Molecular and epigenetic regulations and functions of the LAFL transcriptional regulators that control seed development

L. Lepiniec, M. Devic, T. Roscoe, D. Bouyer, D.-X Zhou, C. Boulard, S. Baud, B. Dubreucq

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Lepiniec L.[1]*, Devic M.[2,5], Roscoe T.J.[2,5], Bouyer D.[3], Zhou D-X.[4], Boulard C.[1], Baud S.[1] and Dubreucq B.[1]

[1] IJPB (Institut Jean-Pierre Bourgin), INRA, AgroParisTech, CNRS, Université Paris-Saclay, RD10, 78026 Versailles, France

[2] Régulations Epigénétiques et Développement de la Graine, ERL 5300 CNRS-IRD UMR DIADE, IRD centre de Montpellier, 911 avenue Agropolis BP64501, 34394 Montpellier, FRANCE

[3] Institut de Biologie de l'ENS, CNRS UMR8197, Ecole Normale Supérieure, 46 rue d'Ulm, 75230 Paris cedex 05, France

[4] Institute of Plant Sciences Paris-Saclay (IPS2), Université Paris Sud 11, Université Paris-Saclay, 91405 Orsay, France

[5] Present address: Sorbonne Universités, Université Pierre et Marie Curie (Paris 06) & Centre National pour la Recherche Scientifique CNRS UMR 7621, Laboratoire d'Océanographie Microbienne, Observatoire Océanologique, F-66650, Banyuls-sur-mer, FRANCE

* Lepiniec L to whom correspondence should be addressed, Loic.Lepiniec@inra.fr

Abstract

The LAFL (i.e. LEC1, ABI3, FUS3, and LEC2) master transcriptional regulators interact to form different complexes that induce embryo development and maturation, and inhibit seed germination and vegetative growth in Arabidopsis. Orthologous genes involved in similar regulatory processes have been described in various angiosperms including important crop species. Consistent with a prominent role of the LAFL regulators in triggering and maintaining embryonic cell fate, their expression appears finely tuned in different tissues during seed development and tightly repressed in vegetative tissues by a surprising high number of genetic and epigenetic factors. Partial functional redundancies and intricate feedback regulations of the LAFL have hampered the elucidation of the underpinning molecular mechanisms. Nevertheless, genetic, genomic, cellular, molecular, and biochemical analyses implemented during last years have greatly improved our knowledge of the LALF network. Here we summarise and discuss recent progress, together with current issues required to gain a comprehensive insight into the network, including the emerging function of LEC1 and possibly LEC2 as pioneer transcription factors.

Keywords

Genetics and epigenetics regulations, pioneer factors, transcriptional network, LAFL regulators, seed development and maturation.

Content:

1 Introduction

1.1 Structure, evolution, and expression of the *LAFL* genes

The few hundred genes highly and specifically expressed during the different phases of seed development (e.g. embryogenesis, maturation, and acquisition of desiccation tolerance and dormancy) emphasize the importance of transcriptional regulations for proper seed formation (Harada and Pelletier, 2012). The *LAFL* genes namely *LEC1, ABI3*, *FUS3,* and *LEC2,* encode master transcriptional regulators that induce and maintain these different phases of seed development and repress precocious seed germination and vegetative growth in Arabidopsis (Braybrook and Harada, 2008; Santos-Mendoza et al., 2008; Verdier and Thompson, 2008; Roscoe et al., 2015; Carbonero et al., 2016; Fatihi et al., 2016; Gonzalez-Morales et al., 2016; Boulard et al., 2017). LEC1 (LEAFY COTYLEDON1) is a member of the NF-YB protein family (NF-YB9, HAP3 subunit of the CCAAT-box binding factors) and has a closely related homolog LEC1-like (L1L, NF-YB6) (Lotan et al., 1998; Kwong et al., 2003; Zhao et al., 2016; Boulard et al., 2017; Swain et al., 2017). ABI3 (ABSCISIC ACID INSENSITIVE3), FUS3 (FUSCA3), and LEC2 (LEAFY COTYLEDON2) belong to the plant specific family of B3 domain Transcription Factors (TFs) and thus are also named "AFL-B3" regulators (Giraudat et al., 1992; Baumlein et al., 1994; Keith et al., 1994; Luerssen et al., 1998; Stone et al., 2008; Swaminathan et al., 2008; Carbonero et al., 2016; Devic and Roscoe, 2016; Fatihi et al., 2016; Boulard et al., 2017; Lehti-Shiu et al., 2017).

Homologs of *LEC1, ABI3, and FUS3* have been identified in all the angiosperms studied so far, including important crops such as rice, maize, wheat, soybean, or barley (Verdier and Thompson, 2008; Wang et al., 2012; Peng and Weselake, 2013; Cagliari et al., 2014; Carbonero et al., 2016; Fatihi et al., 2016; Boulard et al., 2017; Manan et al., 2017; Pelletier et al., 2017; Sun et al., 2017; Zhiguo et al., 2018). Some of these homologs have been functionally characterized in monocots, and in lower plants, such as *ABI3* in the moss *P. patens* (Tan et al., 2017). LEC2 seems to be found only in dicots (Li et al. 2010). Although the existence of *LEC2* orthologs in monocots has been suggested based on sequence similarity, their function might be slightly different (e.g. ZmAFL4, which is expressed in maize endosperm and involved in carbon metabolism and starch accumulation) or remains to be investigated (Grimault et al., 2015; Carbonero et al., 2016; Boulard et al., 2017). Moreover, another LEC2 related sequence specific to monocot, namely "IDEF", has been describes and could be a monocot ortholog (Li et al., 2010). As things stand, it is therefore unclear whether LEC2 arose from a duplication of ABI3 in dicots after the divergence of monocots and dicots, or if gene duplication occurred before this divergence, with rapid evolution or loss of *LEC2* in monocots. Moreover, *ABI3*, which is the putative common ancestor of the *AFL-B3* (Li et al., 2010), shares some functional similarities with *LEC2* (Baud et al., 2016). As a consequence, *LEC2* may be dispensable in most plants where *ABI3* or some closely related proteins could fulfil similar function.

The expression patterns of the *LAFL* genes have been extensively described in Arabidopsis (Lotan et al., 1998; Kwong et al., 2003; Gazzarrini et al., 2004; Ng et al., 2004; To et al., 2006; Santos-Mendoza et al., 2008; Sreenivasulu and Wobus, 2013; Roscoe et al., 2015; Boulard et al., 2017; Tao et al., 2017). *LAFL* are expressed in seed and, under normal physiological conditions, none of the mRNAs is

detected above the background noise outside of the seed (http://bar.utoronto.ca). Nevertheless, a few studies report some *LAFL* promoter activity in vegetative tissues and abnormal vegetative phenotypes of *lafl* mutants. FUS3 and LEC2 could be involved in lateral root formation (Tang et al., 2017) and LEC1 in hypocotyl elongation in dark-grown seedlings (Junker and Baumlein, 2012; Huang et al., 2015a). More recently, it has been demonstrated that LEC1 plays a role in the activation of the flowering gene *FLC*, but also that *LEC1* is expressed only in seed and induces *FLC* early during embryo development (Tao et al., 2017). Thus, the activation of *LAFLs* during vegetative development may be restricted to specific cells or stress conditions such as wounding or skotomorphogenesis (Junker et al., 2012; Huang et al., 2015b; Iwase et al., 2015; Swain et al., 2017). In Arabidopsis seed, *LAFL* mRNAs are detected early during embryo development: *LEC1* and *LEC2* peak about a week after pollination, whereas *ABI3, FUS3,* and *L1L* peak one week later. It is to be noted that *FUS3* would be mainly expressed in the epidermis (Gazzarrini et al., 2004; Tsuchiya et al., 2004). These specific and overlapping expression patterns are fully consistent with the partial redundancy of the *LAFL* described below. The *LAFL* transcripts are also detected in the endosperm although metabolic and developmental fates are different from that of the embryo. It suggests that additional regulators are required to specify the proper accumulation of different storage compounds in the two zygotic tissues.

1.2 Redundant and partially overlapping functions in controlling seed development

Genetic analyses have revealed the partially overlapping and synergistic functions of the *LAFL* genes during seed development (Raz et al., 2001; To et al., 2006; Braybrook and Harada, 2008; Santos-Mendoza et al., 2008; Verdier and Thompson, 2008; Yamamoto et al., 2014; Roscoe et al., 2015; Carbonero et al., 2016). The *lafl* mutants are all affected in seed reserve deposition, but the severity of the phenotypes increases in double and triple mutants, suggesting redundancy and synergism among the LAFL proteins. Consistently, the LAFL share several common targets(Yamamoto et al., 2010; Chiu et al., 2012; Monke et al., 2012; Wang and Perry, 2013; Baud et al., 2016). Moreover, effective complementation of either combinations of double mutants or the triple mutants by expression of individual *AFL-B3* confirms a functional redundancy among these regulators (To et al., 2006; Roscoe et al., 2015).

Nevertheless, clear differences have been reported in the level and specificity of the control exerted by each LAFL over the accumulation of fatty acid (FA), triacylglycerols (oil), and storage proteins, vascular differentiation, flavonoid and chlorophyll degradation, or desiccation tolerance for instance (Parcy et al., 1997; Lopez-Molina et al., 2002; Vicente-Carbajosa and Carbonero, 2005; Braybrook et al., 2006; To et al., 2006; Braybrook and Harada, 2008; Santos-Mendoza et al., 2008; Verdier and Thompson, 2008; Monke et al., 2012; Delmas et al., 2013; Yamamoto et al., 2014; Roscoe et al., 2015; Carbonero et al., 2016). That some ectopically expressed individual *AFL-B3* essentially restores reserve accumulation and morphology to *afl* embryos but not tolerance to desiccation nor dormancy also reveals that mid- and late-maturation programs are uncoupled and under the control of distinct LAFL. Conversely, failure to complement single *afl* mutants may also be due to aberrant vegetative development as a consequence of an excess of AFL factors (Roscoe et al., 2015). Taken together

these results suggest the existence of upper and lower thresholds of AFL-B3 pool required for proper seed development. Thus, although conservation of the B3 DNA-binding domain is essential for determining embryo morphology and accumulation of reserves, functional specialisation among the AFLs is likely to be determined by their distinct expression patterns and combinatorial interactions with other regulators as described hereafter in this review.

Mutations of the *LAFL* genes not only affect seed maturation but have also a more global developmental impact: the embryo bypasses the maturation phase and immediately enters vegetative development (Raz et al., 2001; Santos-Mendoza et al., 2008; Yamamoto et al., 2014). The cotyledons display characteristic traits of leaves, such as trichome development, anthocyanin accumulation, and precociously develop stomata and xylem elements, hence the name "Leafy Cotyledon" (Meinke, 1992). This diversity of mutant phenotypes could result from this homeotic modification. In *lec1*, the precocious expression of a seedling specific gene (*i.e*. *PYK10*) can be detected very early at the globular-heart transition stage of embryo development, before the activation of seed maturation genes (Yamamoto et al., 2014). This suggests that the heterochronic expression of seedling genes in *lec1* embryos is not only a consequence of a defect in the maturation program, but also results from the inability to maintain their repression during early embryogenesis. Consistent with this developmental role, the ectopic expression of *LEC1* and *LEC2* in vegetative tissues is sufficient to trigger the seed maturation program and somatic embryogenesis, whereas *lec1* and *lec2* mutants have lower ability to undergo somatic embryogenesis (Lotan et al., 1998; Stone et al., 2001; Gaj et al., 2005; Santos Mendoza et al., 2005; Suzuki et al., 2007; Stone et al., 2008; Gao et al., 2009; Junker and Baumlein, 2012; Feeney et al., 2013; Guo et al., 2013; Jia et al., 2013; Wojcikowska et al., 2013; Jia et al., 2014; Zhang et al., 2014b; Ikeuchi et al., 2015; Nowak and Gaj, 2016; Horstman et al., 2017; Magnani et al., 2017; Mozgova et al., 2017; Wójcik et al., 2017; Lee and Seo, 2018). This LEC1/LEC2 activity probably explains why the induction of the *LAFL* genes is accurately controlled during seed development and expression strongly repressed during vegetative growth.

2 Functions of the LAFL proteins

2.1 How the LAFL trigger seed maturation

Induction of seed maturation genes by the LAFL has been extensively analysed and the understanding of the molecular mechanisms involved has considerably progressed recently (Carbonero et al., 2016; Devic and Roscoe, 2016; Fatihi et al., 2016; Boulard et al., 2017). Briefly, the three AFL-B3 proteins bind consensus "RY" DNA motifs (core sequence 5'-CATG-3'), but with different specificities regarding the nucleotides flanking this core sequence. We have recently demonstrated that both LEC2 and ABI3 proteins can cooperate with LEC1 in multi-protein complexes to activate their target promoters (Baud et al., 2016; Boulard et al., 2018). On the contrary, no direct cooperative effect of LEC1 with FUS3 could be demonstrated, suggesting that the molecular mechanisms involving FUS3 are different, unless it requires other cis-DNA sequences and protein partners.

In addition to RY motifs, the presence of E/G-Box elements (5'-CANNTG-3'/ 5'-CACGTG-3') is also necessary for proper activation of the AFL-B3 target promoters *in planta* (for references see Abraham et al., 2016; Baud et al., 2016). It suggests that the AFL-B3 can cooperate with bHLH or bZIPs TFs that bind E/G-Box elements (see Plant Transcription Factor Database, http://planttfdb.cbi.pku.edu.cn), as demonstrated for ABI3 with bZIP10/25/53 (Lara et al., 2003; Alonso et al., 2009). In addition, other TFs of the MYB, MADS, DOF or AP2 families are also involved in the activation of maturation genes either as protein partners or as targets mediating the control exerted by the AFL-B3 toward downstream secondary targets. These transcription factors modulate and / or act in combination to specify the function of the LAFL, providing variations in the end-targets of the regulatory cascade and contributing to specify the type of storage compounds accumulated (i.e. oil, starch, or seed storage proteins) in the different zygotic tissues composing the seed (embryo or endosperm). Among the important TF targets of the LAFL are *WRINKLED1* (*WRI1*) and two closely related *MYB115/118* genes that play key roles in the regulation of oil deposition, controlling fatty acids biosynthesis and desaturation, respectively (Braybrook et al., 2006; Baud et al., 2009; Wang and Perry, 2013; Troncoso-Ponce et al., 2016; Ettaki et al., 2018).

Finally, the NF-YB LEC1 and L1L can interact with NF-YA and C-subunit proteins, as well as with other proteins including LEC2, ABI3, and some bZIPs (Yamamoto et al., 2009; Boulard et al., 2017; Gnesutta et al., 2017; Boulard et al., 2018). In association with these latter proteins LEC1 binds to genes that are involved in lipid metabolism and hormone signalling (Mu et al., 2008; Yamamoto et al., 2009; Junker et al., 2012; Mendes et al., 2013; Baud et al., 2016). This is consistent with the results of a recent genome-wide survey demonstrating that LEC1 controls distinct gene sets with different cis-regulatory elements from early to late stages of seed development (Pelletier et al., 2017).

2.2 LAFL and hormonal signalling

One important function of the LAFL relates to various plant hormone metabolisms and/or signalling, (Junker and Baumlein, 2012; Jia et al., 2014). For instance, during embryogenesis, the control of auxin biosynthetic genes by LEC1 and LEC2 could play a role in maintaining or inducing a totipotent cell state (Stone et al., 2008; Wojcikowska et al., 2013; Wójcikowska and Gaj, 2015). FUS3 also interacts with LEC2 to induce auxin biosynthesis during lateral root formation (Tang et al., 2017) and auxin induces *FUS3* expression (Gazzarrini et al., 2004) in a positive feedback loop regulation. FUS3 controls the critical ABA/GA balance by repressing GA biosynthesis and by promoting ABA accumulation, whereas ABI3 rather integrates ABA signalling (Curaba et al., 2004; Gazzarrini et al., 2004; Wang et al., 2004; Braybrook et al., 2006).

Moreover, LAFL activities are themselves controlled by hormone signaling through positive or negative feedbacks loop involving ABA and GA, respectively. Indeed, ABA has a positive role in the expression of *ABI3* (Lopez-Molina et al., 2002; Cheng et al., 2014), while GA has been shown to repress *FUS3* expression (Curaba et al., 2004; Gazzarrini et al., 2004; Wang et al., 2004; Braybrook et al., 2006). The transcription factor WRKY41, which induces the expression of *ABI3* during seed maturation, is negatively regulated by ABA (Kanno et al., 2010; Ding et al., 2014). In addition, ABA

and GA modulate ABI3 and FUS3 activity through post-translational regulations (Gazzarrini et al., 2004; Zhang et al., 2005). Last, brassinosteroids are involved in repression of *ABI3* expression and weak ABA sensitivity during germination (Ryu et al., 2014).

2.3 How the LAFL maintain embryogenic cell fate and repress vegetative development

While the function of the LAFL as positive regulators of seed maturation genes has been well establish their active role in repressing germination, seedling and vegetative development remains to be thoroughly investigated (Yamamoto et al., 2014; Yoshii et al., 2015). As described above, LAFL regulators are involved in the control of the hormonal ABA/GA balance. An elevated ratio such as the one found in embryo promotes maturation, whereas a lower ratio is associated with the activation of seedling development as in *lec1* or *fus3* (Curaba et al., 2004; Gazzarrini et al., 2004; Yamamoto et al., 2014; Liu and Hou, 2018). Interestingly, the precocious expression of PYK10 observed in lec1 and fus3 embryos is also found in ga deficient backgrounds (i.e. ga1 lec1 and ga1 fus3 double mutants), suggesting that the elevated gibberellin level found in lec1 or fus3 simple mutants is not the cause of the heterochronic gene expression in these mutants (Yamamoto et al., 2014). Moreover, there is no strict overlapping between the timing of *LAFL* expression and appearance of the abnormal phenotypes in corresponding *lafl* mutants. These observations together with the stochastic and cell-autonomous expression of *PYK10* in *lafl* mutant embryos and modification of the *PYK10* locus by histone modifications suggest the involvement of LAFL in epigenetic mechanisms repressing post-germination genes during embryogenesis.

3 LAFL gene regulations

3.1 Multiple transcriptional feedback regulations

Genetic analyses have shown that LAFLs are components of regulatory loops inducing their own expression. LEC1 and LEC2 can activate their own expression as well as the expression of *FUS3* and *ABI3*, depending on the tissue considered (To et al., 2006; Suzuki and McCarty, 2008; Monke et al., 2012; Wang and Perry, 2013; Zhu et al., 2013; Carbonero et al., 2016; Devic and Roscoe, 2016; Fatihi et al., 2016; Boulard et al., 2017; Pelletier et al., 2017). ABI3 and FUS3 also activate their own transcription. These positive feedback regulations probably contribute to enhance the activity of the network during seed development and are consistent with the occurrence of a minimum threshold for AFL-B3 pool to function (Santos-Mendoza et al., 2008; Yamamoto et al., 2014; Devic and Roscoe, 2016).

Several additional transcriptional regulatory feedbacks have been identified that affect the expression of the *LAFL* genes (Figure 1). For instance, LEC2 and FUS3 activate *AGL15* that in turn activates the *LAFL* genes in a positive loop (Zhu and Perry, 2005; Braybrook et al., 2006; Zheng et al., 2009; Wang and Perry, 2013; Jia et al., 2014). However, this positive feedback loop is limited by the fact that AGL15 negatively regulates its own expression. Moreover, the normal *LAFL* expression in *agl15* mutant seeds (Chen et al., 2018) suggests that paralogous MADS proteins could act redundantly with AGL15 to control the network. Another complex regulation involves the closely

related *MYB115* and *MYB118.* Their expression is induced by the LAFL in the endosperm of maturing seeds and they repress in turn *LEC2* expression in the endosperm. What is more, these MYB trigger *LEC1* expression and thus promote somatic embryogenesis when expressed ectopically in vegetative tissues (Wang et al., 2009; Zhang et al., 2009). Nevertheless, neither the *myb118* nor the *myb118 myb115* double mutants display obvious developmental defects (Troncoso-Ponce et al., 2016), suggesting that genetic redundancy also exists between these TFs. Last, the HD-ZIP transcription factors PHAVOLUTA (PHV) and PHABULOSA (PHB) induce *LEC2* during early embryogenesis (Tang et al., 2012b), and BABY BOOM (an APETALA2/ ERE-binding factor) directly binds *LAFL* gene promoters and activates their expression during somatic embryogenesis (Horstman et al., 2017).

Some negative regulations of the *LAFL* genes have been characterized too (Figure 1). The trihelix transcription factors ASIL1/2 inhibit *LEC2* and *FUS3* expression during both early embryogenesis and after germination (Willmann et al., 2011). The homeodomain proteins HIGH-LEVEL EXPRESSION OF SUGAR-INDUCIBLE GENE 2 (HSI2), and HSI2-like1 (HSL1), that have been also referred to as VP1/ABI3-Like1 (VAL1) and VAL2, respectively, inhibit the *LAFL* network in developing seedlings and are required to terminate the embryonic developmental program (Tsukagoshi et al., 2005; Suzuki et al., 2007; Tsukagoshi et al., 2007; Veerappan et al., 2012; Jia et al., 2013; Zhou et al., 2013; Jia et al., 2014; Veerappan et al., 2014; Chhun et al., 2016; Schneider et al., 2016). HSI2 and HSL1 can form homo or heterodimers that can bind promoter regions and inhibit the expression of *LAFL* and other target genes (Chhun et al., 2016). Nevertheless, recent experiments suggest that the dimers would rather bind *AGL15* promoter and indirectly repress the *LAFL* genes (Chen et al., 2018). Interestingly, FUS3 has been shown to activate *HSI2/VAL1* at the end of the maturation phase (Wang and Perry, 2013), triggering its own down regulation. These HSI/VAL proteins contain several domains involved in the interaction with chromatin-modifying proteins or the chromatin itself, highlighting the importance of these regulations for *LAFL* expression, as described below.

3.2 Chromatin-based regulations

Dynamic chromatin modifications and DNA methylation occur during seed development and germination (Muller et al., 2012; Yang et al., 2013; Kawashima and Berger, 2014; Molitor et al., 2014; Bouyer et al., 2017; Kawakatsu et al., 2017; Lin et al., 2017; Narsai et al., 2017). Nevertheless, the significant increase in DNA methylation observed at non-GC sites does not seem to be involved in the control of seed gene expression (Lin et al., 2017). On the contrary, *LAFL* genes are subject to extensive chromatin modification dynamics and numerous reports provide insight into the mechanisms involved (Figure 2). This is especially evident during the germination process, leading to the silencing of *LAFL* genes. In mature embryos, *ABI3* and *LEC2* are associated with H3K4me3, which marks active genes. These marks are replaced by negative H3K27me3 marks upon germination (Muller et al., 2012; Molitor et al., 2014). Hence, removal of histone acetylation (H3ac and/or H4ac), which is associated with active transcription, is a prerequisite to establish and maintain repression. Stable repression is carried out by the Polycomb Repressive Complexes PRC1 and 2 (Mozgova and Hennig, 2015). PRC2 has Histone3 Lysine 27 trimethylase (H3K27me3) activity and PRC1 carries out histone H2A Lysine ubiquitination (H2Aub). The canonical role of PRC1 is to recognize H3K27me3 and

confer chromatin compaction, although this view seems largely oversimplified. For instance, PRC1 also acts independently or upstream of PRC2 in both animals and plants (Gil and O'Loghlen, 2014; Merini and Calonje, 2015). Both PRC1 and PRC2 correspond to several complexes with various specificities and functions depending on protein partners and target genes (Wang et al., 2016; Merini et al., 2017). In addition, LHP1 that has been primarily associated with PRC1 function (Turck et al., 2007; Zhang et al., 2007) was also found to be associated with members of PRC2 (Derkacheva and Hennig, 2014), questioning the explicit separation of both complexes in Arabidopsis. Thus, the relationship between PRC1 and PRC2 may be specific to various complexes and target genes, although both are required for stable repression (Figure 2).

Consistent with the positive role of histone acetylation on *LAFL* expression, chemical inhibition of histone deacetylase (HDACs) activity and mutations in the closely related HDACS, *HDA6* and *HDA19,* maintain elevated H3K9ac and H4Kac over the 5' region of the *LAFL* genes and induce their expression in seedlings (Tanaka et al., 2008; Zhou et al., 2013). Moreover, two other closely related deacetylases (HDA7 and HDA9) negatively regulate seed germination (van Zanten et al., 2014), indicating that HDAC activity is required for both, the termination of seed maturation and the onset of germination programs. HDA19 can be recruited by the brassinosteroid-activated BES1 to repress *ABI3* expression during germination (Ryu et al., 2014). HDA6 and HDA19 would also be recruited respectively by VAL1/HSI2 and VAL2/HSL1 that interact with the H3K4me3 marks (Zhou et al., 2013; Chhun et al., 2016; Schneider et al., 2016). As discussed above, HSI2 and HSL1 could repress *LAFL* genes directly (Chhun et al., 2016) or indirectly through binding to and repression of *AGL15* (Chen et al., 2018). Apart from HSI/VAL, the GRAS-transcription factor SCARECROW-LIKE15 (SCL15) has been involved in conveying HDA19 to *ABI3* after germination, although the HDA19 binding to *ABI3* was unaltered in *scl15* mutant seedlings (Gao et al., 2015).

Several proteins involved in histone methylation have been shown to regulate *LAFL* expression and seed dormancy (Narro-Diego et al., 2017; Nonogaki, 2017). For instance, mutants affected in ATRX7 (H3K4 methyltransferase), EFS/SDG8 (H3K36 di- and trimethylation), EBS (an H3K4me2/3 “reader” interacting with HDA6) or HUB1 and HUB2 (H2B monoubiquitination) display lower *ABI3* expression and dormancy, whereas those affected in H3K9 methylation (*kyp/suvh4*) or H3K4 demethylation (*ldl1* and *ldl2*) display the opposite phenotype (Liu et al., 2007b; Bassel et al., 2011; Zheng et al., 2012; Zhao et al., 2015; Narro-Diego et al., 2017). Moreover, although PRC1/2 are not required for proper embryo development, mutations affecting PRC1 or PRC2 lead to a failure in both, the repression of *LAFL* expression and directly switch from embryo to seedling development (Chanvivattana et al., 2004; Makarevich et al., 2006; Aichinger et al., 2009; Bratzel et al., 2010; Chen et al., 2010; Berger et al., 2011; Bouyer et al., 2011; Muller et al., 2012; Tang et al., 2012a; Zhang et al., 2012; Deng et al., 2013; Yang et al., 2013; Molitor et al., 2014; Ikeuchi et al., 2015; Mozgova et al., 2015; Xiao and Wagner, 2015; Feng et al., 2016; Trindade et al., 2017; Xiao et al., 2017; Lee and Seo, 2018). Similar accumulation of seed proteins in vegetative tissues is also observed when the RETINOBLASTOMA-RELATED1 (RBR1) that is required to maintain the PRC2-dependent repression of *LEC2* and *ABI3* is affected (Gutzat et al., 2011; Kuwabara and Gruissem, 2014) and in mutant of the SWI2/SNF2

chromatin remodelling ATPase BRAHMA (BRM) (Tang et al., 2008), although BRM has been shown to controls the transition from juvenile to adult vegetative phase antagonistically to PRC2 (Xu et al., 2016).

PRC1 complexes initiate the repression of the embryonic program allowing H3K27me3 deposition and the establishment of more stable irreversible repression (Calonje, 2014; Mozgova et al., 2015; Xiao and Wagner, 2015; Zhou et al., 2017). LHP1 seems to have only a limited impact on this developmental switch (Wang et al. 2016), despite its binding to *ABI3* (Molitor et al. 2014). Besides its interaction with H3K4me3 and HDACs, VAL/HSI and ALFIN1- like (AL) proteins also recruit PRC1 to seed maturation loci such as *LEC2* (Bratzel et al., 2010; Yang et al., 2013; Molitor et al., 2014; Questa et al., 2016; Yuan et al., 2016; Merini et al., 2017; Chen et al., 2018; Lee and Seo, 2018). Consistently, *hsi2, hsl1*/*val1, and val2* mutants show reduced H3K27me3 and H2Aub levels at *LAFL* genes during germination. It is also likely that the PHD domain of HSI2 alone can repress target genes by binding with the repressive H3K27me3 mark (Veerappan et al., 2014). Therefore, this module involving the two PHD-domain proteins HSI2/VAL1 and ALFIN1-like (AL) might directly trigger the switch from an active (H3K4me3 marked) to a repressed, (H3K27me3 marked) state for *LAFL* genes. However, AL binding to *ABI3* was only detected at the end of the germination process (Molitor et al., 2014), suggesting that AL is implicated in maintenance of repression rather than in the initiation of the process.

The key role of HSI2/VAL1 in mediating *LAFL* silencing is emphasized by its interaction with MSI1 (Mehdi et al., 2016), a WD protein that is thought to bridge PRC1 and PRC2 function (Derkacheva et al., 2013). Beside its association with PRC2 and PRC1, MSI1 is also a member of the histone H3-H4 chaperone CAF1 complex (Ramirez-Parra and Gutierrez, 2007) and it needs to be determined how its association with HDA19 (Mehdi et al., 2016) and HSI2 (Chen et al., 2018) relates to these two distinct functions. PRC2 recruitment involves specific Polycomb Responsive Elements (PREs) that trigger binding of PRC2-associated transcription factors and subsequent H3K27me3 deposition. A first PRE motif named "*RLE*" (*Repressive LEC2 Element*) has been identified and functionally characterized, that is responsible for *LEC2* stable repression after the embryonic stage (Berger et al., 2011; Xiao et al., 2017). An orthologous sequence has been identified in the *FUS3* promoter (Devic M, unpublished) suggesting a common regulatory module of PRC2-mediated *LAFL* repression. The requirement for dynamic chromatin remodeling is further supported by recent reports on the synergistic activity of the normally antagonistically acting EMBRYONIC FLOWER 1 (EMF1) and trithorax group (TrxG) proteins *ATX1* and *ULT1* (Kim et al., 2012; Xu et al., 2018). Unexpectedly, the loss of TrxG enhances the de-repression of *LAFL* observed in *emf1* single mutants. Although there is no direct correlation with TrxG-associated H3K4me3 levels, both ATX1 and ULT1 bind to *LEC2* and *ABI3* locus and also interact with EMF1, suggesting a concerted activity to repress *LAFL* after embryogenesis (Xu et al., 2018). Alternatively, both PcG-dependent repression of embryo-specific transcripts, as well as elevated activation of post-embryonically expressed genes requiring TrxG function might be necessary to efficiently switch from seed to seedling development. This scenario would be in agreement with the highly dynamic chromatin modification changes observed during

this transition process.

Two ATP-dependent chromatin-remodelling proteins of the CHD3 family PICKLE (PKL) and PKR2 are also involved in the regulation of *LAFL* expression during germination and in vegetative tissues. The mutants fail to repress *LAFL* and display reduced H3K27me3 marking during germination (Dean Rider et al., 2003; Henderson et al., 2004; Zhang et al., 2008; Aichinger et al., 2011; Zhang et al., 2012; Han et al., 2015; Carter et al., 2016), emphasizing a requirement for active chromatin remodeling to switch between H3K4me3 and H3K27me3. Importantly, PKL has also been found to negatively regulate H3K27me3, underlining its role as nucleosome remodeler instead of being a constitutive polycomb cofactor (Aichinger et al., 2011; Ho et al., 2013; Jing et al., 2013). Moreover, it has been recently demonstrated that PKL significantly affects RNA-directed DNA Methylation (RdDM) at specific target loci, suggesting that it may be required to create a chromatin environment that influences non-coding RNA production, DNA methylation and transcriptional silencing (Yang et al., 2017a). Last, PKL has been implicated in GA signalling (and thus, germination) by interacting with DELLA proteins that are master repressors of GA signalling (Zhang et al., 2014a).

Less information is available about the role of chromatin dynamic in activating *LAFL* genes during seed development. Nevertheless PKL (CHD3 family) and paralogous CHR5 (CHD1 family) act antagonistically during seed maturation for establishing an active chromatin state allowing proper induction of *ABI3* and *FUS3* (Shen et al., 2015). In addition, mutations of *CLF* (which encodes a PRC2 subunit) lead to ectopic expression of *AGL15*, *FUS3*, and *ABI3* associated with enhanced accumulation of oil in seed (Liu et al., 2016). Moreover, consistent with the chromatin repression of *LAFL* expression and the role of LEC1 and LEC2 in somatic embryogenesis previously described, mutations affecting *PKL* or *VAL1* induce *LAFL* expression and improve the efficiency of somatic embryogenesis (Horstman et al., 2017). Similarly mutation affecting *FIE* an essential component of PRC2, results in abnormal vegetative development that resembles somatic embryogenesis (Bouyer et al., 2011). These results are fully consistent with the simultaneous repression of *LAFL* expression and loss of zygotyc embryo potential for somatic embryogenesis that occur during germination (Horstman et al., 2017; Mozgova et al., 2017).

3.3 Structure function relationships of *LAFL* promoters

Molecular dissections and functional analyses of promoter regions and the subsequent identification of important *cis*-elements and *trans*-acting factors have brought further information on the regulations of *ABI3* (Ng et al., 2004) and *LEC2* (Berger et al., 2011), but are still missing for *FUS3* or *LEC1*. The promoter region necessary for high and accurate expression of *ABI3* is unusually long (Ng et al., 2004). Fragments of 4.6 or 3.6 kb upstream the transcription start site, provide the best level of activation. Loss of a further 1.6 kb results in a severe decrease of promoter activity. Deletion of an additional 1.2 kb almost completely abolishes *ABI3* transcription. Equally, the 5'UTR of *ABI3* mRNA of 541 bp (previously estimated at 519 bp) is much longer than the average size of 155 bp for Arabidopsis genes (Srivastava et al., 2018). This sequence has a negative effect on *ABI3* expression, probably affecting translation. It has been shown that the homeodomain transcription factors BLH1

and KNAT3 activate *ABI3* gene expression (Kim et al., 2013), whereas RAV1, a TF of the B3-Family represses *ABI3* expression upon seed imbibition (Feng et al., 2014). But only a few functional *cis*-elements and their corresponding factors (e.g WRKY41) have been thoroughly characterised in the *ABI3* promoter to date (Ding et al., 2014).

All the regulatory elements of the promoter required for proper expression of *LEC2* are present within the 447 bp sequence upstream the translation start codon. The developmental regulation of *LEC2* expression results from a combination of activating and repressive mechanisms involving at least three distinct *cis*-regulatory elements located within the proximal *LEC2* promoter (Berger et al., 2011). Two of them, a *CArG* regulatory elements recognized by MADS box transcription factors and a GAGA box bound by plant BPC (BASIC PENTACYSTEINE) proteins are essential for the transcriptional activation of *LEC2* during seed development. The third element named *RLE (Repressive LEC2 Element)* was the first plant functional homolog of a PRE element (Berger et al., 2011). PREs have been extensively characterized in Drosophila and are necessary and sufficient for H3K27me3 deposition and transcriptional repression (Ringrose and Paro, 2007), which also characterizes the *RLE*. Chromatin Immunoprecipitation (ChIP) experiments suggested that both RLE and GAGA element provide sites for PRC2 binding, although the factor binding these elements remain to be identified (Berger et al., 2011; Deng et al., 2013; Hecker et al., 2015). BPC factors are also necessary for the repressive action of PRC1 (Hecker et al., 2015) and PRC2 (Mu et al., 2017a). In cucumber, BPC factors bind the *ABI3* promoter to repress its activity and control seed germination (Mu et al., 2017b). The repressive action of BPC together with PRC2 could therefore constitute a widespread mechanism restricting the expression of *LAFL* to seed development (Mu et al., 2017a).

Similarly, the expression of *FUS3* is regulated by positive and negative *cis*-elements. A 2 kb upstream sequence of the *FUS3* gene contains all the necessary elements allowing proper *FUS3* expression and to complement the *fus3-3* mutation (Gazzarrini et al., 2004), and a shorter sequence of 500 bp is also sufficient (Roscoe et al. unpublished). Several activating transcription factors directly recognising the promoter have been identified mainly by ChIP (as described above, e.g. AGL15/18, LEC2, or LEC1). Sequences recognised by factors participating in epigenetic regulation of *FUS3* have also been characterised. MEDEA, a subunit of PRC2, binds to the promoter of *FUS3* on a defined 126 bp fragment (Makarevich et al., 2006). Binding of this PRC2 subunit is thought to repress *FUS3* expression in the endosperm. On the contrary, binding of CHR5 around the transcription start site regulates nucleosomes occupancy and enhances *FUS3* expression (Shen et al., 2015).

No detailed analyses of the *LEC1* promoter have been performed, although a genomic fragment of 3.4 kb, which includes 2.5 kb of promoter, was sufficient for complementation of the *lec1* phenotype (Lotan et al., 1998). On this line, about 2kb of *LEC1* promoter allowed the complementation of *lec1* with *L1L* (Kwong et al., 2003). To gain further insight into the transcriptional regulations of *LAFL* expression, the promoter-deletion studies could be combined with a comprehensive mapping of transcription factor-promoter interactions for the four LAFL regulators (Castrillo et al., 2011; Gaudinier et al., 2017). Since there is a positive correlation between the number of TF bound to a

promoter and its developmental importance, we can expect to identify supplementary TF binding to the *LAFL* promoters. For example, more than 30 TF recognise the promoters of *REV* and *PHB* in comparison to the average number of 3.36 TF per promoter reported in Arabidopsis (Brady et al., 2011; Gaudinier et al., 2011). These results should allow a comparison of the transcriptional regulation of *LAFL* genes and discriminate the common *cis*-elements/*trans*-acting factors from the specific ones.

3.4 Post-transcriptional and post-translational regulations

Overall, miRNA are involved in the control of several aspects of plant development, including seed and embryo development (for review see Seefried et al., 2014; D'Ario et al., 2017; Rodrigues and Miguel, 2017). For instance, loss of *DICER-LIKE 1* (*DCL1*), the major RNA helicase involved in *miRNA* processing leads to embryo lethality (Golden et al., 2002; Park et al., 2002). During early embryogenesis, *miR156* represses *LEC1, FUS3* and *ABI3* expression by silencing *SPL10* and *SPL11*, therefore preventing precocious onset of the maturation program (Nodine and Bartel, 2010; Willmann et al., 2011; Vashisht and Nodine, 2014). It is not clear whether *LAFL* are direct targets of SPL10/11. The HD-ZIP PHABULOSA (PHB), which recognises *cis*-elements in the *LEC2* promoter, and PHAVOLUTA (PHV) are targets of *miR166* (Tang et al., 2012b). It is hypothesised that *miR156* could act together with miR166 to repress the maturation phase in the leaves. In addition, *miR165/166* together with *miR160* (which targets the AUXIN RESPONSE FACTORS ARF10, ARF16, and ARF17) contribute to the control of somatic embryogenesis involving LEC2 (Wójcik et al., 2017) and the repression of ARF10 by *miR160* is involved in the regulation of ABA-responsive genes (Liu et al., 2007a). The regulation of *LAFL* by miRNA is an integral part of the network's feedback regulatory loop. For instance, *miRNA156* is activated by FUS3 (Wang and Perry, 2013) as well as AGL15/AGL18 (Serivichyaswat et al., 2015). In return, AGL15/18 activate *ABI3*, *FUS3* and *LEC2* (Zheng et al., 2009), but are also activated by FUS3 (Wang and Perry, 2013) and LEC2 (Braybrook et al., 2006).

Alternative splicing has been shown to produce truncated ABI3 proteins in numerous species including Arabidopsis, tomato, wheat, rice, pea or flax (McKibbin et al., 2002; Gao et al., 2013; Wang et al., 2018). Developmentally regulated alternative splicing may contribute to a fast inhibition of the seed maturation program in germinating Arabidopsis seed (Sugliani et al., 2010). The mis-splicing of *VP-1*, the wheat *ABI3* ortholog, yields truncated polypeptides that could be involved in the susceptibility to pre-harvest sprouting (McKibbin et al 2002). In pea, several ABI3 isoforms with different in vitro activity were identified (Gagete et al., 2009; Gao et al., 2013). But in most cases, because ABI3 is a multi-functional protein with several acidic and basic sub domains (A1, B1, B2, B3), the biological significance of *ABI3* alternative splicing remains to be further investigated.

Last, posttranslational controls involving protein phosphorylation and ubiquitination play an important role in hormone signalling, including ABA (for review see Yang et al., 2017b). Among the LAFL, ABI3 and FUS3 have been shown to be short-lived proteins. ABI3 is ubiquitinated after interaction with ABI3-INTERACTING PROTEIN 2 (AIP2) E3 ligase and subsequently degraded by the proteasome (Zhang et al., 2005). FUS3 contain a C-terminal PEST domain necessary for targeting to

degradation and for FUS3 function, since C-terminal truncated FUS3 is unable to complement the *fus3* phenotype (Lu et al., 2010). On the contrary, when phosphorylated at the N-terminus by AKIN10, FUS3 is stabilised (Tsai and Gazzarrini, 2012). This phosphorylation of FUS3 is necessary for proper embryogenesis and integration of environmental cues (Chan et al., 2017). FUS3 also interacts with AIP2 leading to its degradation during early embryogenesis preferentially in the protoderm (Duong et al., 2017). Altogether, these positive and negative post-translational mechanisms allow a rapid adjustment of the protein level within the cell. At present, it is not known whether LEC1 and LEC2 stability are also regulated.

4 Conclusions

The LAFL regulators fulfil crucial functions in angiosperms promoting embryogenesis and seed maturation, and preventing precocious germination. These multiple roles of the LAFL directly activating maturation genes, maintaining embryonic cell fate and repressing vegetative growth, and their partial functional redundancy with intricate cross-talks and feedback loop regulations have hampered the elucidation of the underlying molecular mechanisms. Recent progress has been made by combining various genetic, molecular, cellular, *in vivo*, and *in vitro* approaches. However, our understanding of the network remains limited by the paucity of information available on the spatio-temporality of these multiple controls. It is crucial to apply novel approaches to monitor the molecular events (i.e. chromatin and transcriptome profiling, proteins interactions, hormone levels) directly within the seed, in a cell-specific manner. Recent developments combining INTACT or laser-assisted micro-dissection with RNA-seq, ATAC- and CHIP-seq (Belmonte et al., 2013; Buenrostro et al., 2015; Chan et al., 2016; Kelsey et al., 2017; Libault et al., 2017; Magnani et al., 2017; Sakai et al., 2018; Sijacic et al., 2018) may provide more precise and quantitative data to draw a comprehensive and predictive model of the LAFL network.

Consistent with the key functions of the LAFL regulators, their activity is tightly controlled by numerous epigenetic, transcriptional, post-transcriptional and post-translational regulators. This is well exemplified at the transcriptional level, by the co-existence of positive and negative regulatory elements in the promoter of *LEC2* and *FUS3* that provides robust transcriptional control based on competition between activating and repressing complexes. Altogether, these mechanisms finely tune LAFL activity throughout seed development (*i.e*. the strong and cell specific expression in the embryo followed by an inhibition during late maturation and vegetative growth). It would be interesting to investigate whether the LAFL network or any of its components respond to environmental changes, and possible participate to seed resilience to biotic or abiotic stresses. This could provide interesting targets for crop improvement under current climate change (Fatihi et al., 2016).

Although it is well established that *LAFL* repression is accompanied by elevated chromatin dynamics, this picture has been derived mainly from studies in post-embryonic tissues and we still lack information how and when the repression is initiated during the seed maturation phase. Moreover,

little is currently known about the role of chromatin modifying mechanisms that are involved in *LAFL* activation during embryogenesis. Thus, it would be interesting to further investigate the epistatic relationship between LAFL and chromatin regulators, by establishing mutant combinations and monitoring their phenotypic and molecular consequences. In addition, it may be of interest to use inducible alleles of LAFL or chromatin regulators, allowing to ectopically activating wild-type or dominant negative regulators, to precisely dissect their role with clear temporal resolution.

Several results suggest that LEC1 and LEC2 may act both, as direct transcriptional activators and as pioneer factors (Mayran and Drouin, 2018; Zaret, 2018) able to trigger chromatin modifications and binding of different TFs. Indeed, LEC1 and LEC2 have homeotic functions often associated with chromatin modifications: they maintain embryonic cell fate and repressed precocious expression of seedling genes (e.g. *PYK10*), their ectopic expression is sufficient to trigger somatic embryogenesis, and *LEC2* is involved in the maturation program triggered in *prc2* mutant (Ikeuchi et al., 2015). Moreover, several LEC1/LEC2 target genes (e.g. FUS3 or ABI3) contain PREs and/or are induced in prc mutants (Merini et al., 2017; Xiao et al., 2017), and PRC1 and VAL1/2 co-regulate a high number of genes the promoters of which are significantly enriched in both RY and G-box elements (Wang et al., 2016; Merini et al., 2017) found in LEC1/LEC2 target promoters.

Different NF-Y complexes have been shown to act as pioneer factors in animals or in plants (Hou et al., 2014; Belluti et al., 2018). Therefore, LEC1, as component of NF-Y complexes, may act as pioneer TFs in diverse developmental processes (Pelletier et al., 2017). This hypothesis has been recently nicely supported by the discovery that LEC1 promotes the initial establishment of an active chromatin state at a flowering locus (*FLC*) during early embryo development (Tao et al., 2017). LEC1 directly binds *FLC,* promotes H3K4me3 and H3K36me3 deposition and inhibits antagonist H3K27me3 by engaging EFS/SDG8 and SWRIc, a chromatin remodelling complex (Tao et al., 2017). This conclusion is fully consistent with the lower expression of *ABI3* (a LEC1 target) observed in sdg8 mutant. In addition, it has been shown that *FLC* is also regulated by PRC1 and VAL1/VAL2 (Questa et al., 2016; Yuan et al., 2016).

Taken together these results strongly support that LEC1 and possibly LEC2 act as pioneering factors by direct binding to DNA and recruiting different chromatin regulators that remain to be characterized. Last, *VAL1* and *RING1b* (encoding a PRC1 subunit) are direct targets of FUS3 (Wang and Perry, 2013) and *PKL* expression is enhanced when *LEC1* is over-expressed (Mu et al., 2008), suggesting that LAFL could have a role in inducing the chromatin negative feedback regulation of their own expression, at the end of the maturation phase.

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Figure Legends

Figure 1 Overview of the dynamic regulation of *LAFL* gene expression. Brown and green boxes contain activators and inhibitors of LAFL genes, respectively. They are grouped according to demonstrated genetic or molecular interactions or functions (target genes, signalling pathways). Arrows indicate demonstrated or putative sequential order of activity during seed development. Details and references are given in the text.

Figure 2. Chromatin based mechanisms of LAFL silencing. Model describing interactions at the *LAFL* gene location, leading form a transcriptionally active state (H3K4me3 and H3/H4ac marked) to a repressed state (H3K27me3 marked). The VAL1/2 B3 domain transcription factor has DNA-sequence specific binding activity and contains repressory as well as a PHD chromatin binding domain and serves as a hub to recruit chromatin modifying components, such as HDACs as well as PRC1 and PRC2 components (red colour key). Recruitment of PRC1 to *LAFLs* is also mediated by the H3K4me3 binding AL PHD domain protein. EMF1, a PRC1/2 co-factor interacts with the TrxG proteins ULT1 and ATX1 (H3K4me3 mediating activity not shown for *LAFLs*), which synergistically add to H3K27me3 (PRC2)-mediated *LAFL* repression. The CHD chromatin remodeller PKL is able to shift nucleosome position and possibly involved in changing chromatin conformation. Once repression is established, H3K27me3 and H2Aub (PRC1-mark) are stably maintained and the repressive complex is permanently bound.

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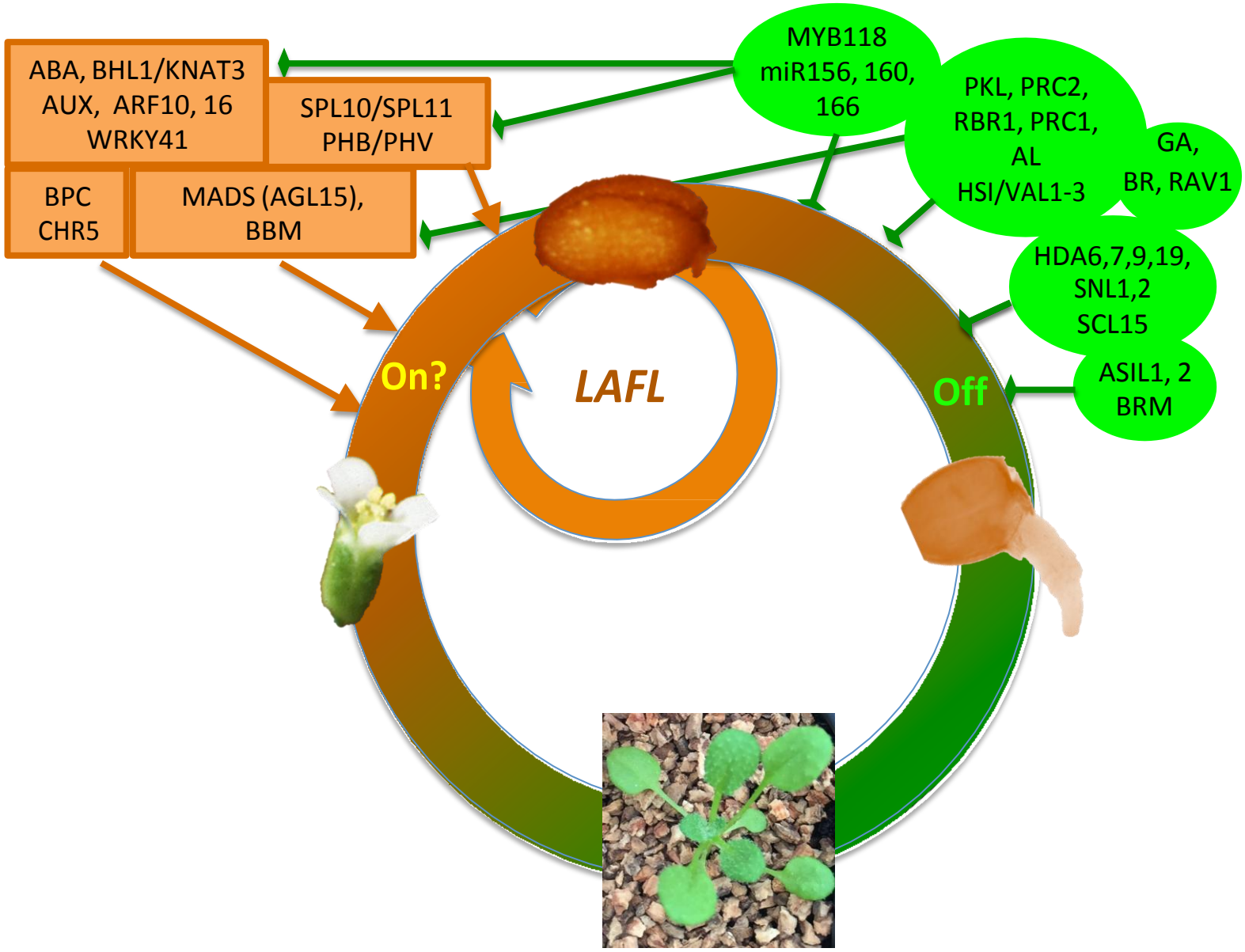
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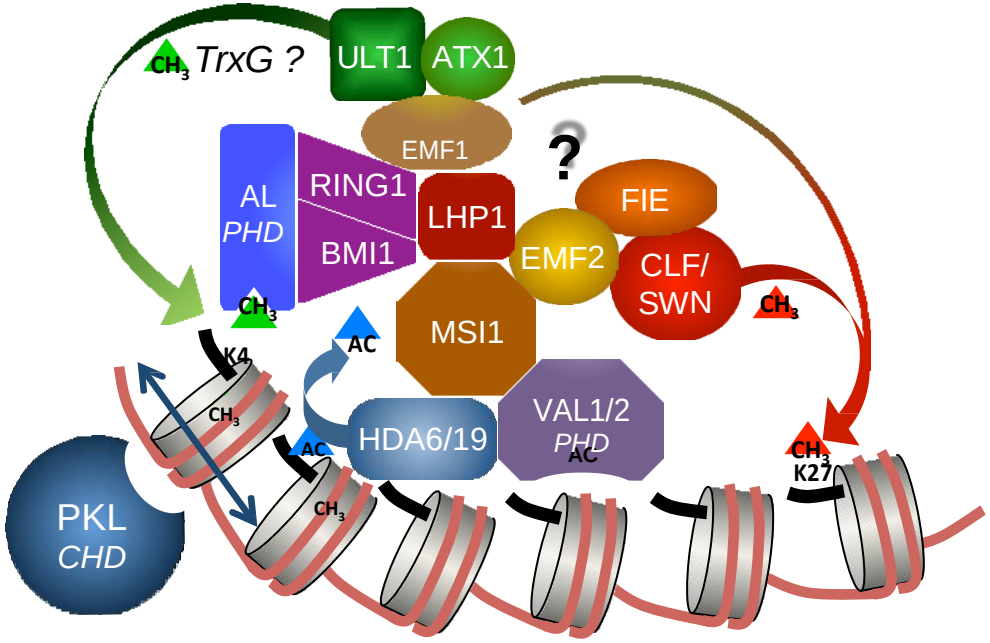

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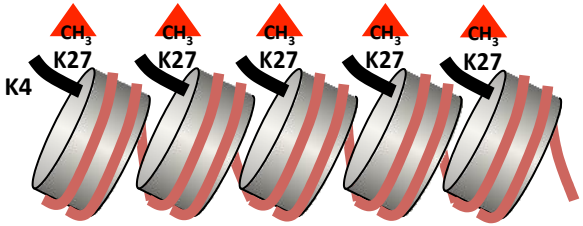

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