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Two Faces of One Seed: Hormonal Regulation of Dormancy and Germination

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ABSTRACT

Seed plants have evolved to maintain the dormancy of freshly matured seeds until the appropriate time for germination. Seed dormancy and germination are distinct physiological processes, and the transition from dormancy to germination is not only a critical developmental step in the life cycle of plants but is also important for agricultural production. These processes are precisely regulated by diverse endogenous hormones and environmental cues. Although ABA (abscisic acid) and GAs (gibberellins) are known to be the primary phytohormones that antagonistically regulate seed dormancy, recent findings demonstrate that another phytohormone, auxin, is also critical for inducing and maintaining seed dormancy, and therefore might act as a key protector of seed dormancy. In this review, we summarize our current understanding of the sophisticated molecular networks involving the critical roles of phytohormones in regulating seed dormancy and germination, in which AP2-domain-containing transcription factors play key roles. We also discuss the interactions (crosstalk) of diverse hormonal signals in seed dormancy and germination, focusing on the ABA/GA balance that constitutes the central node.

Keywords: seed dormancy, germination, ABA, GA, auxin, crosstalk

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INTRODUCTION

Seed dormancy is crucial to plant survival and ensures that seeds germinate only when environmental conditions are optimal. It thus is an adaptive trait in numerous seed-plant species, enabling wild plants to survive under stressful conditions in nature (Finkelstein et al., 2008). Most crops have been domesticated from wild species and show decreased levels of seed dormancy compared with their wild relatives, which ensures higher emergence rates after sowing (Lenser and Theissen, 2013; Meyer and Purugganan, 2013). However, the inappropriate loss or release of seed dormancy results in the rapid germination of freshly matured seeds or even pre-harvest sprouting (vivipary) in crops (Figure 1), causing substantial losses in yield and quality in agricultural production in addition to problems including post-harvest management and subsequent industrial utilization (Simsek et al., 2014).

Induction, maintenance, and thereafter release of seed dormancy are important physiological processes in seed plants. The ecological significance of seed dormancy includes preventing germination out of season, and consequently decreasing competition within species and ensuring plant survival under stressful conditions. As a complex and mysterious biological question, seed dormancy has attracted increasing attention from multi-disciplinary researchers, including plant biologists, crop geneticists, breeders, and food scientists. Nevertheless, it remains ‘‘one of the least understood phenomena in seed biology’’ (Finkelstein et al., 2008), despite considerable progress over past decades (Graeber et al., 2012; Rajjou et al., 2012). In this review, we summarize the mechanisms underlying the regulation of seed dormancy and germination, and focus on the emerging findings concerning the phytohormone network controlling this transition, mostly from studies with the model plant *Arabidopsis thaliana*.

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Figure 1. Representative Image of the Pre-harvest Sprouting Phenotype of Rice in the Field.
Pre-harvest sprouting of crops often occurs when mature plants encounter prolonged rainfall and high humidity during the harvest season, which decreases yields and grain quality and also causes problems in industrial process. Red arrows indicate sprouting seeds on panicles.

DISTINCT PROCESSES OF SEED DORMANCY AND GERMINATION

Seed dormancy and germination has been studied intensively and extensively in the past; however, what constitutes seed dormancy at the molecular level remains largely unknown. Here, we attempt to address this question from a new viewpoint based on recent progress.

Seed dormancy ensures that seeds germinate at the appropriate time. Therefore, during maturation, the embryo must be kept in a quiescent state, mobilizing almost no stored nutrients and undergoing no cell division or elongation. In this quiescent state, germination-promoted genes are not actively expressed. Therefore, the radicle does not penetrate the testa and endosperm. It is now widely recognized that the chromatin structure determines gene expression and thereby regulates multitudinous developmental processes. In recent years, many genes associated with chromatin remodeling have been reported to regulate seed dormancy and germination (Liu et al., 2007; Saez et al., 2008; Wang et al., 2011a; Cho et al., 2012; Zheng et al., 2012). Emerging evidence shows that ABA (abscisic acid) is also involved in chromatin remodeling (Chinnusamy et al., 2008). For example, the histone methyltransferase gene *KYP/SUVH4* is repressed by ABA (Zheng et al., 2012), while histone acetyltransferase *HvGNAT/MYST* is induced by ABA (Papaefthimiou et al., 2010), and the epigenetic regulators *HUB1* and *RDO2* are strikingly up-regulated during the induction of seed dormancy (Liu et al., 2011). These investigations indicated that the epigenetic regulatory-related genes possess key roles during seed maturation, which thereafter affect the seed dormancy establishment process (Figure 2).

We propose that subsequently, during the germination process, seed dormancy may be related to a characteristic chromatin structure in certain regions of chromosomes in the seed, where germination-promoted genes cannot be activated even in the presence of related transcription factors because their binding sites are unavailable due to steric hindrance, with phytohormones also involved in this process. In contrast, dormancy release leading to germination is a process in which the chromatin structure is modified by cold stratification or after-ripening treatments, making the germination-promoted genes available for transcription, resulting in cell elongation and division, seed coat and endosperm rupture, and finally emergence of the radicle when conditions are favorable.

Although dormancy is established during seed maturation, whereas exogenous ABA application (or even maternal ABA in the plant during seed development) only inhibits seed germination but fails to induce seed dormancy; only ABA synthesized by the seed can establish dormancy (Kucera et al., 2005). Thus, the differently localized ABA in plant tissues possesses distinct effects on seed dormancy or germination. In addition, ABI5 is an important positive regulator in the ABA-signaling pathway, and its loss-of-function mutant *abi5* is insensitive to ABA-mediated inhibition of seed germination; however, *abi5* does not show altered seed dormancy (Finkelstein, 1994; Brocard-Gifford et al., 2003; Finkelstein et al., 2008). Furthermore, DOG1 (Delay of Germination 1) is a key player in the induction and maintenance of seed dormancy, but ABA sensitivity is unchanged in *dog1* (Nakabayashi et al., 2012). A new study demonstrated that DOG1 mediates a conserved coat-dormancy mechanism including the temperature- and gibberellin (GA)-dependent pathways (Graeber et al., 2014). Subsequent studies suggested the importance of epigenetic regulation for DOG1. Histone demethylases LDL1 (LYSINESPECIFIC DEMETHYLASE LIKE 1) and LDL2 repress seed dormancy by regulating *DOG1* (Zhao et al., 2015), and chromatin remodeling of DOG1 is also involved in dormancy cycling (Footitt et al., 2015). Furthermore, the histone methyltransferases KRYPTONITE (KYP)/SUVH4 and SUVH5 repress *DOG1* and *ABI3* transcription during seed maturation (Zheng et al., 2012) (Table 1). These studies demonstrated that the DOG1-mediated regulation pathway might be distinct from the ABA and/or GA pathway (Figure 2). These observations suggest that distinct signaling pathways may be adopted in the regulation of seed dormancy and seed germination.

ABA AND GA, THE MAJOR DETERMINANTS: NEWLY EMERGING EVIDENCE

It is widely recognized that ABA and GA are the primary hormones that antagonistically regulate seed dormancy and germination (Gubler et al., 2005; Finkelstein et al., 2008; Graeber et al., 2012; Hoang et al., 2014; Lee et al., 2015a). During seed maturation, endogenous ABA accumulates in the seed, inducing and maintaining seed dormancy and thus preventing