



A study on genetic aspects of seed dormancy

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Abstract

Changes in seed dormancy were selected during the domestication of crop plants from wild-plant species, because some features of dormancy that provide ecological advantages presented agronomic disadvantages within a farmed system. Seed dormancy varies widely among seed batches, even for seeds of the same genotype, indicating large environmental influences. For genetic studies, it is essential to obtain such quantitative data on seed material without the effects of variation in environmental conditions, including the maternal growing and seed storage conditions. The genetics of seed dormancy is as predicted on Mather's theory of genetical architecture for a character that has been subject to strong directional selection. Many studies on the genetics of seed dormancy regulation have concluded that dormancy is a quantitative trait controlled by multiple loci that affect different seed tissues, and often different aspects of seed physiology. Therefore, in this study discus on some research on seed dormancy control by genetic.

Key words: Genetic and seed dormancy

Introduction

Forgive to highest seed yield in agriculture addition to both fertilizer, pest management and seeds with good quality is very important (Shaban, 2013b,c; Azimi et al, and Beyranvand et al, 2013). Many of our crops are reproduced through seeds, and throughout the world large quantities are produced, stored and transported (Shaban, 2013a). Seed dormancy is a temporary failure of a mature viable seed to germinate under environmental conditions that would normally favour germination (Foley, 1998). Plants have evolved several dormancy mechanisms to optimize the time of germination and, by optimizing the distribution of germination over time ('dispersal in time'), seed dormancy enhances survival (Foley, 2001). Since it is a physiological adaptation to environmental heterogeneity, seed dormancy is a primary factor that influences natural population dynamics (Bewley and Black, 1994). Seed dormancy is a typical quantitative trait, since it is controlled by multiple loci and highly influenced by genotype versus environment interactions (Koornneef *et al.*, 2002). Temperature, oxygen, light and moisture are some

environmental factors present during seed development that influence the germination phenotype. Most studies on the genetics of seed dormancy regulation have concluded that dormancy is a quantitative trait controlled by multiple loci that affect different seed tissues, and often different aspects of seed physiology (Foley and Fennimore, 1998). As with many quantitative traits, developmental environment may interact strongly with genotype to produce the resultant phenotype, in this case the fraction of seeds dormant under a particular set of germination conditions (Naylor, 1983). Genetic studies of seed dormancy are complicated by the fact that different tissues within a seed have different parentage. The seed coat or testa and any accessory structures are of strictly maternal origin, while the embryo contains equal representation of maternal and paternal genotypes. Endosperm may also be present; this contains two complements of maternal genes and usually one of paternal genes, depending on the plant family. The longevity of seed viability of domestic crops is a function of genotype, dormancy and storage conditions. Mature plant seeds can be viable before or at their separation from the mother plants, but may not be capable of immediate germination due to dormancy factors. Dormancy is the temporary arrest of seed development before completion of their maturation process. It is a condition during which the growth and development of rice seeds is temporarily suspended and it occurs in the formation period of seeds and continues for certain period of time (Takahashi 1997, Gardner et al. 1985). This review gives an overview of the genetic aspects of control seed dormancy.

Dormancy and genetic

The level of dormancy in seeds is determined by several factors apart from genetic origin, such as maternal environment during maturation, age of the mother plant during maturation and position of the seed on the plant (Fenner, 1991). In addition, the different conditions during natural stratification cause a more or less constant change in dormancy level (Vleeshouwers *et al.*, 1995). These factors often account for a great deal of the variation in germination percentage among populations, and also for the differences in germination between seeds harvested in different years. In some species, embryo genotype has been shown to be of over-riding importance in determining dormancy phenotype, e.g. *Avena fatua* (Jana et al., 1979), *Hordeum vulgare* (Buraas and Skinnes, 1984), and *Eustoma grandiflorum* (Ecker et al., 1994).

Sprouting and seed dormancy

Lack of adequate dormancy results in pre-harvest sprouting in the field under wet weather conditions. Pre-harvest sprouting is the germination of the grains before harvest due to rainfalls and high humidity. During this process, it occurs an increase of α -amylase activity in the endosperm, resulting in the degradation of starch to reduced sugars, which causes great losses to the bakery industry. Tolerance to sprouting damage and embryo dormancy is therefore a highly desirable but complex trait that is regulated by a number of key resistance genes that strongly interact with environmental conditions (Flintham & Gale, 1988). Lawson et al. (1997), who had worked with Australian background, showed that in two recombinant populations PHS was under a simple genetic control involving two genes. A new major gene (PHS) was identified on the long arm of chromosome 4A as controlling the difference between two red-grained cultivars with widely different dormancy (Flintham et al., 2002). Andreoli et al (2006) cleared that two major genes, hereinafter called *A,a* and *B,b*, control seed dormancy, which appears to be recessive to dormancy. Only the homozygous *aabb* is dormant.

The environment and seed dormancy

Key environmental factors like temperature, nitrate, light, water, oxygen, smoke and allelochemicals influence dormancy levels either during seed development on the mother plant or in the soil seed bank. Germination requires specific environmental conditions and Finch-Savage & Leubner- Metzger (2006) state that the sensitivity of seeds to environmental factors changes continuously as a function of variable ambient conditions. Temperature and light perceived during seed maturation have been shown to influence the dormancy level (Chiang et al. 2009).

Induction of seed dormancy

Dormancy is a complex trait because it is influenced by both environmental and endogenous factors. Moreover, the final level of dormancy is determined by the contributions of the different tissues that comprise a seed. Different plant species show a variety of seed structures (Linkies et al. 2010), but as a general rule in angiosperms, the embryo surrounding tissues or seed envelopes (e.g. testa and endosperm) prevent germination by providing a physical barrier for the elongating radicle (Debeaujon, Léon-Kloosterziel & Koornneef 2000). Germination and dormancy depend on the balance between the growth force of the elongating radicle and the resistance strength of the surrounding tissues (Fig. 1c,d). The activities of cell wall remodelling proteins influence the strength of the surrounding tissues (Endo et al. 2012). The induction of seed dormancy is controlled by a diverse group of regulators that act at various levels and that show different degrees of specificity. In this paper, we divided these regulators into four groups involved in seed maturation, hormonal action, dormancy and chromatin regulation (Graeber et al, 2012). Seed dormancy is induced during the seed maturation phase simultaneously with the accumulation of storage compounds, the acquisition of desiccation tolerance and, finally, the quiescence of metabolic activity (Graeber et al, 2012). Several factors, which control seed dormancy indirectly by regulating ABI3, FUS3, LEC1 and LEC2, have recently been identified (Graeber et al, 2012). For instance, maize VIVIPAROUS 8 (VP8) has been shown to regulate these transcription factors and a mutation in this gene causes a viviparous seed phenotype with pleiotropic developmental changes (Suzuki et al. 2008). Numerous genetic studies using abscisic acid and gibberellin biosynthesis and signalling mutants have demonstrated that these two hormones have essential and antagonistic roles in dormancy and germination (Graeber et al, 2012). In particular, the balance between the levels of these two hormones and their respective signalling pathways are important in regulating both induction and maintenance of dormancy, and promotion of germination (Finkelstein et al. 2008). Very recent reports have demonstrated the roles of strigolactones and karrikins (germination-promoting compounds in smoke) in dormancy and germination. Strigolactone signalling is mediated by the F-box protein KARRIKIN INSENSITIVE 1 (KAI1), which is allelic to MORE AXILLARY BRANCHES 2 (MAX2) (Graeber et al, 2012). The kai1/max2 mutant shows increased primary dormancy (Nelson et al. 2011). It has recently been shown that strigolactones modulate the ABA/GA ratio in secondary dormancy control (Toh et al. 2012). Candidate gene approaches by reverse genetics have become increasingly feasible during the last decade by utilizing large transcriptome and proteome data sets (Graeber et al, 2012). Recently, two new seed dormancy factors have been identified in Arabidopsis using high-throughput quantitative RT-PCR. These are DESPIERTO (DEP), which is a C3HC4 RING finger protein and the HDZip gene ATHB20 (Barrero et al. 2010). A mutation in the DEP gene causes lack of dormancy, whereas the athb20-1 insertion mutant shows increased dormancy compared with the wild type. Interestingly, both genes modulate ABA sensitivity. It is noteworthy that the dep mutant is completely non-dormant, similar to the dog1 mutant. The biochemical properties of DEP and identification of its downstream targets will be of great interest (Graeber et al, 2012).

Release of seed dormancy

The molecular mechanisms controlling dormancy release are less well understood compared to those controlling dormancy induction. The fast release of dormancy requires imbibition at species-specific temperatures and is called stratification (Graeber et al, 2012). In general, imbibition at low temperatures releases dormancy in seeds of summer annuals, while high temperatures release dormancy in seeds of winter annuals (Probert 2000). Dormancy can be artificially released by removing constraints (i.e. embryo surrounding tissues) that prevent germination (scarification) or by storing seeds at room temperature under dry conditions (after-ripening). Increased time of after-ripening is associated with a widening of the conditions required for germination, resembling gradual dormancy loss. Several transcriptome analyses showed that afterripening affects transcript abundances in dry seeds, resulting in the selective change of specific transcripts (Leymarie et al. 2007). It is also possible that the quantity and quality of stored mRNAs is changed within the dry seed by mechanisms that do not require an active metabolism. A recent study showed that the selective oxidation of a subset of stored mRNAs is associated with dormancy release in sunflower seeds. Oxidation of mRNA can prevent their translation and lead to changes in the proteome after translation has been restarted during seed imbibition. Interestingly, there seems to be a selective oxidation of mRNAs corresponding to genes involved in stress response (Bazin et al. 2011).

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