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Review

Plant design gets its details: Modulating plant architecture by phase transitions

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ABSTRACT

Plants evolved different strategies to better adapt to the environmental conditions in which they live: the control of their body architecture and the timing of phase change are two important processes that can improve their fitness. As they age, plants undergo two major phase changes (juvenile to adult and adult to reproductive) that are a response to environmental and endogenous signals. These phase transitions are accompanied by alterations in plant morphology and also by changes in physiology and the behavior of gene regulatory networks. Six main pathways involving environmental and endogenous cues that crosstalk with each other have been described as responsible for the control of plant phase transitions: the photoperiod pathway, the autonomous pathway, the vernalization pathway, the temperature pathway, the GA pathway, and the age pathway. However, studies have revealed that sugar is also involved in phase change and the control of branching behavior. In this review, we discuss recent advances in plant biology concerning the genetic and molecular mechanisms that allow plants to regulate phase transitions in response to the environment. We also propose connections between phase transition and plant architecture control.

1. Introduction

1.1. Phase changes: how many transitions are there and what is their importance?

Post-embryonic plant growth and development include three major phases: the juvenile, adult vegetative, adult reproductive, and to go from one phase to the next, plants undergo what is called 'phase change', or 'phase transition'. These are terms commonly used in the literature as a proxy to flowering time, which is the most discussed and studied event in a plant's lifespan, once it has an evident economic and scientific importance (Balanzà et al., 2018; Immink et al., 2012; Jaeger and Wigge, 2007; Parcy, 2005; Poethig, 2003). In nature, the correct timing of the adult vegetative-to-reproductive phase transition may enhance plant reproductive success and fitness. Thus, plants have evolved a complex network for controlling this phase transition in response to environmental and endogenous signals, such as temperature, day length, hormone concentrations, and carbohydrate content (Srikanth and Schmid, 2011). The correct timing for entering the reproductive phase is not only important in an evolutionary context but as well in a scientific and economic context. By controlling flowering time, it is possible to increase the yield flowers, and fruits and grains.

Nevertheless, it is necessary to point out that, during the lifecycle of a plant, there is another phase transition as important as the adult-to-reproductive transition and it is the juvenile-to-adult vegetative phase change, that is neglected most of the times. This is probably due to the short duration and subtle cues of the juvenile phase in the model plant *Arabidopsis thaliana*, and also due to the less evident economical importance, once there is no fruit production involved on this phase.

The juvenile-to-adult phase change is biologically important as it is responsible for establishing the 'body plan', the architecture of the body of each plant species, which affects its biomass production, its interaction with the surrounding environment, and the posterior crop yield. Consider for instance the body architecture of a tree (a perennial ever-growing plant) and that of *Arabidopsis* (an annual herb). In a food production context, crop production is a major issue to be considered, and the plant architecture strongly influences yield. It is also important to understand the timing of this first phase change during the aging of forest trees, due to its impact on wood production, and in studies concerning climate change, once maintaining the juvenile phase (or delaying the reproductive phase) might increase the rate of growth and biomass gain (Etterson, 2001; Wendling et al., 2014). Nonetheless, by

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delaying the reproductive phase of herbaceous plants, it will not turn them into a tree, but they might produce more biomass and more branches, which eventually could result in higher production of flowers and fruits.

If more branches mean a greater production of biomass and flowers, besides phase transitions, the plant architecture also plays an important role in the evolutionary success and crop yield efficiency. Considering an evolutionary background, some herbaceous species which are native from ecosystems with dense vegetation face a large competition for light. Thus, some plants evolved climbing strategies to reach the canopy, which are related to plant architecture (Cutri et al., 2013; Smith and Whitelam, 1997). Several species of climbing plants show an extended juvenile phase in which they self-suport before finding another plant or surfaces to support them on their way to the canopy (Caballé, 2011). Economically, by controlling plant architecture is possible to improve crop yield efficiency (Lemmon et al., 2018; Varkonyi-Gasic et al., 2019). A higher branching pattern (which could be obtained by an extended juvenile phase) could implicate in more floral meristems once the plant reaches its reproductive status, increasing fruit/grain production, which is not always true (Miura et al., 2010). In some species like *Mangifera* spp. and *Citrus* spp., there is a high rate of flower buds and fruit abortion due to the energy demand for the maintenance of the fruits. Studies

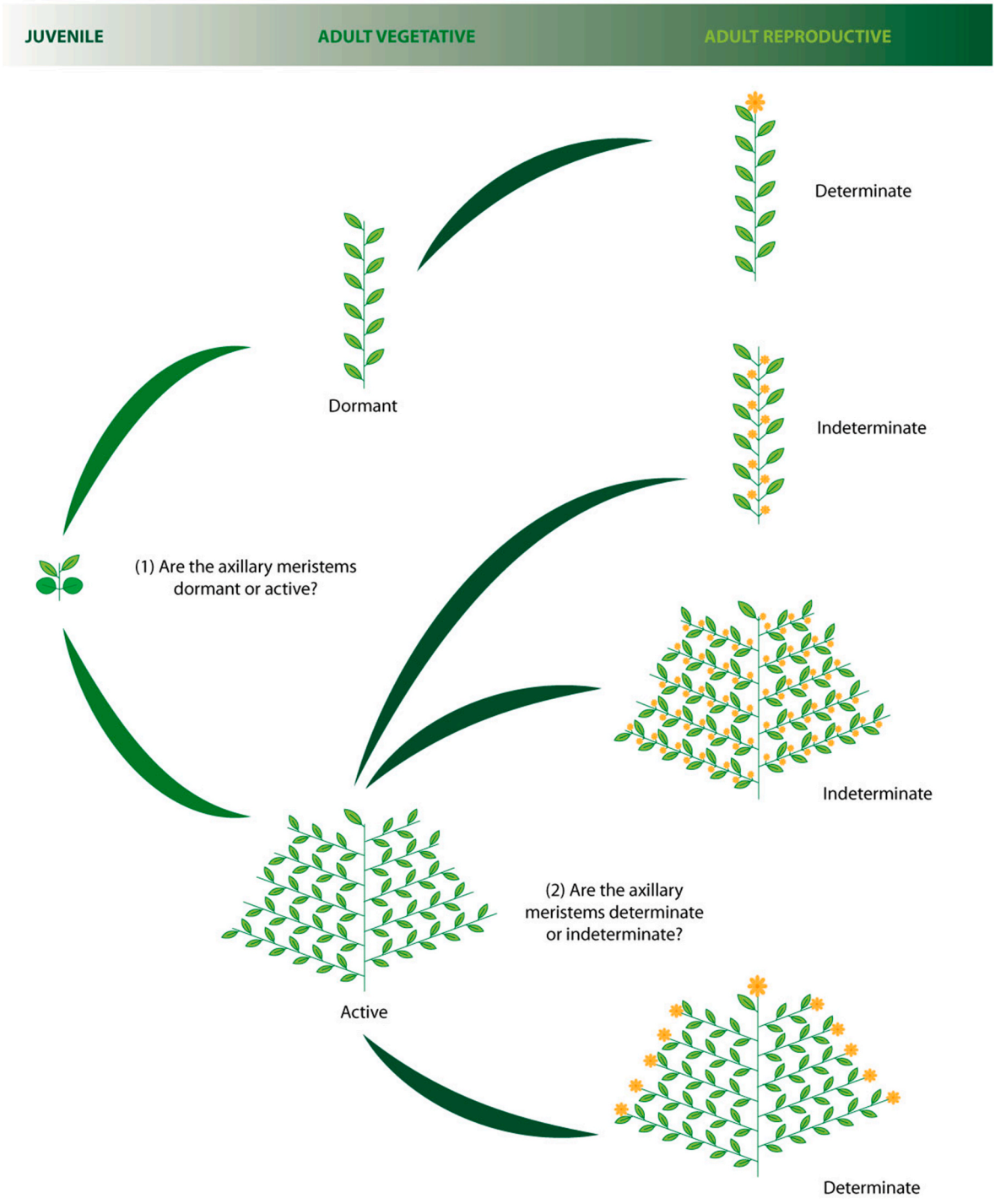

Fig. 1. Meristem decisions. Plant architecture is regulated in space and time by two different developmental decisions **(1)** Are the axillary meristems dormant or active? **(2)** once they are active, would they be determinate or indeterminate?.

concerning the improvement of herbaceous plants like tomato or the 'groundcherry' (*Physalis pruinosa*) have demonstrated that by changing the meristem fate to a determinate growth pattern increased flower and fruit production (Lemmon et al., 2018). Yet, other studies have shown that by CRISPR/Cas9-mediated manipulation, it was possible to transform a climbing woody perennial plant, that has a long juvenile phase before the production of axillary inflorescences into a compact plant with an accelerated production of a terminal flower followed by the fruit development (Varkonyi-Gasic et al., 2019).

1.2. The relationship between phase change and plant architecture

In plants, both flowering and plant architecture are simultaneously controlled by complex networks that are intimately connected and involve environmental and endogenous factors that determine whether to flower and the number of growing apical shoots. Several studies relating flowering time and branching suggest that the genetic networks regulating both phenomena are connected, and hormones could be the "missing link". (Achard et al., 2007; Busch et al., 2011; Chang et al., 2018; Frankowski et al., 2014; Lee et al., 2019; Poethig, 2013; Porri et al., 2012; Rameau et al., 2015; Schmitz et al., 2002; Schumacher et al., 1999; Weng et al., 2016). Nevertheless, other studies suggest that the carbohydrate availability is the major integrator of age, architecture and flowering time control (F. F. Barbier et al., 2015; Mason et al., 2014; Poethig, 2013; Yang et al., 2013; Yu et al., 2015, 2013).

The plant architecture depends on the pattern of shoot branching, which is regulated in space and time by the dynamics of the alternative activation of axillary meristems or their maintenance in a dormant state. Thus, different developmental decisions are at stake: (1) Are the axillary meristems dormant or active? (2) once they are active, would they be determined or indeterminate? (Fig. 1). These decisions are taken by the internal wiring of molecular pathways that are species-specific and that respond to several endogenous and environmental stimuli (Aguilar-Martínez et al., 2007; Blázquez et al., 2006; Janssen et al., 2014; Martins et al., 2018; Su et al., 2011; Teeri et al., 2006).

Among these environmental stimuli, light is an important factor to plant developmental processes, and in nature, the biggest opponent of a plant is other plants. This way, in the aim to increase photosynthesis levels, plants search for the maximum light radiation and quality. To do so, they can modulate the shoot architecture and this process is called 'shade avoidance' (Casal, 2012).

Shade-avoidance responses are a series of developmental changes that affect plant body form and function when there is not enough light. Under these conditions the photosynthetic rate decreases, and as a response, plants carry their energy to elongate their stem (accompanied by an increase in apical dominance) and reduce leaf development (Kebrom, 2017; Q. Wang et al., 2014; Yang and Jiao, 2016). Another component of the shade-avoidance syndrome is the acceleration of flowering. Shade light signals might indicate that the canopy is getting closed with time, so plants, in a desperate attempt to increase the probability of the survival of the specie, produce flowers and therefore seeds (Casal, 2012; Smith and Whitelam, 1997). Thus, shade light signals can modulate gene expression and the responses are triggered by proteins and phytohormones such as auxin and gibberellin (Casal, 2012; Smith and Whitelam, 1997; Tao et al., 2008; Zheng et al., 2016).

Besides light and other environmental stimuli (such as water, and nutrient availability), endogenous signals (such as hormones and sugar content) also can influence meristem decisions. Usually, shoot branching is due to a weak apical dominance, which could be a physiological condition of the plant or due to external conditions. Together they are responsible to determine when and where branching will occur (Beveridge et al., 2003; McSteen and Leyser, 2005). This allows plants to adapt to environmental conditions to which they are submitted (Djennane et al., 2014; Hiraoka et al., 2013; Pierik and Testerink, 2014; Wang and Li, 2006).

Like branching, flowering is also controlled by endogenous and environmental factors. However, differently from branching, flowering only occurs if the plant has already acquired the competence to produce flowers, which means that the genetic and physiological background of the plant is ready to respond to environmental and endogenous stimuli to produce flowers (Balanzà et al., 2018; Immink et al., 2012; Jaeger and Wigge, 2007; Parcy, 2005; Poethig, 2003). This competence is acquired after plants go through the first phase transition in its lifespan (from juvenile to adult phase). Once being an adult, plants can respond to the flowering stimuli and go through the second phase transition (from adult vegetative to adult reproductive phase) and as a result, plants produce flowers. This second phase change can occur only once, if annual, or several times during the plant's life, if perennial (Iwata et al., 2012; Kurokura et al., 2013).

In annual species, after the transition to the adult reproductive phase, all meristems become active and produce flowers and fruits, which is followed by senescence and the death of the plant. Differently, perennial plants must maintain their vegetative growth after flowering, which involves different developmental decisions concerning meristem activity. In perennial species, only a subset of meristems develops into inflorescence meristems during the reproductive phase, which guarantees the maintenance of vegetative growth (i.e. in poplar trees, terminal meristems of branches always remain vegetative) (Brunner and Nilsson, 2004; Yuceer et al., 2003). Additionally, there is also the possibility of meristems to revert from the reproductive phase to the vegetative phase (Amasino, 2009; Friedman and Rubin, 2015).

It has been postulated that the control of these phase transitions is orchestrated by various pathways involving environmental and endogenous cues, that together can be summarized in six main pathways that crosstalk with each other: the photoperiod pathway, the autonomous pathway, the vernalization pathway, the temperature pathway, the GA pathway and the age pathway (Teotia and Tang, 2015). However, some studies have revealed that the sugar content is also involved in phase change control, adding one more pathway to the list (Matsoukas, 2014; Wahl et al., 2013; Yang et al., 2013; Yu et al, 2013, 2015).

In this review, we discuss recent advances in plant biology concerning the genetic and molecular mechanisms that allow plants to regulate flowering time in response to the environment. We also propose connections between the phase transition and plant architecture control.

2. The life pathways of a plant

2.1. The juvenile-to-adult vegetative phase transition

After germination, the first phase of a plant's life is the juvenile phase, where the seedling is not competent to flower even if under the proper stimuli (Bäurle and Dean, 2006; Poethig, 2010). Some morphological, physiological, and genetic aspects are peculiar to this phase.

2.1.1. Morphology, the hallmarks of the first phase transition

The morphology (appearance) of a plant is determined as a response to environmental conditions such as day length, light quality and quantity, water and nutrient content (Ferjani et al., 2007; Gratani, 1996; Matos et al., 2009; Pintado, 1997; Wyka et al., 2007). Besides the external conditions, endogenous cues connected to different developmental phases of a plant's life are also responsible for the control of shoot appearance, which includes leaf morphology. The gradual transition of morphological aspects in plants related to its development is called heteroblasty, and it can be, in most cases, be noticed by the naked eye (Zotz et al., 2011).

Usually, it is possible to distinguish the juvenile phase by leaf shape and its traits. However, in the model plant *Arabidopsis thaliana*, these traits are relatively subtle. Juvenile leaves are less elongated than the vegetative ones, their peduncles are long, and there are no trichomes on the abaxial surface of juvenile leaves, while in adult vegetative leaves they are present (Telfer et al., 1997). On the other hand, several plants present evident different leaf characteristics as they age. In some species,

such as passionfruit (*Passiflora edulis*), and maize, there are notable morphological differences between the two developmental states (Cutri et al., 2013; Poethig, 1988). Juvenile *P. edulis* plants have lanceolate leaves while adult plants have trilobate leaves, and as the plant ages, a meristem capable of producing both a tendril and, after vegetative-to-reproductive phase transition, a flower, are formed in the axil of each leaf (Cutri et al., 2013). In maize, juvenile plants have small leaves with wax in its epidermis, while the adult plants have long narrow leaves, and epidermal hairs replace the production of a cuticle on the leaf blade (Poethig, 1988).

All these modifications occur as a response to the alterations that plants undergo during the first phase transition, which is controlled by physiological and genetic networks.

2.1.2. Physiology: sugar and hormones are the major players

2.1.2.1. Physiological control of juvenile-to-adult phase change. Considering the physiological modifications that lead juvenile-to-adult vegetative phase transition, and as consequence, modifications in leaf morphology, plant nutrition, and metabolism are important players.

The involvement of carbohydrates in the vegetative phase change is long known and it was proposed that the phenomenon of leaf shape modifications is a result of an alteration in the nutritional status of the plant shoot (Allsop, 1952; Allsopp, 1954, 1963; Yang et al., 2013; Yu et al., 2013). This implies that the morphology might be controlled by the physiological conditions of plants. Further studies have shown that plants growing in a deprived nutrient condition or a low light environment produces simple leaves, while exogenous sugar or the supplementation with metabolizable sugars induce the production of larger and more complex leaves with adult traits (Allsop, 1954; Feldman and Cutter, 1970; Njoku, 1956, 1971; Peng et al., 2020; Rijkers et al., 2000).

In short, considering the physiological context, after germination photosynthesis starts and the seedling begins to accumulate metabolites, such as sugars, which are used for plant growth and as signaling molecules (Meyer et al., 2007; Rolland et al., 2006). This allowed the assumption that by reducing the photosynthetic rate, the amount of sugar would be lower, which would lead to the production of juvenile leaves indicating an extension of the juvenile phase. This was confirmed by an experiment with tobacco in which the suppression of *RUBISCO SMALL SUBUNIT* (*RBCS*), a gene related to the photosynthesis machinery, causes a reduction of the photosynthesis rate and resulted in the extension of the juvenile phase (Tsai et al., 1997). Likewise, a defoliation experiment of *Ipomoea caerulea* resulted in an increased production of juvenile leaves (Njoku, 1971). The same effect was observed in woody plants when they suffered severe pruning (Libby and Hood, 1976; Schaffalitzky De Muckadell, 1954). Hence, aside from leaf morphology control, sugars also have a role in the transition control from juvenile to adult vegetative phase.

Hormones are also important players in the control of phase change transitions, and gibberellins (GAs) deserve special attention in this subject. They are diterpene hormones and are well known for their role in internode elongation, the transition from the vegetative phase to the reproductive phase, and seed germination (Ogawa et al., 2003; Richards et al., 2001; Yu et al., 2012). Withal, although GA has its most known role in the second phase transition, it was settled that it affects both phase changes (juvenile-to-adult vegetative and adult vegetative-to-reproductive phase). Studies in maize revealed that in deficient *dwarf* mutants, the juvenile phase is prolonged and there is a delay in the reproductive phase as well (Evans and Poethig, 1995). This is obvious, once extending the juvenile phase plants would take more time to produce flowers. However, in 2014 Yamaguchi and coworkers have demonstrated that GA promotes termination of vegetative development, but inhibits flower formation in *Arabidopsis* (Yamaguchi et al., 2014). Recent studies suggest the interplay between GA and microRNAs, especially miR156, but the details of the molecular mechanisms are still elusive (Yu et al., 2010, 2012).

2.1.2.2. Physiological conditions also afect plant architecture. Besides leaf morphology and phase change control, sugars and hormones are also involved in the control of plant architecture, by regulating the pattern of shoot branching (Barbier et al., 2015; Mason et al., 2014). It is known that sugars, which are used for plant growth, can act both as a substrate for metabolism and as signaling molecules. This gives sugar the status of an important driver of growth (Meyer et al., 2007; Rolland et al., 2006). As signaling molecules, studies have shown that sugars can promote or inhibit plant growth and development (Smeekens et al., 2010). In a high carbon availability, plants have a sugar signaling pathway that includes hexokinase-1 (HXK1), trehalose-6-phosphate (T6P), and target of rapamycin (TOR), however, for starvation, there is another sugar signaling pathway that includes Snf1-related protein kinase 1 (SnRK1) and C/S1 bZIP transcription factors (Smeekens et al., 2010).

Apart from sugar, plant hormones are also responsible for regulating many aspects of plant growth and development. There are several plant hormones involved in the control of shoot branching including auxins, cytokinins (CKs), strigolactones (SLs), gibberellins (GAs), and brassinosteroids (BRs) (Wai and An, 2017; Wang and Jiao, 2018; Yang and Jiao, 2016).

The interactions of endogenous concentrations of auxins (mainly indole-3-acetic acid, IAA) and cytokinins are the responsible for the control of shoot branching, which depends on the level of apical dominance, controlled by the developmental programs of the plant as well as by environmental signals (i.e. light quality and intensity, nutrition, herbivory, gravity, etc.). Auxins and citokinins are known to act antagonistically. Shortly, while auxin maintains the apical dominance by inhibiting axillary meristem outgrowth, CK activates the growth of the axillary meristems (Bartrina et al., 2011; Moubayidin et al., 2009; Rameau et al., 2015; Shimizu-Sato et al., 2009).

According to the classical model, shoot branching is inhibited by the apical dominance, which is a result from the basipetal transport of auxin (produced mainly in young leaves, and the shoot apex) controlled by several proteins, such as the auxin efflux carriers PIN-FORMED (PIN) (Azizi et al., 2015; Müller and Leyser, 2011; Sachs, 1975; Sachs and Thimann, 1967; Shimizu-Sato et al., 2009; Zažímalová et al., 2007). Auxin, in turn, regulates CK transport from roots to the shoot and also suppresses the local biosynthesis of CK in the stem through the regulation of an *ADENOSINE PHOSPHATE-ISOPENTENYLTRANSFERASE3* (*IPT3*) expression, which encodes a key enzyme in CK biosynthesis (Kakimoto, 2001; Shimizu-Sato et al., 2009; Takei et al., 2001).

However, studies have demonstrated that apically derived auxin does not move into the axillary buds, which raised the question of how auxin inhibits those buds (Booker et al., 2003; Sachs and Thimann, 1967). Further, it was proposed that the auxin depletion by the loss of apical dominance is not the factor that triggers axillary bud outgrowth, but it is involved in the transition from the axillary bud release to sustain its growth (Fig. 2A) (Barbier et al., 2015). Besides the decreased auxin flux by the removal of the shoot tip (sink organ), more sucrose (Suc) is transported to the axillary buds (Barbier et al., 2015; Mason et al., 2014). Thus, once apical dominance is broken, there is a subsequent accumulation of sugar, which promotes the increase in CK levels through the induction of *IPT3* expression and the repression of *CYTOKININ OXIDASE* (*CKX4*), resulting in bud outgrowth (Hwang et al., 2012; Kushwh and Laxmi, 2014; Proels and Roitsch, 2009). However, other studies suggest that sugars are the first signal to induce axillary bud outgrowth and that sucrose promotes sustained growth in a cytokinin-independent manner (Barbier et al., 2015). The Suc signal is mediated by the T6P signaling and, in turn, reduces the expression of a branching inhibitor gene called *Branched1* (*BRC1*) (Fig. 2B) (Aguilar-Martínez et al., 2007; Barbier et al., 2015; Mason et al., 2014; Schluepmann et al., 2003). Besides T6P, studies with transgenic *HXK1*-overexpressing *A. thaliana* plants, suggest that the *HXK1* is also

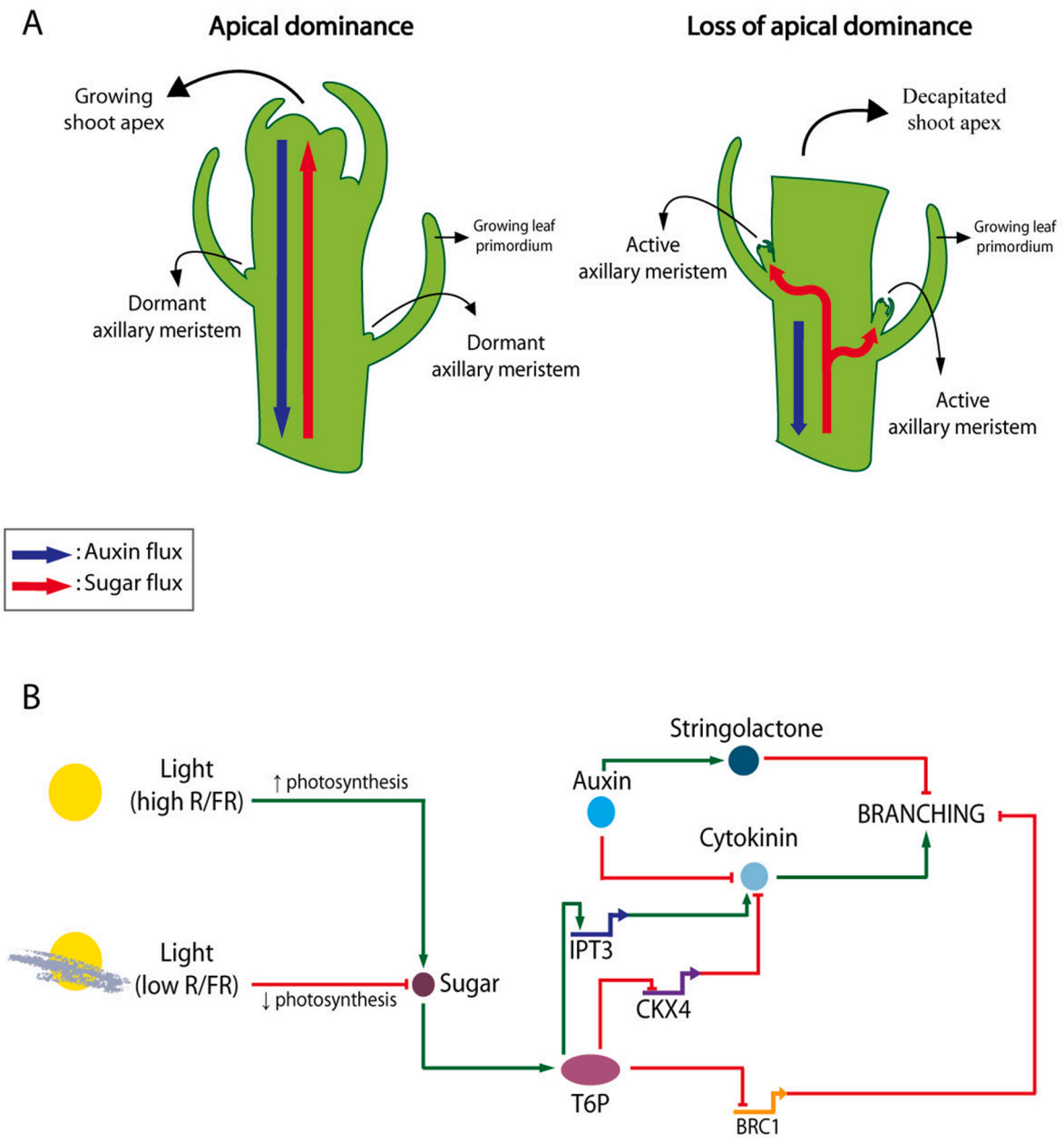

Fig. 2. The role of sugar and hormones in the shoot architecture. A. Apical dominance is a response to sugar and hormone regulation. According to the classical model, shoot branching is inhibited by apical dominance, which is a result of the basipetal transport of auxin. However, other studies suggest that the limited amount of sugar in axillary buds is responsible for the maintenance of apical dominance. Once the shoot tip is lost (e.g. decapitated, which leads to loss of the apical dominance), two processesses occur at the same time: a decrease in the influx of auxin, and an accumulation of sugar in axillary buds, which after achieving a threshold level, breakes the dormancy of the axillary meristem. The loss of the shoot tip interrupts the apical supply of auxin, and auxin concentration is differentially re-distributed along the stem. Upper axillary meristems are affected before the lower ones. In this model sugars are the first signal to induce axillary bud outgrowth and auxin acts later, in the transition from the axillary bud release to sustain its growth. **B.** Diagram of the interplay of light, hormones, and genes in the shoot branching process. Green arrows or red linkers indicate induction or repressive effects, respectively. Hormones and sugar are represented by circles. CKX4: cytokinin oxidase; BRC1: Branched1; T6P: trehalose-6-phosphate; IPT3: adenosine phosphate-isopentenyl transferase 3. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

involved in an increased branching pattern, once these plants lost the apical dominance (Kelly et al., 2012).

In addition to auxin and cytokinin activities, during the past decade studies have uncovered the role of strigolactones (SLs) in the control of shoot architecture (Gomez-Roldan et al., 2008). SLs are synthesized from carotenoid precursors and there are two main explanations of its activity in branching: affecting auxin transport by repressing the gene expression and protein accumulation of PIN auxin transporters (Domagalska and Leyser, 2011); or by targeting *BRC1* expression in the bud meristem (Brewer et al., 2009; Dun et al., 2012). Like auxin, this class of hormones also inhibits axillary buds outgrowth. However, differently from IAA, SLs mechanism of action does not affect CK concentrations in axillary meristems (Brewer et al., 2013; Dun et al., 2012). Experiments with pea plants suggested that SLs and cytokinins actions converge on targeting the *BRC1*, a branching inhibitor gene (Aguilar-Martínez et al., 2007; Braun et al., 2012; Brewer et al., 2009; Dun et al., 2012; Finlayson, 2007). Experiments with sorghum also suggest that light quality and quantity likewise affects SLs levels, once the strigolactone production might be repressed by PHYB (Finlayson et al., 2010).

Considering this new apical dominance model and the potential role

of SLs in this process, in a low light intensity environment (low red/far red (R/FR) ratio) the shoot branching is negatively affected, because in this condition (low R/FR ratio) the photosynthetic rate is decreased, therefore decreasing the supply of sugars to axillary buds. Recent studies have empirically demonstrated this new apical dominance model. In a low R/FR ratio, the expression of *BRC1* in *Arabidopsis* is upregulated and, in a higher R/FR ratio the same gene is rapidly downregulated through the PHYB (PHYTOCHROME B) pathway, which is the major photoreceptor in the R/FR perception (González-Grandío et al., 2013; Holalu and Finlayson, 2017).

Apart from sugar, plant hormones are also responsible for regulating many aspects of plant growth and development. There are several plant hormones involved in the control of shoot branching including auxin, cytokinins (CKs), strigolactones (SLs), gibberellins (GAs), and brassinosteroids (BRs) (Wai and An, 2017; Wang and Jiao, 2018; Yang and Jiao, 2016).

GAs are also involved in the axillary meristem development, although depending on the species, its effects might vary (Davies, 2010; Rameau et al., 2015). Previous studies suggested that in perennial woody plants GA acts as a promoter of shoot branching, while in herbaceous plants such as *Arabidopsis* and rice, GA causes the opposite effect (Elfving et al., 2011; Ni et al., 2015; Oikawa et al., 2004; Rinne et al., 2016; Silverstone, 2001). As *Arabidopsis* is the model plant used as the basis for this review, henceforward GA will be considered only as a repressor of bud outgrowth.

2.1.3. Genetics, the integrator of signals

From a genetic point of view, there are some genes and microRNAs (miRNAs) that are responsible for the modulation of physiological alterations that plants undergo during the juvenile-to-adult vegetative phase change, therefore controlling morphological changes as well.

2.1.3.1. The genetic control of the juvenile-to-adult phase change. Among the vast number of miRNAs described to date, two have a major role in phase transition control, the *miR156* and the *miR172* (Ahsan et al., 2019; Jung et al., 2011; Spanudakis and Jackson, 2014; Wu, 2006; Wu et al., 2009). *miR156* is the master regulator of vegetative phase change in *Arabidopsis* and other flowering plants (Wu, 2006). The *miR172*, in turn, targets the mRNA of *APETALA 2/APETALA 2-like* (*AP2/AP2-like*) genes involved in floral development, which will be discussed forward in this review (Teotia and Tang, 2015; Wu et al., 2009; Zhu and Helliwell, 2011).

Several studies have demonstrated that the expression pattern of *miR156* is intimately connected with the age of the plant and with the regulation of a family of transcription factors (Fouracre and Poethig, 2019; Huijser and Schmid, 2011; Riese et al., 2007; Salinas et al., 2012; Teotia and Tang, 2015; C. Wang et al., 2019). The biological functions of miR156 are executed by its target, the *SQUAMOSA PROMOTER BINDING PROTEIN-LIKE* (*SPL*) transcription factors, which comprises at least 11 of the 17 *SPL* genes in *Arabidopsis* (Birkenbihl et al., 2005; Guo et al., 2008; Wang et al., 2011; Wu, 2006; Wu et al., 2009; Xie et al., 2012). Their expression is inversely proportional to the *miR156* expression as the plant ages (Gordon et al., 2007; Poethig, 2010; Wu et al., 2009).

The *SPL* gene family of are plant-specific transcription factors have been implicated in promoting vegetative and floral phase transitions (Chen et al., 2010; Preston et al., 2016; Shikata et al., 2009; Spanudakis and Jackson, 2014). The SBP-box proteins encoded by *SPLs* all contain a highly conserved DNA-binding SBP domain, which includes approximately 76 amino acid residues and two zinc-binding motifs (Birkenbihl et al., 2005; Salinas et al., 2012).

As plant ages, there is a gradual decline in the *miR156* expression, which is associated with the juvenile traits (Bergonzi et al., 2013; Chuck et al., 2007; He et al., 2018). Also, it has been demonstrated that the overexpression of *miR156* lengthen the juvenile phase, whereas its repression accelerates the appearance of adult traits, corroborating its link to the first phase change control (Gordon et al., 2007). However, the mechanism linking age and the abundance of the *miR156* was poorly understood until Yu et al. (2013) demonstrate that the expression of *miR156* responds to sugar (Yu et al., 2013). Based on the premise that endogenous carbohydrates can affect gene expression, sugar treatment assays have demonstrated that the level of miR156 transcripts decreased and the expression of *SPL9* and *SPL15* (major targets of miR156) increased in plants treated with sucrose, glucose, or maltose, while the sugar-depleted seedlings exhibited a higher expression level of *miR156* (Yu et al., 2013).

Also, analyses of defoliation and photosynthetic mutant assays suggest that sugar from older leaves act as a mobile signal to repress *miR156* expression, which set off the juvenile-to-adult vegetative phase transition in young leaf primordia (Yang et al., 2013; Yu et al., 2013). From the assumption that sugar serves as an endogenous cue for developmental timing in plants, the growth rate must be in synchrony with the metabolic status, so in a low sugar condition, plants should stay longer periods in the juvenile phase. On the other hand, in higher concentrations of sugar the expression of *miR156* is decreased, thus the expression of its targets *SPL9/SPL15* increases, leading to the adult vegetative phase (Fig. 3) (Poethig, 2013; Yu et al., 2013).

Another study with chlorophyll deficient plants show higher levels of *miR156* and an extended juvenile phase and also revealed that in low sugar conditions the *HXK1* gene, helps to keep plants in the juvenile phase (Yang et al., 2013). Other studies have demonstrated that removing leaves from *Arabidopsis* plants resulted in an increased level of miR156 and an extended juvenile phase. The same effect was observed in *Nicotiana benthamiana* plants (Feng et al., 2016; Yang et al, 2011, 2013). These observations strongly suggest that sugar is a major player in the phase change control, and T6P is considered the signaling molecule in high carbon availability, which makes the TREHALOSE-6-PHOSPHATE SYNTHASE 1 (TPS1) an important enzyme in this pathway (Wahl et al., 2013).

Recently, it was demonstrated that in addition to the response to sugar, *miR156* expression is also regulated by another microRNA, the *miR159* (Guo et al., 2017). *miR159* downregulates the expression of *miR156* is mostly through the MYB33 (target of *miR159*) activity. At the same time that MYB33 promotes the expression of *miR156*, it also promotes the expression of one of its targets, *SPL9*, suggesting that miR159-MYB33 interplay is not the major regulator of the juvenile-to-adult vegetative phase change, but it contributes as a fine-tune control of the *miR156* expression and the correct time of phase transition (Fig. 3) (Guo et al., 2017).

2.1.3.2. Genes and morphology. Besides phase transition, *miR156* also participates in the shoot architecture and leaf morphology. Studies have demonstrated that overexpression of *miR156* enhances the shoot branching in *Arabidopsis* through the repression of two *SPL* genes (*SPL9* and *SPL15*) that induce the expression of *BRC1* (Schwarz et al., 2008; M. Wang et al., 2019). However, the sugar that indirectly represses the expression of *BRC1* also induces the expression of those two *SPL* genes that repress branching through the induction of *BRC1*. Thus, the shoot architecture depends on the balance between the indirect repression of *BRC1* by sugar and *BRC1* induction by *SPL9/SPL15*, which are also affected by the sugar content (M. Wang et al., 2019) (Fig. 3).

Concerning the participation of the miR156-SPL pathway in leaf morphology, in *Arabidopsis*, eight SPL genes regulate the differences between juvenile and adult leaves: *SPL2*, *SPL3*, *SPL4*, *SPL5*, *SPL9*, *SPL10*, *SPL11*, and *SPL15* (Gordon et al., 2007; Shikata et al., 2009; Usami et al., 2009). *SPL3*, *SPL4*, *SPL5* are responsible for the regulation of the cell number and size and also for trichome distribution, but do not affect leaf shape (Gordon et al., 2007; Usami et al., 2009). The laminar leaf shape in the vegetative phase is controlled by other 3 *SPL* genes (*SPL2*, *SPL10*, and *SPL11*) which act in a redundant manner (Shikata et al., 2009). Finally, the *Arabidopsis* leaf shape itself is affected by two *SPL* genes,

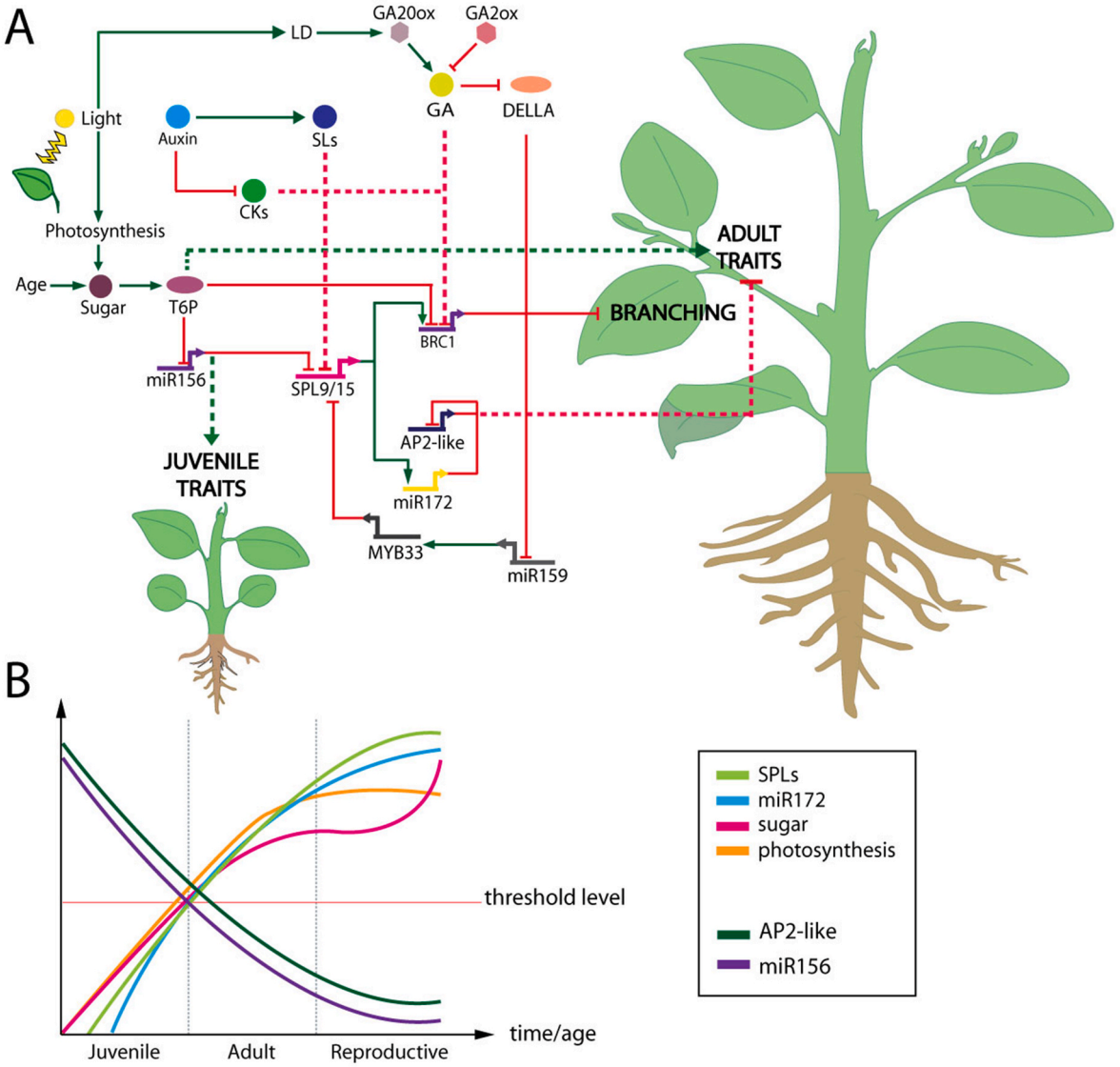

Fig. 3. Summary of the interplay of the juvenile-to-adult vegetative phase transition and the branching regulatory networks. A. The interplay of the juvenile-to-adult vegetative phase transition and the branching regulatory networks. Green arrows or red linkers indicate induction or repressive effects, respectively. Hormones and sugar are represented by circles, proteins by ellipses, and enzymes are represented by hexagons. In *A. thaliana* juvenile traits are rounded leaves with elongated peduncles, and there are no trichomes on the abaxial surface of the leaves, while the adult traits are elongated leaves, with short peduncles and the presence of trichomes. **B.** Graphical representation of levels of photosynthesis rate, sugar concentration, and gene expressions during plant's lifecycle. By the time when these factors reach the threshold level, the juvenile-to-adult vegetative phase change occurs. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

SPL9, and *SPL15*, which are also major players in the control of phase transition (Usami et al., 2009).

The transcription factors *SPL9/SPL15* promote the expression of another microRNA, *miR172*, through the direct binding to its promoter. The *miR172* downregulates two of the six *AP2-like* genes: *TARGET OF EAT 1* and *2* (*TOE1/2)*, by repressing their expression. Therefore, the plant enters the adult vegetative phase, which is characterized by leaves presenting adult traits, which were being repressed by the *AP2-like* gene expression.

The adult vegetative phase follows the juvenile phase, characterizing the first phase transition in a plant's lifespan. The major regulators of this phase change are sugar content, the microRNAs *miR156* and *miR172,* and the *SPL* genes. By this time, plants can respond to floral inductive signals. Once those signals are present, plants undergo the second phase transition: from adult vegetative to adult reproductive phase (Fig. 3) (Bäurle and Dean, 2006).

3. The adult vegetative-to-reproductive phase transition

The flowering induction is controlled by multiple pathways that regulate the expression of genes related to the transition of a vegetative meristem into an inflorescence meristem (Amasino, 2010; Wellmer and Riechmann, 2010). In *Arabidopsis,* these pathways include photoperiod, vernalization (prolonged cold temperature), ambient temperature, gibberellic acid (GA), sugars, the autonomous pathway, and the age pathway (Andrés and Coupland, 2012; Dijken et al., 2004; Galvao et al., 2012; Moon et al., 2003; Srikanth and Schmid, 2011; Wahl et al., 2013). The competence of a plant to respond to any of these flowering inducible signals depends on the status of the other pathways and the plant phase status once it cannot flower during the juvenile phase (Hyun et al., 2017).

3.1. The competence to perceive flowering inductive signals

To respond to signals, first plants need to perceive them. This perception depends on a system that develops and reaches its maturation along with the plant (Hyun et al., 2017). A mature perception system means that physiologically, plants have sufficient carbohydrates to support flowering, which is a highly energy-demanding process for the plant.

As plants age, there is a gradual increase in sugar availability, signaled by the T6P, that besides taking part in the juvenile-to-adult phase change and the regulation of shoot branching, it has also been suggested to play a critical role in controlling the transition to flowering, acting as a possible link between environmental and physiological signals (Fig. 4) (Dijken et al., 2004; Wahl et al., 2013). T6P downregulates flowering repressors, through the downregulation of *miR156* (Bernier et al., 1993; Ponnu et al., 2011; Wahl et al., 2013).

Studies have demonstrated that the activity of the TPS1, which catalyzes the formation of T6P from glucose-6-phosphate and uridine diphosphate (UDP)-glucose, is required for the induction of *FT* in the leaves, even under inductive photoperiod (Cabib and Leloir, 1958; Paul et al., 2008; Ponnu et al., 2011). Thus, plants can ensure the best condition to transition from vegetative to reproductive phase by integrating an environmental signal (e.g. if the day length, is appropriate to flowering) to its physiological status (if the carbohydrate content is enough to support the energy demand of flowering), and both may very depending on the species. Moreover, studies have demonstrated that T6P also acts in the SAM independently of the photoperiod, by affecting the expression pattern of flowering time genes, which might link the energy supply to developmental decisions in the SAM (Wahl et al., 2013).

Shortly, a high sugar concentration implicates in the decrease of the *miR156* expression concomitantly with the constant increase of its targets *SPL* transcription factors expression. These TFs upregulate the transcription of the *miR172*, which is responsible for the activation of *FLOWERING LOCUS T* (*FT*) gene (flowering inductive gene), through the inhibition of its targets, which in *Arabidopsis* are the six *AP2-like* transcription factors: *APETALA-2* (*AP2*), *TARGET OF EAT 1* (*TOE1*), *TOE2*, *TOE3*, *SCHLAFMÜTZE* (*SMZ*), and *SCHNARCHZAPFEN* (*SNZ*). They act as flowering repressor genes at different levels (upstream some floral meristem identity genes, and downregulating *FT* expression) and are

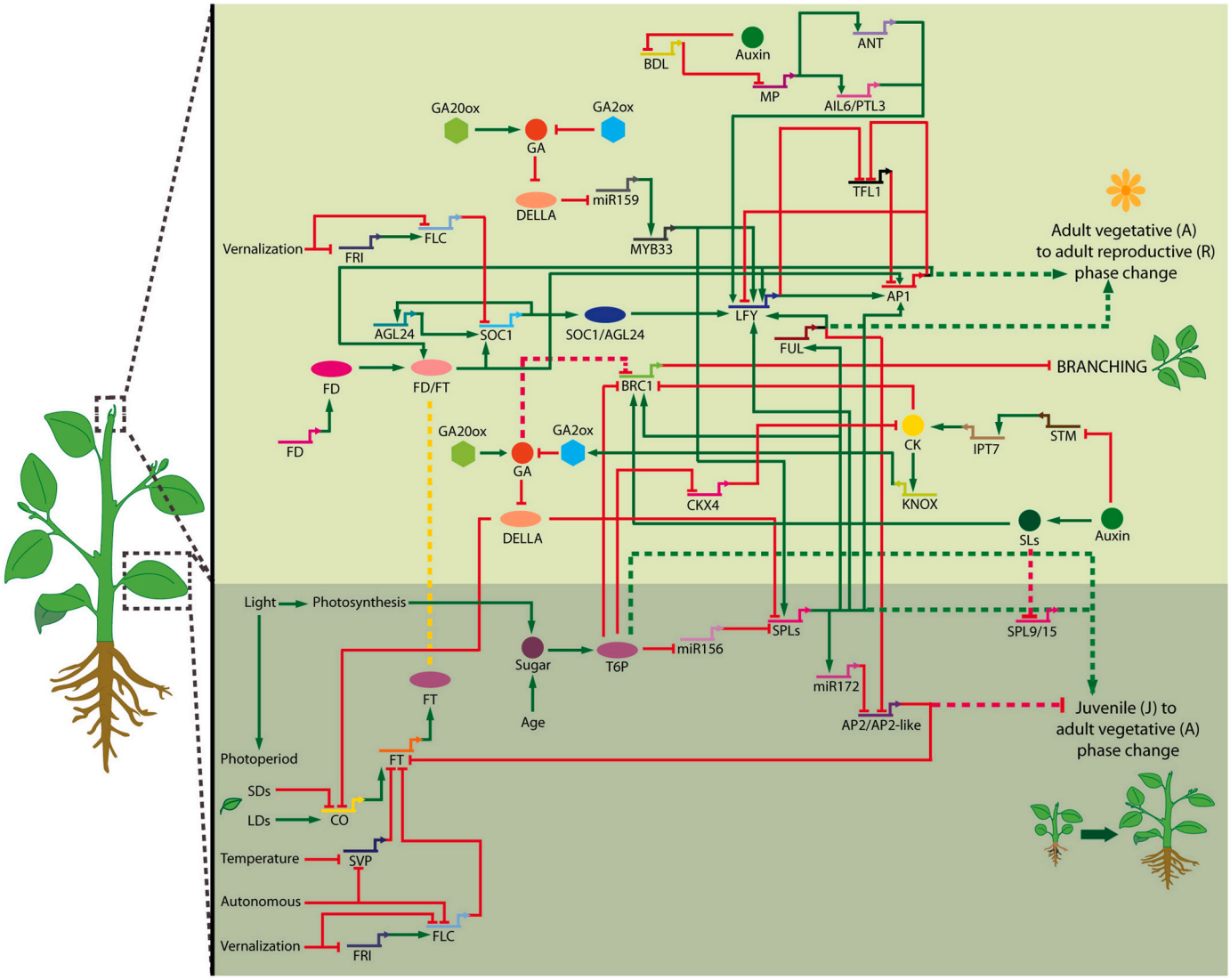

Fig. 4. Flowering induction network and summary of interactions during plant development in both leaves and the shoot apical meristems (SAMs).. Green arrows or red linkers indicate induction or repressive effects, respectively. Dashed yellow linker indicates the transport of FT protein from leaves to SAM. Dashed arrows indicate indirect relations of induction and repression in green and red, respectively. Hormones and sugar are represented by circles, proteins by ellipses, and enzymes are represented by hexagons. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

downregulated by the *miR172* (Fig. 4) (Aukerman and Sakai, 2003; Chen, 2004; Krogan et al., 2012; Yamaguchi and Abe, 2012).

This set of conditions marks the juvenile to adult vegetative phase transition, and henceforward plants can perceive and respond to floral inductive signals (Wu and Poethig, 2006; Wang et al., 2009; Wu et al., 2009; Jung et al., 2011; Aukerman, 2003; Jung et al., 2007; Johannes et al., 2009).

Some studies, however, have demonstrated that the sugar signaling acts upstream of *FT* in the photoperiod pathway, which was demonstrated in an experiment that *ft* mutants almost completely recovered its late flowering after the *CLV3:TPS1* expression. This might mean that the induction of flowering by the age pathway is a 'security mechanism' to ensure that plants produce flowers even in the absence of inductive signals (Wahl et al., 2013).

3.2. Perceiving floral inductive signals

Once reaching the adult phase, plants are able to respond to floral inductive signals (e.g. significative changes in temperature, day lenght, etc). The nature and intensity of the necessary sinals may vary among species and even among varieties of the same species. Under inductive long-day conditions (LDs), the transition from adult vegetative to adult reproductive phase is triggered in *Arabidopsis* plants (Levy and Dean, 1998; Piñeiro and Coupland, 1998). The daylength is perceived in leaves by specialized photoreceptors (phototropin, cryptochromes, and phytochromes) and transducted to *CONSTANS* (*CO*), which activates the expression of *FT* also in the leaves (Andrés and Coupland, 2012; Lariguet and Dunand, 2005; Li and Yang, 2007; Piñeiro and Coupland, 1998; Putterill et al., 1995; Quail et al., 1995). *FT* encodes a protein in the leaves, known as the florigen, that acts as a long-distance signal which then moves through the phloem from the leaves to the shoot apex where it interacts with the *FD* transcription factor (King and Zeevaart, 1973). This complex activates the expression of two MADS-box genes: *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*), and the *AGAMOUS-like 24* (*AGL-24*). *SOC1* and *AGL-24* interact and positively regulate each other (positive feedback loop) (Abe et al., 2005; Andrés and Coupland, 2012; Corbesier et al., 2007; Jaeger and Wigge, 2007; Kobayashi and Weigel, 2007; Lee and Lee, 2010; Wigge, 2005). These two genes are expressed in the shoot apex and activate the expression of *LEAFY* (*LFY*), a flower meristem identity gene (Fig. 4) (Irish, 2010; Lee and Lee, 2010; Schultz and Haughn, 1991).

This set of signals (the increase of *FT-miR172* expression; the decrease of *miR156* expression, both members of the age pathwat), together with the inducible photoperiod, also contributes to the activation of *SPL* genes that upregulates the expression of *LFY* and other floral inductive genes (Wang et al., 2009; Yamaguchi et al., 2009, 2014). However, the induction of flowering in *Arabidopsis* by the photoperiod can be neutralized by several factors that affect the *FT* expression in leaves or act downstream its protein in the shoot apex. These factors include some MADS-box transcription factors that act also as a response to the environmental signals such as temperature, to repress flowering. *FLOWERING LOCUS C* (*FLC*) is one of them. It represses flowering in *Arabidopsis* before plants have been exposed to a prolonged period of cold, which is also called vernalization (Michaels and Amasino, 1999). FLC directly binds to regulatory regions of *FT* and *SOC1*, both promoters of flowering, and repress these genes (Searle, 2006; Sheldon et al., 2006). The expression of *FLC* is upregulated by another gene called *FRIGIDA* (*FRI*) (Michaels and Amasino, 1999; Sheldon et al., 1999).

3.3. The transition to flowering

The perception of flowering inductive signals is followed by the response to these signals triggered by *LFY*, which encodes a transcription factor with homologs in all plant kingdom (Mouradov et al., 1998; Sayou et al., 2014; Shindo et al., 2001; Tanahashi, 2005). In non-flowering plants, its function is associated with the regulation of sporophyte development (Maizel et al., 2005; Tanahashi, 2005). In flowering plants, however, *LFY* is described as one of the main players for the specification of floral meristem (FM) (Bomblies, 2003; Coen et al., 1990; Schultz and Haughn, 1991; Weigel et al., 1992). Phenotype studies of *lfy* mutant plants fail to produce floral meristems and instead presents secondary inflorescence structures with shoot traits replacing flowers (Huala and Sussex, 1992; Schultz and Haughn, 1991; Weigel et al., 1992). Besides, the ectopic expression of *LFY* induces early flower development, which indicates that it is sufficient for specifying floral meristem identity (Weigel and Nilsson, 1995; Simpson and Dean, 2002; Huijser and Schmid, 2011).

There are several floral inductive pathways capable of triggering an *LFY* response. Besides the photoperiod pathway (see 2.2), age, auxin, and GA pathways are also part of this process. The increase of *miR172* expression along with the decrease of *miR156* expression as the plant ages promotes the expression of at least three *SPL* genes that induces *LFY* expression (Hyun et al., 2017).

Concerning the role of auxin in the transition to flowering, as the initiation of any kind of meristem, the floral meristem is initiated after the establishment of a local maximum of this hormone as a result of its polar transport (Benková et al., 2009). When present, auxin triggers the degradation of transcriptional repressor proteins called AUX/IAA, which are responsible for inhibiting the activity of AUXIN RESPONSE FACTORs (ARFs) (Mockaitis and Estelle, 2008). One of these ARFs important for the flower initiation is MONOPTEROS (MP). MP acts downstream of auxin and *mp* mutants do not produce flowers and (Przemeck et al., 1996). Further studies have demonstrated that MP and two other transcription factors (that act in parallel with MP), AINTEGUMENTA (ANT), and AINTEGUMENTA-LIKE6/PLETHORA3 (AIL6/PLT3), directly induces *LFY* expression and there is positive feedback from LFY to the auxin pathway (Yamaguchi et al, 2013, 2016).

Gibberellins also participate in the transition to flower development through the promotion of the termination of vegetative development. Bioactive GAs are perceived by plants and its signal transduction is by binding and activating three GIBBERELLIC INSENSITIVE DWARF (GID1–GID3) receptors, which mediates the degradation of DELLA proteins, the main repressors of the GA signaling pathway, via the 26S proteasome pathway (Griffiths et al., 2006; Harberd et al., 2009; Hirano et al., 2008; Murase et al., 2008; Ueguchi-Tanaka et al., 2005; Willige et al., 2007). Thus, when GA levels are low there is a lower rate of DELLA degradation, which through their DNA binding domain can interact with transcriptional activators, thus blocking their activity. In the opposite situation (in higher levels of GA) GA set off a signal that triggers the degradation of DELLA proteins, releasing the transcriptional activators to promote the expression of their targets (Olszewski et al., 2002).

Previous studies have demonstrated that the GA pathway control flowering through the activity or repression of DELLA proteins (Davière and Achard, 2016; Galvao et al., 2012; Li et al., 2016; Porri et al., 2012; Richards et al., 2001; Silverstone, 2001; Wang et al., 2016). Under long days (LDs), GA promotes flowering through the promotion of the transcriptional activation of *FT* (Porri et al., 2012). The degradation of DELLA proteins promotes the expression of *FT* and *TWIN SISTER OF FT* (*TSF*), which are both flowering time integrator genes (Hisamatsu and King, 2008). This occurs in leaves regardless of the activity of *CO*, which means that the induction of flowering might occur independently of photoperiod. This induction is due to the regulation of *SPL* genes by GA concentrations in both the leaves and at the shoot meristem (Fig. 4) (Galvao et al., 2012).

However, in a noninductive short-day photoperiod (SDs), the *Arabidopsis* floral transition is promoted via the GA pathway, through the regulation of *LFY* expression. In short days, DELLA proteins promote the repression of another microRNA, the *miR159*, which negatively regulates *MYB33* (a *GAMYB-like* gene) that directly binds to GA-response elements (GAREs) that located in the *LFY* promoter (Fig. 4) (Achard et al., 2004; Guo et al., 2017; Jin et al., 2013; Spanudakis and Jackson, 2014). This was demonstrated in studies were GA treatment (and a

consequently DELLA degradation) resulted in a decrease of *miR159* levels, and a consequent increase of its targets. One of them is *MYB33* that, in its turn, binds to GA-response elements (GAREs) located in the *LFY* promoter and induces its transcription (Achard et al., 2004; Jin et al., 2013).

The GA pathway affects flowering through the regulation of other transcription factors, which are described in the recent review of Bao and collaborators (Bao et al., 2020).

3.4. Flower development

The flower development initiates after the induction of floral meristem identity genes such as *APETALA1* (*AP1*), and *FRUITFULL* (*FUL*), which both encodes MADS-box transcription factors (Alejandra Mandel et al., 1992; Langmore et al., 2009). The flowers of *ap1* mutants do not have petals and produce bract-like structures instead of sepals. In the axils of those bract-like whorl organs, new floral meristems arise reiterating this pattern, and as a result, mutants present 'branched flowers'(Irish and Sussex, 1990; Langmore et al., 2009).

Besides *AP1* and *FUL*, other MADS-box genes are also part of the network that promotes the floral meristem identity, including *CAULIFLOWER* (*CAL*) (an *AP1* paralog with partially redundant functions to those of *AP1*, only present in Brassicaceae), *AGAMOUS-LIKE24* (*AGL24*), *SHORT VEGETATIVE PHASE* (*SVP*) and *SUPPRESSOR OF CONSTANS1* (*SOC1*) as previous mentioned (Aukerman et al., 1999; Gregis et al., 2008; Kempin et al., 1995; Langmore et al., 2009; Lawton-Rauh et al., 1999; Lowman, 1999; Melzer et al., 2008; Zhao et al., 2017).

During floral meristem specification, several feedback loops regulate the activity of these genes, which guarantees the floral induction in proper conditions (Fig. 4). In *Arabidopsis*, flowering occurs by the promotion of flower meristem identity genes concomitantly with the repression of an indeterminate shoot fate. To do so, *TERMINAL FLOWER1* (*TFL1*), a gene that acts as a shoot-promoting signal, must be repressed, and those responsible for this repression are the *LFY* and *AP1* genes (Conti and Bradley, 2007; Liljegren et al., 1999; Ratcliffe et al., 1999; Weigel et al., 1992). The balance between *TFL1*, *LFY* and *AP1* expression pattern controls the shoot architecture being that variations on these relations are partly responsible for shoot architecture in angiosperms (Prusinkiewicz et al., 2007).

4. Conclusion

Plants have evolved a wide range of strategies to better adapt to the environmental conditions in which there are inserted. The control of its shoot architecture and the timing of phase changes are major processes that can improve plant fitness, and both are controlled by several pathways, including the photoperiod, age, temperature, and phytohormones.

Based on various studies, we suggest that all of these pathways are connected mainly by sugar content, which can be considered another patwhay because, as we presented in this review, the transition from a juvenile plant to an adult one occurs mainly in response to the level of sugars, which is signalized by the T6P. This signal, when perceived, affects the expression of *miR156*, the major regulator of juvenile-to-adult vegetative phase change. With the decrease of the *miR156* expression, the opposite occurs with its targets, *SPL* transcription factors, that induce the expression of the *miR172*. The expression of these two microRNAs works like a seesaw: while the expression of miR156 decreases, the expression of *miR172* increases. The *miR172* downregulares the expression of two *AP2*-like genes, *TOE1* and *TOE2*, which are responsible for maintaining the plant in its juvenile stage.

The shoot architecture is also modulated as a response to the sugar concentration, once it is a direct consequence of the photosynthetic rate modulated by light quality and quantity. Sucrose affects hormones concentrations, such as cytokinin and strigolactone, and also modulates the expression of genes such as *BRC1*, all of them are somehow involved in bud outgrowth.

Once in the adult vegetative phase, plants are physiological and genetically capable to respond to flowering inductive signals and being present, plants undergo the second phase transition: from adult vegetative to adult reproductive phase, marked by the flower development that initiates after the induction of floral meristem identity genes. In this phase, more branches and an indeterminate pattern of growth might result in more flowers and fruits. Thus, understanding and being able to manipulate the interactions of shoot architecture and the control of phase transitions networks might improve the efficiency of biomass, flowers, and fruit production. This can be done with the use of different approaches, such as gene-editing tools, regulation of hormone and sugar concentrations, and the artificial regulation of environmental conditions.

Contribution

MCD developed the main idea. MCD and HAG researched literature data and wrote the manuscript.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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