Chapter 2
Regulation of Seed Dormancy Cycling in Seasonal Field Environments

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Seeds Select the Habitat, Climate Space and Season in Which to Germinate by Sensing and Responding to the Soil Seed Bank Environment

Seeds are the mobile phase of the plants life cycle; vegetative development is suspended as they transport the plants genetic complement through space and time. In most species this is possible because they can tolerate extreme desiccation and will survive for extended periods in the dry state. However, equally important in the natural world is their ability to exist in an imbibed dormant state, potentially for many years in the soil seed-bank (Finch-Savage and Leubner-Metzger 2006; Footitt et al. 2011). Thus, germination is delayed until they encounter an appropriate habitat, climate space and time of the year suitable for the resulting plant to survive, be competitive and reproduce. This allows multiple species to compete successfully within species-rich natural communities (Baskin and Baskin 1998, 2006; Walck et al. 2011). During their time in the soil seed-bank seeds continually adjust their dormancy status by sensing and integrating a range of environmental signals. The signals related to slow seasonal change can be used for temporal sensing to determine the time of year. In response to these signals seeds alter their depth of dormancy and their sensitivity to other spatial environmental signals. These spatial signals indicate in a more immediate way that conditions are suitable for germination and so trigger the termination of dormancy and therefore induce germination. The response to each of these signals appears to remove successive blocks to germination. However, the process usually needs to be carried out in a set order for it

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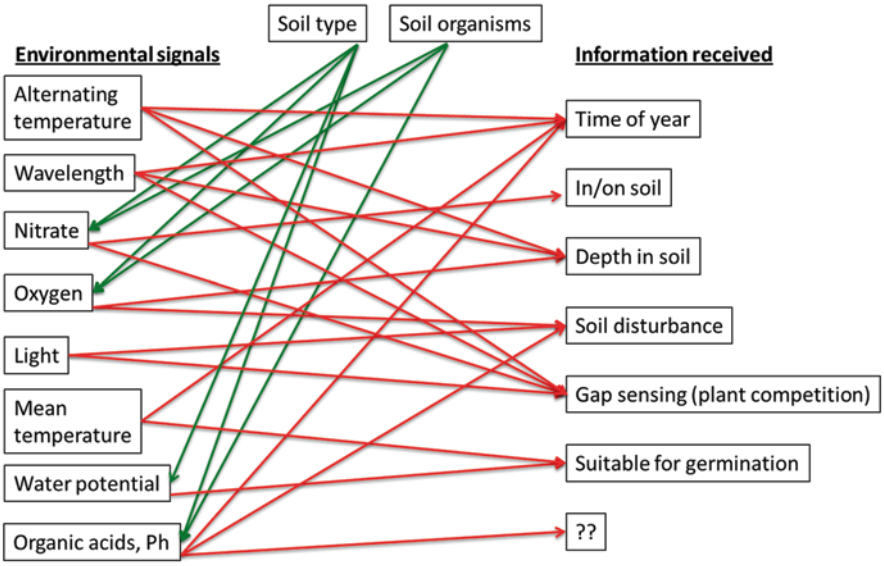

Fig. 2.1 Seeds as environmental sensors: Seeds respond to a wide range of environmental signals that can inform about the time of year (temporal signals) and the suitability of the current environment (spatial signals) for the completion of germination. Seeds are shed onto the soil and become incorporated and so they are also influenced by the physical nature of the soil and the organisms that inhabit it. The figure illustrates the range of environmental signals and how they can potentially inform the seed

to work, that is, spatial signals are only effective if temporal sensing has enhanced sensitivity to them. Thus, a dormancy continuum is proposed that is driven in both directions by environmental signals, and when all layers are removed germination occurs (Finch-Savage and Leubner-Metzger 2006; Finch-Savage and Footitt 2012). In the annual dormancy cycle, if the correct spatial window does not occur sensitivity is lost and the temporal window will close for another year.

A wide range of signals have the potential to inform the seed about its environment (Fig. 2.1). Temperature is the most important signal for temporal sensing (Probert 2000), whereas many signals can be used for spatial sensing to indicate beneficial and adverse conditions for germination, for example, depth in the soil (amplitude of diurnal temperature fluctuation, oxygen, water), soil disturbance (light, oxygen), and vegetation gaps (nitrate, light quality, the degree of diurnal temperature fluctuation) (Baskin and Baskin 2006; Finch-Savage and Leubner-Metzger 2006; Footitt et al. 2011, 2013, 2014). Response to these signals can result in dormancy cycling, which coupled with seed longevity represents a bet-hedging strategy for the short and long-term persistence of native/weed species within the soil seed-bank (Roberts 1964; Evans and Dennehy 2005; Walck et al. 2011; Footitt et al. 2014). Here we will consider the seeds response to temperature as a temporal signal and both light and nitrate as spatial signals.

The underlying sensing and resulting signaling mechanisms to the environment make seeds highly efficient in exploiting distinct habitats and climate spaces (Pons

1989; Saatkamp et al. 2011a, b; Walck et al. 2011). However, the precise response to any environmental signal differs between species, and between ecotypes within species, through adaptation to the habitat and climate space they inhabit. The resulting seasonal seed dormancy cycles and patterns of seedling emergence are well documented as a crucial component of the plants' life cycle that contributes significantly to plant fitness (Donohue 2002; Donohue et al. 2005; Huang et al. 2010; McNamara et al. 2011). It is already recognized that genetic diversity within a species contributes to variation in dormancy and germination phenology; for example in European ecotypes of Arabidopsis (Schmuths et al. 2006; Chiang et al. 2011). As such, dormancy can be seen as contributing to the persistence of genetic diversity (Walck et al. 2011; Lennon and Jones 2011). It is essential that we develop a greater understanding of the mechanisms underlying dormancy cycling and response to environmental signals to determine the impact of future climate change.

Regulation of Dormancy in the Laboratory

Despite the obvious importance of dormancy cycling in the whole life cycle of plants, very little is known about its regulation at a molecular level. In contrast, a great deal is known about mechanisms that influence dormancy loss in short-term laboratory experiments, many of which involve the screening of mutants for altered dormancy and germination (Finch-Savage and Leubner-Metzger 2006; Baskin and Baskin 1998; Finkelstein et al. 2008; Nambara et al. 2010; Graeber et al. 2012). This laboratory-based work has largely used seeds from accessions of the model species Arabidopsis that naturally have limited dormancy. In addition, the seeds used for study have been produced under optimal conditions that tend to minimize dormancy (Kendall et al. 2011). Many of the genes identified have subsequently been found to be involved in the abscisic acid (ABA) and gibberellin (GA) metabolism and signaling pathways (Kucera et al. 2005; Graeber et al. 2012). This has confirmed the central involvement of the ABA/GA balance hypothesis in the seeds ability to interpret the environment and thereby regulate dormancy and germination (Finch-Savage and Leubner-Metzger 2006; Kucera et al. 2005).

Most often, these genes/mechanisms have, for good scientific reasons, been considered in isolation, in constant and therefore simple environments. From these experiments, it is not obvious why so many different mechanisms are required and there is an apparent duplication of function and redundancy. However, in nature seeds have to operate in the complex and variable conditions of the soil seed bank that may require a complexity of subtle dormancy regulation to interpret these conditions. How this complex set of mechanisms is employed by the seed in a coordinated way to regulate dormancy cycling in variable field environments is little understood and until recently unstudied. Our approach to this has been to investigate the molecular ecophysiology of dormancy cycling in field soils using the inherently deeply dormant Arabidopsis ecotype Cape Verdi Isle (Cvi). A key feature in selecting Cvi is that it required exposure to light to remove the final layer of dormancy to allow completion of germination. This absolute requirement for light is important

Table 2.1 Genes studied in the work reported

Gene ID	Annotation
At5g57050	*ABA INSENSITIVE2 (ABI2)*
At3g24650	*ABA INSENSITIVE3 (ABI3)*
At2g40220	*ABA INSENSITIVE4 (ABI4)*
At2g36270	*ABA INSENSITIVE5 (ABI5)*
At2g29090	*CYTOCHROME P450, FAMILY 707, SUBFAMILY A, POLYPEPTIDE 2 (CYP707A2)*
At5g45830	*DELAY OF GERMINATION 1 (DOG1)*
At1g30040	*GIBBERELLIN 2-OXIDASE 2 (GA2OX2)*
At1g15550	*GIBBERELLIN 3 BETA-HYDROXYLASE 1 (GA3OX1)*
At3g05120	*GIBBERELLIN-INSENSITIVE DWARF1 (GID1A)*
At1g18100	*MOTHER OF FLOWERING TIME (MFT)*
At3g24220	*NINE-CIS-EPOXYCAROTENOID DIOXYGENASE6 (NCED6)*
At1g12110	*NITRATE TRANSPORTER 1 (NRT 1.1)*
At2g20180	*PHYTOCHROME INTERACTING FACTOR 3-LIKE 5 (PIL5)*
At4g01026	*ABSCISIC ACID RECEPTOR (PYL7)*
At4g17870	*ABSCISIC ACID RECEPTOR (PYR1)*
At1g14920	*RESTORATION ON GROWTH ON AMMONIA 2 (RGA2)*
At3g03450	*RGA-LIKE 2 (RGL2)*
At5g08590	*SNF1-RELATED PROTEIN KINASE 2.1 (SNRK 2.1)*
At1g10940	*SNF1-RELATED PROTEIN KINASE 2.4 (SNAR 2.4)*
At4g36930	*SPATULA (SPT)*
At1g30270	*CBL-INTERACTING PROTEIN KINASE 23 (CIPK23)*
At1g09570	*PHYTOCHROME A (PHYA)*

experimentally as it allows the separate study of changes in dormancy from downstream changes resulting from the germination process. Our aim was to illustrate how molecular mechanisms identified as controlling dormancy in the laboratory could be seasonally coordinated in seeds buried in field soil to fulfill this process (Footitt et al. 2011). We approached this through gene expression studies targeted at key dormancy regulating genes identified in the laboratory studies described above. We had previously studied the relative importance of these genes for dormancy cycling using full genome arrays of laboratory derived samples that built up the components of dormancy cycling (Cadman et al. 2006; Finch-Savage et al. 2007). We built the study around the dynamic ABA/GA balance and the cohorts of genes that regulate their metabolism, perception, and sensitivity via signaling networks considered central to dormancy and the control of germination completion (radical emergence through the seed coat) (Finch-Savage and Leubner-Metzger 2006; Linkies et al. 2009; Nambara et al. 2010; Bassel et al. 2011; Morris et al. 2011; Dekkers et al. 2013). The genes studied (Table 2.1) and their involvement in the regulation of dormancy are summarized in Fig. 2.2 and the remainder of this section:

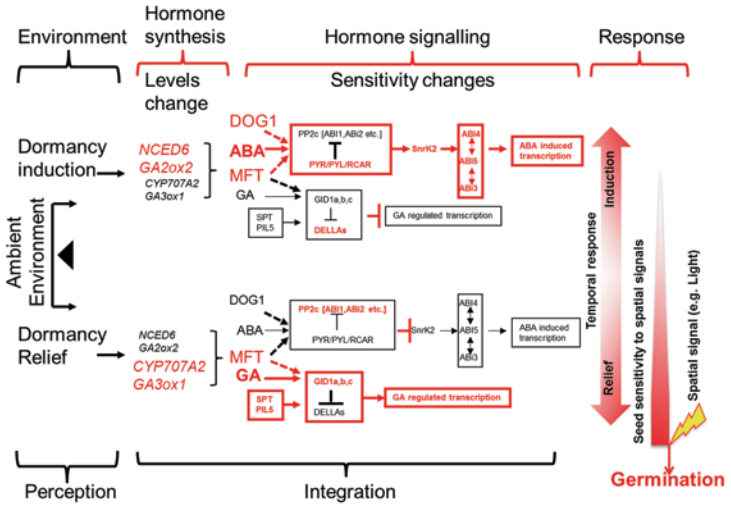

Fig. 2.2 Schematic model for the regulation of dormancy and germination by ABA and GA in response to the environment in Arabidopsis ecotype Cvi: According to this model ambient environmental signals (e.g., temperature, light and nitrate) affect the ABA/GA balance and the sensitivity to these hormones. ABA synthesis and signaling and GA catabolism dominate the induction and deepening of the dormant state, whereas GA synthesis and signaling and ABA catabolism dominate the relief of dormancy and the transition to germination. In the model, when induction or relief are induced by appropriate environmental signals the pathways indicated in red dominate. Change in the depth of dormancy in response to temporal signals alters the requirements for germination (sensitivity to spatial signals, that is, the germination environment); when these signals are perceived in the correct order, all levels of dormancy are removed and germination will proceed to completion. (Model is adapted from Finch-Savage and Leubner-Metzger 2006 and Footitt et al. 2011. Background description to these genes is given in the text)

Hormone Metabolism

GA Active GA levels increase just before radical emergence, suggesting they play a key role in the regulation of germination. The key stages in GA metabolism are now well known (Ogawa et al. 2003). GA3-oxidase is the key enzyme responsible for the final step in GA biosynthesis to produce active GAs. Subsequent degradation is via GA2-oxidase.

ABA Key genes responsible for ABA biosynthesis, degradation, and conjugation during Arabidopsis seed germination are also known and have been described (e.g., Penfield et al. 2006; Okamoto et al. 2006; Holdsworth et al. 2008; Muller et al. 2006; Piskurewicz et al. 2008). NCEDs (Nine-cis-epoxycarotenoid dioxygenases) are the primary regulatory step in ABA synthesis and subsequent inactivation is by hydroxylation (CYP707) or through conjugation with sugars (Nambara et al. 2010). The balance of these processes regulates ABA content. The influence of other hor-

mones, such as ethylene (Linkies et al. 2009), can be significant in the regulation of dormancy and germination, but in general their influence operates through the ABA/GA balance.

Hormone Signaling

Hormone signaling, through repression and de-repression, is a key component of the interacting networks regulating germination (Holdsworth et al. 2008; Kucera et al. 2005). It is now thought that on the ABA side of the balance, the ABA receptors PYR/PYL/RCAR bind to ABA to remove the repression of responses to the hormone by PP2cs (Protein phosphatase 2C; Cutler et al. 2010; Nambara et al. 2010). Removal of PP2c repression allows downstream signaling via SnRK2s to ABRE (ABA-response element) binding transcription factors (ABI3, ABI4, ABI5). On the other side of this balance, DELLA proteins (RGL2, RGA2) repress GA responses and therefore germination potential (Sun and Gubler 2004). DELLAs are degraded to remove this repression on forming a complex with the GA receptor GID1 in the presence of GA (Hartweck 2008).

Environmental Signals and Upstream Regulation

As described above, a diverse range of environmental signals, principally temperature and light, influences these hormone-signaling pathways. Key components of the interaction between these environmental signals and GA are the two phytochrome-interacting bHLH transcription factors, PIL5 and SPT. These repress germination potential, in the dark and at low temperature, respectively. PIL5 represses cell wall modifying genes, *GA3ox1* and *CYP707A2*, and enhances *GA2ox1*, *NCED6*, and *DELLA* expression, while *SPT* represses *GA3ox1* expression (Penfield et al. 2005; Ho et al. 2009). In turn, PIL5 and SPT are inactivated by DELLAs (RGL2 and RGA2) (Penfield et al. 2005). PIF (phytochrome interacting factor) proteins are released when the GID protein–GA complex binds DELLA proteins to target their degradation by the proteosome (Daviere et al. 2008).

Delay of germination 1 (DOG1) is a key regulator of dormancy (Bentsink et al. 2006) and is also closely linked to the impact of temperature on dormancy status (Footitt et al. 2013 etc.) and is thought to alter sensitivity to ABA (Teng et al. 2008). Similarly, mother of flowering time (MFT) is a proposed ABA-induced negative regulator of ABA signaling and is thought to operate as the convergence point of ABA and GA signaling pathways (Xi et al. 2010). Nakamura et al. 2011 reports that MFT expression is regulated in response to temperature and seems to transmit temperature signals to a downstream temperature-signaling cascade to regulate depth of dormancy.

Nitrate is also an important environmental signal in the soil seed bank. Seed dormancy can be released by nitrate in Arabidopsis, but it is not clear whether ni-

trate acts *per se* on seed germination or through the production of N-related signals (Alboresi et al. 2005). However, nitrate accelerates the decrease in ABA prior to completion of germination (Ali-Rachedi et al. 2004) via induction of the catabolic ABA gene *CYP707A2* (Matakiadis et al. 2009).

We have argued (Footitt et al. 2013) that the response of seeds to nitrate in the soil seed bank appears to act via CIPK23 phosphorylation/dephosphorylation of NRT1.1 and the response to light acts via PHYA. PHYA targets ABRE containing promoters and could be involved in the ABA signaling response (Chen et al. 2014).

Regulation of Dormancy Cycling in the Field Soil Seed Bank

The Arabidopsis ecotype Cvi exhibits the life cycle of a winter annual, by germinating in autumn and overwintering as a seedling rosette to produce dormant seeds in late spring that use the warmth of summer to relieve dormancy. Seeds that do not get exposed to environmental conditions that remove the final layer of dormancy and therefore induce germination completion enter a dormancy cycle and over winter as a seed. We studied the seeds' response to the soil seed bank conditions following sowing in late spring (May; Footitt et al. 2014) and autumn (October; Footitt et al. 2011, 2013). Seeds from the same harvest were used for both sowings and these were processed and stored at −80 °C to minimize physiological change. The seeds were buried under the soil surface in mesh bags and exhumed at intervals (methods described in Footitt et al. 2011) for both physiological analysis and measurement of gene expression by quantitative PCR or Nanostring technology (Footitt et al. 2011, 2013, 2014). Seed samples for the latter were exhumed and prepared in the dark.

Seeds that were sown in spring at their natural time of shedding entered a shallow dormancy cycle dominated by spatial sensing that adjusted germination potential to the maximum when soil environment was most favorable for germination and seedling emergence upon soil disturbance (Footitt et al. 2014). This behavior differed subtly from that of seeds sown in autumn and overwintered in the soil seed bank (Footitt et al. 2011, 2013) and this difference spreads the period of potential germination in the seed population (existing seed bank and newly dispersed). As soil temperature declined in autumn, seeds denied conditions required to remove all layers of dormancy and therefore germination completion (e.g., light) entered deep dormancy and the process of dormancy cycling. These spring-sown seeds then become part of the persistent seed bank. Seeds that were sown in autumn represent this cohort of the soil seed population and it is the behavior of these seeds we describe below.

Seed behavior was monitored over a complete year following sowing, and throughout soil temperature and moisture content at sowing depth were monitored. A clear seasonal temperature pattern was recorded (Fig. 2.3a) with temperature declining in winter, rising in spring, peaking in summer, and then declining toward autumn. The depth of seed dormancy, estimated as the afterripening time required

to achieve 50 % germination at 20 °C (AR50), was negatively correlated ($P < 0.001$) with this temperature pattern (Fig. 2.3a). The depth of dormancy, which was already significant, rose sharply in the imbibed seeds following sowing as temperature decreased going into winter and then declined equally quickly in spring. Interestingly, AR50 declined from more than 150 days to less than 50 days in less than one month in the moist warming soils. By mid-July seeds required only exposure to light to remove the final layer of dormancy allowing seeds to proceed to germination completion (radicle emergence). Germination potential, measured by exposing exhumed seed to light, reached a peak in all temperatures tested at this time (Fig. 2.3f). Consequently, in a parallel experiment where seeds sown in the surface layer of soil were disturbed regularly, there was a flush of seedling emergence in early August following a period of germination and pre-emergence seedling growth (Fig. 2.3f).

Over this annual cycle we followed the expression of the genes described above to shed light on the regulation of dormancy cycling and the deployment of dormancy mechanisms identified in the laboratory. Figures 2.3b–e illustrates the patterns of expression we found in key genes. Both *DOG1* and *MFT* had expression patterns that were, like depth of dormancy, negatively ($P < 0.001$) correlated with soil temperature (Fig. 2.3b). *DOG1* is not directly associated with ABA, but is altered by environmental conditions (Chiang et al. 2011) and may enhance ABA sensitivity (Teng et al. 2008). ABA concentration is linked to dormancy level in Cvi (Al-Rachedi et al. 2004), but we did not find a positive relationship between increasing AR50 and ABA concentration. Indeed, ABA concentration increased with AR50 to *c.* 50 days, but then reached a plateau as AR50 continued to increase (Footitt et al. 2011). In contrast, *DOG1* expression had a positive relationship with AR50 up to the highest recorded AR50 of *c.* 200 days. Footitt et al. (2011) suggested that ABA concentration is important to the control of dormancy as seen in the laboratory, but as deep dormancy is induced in the field, *DOG1* expression may be the dominant factor enhancing ABA sensitivity. They also suggest that MFT, as an ABA-induced germination repressor (Nakamura et al. 2011), also functions in this aspect of dormancy regulation.

On the other side of the hormone balance, the expression pattern of *GA3ox* repressors *SPT* and *PIL5* was positively correlated with temperature and therefore negatively correlated with depth of dormancy, but this correlation was only significant for SPT ($P < 0.01$; Fig. 2.3c). Their expression tended to peak when germination potential was highest. Similarly, expression of the *DELLA* genes *RGA2* and *RGL2* was low over winter (both negatively correlated with temperature ($P < 0.01$)) and increased to a peak as dormancy was lowest ($P < 0.01$) and germination potential peaked (Fig. 2.3d). At first sight, this coincidence of high germination potential and peak expression of germination repressors appears counterintuitive. However, it must be remembered that these seeds are still dormant in the soil and germination must not occur until they are exposed to the correct spatial signal (e.g., light) to indicate soil disturbance/absence of plant competition. Nevertheless, on exposure to light the response must be rapid so that germination completion and seedling emergence can take place while conditions are suitable. Thus, in winter, deep dormancy determined by sensitivity to ABA prevents any response to light. In contrast, during

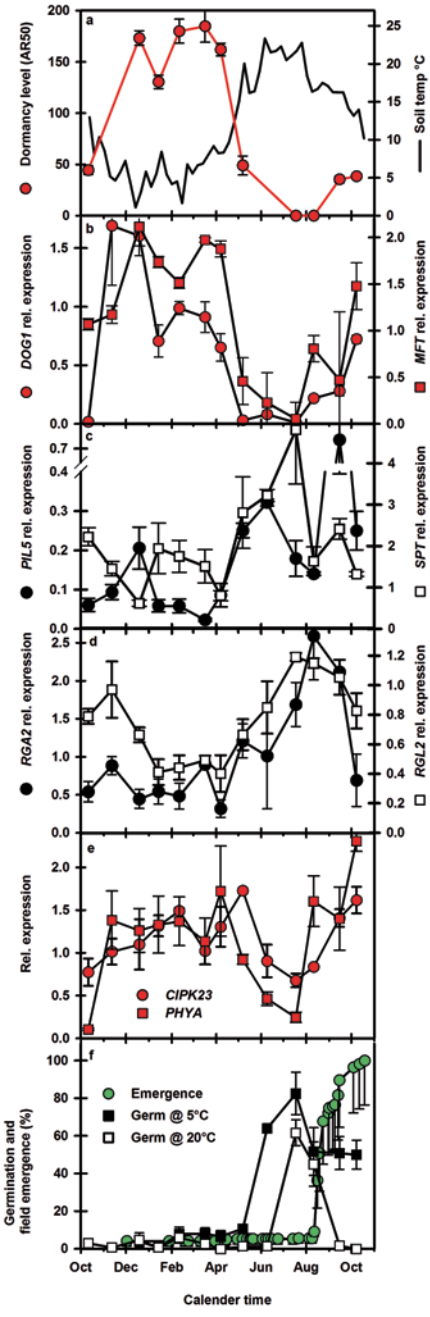

Fig. 2.3 Dormancy cycling in the soil seed bank: Changes in Arabidopsis Cvi seeds following sowing into the soil seed bank are shown. Gene expression is measured in seeds that have been flash frozen at − 80 °C immediately after exhumation in the dark. The seeds are not exposed to light until placed in germination experiments for recording germination potential following soil disturbance. **a** The patterns of mean soil temperature at sowing depth and depth of seed dormancy measured as AR50 (length of after-ripening at 20 °C that enables 50% of the population to germinate at 20 °C). Gene expression is shown for **b** *MFT* and *DOG1*, **c** *PIL5* and *SPT*, **d** *RGA2* and *RGL2*, **e** *CIPK23* and *PHYA*. **f** Potential germination at 5 and 20°C and seedling emergence recorded in adjacent experimental field plots that were disturbed at regular intervals to expose the buried seeds to light. Error bars indicate SEM; $n = 3$. (The figure is adapted from Footitt et al. 2011, 2013)

summer when shallow dormancy results from GA synthesis and signaling repression, seeds are sensitive to light. In the latter case, exposure to light dramatically enhances expression of *GA3ox* (Cadman et al. 2006), resulting in synthesis of GA that binds to DELLAs removing repression of GA signaling. A temporal separation of ABA- and GA-related dormancy mechanisms is revealed, which allows accurate timing of germination completion through dormancy cycling in a seasonal environment. Throughout this cycle, the expression of other genes related to hormone synthesis and signaling were consistent with the operation of the hormone balance described in Fig. 2.2 (Footitt et al. 2011, 2013, 2014).

Temporal sensing, therefore, facilitates the completion of germination to occur at the optimum time of year. However, as discussed above this should only take place if environmental conditions are suitable as indicated by a range of spatial signals (Fig. 2.1) of which light and nitrate have received most attention and are potentially the most important (discussed in Footitt et al. 2013). The response to temporal (seasonal) signals is, therefore, to alter sensitivity to these spatial signals. Footitt et al. (2013) argue that the response to nitrate appears to act via *CIPK23* phosphorylation/dephosphorylation of *NRT1.1*. NRT1.1 is a duel affinity nitrate transporter with the low- or high-affinity function dependent upon the phosphorylation status of threonine-101 (Ho et al. 2009) and is considered to be a nutrient transceptor (duel nutrient transport/signaling function; Gojon et al. 2011). Potentially the signaling and transport function of NRT1.1 may be uncoupled through the action of CIPK23 to reduce sensitivity to nitrate and enhance dormancy (Footitt et al. 2013). They further argue from dormancy-associated expression patterns (Cadman et al. 2006; Finch-Savage et al. 2007) that the response to light is determined via PHYA. *PHYA* expression was negatively correlated with *GA3ox1* expression (Footitt et al. 2013) consistent with the reports of reduced *GA3ox1* expression and GA levels when *PHYA* is overexpressed (Jordan et al. 1995; Foo et al. 2006). Expression patterns of *PHYA* and *CIPK23* were similar being higher in winter, lower in summer, and reaching a minimum in July when germination potential peaked (Fig. 2.3e, f). Thus, in the field when this temporal and spatial sensing overlapped with ambient environmental conditions, dormancy was removed and seeds progressed to germination completion and seedling emergence.

In this work, we were able to show the temporal coordination of the major signaling networks identified in the laboratory that regulate seed dormancy in an ecological context in the field. This highlighted that seeds in the seed bank are capable of adjusting the depth of dormancy through temporal sensing (identifying the correct season and climate space for emergence) and spatial sensing (identifying signals indicating suitable conditions to terminate dormancy and complete germination). Dormancy and the expression of dormancy-related genes were highly sensitive to the soil environment, and molecular and physiological changes could be equated to changes in sensitivity to soil temperature history, nitrate, light, and gibberellins. This illustrates dormancy as a continuum with layers of dormancy being progressively removed by environmental signals until only light is required, in the absence of which seeds remain dormant and enter into another dormancy cycle as the seasons change (Footitt et al. 2011, 2013, 2014; Finch-Savage and Footitt 2012).

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