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Review article

Deciphering and modifying LAFL transcriptional regulatory network in seed for improving yield and quality of storage compounds

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ABSTRACT

Increasing yield and quality of seed storage compounds in a sustainable way is a key challenge for our societies. Genome-wide analyses conducted in both monocot and dicot angiosperms emphasized drastic transcriptional switches that occur during seed development. In *Arabidopsis thaliana*, a reference species, genetic and molecular analyses have demonstrated the key role of LAFL (LEC1, ABI3, FUS3, and LEC2) transcription factors (TFs), in controlling gene expression programs essential to accomplish seed maturation and the accumulation of storage compounds. Here, we summarize recent progress obtained in the characterization of these LAFL proteins, their regulation, partners and target genes. Moreover, we illustrate how these evolutionary conserved TFs can be used to engineer new crops with altered seed compositions and point out the current limitations. Last, we discuss about the interest of investigating further the environmental and epigenetic regulation of this network for the coming years.

Contents

1. Introduction 199
2. Master regulators of seed development and maturation 199
   2.1. *LAFL* genes 199
   2.2. Epigenetic regulation of *LALF* genes expression 199
   2.3. LAFL targets and partners 200
3. Engineering new crops with modified storage reserves 200
   3.1. LAFL functions are conserved in angiosperms 200
   3.2. Using *LALF* and *WRI* genes for engineering crops 202
4. Conclusions and prospects 202
   Acknowledgments 202
   References 202

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1. Introduction

Seed and grain productions are strategic for agriculture, food supply, economy and, more broadly, humankind. They provide half of the world's intake of dietary proteins, oils and starch and green renewable carbon resources (e.g. oil or starch) that represent an alternative to fossil hydrocarbon chains for chemical industries. Thus, there is a need to develop new strategies to modify the composition of oil, storage proteins or carbohydrate in crops for specific industrial uses. These issues are compounded by the dwindling of arable lands and the forecasted climate change, characterized by an increase in average temperature, already impacting negatively seed production [1]. In this context, increasing yield and quality of seed storage compounds represents a global key challenge not only for agriculture but, more broadly, for our societies and industries.

Seed development in both monocots and dicots has been well described [2]. Proper seed formation requires the coordinated development of diploid embryo, triploid endosperm, and maternal testa, as well as inter-compartmental signaling [3]. In exalbuminous species, such as Arabidopsis, embryo growth occurs at the expense of the endosperm, which progressively degenerates. Finally, the embryo, which is surrounded by a one-cell layer of remaining endosperm (i.e. the aleurone layer), fills the mature seed (Fig. 1). Contrastingly, mature cereal grains are mainly composed of a large endosperm, whereas the embryo remains small in size. During seed maturation, photo-assimilates are converted into storage compounds in the form of oil, storage proteins, or starch. The nature and location of these reserve sin the mature seeds are highly variable depending on the species considered [2] (Fig. 1). Starch constitutes the main reserve in the endosperm of cereal grains [4,5]. Embryos can store mainly oil (e.g. rapeseed), proteins (e.g. soybean) or starch (e.g. pea) [6,7]. Some species (e.g. sugar beet or coffee) accumulate their reserves in a perisperm originating from the maternal nucellus [8].

Arabidopsis seed development and maturation have been extensively described [9]. The metabolic routes leading to the accumulation of the main storage compounds (i.e. oil or proteins), which determine seed quality, are well characterized too [7]. Genome wide transcriptional profiles have been established at different stages of seed development [2,10]. Besides an overall elevated transcriptional activity observed during the maturation phase, these analyses have revealed that the late embryonic phase is characterized by a fundamental shift in gene expression profiles, with many genes being specifically active during this phase. This has been confirmed in other plants including cereals such as maize and rice [11,12]. Taken together these results suggest that important transcriptional switches are required to trigger and orchestrate the maturation process and the accumulation of storage compounds, which define to a large extent seed quality. Consistent with this idea, a complex network of transcriptional regulators that controls seed maturation has been characterized and is presented below.

2. Master regulators of seed development and maturation

Several seed transcriptional regulators have been identified and characterized in Arabidopsis as well as in other plant species. They control different aspects of seed growth and development [13]. Most of them regulate testa or early endosperm and embryo development, and/or are involved in hormone signaling and cell division [14,15]. Here we focus on the members of the LAFL regulatory network that controls seed maturation, their protein partners and their target regulatory genes.

2.1. LAFL *genes*

The Arabidopsis *LAFL* genes code for LEAFY COTYLEDON1 (LEC1), ABSCISIC ACID INSENSITIVE3 (ABI3), FUSCA3 (FUS3), and LEC2, respectively [16–19]. Arabidopsis LEC1, one of the first characterized regulators of seed development, is a member of the NF-YB protein family (HAP3 subunit of the CCAAT-box binding factors). ABI3, FUS3, and LEC2 belong to the conserved, plant specific, family of B3 domain transcription factors and thus are collectively named AFL-B3. *LEC1* is expressed in developing seeds at early stages of embryogenesis from the globular to the bent-cotyledon stages whereas *LEC2* is weakly and specifically expressed at early stages of embryogenesis, with highest mRNA levels detected between the pre-globular and bent-cotyledons stages (Fig. 2). By contrast, *ABI3* and *FUS3* accumulate during late stages of embryogenesis. Interestingly, *LAFL* transcripts are also detected in the endosperm, suggesting that additional regulators are required to specify the proper accumulation of different storage compounds in the endosperm and embryo (see Section 2.3).

Characterization of *lafl* single and multiple mutants has demonstrated that the LAFL proteins have partially overlapping and synergistic functions during embryogenesis and seed maturation [16–20]. The structural similarity between the AFL-B3 proteins and their resulting ability to bind similar DNA cis-elements (see Section 2.3) account for this functional redundancy. In addition, the expression of *FUS3* and *ABI3* is controlled by a network of partially redundant regulations that involves LEC1 and LEC2 and, more surprisingly, FUS3 and ABI3 themselves, through positive regulatory feedback loops (Fig. 3). Moreover, we have recently demonstrated that the AFL-B3 proteins can take part in multi-protein complexes, which include LEC1 or LEC1-like, to control the expression of their target genes [21]. Nevertheless, the different LAFL also have more specific functions [17,19][17,19 and references therein]. For instance, ABI3 is involved in chlorophyll degradation in late maturing seeds [22], but has only a limited impact on oil synthesis and accumulation, in comparison to FUS3 [19]. Conversely LEC1, LEC2, and FUS3, but not ABI3, control embryo identity, and loss of their function induces the formation of true leaves in embryos (instead of cotyledons) that display trichomes and accumulate anthocyanin (instead of storage compounds) [17]. Finally, ectopic expression of *LEC1* and *LEC2* in vegetative cells can induce somatic embryogenesis and *lec1* and *lec2* mutants have lower ability to undergo somatic embryogenesis [23,24 and references therein] suggesting an essential role of these genes in embryonic differentiation. This surprising ability of *LEC* genes to induce somatic embryogenesis provides a possible explanation for the establishment of strong and apparently redundant genetic and epigenetic mechanisms that repress their expression in vegetative tissues [25][25 and references therein].

2.2. *Epigenetic regulation of* LALF *genes expression*

LALF are repressed in vegetative tissues by Polycomb Repressive Complexes (PRC) 1 and 2 that are responsible for histone H2A monoubiquitination and trimethylation of histone H3 on lysine 27 (H3K27me3), respectively [26–30]. Consistent with these results, *FUS3* and *ABI3* are de-repressed in *curly leaf* (*clf*) mutant seeds (*CLF* encodes one of the methyltransfererase of PRC2 complexes), leading to ectopic accumulation of oil and enhanced seed growth [31]. Moreover, *LAFL* genes are also marked by the activating H3K4me3 modification in seed, indicating that both, their inhibition as well as their activation, are tightly regulated by chromatin-based mechanisms [29,32]. Consistently, two chromatin remodelers of the CHD3 family (PICKLE and PRK2) are also involved in H3K27me3-dependent *LAFL* repression, although how these proteins affect PRC2 function remains a matter of debate [33–35]. Conversely, proper induction of *ABI3* and *FUS3* in seeds, requires another chro-