvest sprouting, a serious problem in cereals including rice, wheat, barley, and maize (17), and nondormant mutants can have reduced seed longevity (36).

All types of dormancy impose a delay between seed shedding and germination, but the underlying causes may vary. This variety has been classified in terms of whether germination is inhibited owing to embryonic immaturity or physical or physiological constraints, and whether the controlling structure or substances are embryonic or in the surrounding tissues of the seed, i.e., coat-imposed (reviewed in 50). Whereas primary dormancy is acquired during seed maturation, imbibed after-ripened seeds exposed to unfavorable temperature conditions or lacking adequate light or nitrate may enter a state of secondary dormancy. Furthermore, seeds may undergo seasonal dormancy cycling if conditions are suboptimal, progressively gaining or losing dormancy until they eventually germinate or die. Although physiologically fully dormant seeds cannot germinate regardless of environmental conditions, seeds with intermediate dormancy germinate slowly or under a narrower range of light and temperature conditions (82).

Strictly defined, germination is the initial emergence of the radicle from the seed coat. This process requires that the plant embryo leave the quiescent state, mobilize stored nutrients, overcome the barrier of surrounding tissues, and resume cell elongation, cell division, and development. Dormancy may result from blocks in any of these processes. Because the forms of seed dormancy are defined on the basis of mechanisms of dormancy release, scored in terms of increased germination, it is controversial whether some of the effective signals induce dormancy release or the subsequent germination.

Dormant seeds that exhibit seed coat–imposed dormancy will germinate following damage or removal of the seed coat. Although some species require scarification or fire for dormancy release, and thus appear to show seed coat–imposed dormancy (reviewed in 82), smoke contains dormancy-breaking compounds, including nitrogen oxides (96) and a butenolide (52), suggesting that fire also stimulates germination by biochemical mechanisms.

The precise mechanisms underlying dormancy breaking by after-ripening remain elusive but have been correlated with changes in gene expression, enzyme activity, and hormone accumulation, suggesting that biological processes such as transcription and translation can occur in dry seeds (22, 49, 108). Several dormancy-breaking signals have additive or synergistic effects, suggesting that they might affect key regulators of dormancy similarly. This possibility raises several questions: What are these key regulators; do the different types of physiological dormancy reflect different underlying molecular mechanisms; and do different depths of dormancy reflect quantitative or qualitative differences? This review focuses on the balance between plant hormone signaling pathways regulating dormancy and on the molecular mechanisms by which plants control the induction and release of seed dormancy.

REGULATION OF DORMANCY BY HORMONES AND OTHER SMALL MOLECULES

Dormancy Induction and Maintenance

Potential regulators of dormancy have been identified as substances whose accumulation in seeds correlates with the depth of dormancy, and by genetic analyses of lines with different degrees of dormancy.

Abscisic Acid

Several lines of evidence have established that abscisic acid (ABA) induces dormancy during embryo maturation (reviewed in 48, 50, 51, 97, 127). ABA can inhibit germination and its

accumulation correlates with the onset of dormancy. Dormancy is reduced in seeds that are ABA-deficient owing to mutations, chemical inhibition of biosynthesis, or sequestering of ABA by antibodies expressed in seeds, whereas overexpression of ABA biosynthetic enzymes leads to enhanced dormancy (110, 127). Genetic studies have shown that ABA produced by maternal tissues or supplied exogenously is not sufficient to induce dormancy (50, 51), implying that this is a form of embryo-controlled dormancy dependent on ABA synthesis in the embryo and/or endosperm.

Although ABA decreases to relatively low levels in dry dormant seeds owing to catabolism by specific ABA 8′-hydroxylases encoded by the cytochrome P450 CYP707A family (136), dormancy is maintained by renewed accumulation of ABA following imbibition of dormant seeds (2, 70, 103). Expression and genetic analyses of specific genes encoding key enzymes in ABA metabolism have identified members of the *AtNCED* (9-*cis*-epoxycarotenoid dioxygenase) family (*NCED6* and *NCED9*) that are essential for ABA synthesis in both the embryo and endosperm during dormancy induction (105). Additional studies have identified specific genes correlated with dormancy maintenance [*NCED6*, *NCED9*, and *ZEP* (zeaxanthin epoxidase)] and dormancy release via ABA catabolism (*CYP707A2*) (22, 121). Thus, seed ABA levels and the resulting dormancy are controlled by the combined action of differentially expressed enzymes involved in several steps of both synthesis and catabolism.

Dehydration and Desiccation Tolerance

The vast majority of dormant seeds are "orthodox" in that ABA induces desiccation tolerance late in seed development, permitting their survival for long periods in a dry state. In contrast, "recalcitrant" seeds are not desiccation tolerant and this is correlated with the generation of active oxygen species (AOS) and the occurrence of oxidative damage during dehydration (6, 12), leading to the suggestion that desiccation tolerance depends in part on the ability to scavenge AOS compounds by antioxidant defense systems. Most maturing seeds have increased catalase activity (6, 12) and accumulate glutathione, peroxiredoxins (79, 162), or even storage proteins serving as substrates for oxidative carbonylation (6, 93). Although glutathione production in the embryo appears to be essential for maturation (23) and overexpression of peroxiredoxins confers hypersensitivity to several germination-inhibiting stresses, peroxiredoxin levels do not correlate with depth of dormancy (80).

In addition to effects on cellular oxidation state, desiccation tolerance involves formation of an intracellular "glassy state" due to supersaturation by a combination of sugars and late embryogenesis abundant (LEA) proteins (reviewed in 12). Despite the correlation between LEA proteins and desiccation tolerance, LEA transcript levels do not appear to be correlated with dormancy (8, 49).

NCED: 9-*cis*-epoxycarotenoid dioxygenase

LEAs: late embryogenesis abundant proteins

Seed Coat Components

Genetic variation in structure and/or pigmentation of the seed coat (testa) or surrounding layers, such as the pericarp of grains, leads to altered dormancy and seed longevity in many species (44, 72, 170). The testa both protects the embryo from environmental damage and restricts its growth prior to germination. Mutations affecting pigmentation include disruption of pigment biosynthetic genes, transporters involved in compartmentation of the pigments, and transcription factors that regulate production of the biosynthetic enzymes (reviewed in 106).

Seed coat pigments are generally phenolic compounds such as flavonoids. The pigments accumulated depend on the species; red phlobaphenes are common in grains, isoflavones are common in legumes, and proanthocyanidins and flavonol glycosides are present in *Arabidopsis* (reviewed in 44). The proanthocyanidins are generally present as

GAs: gibberellins

polymers that become cross-linked to wall components by oxidation during seed maturation, resulting in thicker cell layers that provide greater mechanical restraint and reduced permeability to water, gases, and hormones. In addition, as efficient antioxidants, flavonoids can further restrict oxygen availability, thereby inhibiting metabolic processes of after-ripening and germination, e.g., oxidative degradation of ABA. Light sensing both regulates and is regulated by seed pigments. The degree of pigmentation depends on the photoperiod experienced by the maternal plant during seed set and highly pigmented seeds filter light reaching the embryo, although only UV light has been shown to be absorbed by flavonoids (187).

Flavonoids also provide protection from antimicrobial and antiherbivory functions, UV radiation, and possibly oxidative damage. Consequently, breeding programs that attempt to separate characters associated with seed color, dormancy, and longevity suggest that these characteristics may not be separable. For example, mapping of some loci affecting seed color, dormancy, and shattering characteristics of rice shows very tight linkage such that these traits are referred to as a domestication block; some of these traits may be controlled by a single locus (90). However, whereas pigmented seeds are generally more dormant, additional characters such as hormone levels or sensitivity can enhance dormancy of nonpigmented seeds (53, 60, 182).

Dormancy Release and Germination

Dormancy release is accomplished by diverse mechanisms that include complex interactions with the environment mediated by phytohormones and other small molecules, and is believed to select conditions for germination that are most conducive for plant survival. The discussion herein is restricted to the molecular mechanisms of dormancy release by gibberellins, brassinosteroids, ethylene, reactive oxygen species, and nitrogen-containing compounds such as nitrate and nitric oxide (NO).

Gibberellins

Gibberellins [e.g., gibberellic acid (GA)] are a family of 136 tetracyclic diterpenes, a small subset of which are active as plant hormones and known to stimulate seed germination in a wide range of plant species; the predominant active GA depends on the species (reviewed in 172). Gibberellins stimulate germination by inducing hydrolytic enzymes that weaken the barrier tissues such as the endosperm or seed coat, inducing mobilization of seed storage reserves, and stimulating expansion of the embryo (reviewed in 17). GA may also stimulate germination via the transition from embryonic to vegetative development, in part mediated by the chromatin remodeling factor PICKLE (PKL) (81). GA stimulation of this transition is suggested by enhancement of the *pkl* embryonic root phenotype by GA biosynthesis inhibitors, and by GA-stimulated disappearance of the embryonic identity protein FUSCA 3 (FUS3), which positively regulates ABA synthesis and negatively regulates GA synthesis (62, 81).

The role of gibberellins in dormancy release is controversial. Although GA accumulation is associated with dormancy release and/or germination, GA treatment alone does not stimulate germination in all species or in fully dormant *Arabidopsis* seeds (2, 16, 46). A decrease in ABA levels may be required before GA levels and sensitivity can increase (2, 88). However, sensitivity to both GA and light increases as after-ripening progresses in *Arabidopsis* (45) and increased GA levels mediate the dormancy-relieving effect of moist chilling in *Arabidopsis* (188). Light promotes GA synthesis, and light and GA promote the degradation of ABA in imbibing lettuce seeds (175). The accumulation of the GA biosynthetic gene *GA3ox2* (*GIBBERELLIN 3 OXIDASE*) transcript increased 40-fold in after-ripened seeds whereas the GA-deactivating enzyme *GA2ox1* (*GIBBERELLIN 2 OXIDASE*) was expressed at the highest levels in the highly dormant seeds of *Arabidopsis* ecotype Cvi (Cape Verde Islands) (49). Stratification led to

increased expression of the GA biosynthesis genes *GA20ox1* (*GIBBERELLIN 20 OXIDASE*), *GA20ox2*, and *GA3ox1* and decreased expression of the GA catabolic gene *GA2ox2* (188). Taken together, GA may not trigger the onset of after-ripening, but it may be necessary though not sufficient for seed dormancy release and germination.

GA is critical for seed germination in the dicots *Arabidopsis* and tomato because mutants with defects in genes encoding GA biosynthetic enzymes fail to germinate (122, 163 and references therein). Nongermination of GA mutants is relieved by exogenous application of GA, mutations affecting ABA biosynthesis or signaling, or removal of the mechanical restraint of the testa and endosperm. Thus, GA mutants fit the classical definition of dormant, suggesting that GA synthesis is one step required to release dormancy. A dramatic increase in bioactive GA_4 level is observed just before initial radicle protrusion, consistent with the timing of GA-responsive gene induction (33, 131). GA synthesis depends on the movement of precursors within the embryo: The step catalyzed by the *Arabidopsis GA1* locus occurs in provascular tissue, but subsequent steps occur in cortex and endodermis (131). Unlike dicots, cereals do not require GA for germination. For example, mutations in the rice GA receptor *OsGID1* (*Oryza sativa GIBBERELLIN-INSENSITIVE DWARF 1*) display decreased α-amylase production, but allow germination (177), whereas loss of all three *Arabidopsis GID1* homologs blocks germination (185). This discrepancy may reflect differences in the structure, pigmentation, and composition of these seeds, or as yet undetected sources of redundancy in rice GA signaling.

Brassinosteriods

Brassinosteriods (BRs) are plant steroid hormones involved in stem elongation and leaf unfurling (reviewed in 38) that also promote seed germination. Although BR biosynthesis and signaling mutants germinate well, they are more sensitive than wild-type to ABA inhibition of seed germination, suggesting that these mutations decrease germination potential (39, 164). In *Arabidopsis*, epibrassinolide (EBR) and brassinolide (BL) application overcomes nongermination of GA biosynthetic and *sleepy 1* (*sly1*) GA signaling mutants (164), but BR and GA stimulate tobacco seed germination by different mechanisms (107). Although both BRs and GA promote endosperm rupture of nonphotodormant tobacco seeds imbibed in the dark, only GA induces the class I β-1,3 glucanase (bGLU I) activity that is critical for endosperm rupture of photodormant tobacco seeds (107). Furthermore, microarray analysis shows that both BR and GA induce the expression of cell elongation-associated genes, but these hormones induce the expression of distinct expansin family members (65). Thus, BRs may promote seed germination by directly enhancing the embryo growth potential in a GA-independent manner (107).

Ethylene

Ethylene promotes dormancy breaking through interactions with ABA signaling. Seed of *ethylene resistant* (*etr*) *1* receptor mutants and *ethylene insensitive* (*ein*) *2/enhanced response to aba* (*era*) *3* mutants display increased dormancy correlated with increased sensitivity to ABA in seed germination and increased ABA synthesis (9, 33, 64). In contrast, *constitutive triple response* (*ctr*) *1* mutations and treatment of wild-type seeds with the ethylene precursor 1-aminocyclopropane-1-carboxylic acid (ACC) result in decreased sensitivity to ABA (64). The *etr1-2* mutant also overaccumulates GA, suggesting that the increased dormancy due to ABA overaccumulation is partly compensated for by increased GA accumulation. Thus, ethylene stimulation of seed germination may occur via antagonism of ABA signaling.

GA(n)ox: gibberellin (n) oxidase

GID: gibberellin-insensitive dwarf

Germination potential: likelihood of successful germination due to combination of decreased restrictive properties of tissues surrounding embryo or radicle tip, and increased ability of embryo to produce sufficient turgor to push through these tissues

Nitric Oxide, Nitrate, and Active Oxygen Species

Many nitrogen-containing compounds, including NO gas, nitrite (NO_2^-), nitrate (NO_3^-), nitrogen dioxide, ammonium, azide, and cyanide, promote dormancy release and seed germination in many species, possibly as a means of sensing soil N availability (reviewed in 15). Even nitrate provided during seed development via the maternal plant leads to lower dormancy (1). Interconversion of these compounds and the lack of specificity for NO donors, scavengers, and detectors complicates analysis of their mechanism(s), but they may act as sources of NO or by similar mechanisms as NO (14, 15).

Enzymatic NO production in plants occurs primarily via nitrate reductase (NR) from nitrite and nitrate (reviewed in 15) and possibly a plant-specific nitric oxide synthase whose identity is still ambiguous (42). However, although *Arabidopsis NR1* expression is correlated with dormancy-breaking conditions (49), seeds of NR-deficient plants accumulate nitrate and have reduced dormancy (1). NO can also be produced by the oxidizing environments of glyoxysomes engaged in lipid catabolism (41), and nonenzymatic NO production may occur in the apoplast or soil where nitrite can be converted to NO at low pH (13).

These nitrogen-containing compounds may promote seed germination via effects on metabolism, oxidation state, or signaling (15). Nitrate and dormancy-relieving conditions induce the expression of some genes that encode enzymes in the pentose phosphate pathway, which may provide essential nutrients for germination (49). NO, nitrite, and nitrate may stimulate the pentose phosphate pathway, and therefore germination, by increasing oxidation of NADPH to $NADP^+$, a limiting electron acceptor. NO inhibition of catalase may lead to increased accumulation of H_2O_2 from the β-oxidation of stored fatty acids in seeds. Accumulated H_2O_2 may stimulate germination by acting as a substrate for peroxidases, leading to oxidation of NADPH to $NADP^+$ or by causing breakdown of ABA (reviewed in 6). Nitrate can also modify hormone levels by inducing the expression of enzymes that catalyze ABA deactivation (*CYP707A2*) and GA biosynthesis (*GA3ox1*) (49). In *Arabidopsis*, NO may stimulate germination by causing vacuolation and cell wall weakening of the aleurone layer (14).

PROTEINS REGULATING DORMANCY INDUCTION AND MAINTENANCE

Genetic studies have led to the identification of numerous loci that encode dormancy-regulating proteins (reviewed in 11). Mutations in many of these loci have pleiotropic effects, indicating that the specific events leading to the dormant state are only a subset of the downstream effects of these regulators. The proteins encoded by these loci include standard signaling molecules such as G proteins and G protein–coupled receptors, transcriptional regulators, and protein phosphatases or kinases regulating activity of the transcription factors via effects on phosphorylation state. Additional proteins regulate transcription factor activity, stability, or localization via other modifications. Numerous other loci display altered ABA or stress sensitivity at germination, but we focus on the relatively few that affect dormancy, as reflected in requirements for stratification or after-ripening.

Transcriptional and Posttranscriptional Regulators

A variety of mutants with major defects in seed maturation, e.g., the *Arabidopsis leafy cotyledon* class (*lec1*, *lec2 and fus3*) and *ABA-insensitive* (*abi*) *3* mutants, result in nondormancy (reviewed in 50, 51). All these loci encode transcriptional regulators, so it is tempting to suggest that some of their regulatory targets are essential to dormancy. However, because they constitute part of a regulatory hierarchy

including both cross- and autoregulation (78, 173), many of their effects on maturation are likely indirect. Furthermore, because these highly pleiotropic mutants do not produce viable desiccation-tolerant seed, many of the genes misexpressed in these mutants probably reflect the failure to reach the stage of development when cues inducing dormancy begin. In this respect, the weaker *abi3* alleles provide a more promising approach toward a molecular fingerprint for dormancy because these mutants produce viable dry seeds yet are still nondormant. Although two additional classes of transcription factors, encoded by the *ABI4* and *ABI5* loci, regulate germination in response to ABA and stresses (51), they do not appear to affect dormancy significantly.

Further evidence suggesting the importance of *ABI*-like genes in dormancy regulation comes from studies of their orthologs in other dicots, cereals, and even conifers (50, 97). Although most crops have reduced dormancy compared with their wild relatives, specific cereal cultivars exhibit different degrees of dormancy, and comparison of these lines reveals some positive correlations between the depth of dormancy and high expression of the *Vp1* (*Viviparous*) genes (*ABI3* orthologs) from wild oats (95) and wheat (126), but not sorghum (26). However, wheat and barley *Vp1* genes appear to be mostly misspliced even in ancestral varieties of wheat, such that little functional protein is produced, leaving these species predisposed to preharvest sprouting (117). *ABI5* homologs are also more highly expressed in more dormant cultivars of sorghum (154). Despite these correlations, demonstration of a significant role in controlling seed dormancy would require functional evidence, e.g., coincidence with dormancy quantitative trait loci (QTL) between these cultivars or alteration of dormancy by modified expression of either gene in otherwise isogenic lines. Although transcriptional profiling studies of dormancy cycling in the highly dormant Cvi ecotype of *Arabidopsis* show a preponderance of ABA-responsive element (ABRE)-containing genes correlated with dormancy, *Arabidopsis* differs from cereals in that none of the *ABI* transcription factors are included in this set and *ABI4* actually appears in the after-ripening set (8, 22, 49). Only the basic leucine zipper domain (bZIP) *ABA-RESPONSIVE ELEMENT BINDING PROTEIN* (*AREB*) *2/ABRE BINDING FACTOR* (*ABF*) *4* is enriched in dormant states (22), but mutant analyses have not tested its role in dormancy.

Unlike the highly seed-enriched expression of these *ABI* loci, the *DOF* (*DNA-binding with one zinc finger*) *AFFECTING GERMINATION* (*DAG*) *1* and *DAG2* loci encode highly similar transcription factors that are both expressed exclusively in maternal vascular tissue and have opposing effects on seed germination potential (75, 139). Double mutant analyses showed that DAG1 acts epistatically, leading to the suggestion that DAG1 activates maternal genes that promote dormancy and DAG2 represses this action, by binding to either the same *cis*-elements or the DAG1 factor. Although the specific targets of the DAG transcription factors have not yet been identified, the products of their target genes could modify characteristics of either the embryo or surrounding tissues. Whereas the DAG genes appear to be coregulated during normal growth, environmentally induced changes in their relative expression or protein accumulation might regulate seed sensitivity to germination-inducing signals such as light and GAs.

Recent studies have also provided genetic evidence for transcriptional control via effects on chromatin structure. The *REDUCED DORMANCY* (*RDO*) *4* locus (142) encodes a C3HC4 RING finger protein with homology to histone-modifying enzymes of other species (113). The mutant fails to monoubiquitinate histone H2B and the locus was consequently renamed *HISTONE MONOUBIQUITINATION* (*HUB1*). Defects in a close homolog, designated *HUB2*, also result in decreased dormancy. The single and double mutants have similar phenotypes, leading to the suggestion that these proteins

ABI: abscisic acid insensitive

Quantitative trait loci (QTL): genes that contribute quantitative variation to complex traits regulated by multiple unlinked loci

RDO: reduced dormancy

function as part of a heterotetramer, required for monoubiquitination, that is associated with actively transcribed genes. Consistent with this emphasis on transcription elongation, the *RDO2* locus encodes the transcription elongation factor S II (TFIIS) (63). TFIIS factors can enhance elongation by promoting cleavage and reactivation of nascent transcripts whose elongation is blocked under difficult conditions in yeast and mammalian cells (186). Similar blocks may occur in a drying or dry seed. However, how this control mechanism specifically targets histone modification and enhanced transcription to dormancy-related genes remains unclear.

Dormancy-related gene expression is also regulated posttranscriptionally. Early studies of potential dormancy marker genes demonstrated that transcript stability was enhanced far more than transcription rate in dormant versus after-ripened seeds (109), and transcriptional profiling demonstrated the importance of translational control during after-ripening of *Nicotiana plumbaginifolia* (18). Researchers have identified mutations resulting in increased dormancy and/or ABA hypersensitivity at germination that disrupt RNA capping [*ABA-hypersensitive* (*abh*) *1*]; miRNA biosynthesis [*hyponastic leaves* (*hyl*) *1*]; mRNA splicing, export, and degradation [*supersensitive to ABA and drought* (*sad*) *1*]; and degradation of polyadenylated RNA [*ABA hypersensitive germination* (*abg*) *2*] (reviewed in 101, 128). Although the hypersensitivity of the mutants implies that these proteins are negative regulators of the ABA response, some of these proteins are induced by ABA or stresses (128), and therefore may participate in a negative feedback loop.

Protein Phosphatases and Kinases

Nondormant *Arabidopsis abi* mutants also include two dominant mutations in the *ABI1* and *ABI2* loci (99), which encode closely related members of the A group of the protein phosphatase 2C (PP2C) family (157). Significantly, although these mutants have greatly reduced phosphatase activity, knockout mutations in these loci result in the opposite phenotype: weak hypersensitivity to ABA and hyperdormancy (119). Recent studies of this clade of PP2Cs have demonstrated that knockout/knockdown mutants in several family members result in increased dormancy, which is most pronounced for the family members that are most highly expressed at seed maturity, i.e., *abg1* and *abg3*, or for double mutants (129, 191), reflecting some redundancy. A homologous PP2C from beech also negatively regulates dormancy when transferred into *Arabidopsis* (69), but one family member appears to act as a positive regulator of ABA response, mediated in part by reduction of GA levels (153). Pernas and coworkers (146) recently reported a similar ABA-hypersensitive hyperdormant phenotype for a mutant with a disrupted PP2A catalytic subunit. Expression of many of these protein phosphatases is induced by ABA, providing a negative feedback mechanism to attenuate ABA response. However, other PP2As either positively regulate (102) or have no effect on ABA response (146).

Each of these PP2Cs probably has numerous substrates, but relatively few have been identified (35). These substrates include transcription factors implicated in the drought/stress response (83), protein kinases (190), calcineurin B–like calcium sensors (77), a glutathione peroxidase (120), and fibrillin, a stress-induced photoprotective plastid protein (189). The dominant negative mutant PP2Cs fail to interact with protein kinases of the SALT OVERLY SENSITIVE (SOS) 2 family, but the mechanistic significance of this interaction remains unclear (135). The function of at least some of these PP2Cs is modified by phosphatidic acid binding (35) or H_2O_2 exposure (118). The latter provides a mechanism by which ROS production in imbibed seeds can modify transcription profiles (118). Although the specific interactions and mechanisms have not been demonstrated, this hypothesis is consistent with the recent identification of dozens of genes whose

expression depends on AHG1 and/or AHG3 (129).

The activity of many of the bZIP factors is controlled by their phosphorylation states. A mitogen activated protein (MAP) kinase cascade activates ABI5 (115), possibly indirectly. Members of the calcium-dependent protein kinase (CDPK) and SNF1-related protein kinase (SnRK) families of serine-threonine protein kinases interact directly with multiple members of the AREB/ABF/ABI5-class of bZIPs in vitro, although in vivo interactions have not been demonstrated for most (29, 34, 57, 59, 98). Several of these kinases and transcription factors are differentially expressed, and the specific interactions may be partially dictated by which proteins are coexpressed. Phosphorylation of these bZIPs is correlated with their activation and, consistent with this, the SnRK2.2 and SnRK2.3 family kinases are redundant positive regulators of dormancy, ABA sensitivity of germination, and some gene expression (57). In contrast, a nonorthologous family member from rice, OSRK1/SAPK6, shows reduced sensitivity to ABA at germination (29). Additional kinases are also correlated with dormancy, but their specific substrates are unknown (19, 92). One of the best known interactions that affects germination potential is phosphorylation of the wheat bZIP factor TaABF by the SnRK PKABA1 (94), leading to both repression of GA-induced genes, including the transcription factor GA-Myb, and activation of ABA-induced genes (67, 68).

Regulation of Protein Stability and Localization

The function of some ABI transcriptional regulators is also controlled by stabilization against proteasomal degradation. ABI3 expression is high in seeds and quickly turned off in most tissues following germination (21, 140, 173), but is also targeted for destruction by the E3 ubiquitin ligase ABI3 INTERACTING PROTEIN (AIP) 2, which is expressed throughout development (195). Whether accumulation of AIP2 protein reflects AIP2 transcript levels remains unclear, but if so AIP levels are either insufficient to induce ABI3 destruction prior to germination or ABI3 synthesis exceeds destruction at this stage. However, even strong overexpression of AIP2 has relatively mild effects on ABA sensitivity, resulting in a similar phenotype to the weak allele *abi3-1*.

Although the specific ubiquitin ligase(s) involved in tagging ABI5 have not yet been definitively identified, mutations in four loci alter ABI5 stability and consequently germination kinetics: *RPN10* (161), *KEEP ON GOING* (*KEG*) (166) *SALT- AND DROUGHT-INDUCIBLE RING FINGER 1* (*SDIR1*) (196), and *ABI FIVE BINDING PROTEIN* (*AFP*) (114). Despite their effects on ABA sensitivity of germination, all these mutants are similar to *abi5* in that none have significant effects on dormancy.

Lipid modification of proteins by farnesylation can promote membrane attachment, protein stabilization, or protein-protein interactions in plants (61). *ERA1*, which encodes the β subunit of protein farnesylase, was first identified in a screen for enhanced response to ABA (hence the name, *ERA*) at germination (43), and was subsequently shown by epistasis analyses to act downstream of the ABI phosphatases but upstream of the ABI transcription factors in ABA signaling (21). *era1* mutants are highly pleiotropic, displaying hypersensitivity to ABA for multiple responses including dormancy, and defects in meristem regulation, which probably reflect ERA1's role in farnesylating multiple targets. Searches for C-terminal farnesylation targets have identified 119 potential substrates (61) including G protein γ subunits (192), suggesting ERA1 might control G protein responsiveness.

Abscisic Acid Perception

Three distinct ABA receptors have been identified in the last two years by biochemical or reverse genetic approaches (84): an RNA

binding protein that regulates flowering, FLOWERING TIME CONTROL A (FCA) (152); a subunit of Mg chelatase involved in chlorophyll biosynthesis and plastid-to-nuclear retrograde communication, ABAR/CHLH/GENOMES UNCOUPLED 5 (GUN5) (158); and a G protein–coupled receptor (GCR2) (112). Only the latter two appear to regulate seed responses to ABA, and only mutations affecting ABAR have severe defects in seed development. *ABAR* knockout lines are embryonic lethal plants that lack storage proteins and lipids, but can't be readily analyzed biochemically owing to a lack of homozygotes. *ABAR* knockdown lines display ABA-resistant germination and reduced expression of *LEA* genes and the *ABI* and *MYB* transcription factors that positively regulate ABA response, but overexpress the *ABI* PP2Cs that function as negative regulators of ABA response. In this respect, the ABAR/CHLH/GUN5 receptor appears to play a central role in signaling that mediates ABA responses of seeds, but its effects on dormancy per se have not been characterized.

G protein involvement in ABA regulation of germination was first demonstrated genetically by the observation that a mutant in the sole G protein α subunit (*GPA1*) of *Arabidopsis* was hypersensitive to ABA for inhibition of germination (179), implying that GPA1 inhibits this ABA response despite the fact that it appears to promote stomatal response to ABA (138). GPA1 was subsequently shown to interact with a putative G protein–coupled receptor (GCR1) that also inhibited ABA response (40), but whose ligand was unknown. The G protein β subunit also negatively regulates ABA inhibition of germination (138). Recently, a complete signal transduction chain comprised of GCR1, GPA1, Pirin1 (an iron-containing member of the cupin superfamily), and a CCAAT box binding protein complex (nuclear factor Y) including LEC1 was described as mediating both ABA inhibition of germination and the slight induction of Lhcb (light harvesting complex/chlorophyll binding protein) expression by either blue light or ABA (184). However, whether this also reflected a change in dormancy level remained unclear. Liu and coworkers (112) recently found a second G protein–coupled receptor (GCR2) that interacts with GPA1, binds ABA, and functions as a positive regulator of ABA signaling. Mutations in GCR2 have a surprisingly mild phenotype for a receptor, leading to the suggestion that it may act redundantly with other G protein–coupled receptors present in plants. Both GCR1 and GCR2 appear to be regulators of ABA response whose interactions with the G protein complex either promote (GCR1) or repress (GCR2) its inhibitory effects on germination.

PROTEINS REGULATING DORMANCY RELEASE

Transcriptional Regulation of Gibberellin Biosynthesis by SPT, PIL5, BME3

GA synthesis is regulated developmentally and in response to environmental signals. For example, the GATA zinc finger protein BLUE MICROPYLAR END 3 (BME3) promotes expression of the GA biosynthesis genes *GA3ox1* and *GA20ox3* in the micropylar end of the seed, which stimulates breakdown of barrier tissues prior to radicle emergence; the importance of these GA biosynthesis genes is demonstrated by the delayed seed germination of *bme3* mutants (111). Changes in expression of GA biosynthetic genes in response to cold imbibition and light are regulated by two basic helix-loop-helix (bHLH) transcription factors, SPATULA (SPT) and PHYTOCHROME-INTERACTING FACTOR 3–LIKE 5 (PIL5) (132–134, 144). SPT inhibits expression of two *GA3ox* genes (*GA3ox1* and *GA3ox2*) during seed imbibition in the cold. The reduced dormancy of *spt* mutants is more pronounced in the light, suggesting SPT-mediated cross talk between light and cold regulation of dormancy. PIL5 prevents seed germination in the dark at low temperature by repressing expression

of *GA3ox1* and *GA3ox2* genes, whereas it induces expression of the *GA2ox2* catabolic gene during imbibition in the dark (134, 144). PIL5 protein disappears in the light, apparently owing to phytochrome-stimulated degradation by the 26S proteasome (134). This suggests that stratification promotes germination by increasing the potential for bioactive GA accumulation. PIL5 also appears to repress GA responses by stimulating expression of the DELLA repressors of GA response, *REPRESSOR OF GA1-3* (*RGA*) and *GA-INSENSITIVE* (*GAI*), through direct binding to their promoters (133), suggesting that DELLA proteins may be involved in environmental control of seed dormancy.

The DELLA Repressors of Gibberellin Signaling and Seed Germination

DELLA proteins are negative regulators of diverse GA responses (reviewed in 169); GA stimulates germination by causing the disappearance of DELLA proteins (5, 176). The DELLA family is defined by an N-terminal DELLA domain required for GA regulation and a C-terminal GRAS [GAI (GA-INSENSITIVE), RGA (REPRESSOR OF GA 1–3), and SCARECROW] domain required for function of these putative transcription factors (145, 150). Mutations in the DELLA domain result in gain-of-function GA signaling phenotypes resembling GA deficiency, whereas loss-of-function mutations in the GRAS domain typically result in an enhanced or constitutive GA response (reviewed in 172). Although a single DELLA gene exists in rice and barley, the *Arabidopsis* genome contains five DELLA genes.

RGL (*RGA-LIKE*) *2* appears to be the major DELLA protein regulating seed germination because loss of *RGL2* function partly restores germination in GA-deficient seeds (104, 176), but single mutations in the other family members are not sufficient. However, combinations of *RGA*, *GAI*, and *RGL1* can enhance this seed germination rescue (25). *RGL2* mRNA and protein levels increase during cold imbibition, then decrease rapidly as germination approaches after transfer to 23°C (5, 7, 104). RGL2 protein disappears within 5 h of GA application to imbibing GA-deficient (*ga1-3)* seeds, long before germination occurs (176). This pattern of expression suggests that RGL2 is a potent repressor of seed germination whose repression is lifted by GA-stimulated DELLA degradation. Conversely, ABA stabilizes at least one DELLA, an RGA::GFP protein fusion (143). Recent evidence suggests that DELLA proteins promote seed dormancy through inhibition of cotyledon expansion prior to germination (143), possibly by repressing expression of hydrolytic and wall-modifying enzymes.

GA relieves DELLA repression of seed germination through proteolysis by the 26S proteasome, triggered via polyubiquitination by the $SCF^{SLY1/GID2}$ (Skp1-Cdc53/CUL-1/F-box protein, in this case SLY1/GID2) E3 ubiquitin ligase (5 and references therein). *Arabidopsis* SLY1 and rice GA-INSENSITIVE DWARF 2 (GID2) are homologous F-box subunits of an SCF E3 ubiquitin ligase that ubiquitinates DELLA proteins, thereby targeting them for destruction in the presence of GA (47, 56, 116, 156). The GA signal is received by the *GID1* GA receptor(s) (71, 87, 124, 177, 185). The GID1 protein undergoes a GA-dependent interaction with DELLA proteins, which promotes the interaction of DELLA with the F-box protein (71, 178).

DELLA function may be regulated by mechanisms other than destruction. Although rescue of *sly1* dormancy by *RGL2* mutations suggests that *sly1* dormancy is due to RGL2 overaccumulation, weak alleles and long after-ripened *sly1* seeds are able to germinate, although they retain high levels of RGL2 and RGA protein (5). This suggests that the RGL2 protein that accumulates in *sly1* mutant seeds is not fully active as a repressor of GA signaling. RGL2 may be subject to additional posttranslational regulation or germination may be regulated by parallel

pathways. RGL2 is subject to phosphorylation, and phosphomimic mutations stabilize the RGL2 protein (85, 86). Mutations in the *Arabidopsis* O-linked β-N-acetylglucosamine (O-GlcNAc) transferase SPINDLY (SPY) lead to GA-independent germination and increased stability of DELLA proteins, suggesting that O-Glc-NAc modification may both activate DELLAs and stimulate their turnover (89, 159, 160). An alternative pathway mediating GA effects on dormancy release is suggested by microarray analyses demonstrating DELLA-independent GA-induced gene expression during stratification (24). Because *sly1* mutants are ABA hypersensitive, ABA catabolism may constitute one parallel pathway (167).

Role of Reserve Mobilizing Enzymes in Germination Progression

Embryos must mobilize carbon and energy sources to germinate and grow. In dicots, lipid reserves consist predominantly of triacylglycerol (TAG). During germination, TAG is hydrolyzed by lipases, and the resulting fatty acids pass into the peroxisome where β-oxidation and the glyoxylate cycle provide energy and carbon skeletons for postgerminative growth (66). Genetic studies emphasize the importance of TAG breakdown via lipid catabolism in regulating dormancy release and/or germination: Mutants defective in lipid catabolism and β-oxidation [*kat2* (ketoacyl-CoA thiolase-2) and *acx1acx2* (acyl-CoA oxidase) or *csy2csy3* (citrate synthase) double mutants] (54, 147, 148) or import of very long chain fatty acids into peroxisomes (*cts*, *COMATOSE*) (55, 155, 171) are deeply dormant. Although *cts* seed germination is not rescued by stratification, after-ripening, GA treatment, or *rgl2* mutations (28), removal of the seed coat rescues germination and *transparent testa glabrous* (*ttg*) mutants rescue germination upon after-ripening. However, loss of two long-chain acyl-CoA synthetases (LACS) in the *lacs6 lacs7* double mutant causes failure in TAG breakdown without blocking germination (54, 58), suggesting that breakdown of long-chain acyl-CoA esters may not be required for seed germination. After-ripening of wild-type Landsberg *erecta* (L*er*) and *cts* mutant seeds results in similar changes in gene expression (28, 54, 55), suggesting that the failure of β-oxidation in *cts* mutants may block production of a germination-promoting factor or breakdown of a germination-inhibiting factor (55).

GENETIC ANALYSES OF NATURAL VARIATION THAT AFFECTS DORMANCY

The genetic approaches described above identify regulators with either relatively minor effects on dormancy, in some cases due to redundancy, or those with such dramatic pleiotropic effects that loss of function would not be sustained in wild populations. Although some of these are candidate loci to explain natural variation resulting from more subtle changes in function or expression, studies of natural variation offer an opportunity to identify new regulators with ecological relevance in wild populations. This strategy identifies multiple QTL and their relative contribution to dormancy based on mapping and cosegregation analysis of progeny from crosses between accessions differing in dormancy characteristics. This approach has been applied to *Arabidopsis* (3, 37, 180) and a variety of crops (4, 60, 73, 76, 137, 170).

Many of the dormancy QTLs identified in *Arabidopsis* using multiple recombinant inbred lines (RILs) populations colocalize. Fourteen QTLs identified from a cross of weakly dormant L*er* and Columbia (Col) include overlaps with four from a cross between L*er* and stress-tolerant Shakdara (Sha) and seven from a cross between L*er* and the highly dormant Cvi (3, 37, 180), indicating surprisingly few loci account for much natural variation. Genetic interactions were observed between some QTLs, and several QTLs altered additional seed characteristics including longevity, sugar content, and stress tolerance.

The L*er*/Cvi QTLs were introgressed into the L*er* background to permit fine-mapping of individual *DELAY OF GERMINATION* (*DOG*) loci in near-isogenic lines (3). Some of these have major effects on dormancy, whereas others can be scored only under certain conditions (11).

Although several of the *Arabidopsis DOG* loci map near known seed trait loci, none have yet been colocalized and only *DOG1* has been cloned (10). This gene is expressed only in seeds, appears to be essential and specific for dormancy, and requires ABA for its action, but encodes four alternatively spliced transcripts that encode proteins of unknown function, so its mechanism of action is still unknown. Comparison of the *DOG1* locus among accessions of varying dormancy suggests that the major differences are due to altered expression levels, not specific functional differences in the coding sequence. Although four close homologs are tightly clustered on chromosome 4, none have any obvious effect on dormancy. However, only one homolog (*DOG1L4*) shows a similar expression pattern to *DOG1* (22, 123, 174), and its contributions might be masked by redundancy.

QTL mapping has been used extensively in studies of dormancy and preharvest sprouting in cereal grains. The Gramene QTL Database (**http://www.gramene.org/qtl/index.html**) lists 164 seed dormancy QTLs (qSD, qDOR, or Sdr loci) for rice, representing at least 30 loci identified in a variety of crosses among cultivars or with wild relatives (74, 91, 183). Although several of these are closely linked to loci affecting weedy characteristics such as shattering, awn length, black hull color, and red pericarp color, *qSD12* appeared unlinked and therefore presents a potentially valuable target for modifying dormancy without associated deleterious effects (74). However, no molecular information is yet available for this gene. *Sdr4* was recently cloned and analysis of insertion lines in an *Arabidopsis* homolog indicates that this gene's function in germination is conserved across monocots and dicots (168).

Additional QTLs in barley, wheat, and sorghum have been fine mapped for identification of linked markers for breeding (100) and testing for cosegregation with candidate regulators such as the orthologs of ABA signaling genes (26, 125). Although expression levels of known ABA response loci are sometimes correlated with dormancy state (95, 117, 126), no base pair changes in ABA signaling genes have yet been shown to cosegregate with the dormancy QTLs. Recent studies have tested whether the same loci regulate dormancy in cultivated and wild barley, as a means of possibly identifying loci in the wild relatives that have been lost during domestication (181). However, the molecular identities of the QTLs have not yet been identified.

DOG: delay of germination

-OMICS APPROACHES

Attempts to provide molecular fingerprints for dormancy and germination have used transcriptional profiling to compare seeds exposed to conditions resulting in different states of dormancy (18, 22, 49) and to follow the progression of germination in seeds with varying germination potential (24, 27, 28, 123, 129, 131, 149, 194). These studies have identified gene classes that are enriched in dormant versus nondormant seeds, as well as classes that are variably expressed in both states (22, 49). They have also shown that expression of these classes is more tightly correlated with the depth of dormancy than environmental conditions, suggesting that the diverse signals regulating dormancy are integrated through some common mechanisms. Profiling of germinating seeds has shown that: (*a*) major changes in gene expression occur between 1 and 3 h after imbibition of *Arabidopsis* seeds (149); (*b*) genes that are highly expressed in seeds and rapidly downregulated and/or classified as GA-repressed are enriched for ABREs in their promoters (123, 131); and (*c*) only half of the GA-regulated genes are regulated by the DELLA class repressors and these include a significant number of genes involved in responses to ABA, auxin, or

ethylene (24). Surprisingly, although DELLA proteins repress GA-inducible gene expression, direct targets identified by chromatin immunoprecipitation experiments with a stabilized DELLA protein are mostly upregulated, suggesting that DELLA repression is mediated by the induction of downstream repressors (193).

Most dormancy-enriched transcripts are present in both the dry and imbibed states, and overlap significantly with ABA- and stress-regulated transcriptomes (22). Specifically, gene expression in dormant *Arabidopsis* seeds is enriched for those involved in ABA synthesis, GA catabolism, and stress response [e.g., LEAs and small heat shock proteins (sHSPs)], and repressed for translation capacity. Profiling by cDNA-amplified fragment length polymorphism (AFLP) analysis in *N. plumbaginifolia* provided a more limited view than microarray analyses, but also showed a correlation between dormancy, ABA synthesis, repression of protein synthesis, and enhanced cell rescue/defense and transport facilitation (18). The *Arabidopsis* studies also identified 30 genes that show higher expression in all dormant states than in afterripened states, making these genes strong candidate regulators of dormancy (49). These candidates include protein kinases and phosphatases, transcription factors, and six genes of unknown function, including *DOG1*.

Despite these similarities among dormant states, the transcriptome changes significantly in seeds transitioning from primary to secondary dormancy. Genes of the metabolism and energy group showed particularly high variability, with 84 genes specific to distinct dormant states. Several transcription factors and regulators of the cell cycle and DNA processing also show increases in expression during secondary dormancy (22).

Both dormant and nondormant states differentially express many genes in the metabolism and transcription regulation classes. The state-specific expression of select transcription factors, histones, and histone-modifying enzymes suggests that a global change in chromatin structure may be associated with the dissimilar expression programs of dormant and nondormant seeds (22).

Classes of genes correlated with nondormancy include those involved in cell organization and biogenesis, proteolysis (essential for reserve mobilization), and many protein synthesis factors, such as ribosomal proteins and initiation and elongation factors (18, 22), consistent with the essential role of translation in radicle emergence during germination (151). Changes in hormone metabolism are also correlated with increased expression of specific family members required for ABA catabolism (CYP707A2) and GA synthesis (GA3ox2) (22).

Radicle emergence requires both decreased mechanical resistance of the covering tissues and increased internal force from embryo expansion. Tomato germination is correlated with GA-induced mRNA expression of enzymes involved in breakdown of the endosperm cap at the micropylar end, including the germination-specific endo-β-mannanase gene *LeMAN2* and the xyloglucan endotransglycosylase/hydrolase gene *LeXET4* (31, 130, 165). During *Arabidopsis* germination, GA induces the expression of genes involved in the induction of cell division and cell wall expansion and modification, such as xyloglucan endotransferase/hydrolases (XTH), expansins, and pectin methylesterases (131). Expansins and XTH enzymes are believed to stimulate cell elongation by loosening cell walls. *AtXTH5* (131) and *LeEXP8* (30) are both expressed prior to germination in the cortex of the embryonic axis, which suggests that they may promote cell wall expansion in the embryonic axis leading to radicle emergence. Similarly, *Arabidopsis* genes involved in cell wall modification are enriched in afterripened states compared with dormant seeds (22).

Partial release of dormancy, by short afterripening and imbibition under conditions that do not allow germination, affects expression of the same gene groups as full release, but the extent of upregulation and downregulation is

intermediate between the levels in dormant and nondormant seeds (49), showing that the regulation of dormancy is not a simple on/off switch. Further evidence for this continuum is provided by comparisons of fresh versus after-ripened seeds, which indicated that even nondormant mutants (due to ABA deficiency or insensitivity) undergo after-ripening, reflected in changes in levels of thousands of transcripts (27). However, this study compared transcripts in 24 h imbibed seeds, so whether the observed changes truly reflect after-ripening or the rapid changes occurring upon imbibition is unclear.

In contrast to the comprehensive datasets available with microarrays, proteomic studies characterize only the most abundant proteins that differ in dormant versus nondormant seeds, i.e., only ~5% as many gene products as assayed by microarrays. However, proteomic studies reflect posttranscriptional effects and may give a more accurate view of cellular metabolic and developmental potential. During dormancy breaking in beech seeds, which requires three months of cold stratification, the translation elongation factor Tu is upregulated, as are several chaperones and energy/metabolism-related proteins. In contrast, delay of dormancy release by ABA treatment is associated with the downregulation of specific translation elongation and initiation factors (141), consistent with transcriptome studies suggesting that dormancy regulates translation capacity. However, measurements of ^{35}S-methionine incorporation indicate that protein translation occurs at similar rates in dormant and nondormant imbibed *Arabidopsis* Cvi seeds, but the identities of the de novo synthesized proteins differ significantly (32). This study also showed that although ABA prevents germination in nondormant seeds, it does not inhibit protein synthesis. Furthermore, the proteomes of truly dormant and ABA-treated nondormant seeds are significantly different, consistent with recent comparisons showing that transcriptomes of ABA-treated after-ripened seeds are more similar to those of untreated after-ripened seeds than those of dormant seeds (27). Finally, only one of the differentially regulated genes identified in *Arabidopsis* Cvi transcriptome studies showed comparable differential expression at the protein level, leaving open the possibility that (post) translational regulation plays a key role in dormancy and dormancy release.

SUMMARY POINTS

1. Potential "dormancy genes" have been identified through correlations between dormancy state and expression levels or gene function, based on mutant or quantitative trait loci (QTL) analysis. Although some potential regulators correlate in both expression and function [e.g., DELAY OF GERMINATION 1 (DOG1)], many "dormancy genes" identified by correlation are not required for dormancy per se (e.g., stress-induced genes) and some dormancy-regulating genes are not expressed in seeds [e.g., DOF (DNA-binding with one zinc finger) AFFECTING GERMINATION (DAG) 1 and 2]. Furthermore, correlations in expression are observed for factors that antagonize each other as well as for those that promote the same process.
2. QTL mapping has identified at least a dozen loci in each species analyzed that contribute to natural variation in dormancy. Classical and reverse genetic studies have identified many more loci that can modify dormancy or hormone sensitivity affecting germination.
3. Abscisic acid (ABA) is the major hormone involved in induction and maintenance of dormancy, but not all mediators of ABA response control dormancy and some

dormancy regulation is ABA-independent, e.g., that due to testa structure or pigmentation. Furthermore, ABA inhibition of germination in nondormant seeds does not phenocopy dormancy at the transcriptome or proteome level.

4. Increased gibberellin (GA) levels and sensitivity are implicated in dormancy release, and are especially important for subsequent germination and seedling growth via effects on softening of restrictive tissues and promoting reserve mobilization. DELLA repressors of GA signaling are stabilized by ABA and phosphorylation, and destabilized by O-Glc-NAc modification and GA. GA perception leads to proteasomal degradation of DELLA proteins.
5. ABA and GA levels are regulated by the balance of synthesis and catabolism due to environmentally regulated expression of specific isozymes, which sometimes requires transport of intermediates between tissues. Regulation by ethylene, brassinosteroids, reactive oxygen species, and nitric oxide or nitrate are partly mediated by effects on the ABA:GA balance, but the latter four also have independent effects.
6. Transitions between various states of dormancy are active processes that involve changes in gene expression even in dry after-ripening seeds. The expression of dormancy-associated genes correlates quantitatively with depth of dormancy.
7. Comparison of dormant versus nondormant transcriptomes and proteomes indicates regulation at multiple levels (e.g., transcript accumulation, translation, protein stability, and modifications affecting activity).

FUTURE ISSUES

1. Functional testing of the role in dormancy for loci identified as showing tightest correlations between expression and dormancy state is required.
2. Cloning of additional QTLs and comparative analyses across species to identify conserved versus novel mechanisms of dormancy regulation is necessary.
3. The timing of requirement for GA synthesis or action in after-ripening versus stratification should be clarified.
4. Researchers should distinguish between the role of ABA signaling loci in ABA response versus dormancy.

DISCLOSURE STATEMENT

The authors are not aware of any biases that might be perceived as affecting the objectivity of this review.

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