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REVIEW

# Plant responses to high temperature: a view from pre-mRNA alternative splicing

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## Abstract

***Key message*** **This review focused on the recent breakthroughs in plant high temperature responses from an alternative splicing angle.**

**Abstract** With the inevitable global warming, high temperature triggers plants to change their growth and developmental programs for adapting temperature increase. In the past decades, the signaling mechanisms from plant thermo-sensing to downstream transcriptional cascades have been extensively studied. Plenty of elegant review papers have summarized these breakthroughs from signal transduction to cross-talk within plant hormones and environmental cues. Precursor messenger RNA (pre-mRNA) splicing enables plants to produce a series of functional un-related proteins and thus enhances the regulation flexibility. Plants take advantage of this strategy to modulate their proteome diversity under high ambient temperature and elicit developmental plasticity. In this review, we particularly focus on pre-mRNA splicing regulation underlying plant high temperature responses, and will shed new light on the understanding of post-transcriptional regulation on plant growth and development.

**Keywords** Alternative splicing · Circadian clock · Flowering time · PIF4 · Spliceosome · Thermomorphogenesis

## Introduction

Plants are able to monitor subtle temperature changes including diurnal and seasonal temperature variations and then to coordinate their rhythmic growth. Moreover, with the global temperature increase, plants have to adjust their developmental programs for adapting to the warming world. For example, high ambient temperature triggers leaf petiole elongation and up-ward growth to cool down the leaf surface temperature (Gray et al. 1998; Crawford et al. 2012). High temperature also promotes floral initiation (Kumar et al. 2012), which helps plants to accelerate propagating their next generations and maintain their genetic information inherited to avoid the harsh environment. These morphological changes under high ambient temperature are collectively termed thermomorphogenesis (Casal and Balasubramanian 2019).

One of the central regulators underlying plant thermomorphogenesis is the bHLH transcription factor PHYTOCHROME INTERACTING FACTOR4 (PIF4). PIF4 loss-of-function mutants (*pif4*) display reduced temperature-induced hypocotyl and petiole elongation, indicating that PIF4 is a fundamental positive regulator (Koini et al. 2009). It has been well documented that PIF4 stimulates auxin biosynthesis through directly binding to the promoters of *YUCCA8* (*YUC8*) in *Arabidopsis thaliana* (Sun et al. 2012). High temperature induces *PIF4* transcription and also increases PIF4 protein stability to enhance PIF4 activity. In 2016, two elegant articles demonstrated that the plant red/far-red light photoreceptor phytochrome B (phyB) is the long-sought plant thermosensor (Jung et al. 2016; Legris et al. 2016). The photo-activated phyB photoreceptor interacts with PIF4 and then promotes PIF4 phosphorylation and degradation (Pham et al. 2018). High temperature directly represses phyB activity through promoting the transition from the active form of phyB to its inactive form, which releases its repression on PIF4.

In addition to the PIF4-dependent transcriptional control, a recent genome-wide RNA-sequencing (RNA-seq) experiments have shown that more than 74.3% of the differentially

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alternatively spliced genes (DASGs) were controlled by PIF4 as well (Jin et al. 2020), which suggests that high temperature also triggers post-transcriptional regulations to coordinate plant growth and development in a PIF4-dependent manner.

In this review, we will highlight the recent progresses on precursor messenger RNA (pre-mRNA) splicing regulation in *A. thaliana* high temperature responses. Readers are also encouraged to read transcriptional regulation during thermomorphogenesis in these recent published reviews and their cited literatures as references (Quint et al. 2016; Casal and Balasubramanian 2019; Jin and Zhu 2019a).

## Pre-mRNA splicing process

In eukaryotes, splicing removes introns from pre-mRNA to produce mature mRNA molecules. However, two or more mature mRNAs could be produced from one precursor mRNA, which is termed alternative splicing (Lorkovic et al. 2000). Alternative splicing is beneficial for expanding the proteome diversity, which occurs in almost 95% of human genomes and about 61% in *A. thaliana* genomes (Capovilla et al. 2015). In general, alternative splicing includes five different types: (1) retention of introns; (2) skipping of exons; (3) mutual exclusion of exons; (4) alternative splicing site selection at the 5′ end; (5) alternative splicing site selection at the 3′ end (Reddy et al. 2013).

Spliceosome assembly is the prerequisite for splicing. Spliceosome is mainly composed of small nuclear ribonucleoprotein (snRNP) complexes (U1, U2, U4, U5 and U6). The five snRNP complexes recognize and assemble on introns, which contain three separated regions (5′ splicing site (5′SS) with conserved GU, branch point (BP) with a conserved A, 3′ splicing site (3′SS) with conserved AG) (Reddy et al. 2013). Non-snRNPs including heterogeneous nuclear ribonucleoproteins (hnRNPs), serine/arginine-rich proteins (SR proteins), NineTeen complex (NTC) and NTC related complex (NTR) are also participating the composition of spliceosome (Wan 2018).

The ATP-required Pre-mRNA splicing is executed with the following steps: (1) U1 firstly recognizes the 5′SS (Reddy 2001). SR proteins identify and bind to exonic splicing enhancer (ESE) to promote U1 recognizing the 5′ splice sites (Cáceres and Kronblihtt 2002). In contrast, hnRNPs identify and bind to the exonic splicing silencer (ESS) to abrogate U1 recognizing the 5′SS (Cáceres and Kronblihtt 2002). (2) U2 associates with BP to form the spliceosome precursor (Reddy 2001). (3) U4/U5/U6 interact with the spliceosome precursor to complete spliceosome assembly (Reddy 2001). (4) The catalytic activation of the spliceosome takes place in two steps through transesterification reactions. U2 recruits NTC and NRC to release U1 and U4 and then breaks the phosphodiester linkage at the splice site to form two adjacent exons with a lariat containing introns (Wan 2018). (5) The intron and snRNPs are released from the spliceosome complex. The 5′ direction exon attacks the lariat structure to remove and degrade introns, and then the two exons are connected to complete the splicing process (Reddy 2001) (Fig. 1).

SR proteins are one of the best characterized non-snRNP proteins in spliceosome (Reddy et al. 2013). There are twenty SR proteins in *A. thaliana* (Table 1). SR proteins contain one or two RNA binding domains (RBDs) and serine/arginine-rich repeat domain at the C-terminus (Reddy 2001). SR proteins recruit other splicing-related factors through protein–protein or protein-RNA interactions (Reddy 2001). SR proteins participate in the regulatory targets to control splicing efficiency (Reddy et al. 2013). SR family proteins function in recognizing the ESEs to activate adjacent 3′ splice site (Cáceres and Kronblihtt 2002). In *A. thaliana*, SR34 is the first characterized SR protein, which shows significant homology to the human splicing factor SF2/ASF (Lambermon et al. 1995). SR phosphorylation is crucial for spliceosome assembly and splicing site selection (Mermoud et al. 1994). It has been reported that phosphorylation of *A.thaliana* SR proteins (SCL33, SR45, RSZ21, RSZ22) increased the interaction between SR proteins and U1-70 K in the early stages of spliceosome assembly (Golovkin and Reddy 1999).

hnRNP also functions in the selection of splicing site through competing with SR proteins to bind pre-mRNA. For example, hnRNP A1 randomly binds to pre-mRNA and interferes U1 snRNP moving to the distal 5′ splice site, while SF2/ASF interferes with hnRNP A1 to enhance the binding ability of U1 snRNP at 5′ splice sites (Eperon et al. 2000). There are six hnRNP-like proteins in the *A.thaliana* genome according to sequence similarity comparing with metazoan hnRNP (Lorkovic et al. 2000) (Table 2). Most of the hnRNP proteins have at least one RNA binding domain and an auxiliary C-terminal domain. Half of the hnRNP proteins are Glycine (Gly)-rich in C-terminus, and the another half enrich in Asparagine (Asn), Gly and Serine (Ser) in their C-terminus (Lorkovic et al. 2000). These plant hnRNP seems to have conserved functions as in animals (Chan and Black 1997; Chou et al. 1999; Krecic and Swanson 1999), but the detailed mechanisms are awaiting to be explored.

Temperature has a direct effect on the core splicing machinery. For example, *U2 Auxiliary Factor 65A* (*AtU2AF65A*), which encodes a spliceosome component, has three splicing variants. High temperature induces its intron retention form, while low temperature promotes intron removal (Verhage et al. 2017; Cavallari et al. 2018). AtU2AF65A promotes the floral initiation, therefore the alternative splicing of *AtU2AF65A* probably integrates the temperature cue with flowering time control in *A. thaliana*

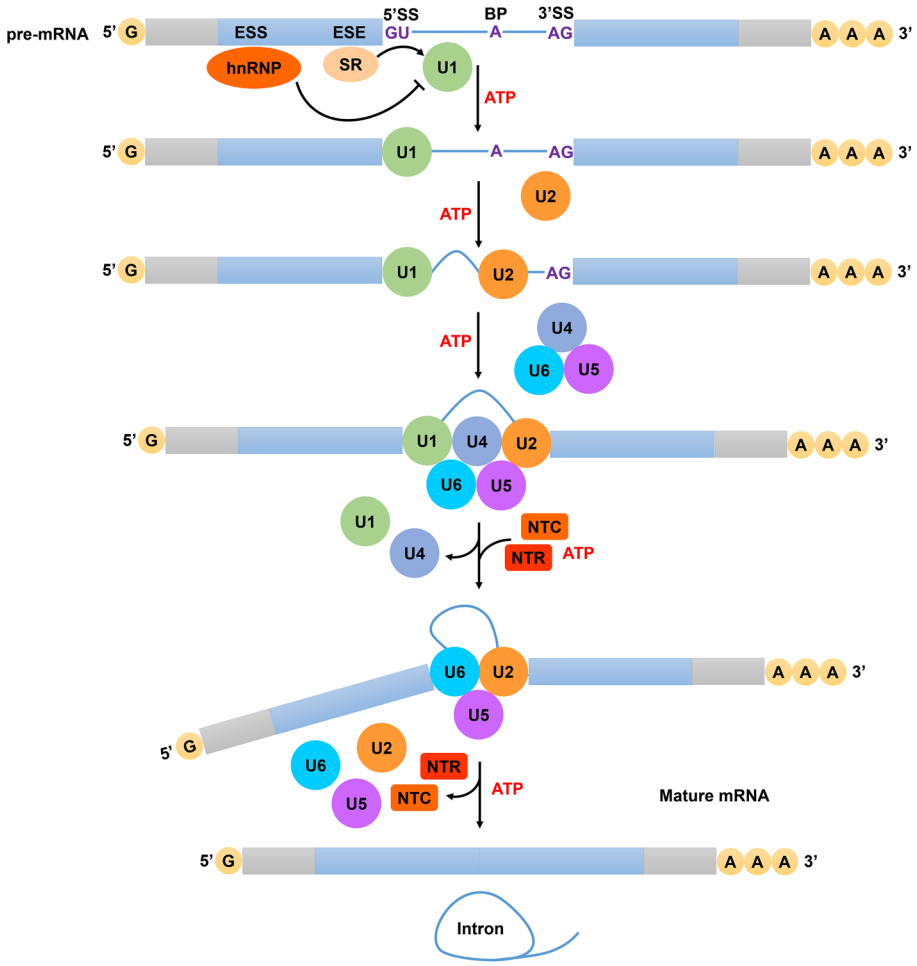


**Fig. 1** Spliceosome assembly and splicing process. U1 snRNP firstly recognizes the 5′SS in an ATP-dependent manner, and then U2 binds to BP to form a spliceosome precursor. SR and hnRNP bind to ESE or ESS to regulate splicing initiation, respectively. U4/U5/U6 interact together to assemble into the spliceosome. NTC and NTR interact with U2 snRNP and result in complex structural rearrangements to release U1 and U4 from the complex. U2/U5/U6 complex executes the splicing reaction and finally all these spliceosome components and introns are separated from the mature mRNA. G indicates G-capped RNA; AAA indicates 3′ poly (A) tail

(Verhage et al. 2017; Cavallari et al. 2018). In addition, high temperature induces the expression of *STABILIZED1* (*STA1*, encodes an U5-snRNP-interacting protein) to increase its splicing activity, and then to maintain heat-inducible transcription factor *HEAT SHOCK TRANSCRIPTION FACTOR A3 (HSFA3)* appropriately spliced (Kim et al. 2017).

## PIF4 controls alternative splicing during thermomorphogenesis

Thermomorphogenesis is largely dependent on transcription factor PIF4. RNA sequencing (RNA-seq) analysis on long-day-grown seedings treated with high ambient temperature show that 1740 and 913 genes are differentially expressed in Col-0 and *pif4-2* mutants, respectively. Moreover, in the 1740 differentially expressed genes in Col-0, 1501 of them (86.3%) are relied on PIF4. These results further confirm that PIF4 governs the transcriptional landscape in thermomorphogenesis. RNA-seq results also show that high temperature induces 870 and 696 genes to occur differentially alternative splicing events in Col-0 and *pif4-2* mutants, respectively, which indicate that PIF4 is also necessary for alternative splicing during thermomorphogenesis (Jin et al. 2020). Consistently, the high temperature-induced intron retention events are reduced in spliceosome component mutants, such as SNW/Ski-interacting protein (SKIP) deficient mutants *skip-1*. Similar to *pif4-2*, *skip-1* mutants are partially insensitive to high temperature treatment in hypocotyl elongation assay, which indicates that alternative splicing is required for proper thermomorphogenesis establishment (Jin et al. 2020).

RNA-seq data further illustrate that 79.9% of the PIF4-dependent DASGs are also relied on HOOKLESS1 (HLS1) (Jin et al. 2020). HLS1 was originally identified as a positive regulator in apical hook development and was recently shown to be necessary for thermomorphogenesis in *A. thaliana* (Lehman et al. 1996; Jin and Zhu 2019b). Addition to regulate pre-mRNA splicing, HLS1 also participates in the PIF4-regulated transcriptome. HLS1 physically interacts with PIF4, and occupies the PIF4 binding sites in *YUCCA8* promoter region, which indicates that PIF4 and HLS1 act together to regulate both transcription and post-transcription

**Table 1** List of SR Proteins in *Arabidopsis thaliana*

| Locus | Product name | Description (Asterisk represents the results based on sequence alignment) | References |
|---|---|---|---|
| At1g09140 | SR30 | Modulate alternative splicing | (Lopato et al. 1999a) |
| At1g02840 | SR34 | Splicing factor; promote splice site selection | (Lopato et al. 1999a) |
| At3g49430 | SR34A | Involved in intron recognition and spliceosome assembly* | |
| At4g02430 | SR34B | Involved in intron recognition and spliceosome assembly* | |
| At1g16610 | SR45 | Involved in 5′ and 3′ splicing site selection of introns; involve in flower petal development and root growth; negatively regulate ABA signaling | (Zhang and Mount 2009) (Carvalho et al. 2010) |
| At1g07350 | SR45A | Act as splicing factor; bridge the 5′and 3′components of the spliceosome | (Tanabe et al. 2009) |
| At1g23860 | RSZ21 | Involved in intron recognition and spliceosome assembly* | |
| At4g31580 | RSZ22 | Involved in alternative splicing | (Lopato et al. 1999b) |
| At2g24590 | RSZ22A | Involved in intron recognition and spliceosome assembly* | |
| At5g64200 | SC35 | Involved in intron recognition and spliceosome assembly | (Thomas et al. 2012) |
| At5g18810 | SCL28 | Involve in intron recognition and spliceosome assembly* | |
| At3g55460 | SCL30 | Involve in intron recognition and spliceosome assembly*; active at the 5′splice sites* | |
| At3g13570 | SCL30A | Involve in intron recognition and spliceosome assembly | (Thomas et al. 2012) |
| At1g55310 | SCL33 | Involve in intron recognition and spliceosome assembly | (Thomas et al. 2012) |
| At3g53500 | RS2Z32 | Involve in intron recognition and spliceosome assembly* | |
| At2g37340 | RS2Z33 | Splicing factor | (Kalyna et al. 2003) |
| At3g61860 | RS31 | Involve in alternative splicing* | |
| At2g46610 | RS31A | Involve in intron recognition and spliceosome assembly* | |
| At4g25500 | RS40 | Involve in primary miRNA processing and pri-miRNA biogenesis | (Chen et al. 2015) |
| At5g52040 | RS41 | Involve in primary miRNA processing and pri-miRNA biogenesis | (Chen et al. 2015) |

**Table 2** List of Six hnRNP-like Proteins in *Arabidopsis thaliana*

| Locus | Protein name | Functions | References |
|---|---|---|---|
| At4g14300 | UBP1 | Interact with the U-rich introns and 3′UTR sequence | (Filipowicz et al. 2000) |
| At5g40490 | hnRNP A | Alternative pre-mRNA splicing; alter the distal alternative 5′ splice site; antagonizes SR protein | (Krecic and Swanson 1999) (Eperon et al. 2000) (Lorkovic et al. 2000) |
| At5g47640 | hnRNP B | Pre-mRNA mature in plant | (Krecic and Swanson 1999) |
| At2g33410 | hnRNP F | Interact with nuclear cap-binding complex; affect the efficient of RNA splicing in *vitro* | (Gamberi et al. 1997) (Chou et al. 1999) |
| At4g26650 | hnRNP H | Interact with nuclear cap-binding complex; affect the efficient of RNA splicing in *vitro*; bind to the splicing enhancers or silences | (Gamberi et al. 1997) (Chou et al. 1999) (Chou et al. 1999) |
| At1g43190 | hnRNP I (PTB) | Select 3′ site; alternative pre-mRNA splicing | (Chan and Black 1997) (Krecic and Swanson 1999) |

(Jin et al. 2020) (Fig. 2). However, how does PIF4-HLS1 module controls alternative splicing is still unknown.

## Alternative splicing in high temperature responsive plant development

A number of microRNAs (miRNAs) respond to heat stress (temperature range from 34 to 40 °C) to modulate plant growth and development, such as leaf morphology and floral organ development (Zhao et al. 2016). For example, heat stress promotes *miR167h* to repress the expression of *AUXIN PRSPONSE FACTOR 8*, modulating floral organ development (Zhao et al. 2016). miR400 is located in the first intronic regions of *At1g32583*, which belongs to intronic miRNAs. Plants overexpression of *MIR400* gene display lower germination rates and reduced root growth and hypocotyl elongation (Yan et al. 2012). Heat stress regulates *MIR400* expression through alternative splicing. Under heat stress, the selection of downstream 5′ splice donor site

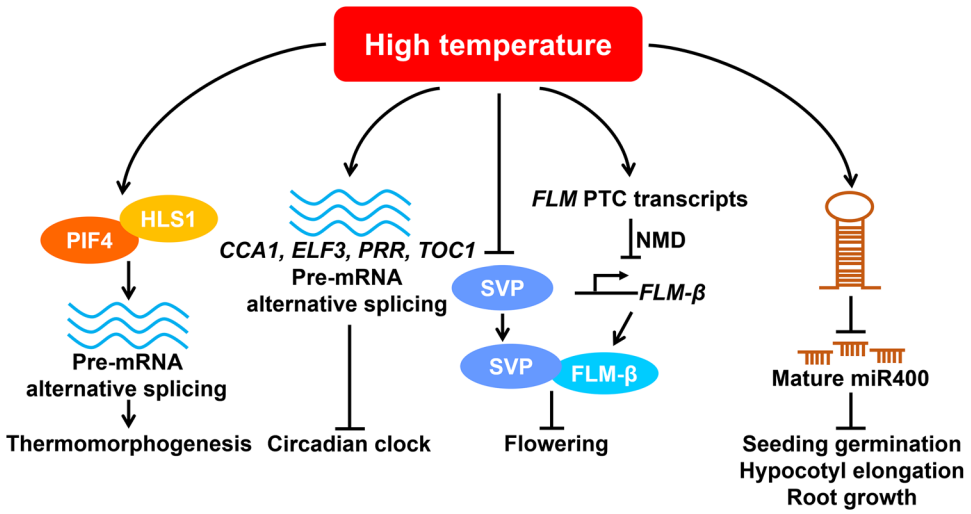


**Fig. 2** Alternative splicing in high temperature responses. High temperature triggers pre-mRNA splicing in a variety of biological responses. PIF4 and HLS1 control alternative splicing during thermomorphogenesis. Several clock related genes are alternatively spliced to regulate the clock period. High temperature triggers premature termination codons (PTC) contained *FLM* splicing variants transcription and further nonsense-mediated mRNA decay (NMD) to reduce *FLM*-β expression. High temperature also promotes SVP protein degradation to further release the SVP-FLM-β repression on *FT* transcription and stimulates flowering. High temperature modulates seed germination and cell elongation through repressing the formation of mature miR400

disturbs the splicing of the retention region of the first intron containing miR400, therefore inhibiting mature miR400 production. Hence, although primary *MIR400* transcription level is upregulated, heat stress decreases mature *miR400* levels to control plant seed germination and cell elongation (Brown et al. 2008; Yan et al. 2012) (Fig. 2).

There are also some reports showing the correlation between temperature responsive alternative splicing events and their impacts on plant development. For example, a temperature-sensitive mutant (*short rediffierentiation defective 2-1, srd2-1*) exhibits lateral root growth and cell proliferation defects (Ohtani et al. 2005; Ohtani et al. 2010). SRD2 stimulates the transcription of small nuclear RNAs (snRNAs), which are involved in Pre-mRNA splicing. Similarly, another mutant called *root initiation defective 1-1* (*rid1-1*) also displays temperature responsive tissue culture defects (Ohtani et al. 2013). *RID1* encodes a DEAH-Box RNA helicase, which participates in the Pre-mRNA splicing process. Taken together, these results suggest that high temperature responsive splicing events play a key role for plant development.

Warm temperature also promotes plant flowering. FLOWERING LOCUS T (FT) acts as florigen to promote floral initiation in *A. thaliana*. Previous studies have shown that PIF4 binds to *FT* promoter and induces *FT* transcription under high ambient temperature to stimulate flowering (Kumar et al. 2012), but this mechanism is still under debate (Fernández et al. 2016). MADS-box transcription factors FLOWERING LOCUS M (FLM) and SHORT VEGETATIVE PHASE (SVP) are reported as negative regulators of flowering through repressing *FT* expression (Lee et al. 2007; Scortecci et al. 2001). Mutation of *FLM* and *SVP* reduced plants sensitivity to temperature (Balasubramanian et al. 2006; Lee et al. 2007) and *FLM* is subject to temperature-dependent alternative splicing (Scortecci et al. 2001). SVP and FLM have been reported to interact with each other (Posé et al. 2013). The interaction capacity between different forms of FLM proteins and SVP contributes to the flowering time variations. Previous study demonstrates that the ratio between *FLM-β* and *FLM-δ* (two different splicing variants of *FLM*) expression levels significantly decreases under warm temperature. They argue that FLM-β interacts with SVP to repress flowering, while the interaction between FLM-δ and SVP induces flowering (Posé et al. 2013). Although this model nicely elucidates how temperature regulates flowering through alternative splicing, subsequent experimental evidences show that high temperature also induces the production of many non-canonical *FLM* transcripts (in addition to the *FLM-β* and *FLM-δ*), which contain premature termination codons (PTC) (Sureshkumar et al. 2016). It is well-known that nonsense-mediated mRNA decay (NMD) is a conserved and effective mRNA surveillance mechanism, which selectively degrades mRNAs with PTC and protects cells from the potentially deleterious effects of truncated proteins (Maquat 2004). Consistently, total *FLM* expression levels are increased in *A. thaliana* NMD factor mutants *upframeshift* (*upf*). Therefore, high temperature represses the total *FLM-β* expression through NMD pathway and FLM-β is actually the active flowering repressor (Sureshkumar et al. 2016). Recently, Capovilla et al. took advantage of CRISPR-Cas9 approach to generate

*FLM* mutants which lacking either the 2nd (FLM-ΔE2, *FLM-δ*) or 3rd exon (FLM-ΔE3, *FLM-β*) of *FLM*. Comparing with *flm* loss-of-function mutants, only expression of *FLM-δ* could not trigger earlier flowering phenotypes. In contrast, FLM-ΔE3 plants exhibit late flowering. These results suggest that the endogenous FLM-δ is unlikely to be a floral inducer (Capovilla et al. 2017). On another side, high temperature also promotes SVP protein degradation to further release the SVP-FLM-β repression on *FT* expression (Lee et al 2013) (Fig. 2).

## Alternative splicing in the regulation of circadian clock

Plant endogenous clock ensures plants to anticipate the daily environmental changes and adjusts their growth and metabolism in advance for obtaining the best fitness. In *A. thaliana*, the core circadian oscillators form interconnected feedback loops. CIRCADIAN CLOCK ASSOCIATED 1 (CCA1) and LATE ELONGATED HYPOCOTYL (LHY) peak at dawn. In the morning, CCA1/LHY repress *TIMING OF CAB EXPRESSION1* (*TOC1*) and evening complex (EC). TOC1 also represses the transcription of the EC complex (Pokhilko et al. 2012). Meanwhile, CCA1/LHY activate *PSEUDO-RESPONSE REGULATOR* (*PRR*) family members. From dawn to dusk, PRR9/7 suppress the expression of *CCA1/LHY* (Farré et al. 2005). In the evening, EC negatively regulates *PRR9* to release their repression on *CCA1/LHY* and maintain *CCA1/LHY* expression peaks in the early morning (Mizuno and Nakamichi 2005; Pokhilko et al. 2012).

Temperature controls the splicing events in the central clock oscillators (Fig. 2). For instance, transcription factor CCA1 has been identified to have two isoforms: CCA1 α and CCA1 β. CCA1 α and LHY form protein heterodimers to regulate circadian rhythms through enhancing the DNA binding capacity. However, high temperature induces *CCA1 β* pre-mRNA splicing to produce a protein without MYB DNA binding domain (Seo et al. 2012) (Fig. 3). CCA1 β competes with CCA1 α and LHY, producing non-functional heterodimers (Seo et al. 2012). For example, CCA1 directly binds to the conserved CCA1 binding site (CBS, AAAAATCT) in their target gene promoters, such as *TOC1* and *CCA1 HIKING EXPEDITION* (*CHE*) to negatively regulate their expression (Pruneda-Paz et al. 2009). However, chromatin immunoprecipitation (ChIP) assays show that the binding capacity is significantly reduced in plants overexpression of *CCA1 β* (Seo et al. 2012). Other core circadian regulators such as ELF3, TOC1, PRR7 and PRR9 also have different splicing variants (Capovilla et al. 2015) (Fig. 3). High temperature induces intron retention of *ELF3* and *TOC1*, resulting in their transcripts are degraded through the nonsense-mediated mRNA decay pathway (Kwon et al. 2014). More interestingly, ELF3 has been reported to be a thermosensor very recently. High temperature triggers ELF3 proteins phase separation through their intrinsic polyglutamine repeat and therefore inhibits their activity (Jung et al. 2020). Similar to *ELF3* and *TOC1,* high temperature also increases the intron retention of *PRR7* and *PRR9* (Kwon et al. 2014).

Although circadian clock maintains a relatively stable rhythm under a broad temperature range, known as temperature compensation (Gil and Park 2019). Spliceosome component *skip-1* mutants display prolonged period length under low temperature (Wang et al. 2012). As we stated above, SKIP is required for controlling high temperature-responsive alternative splicing (Jin et al. 2020), it is thus deserved to further explore what kinds of clock-related events are affected for causing its circadian defects.

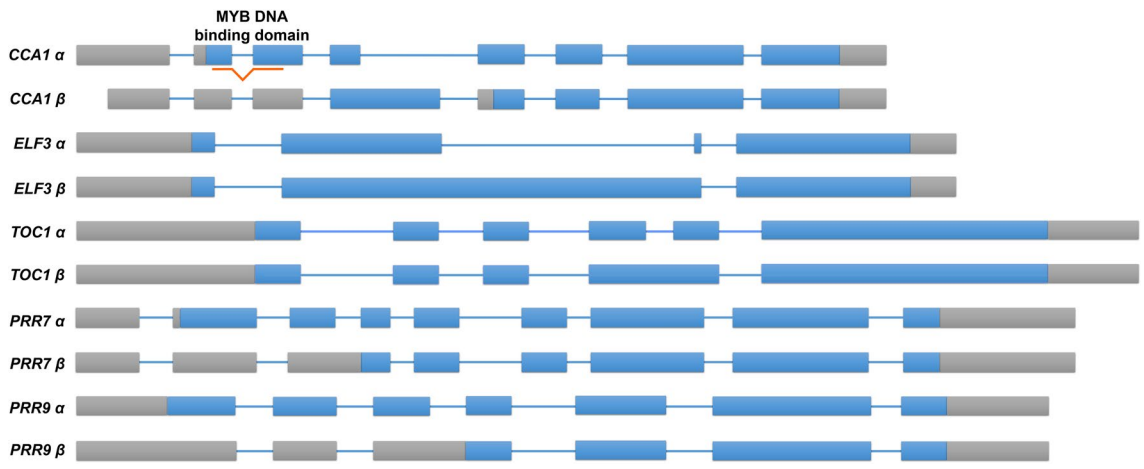


**Fig. 3** Schematic illustration of representative clock gene splicing forms. Grey box indicates untranslated region (UTR); Blue box shows exon; Blue straight line represents intron. The MYB DNA-binding domain encoded in *CCA1 a* first two exons is depicted with red lines

## Perspectives

Many studies have demonstrated that high temperature elicits alternative splicing events and these post-transcriptional regulations have diverse biological consequences. As we reviewed above, plants take advantage of alternative splicing to broaden its regulatory complexity when they are grown under high ambient temperature. In fact, these events exist from seed germination to flowering, covering almost all the major developmental stages during plant life cycle. Although there are plenty of breakthroughs in this field, there are still several mysteries awaiting to be answered in future.

1. How does temperature information signal to the alternative splicing machinery is unknown. It seems that there is a missing link between thermosensor (phytochrome) and the spliceosome. A recent study in mammals provides a possible clue for this question. It was shown that CDC-like kinases (CLKs) activity is very sensitive to temperature increase, even in in vitro phosphorylation assays. CLKs directly phosphorylate SR proteins (Colwill et al. 1996a, b; Haltenhof et al. 2020) and govern the global alternative splicing landscape (Haltenhof et al. 2020). There are three CLK homologs in *A. thaliana* (Yun et al. 1994) and one of them has already been shown to interact and phosphorylate SR proteins in plants (Golovkin and Reddy 1999). Therefore, it is intriguing to test whether *A. thaliana* CLKs function in transmitting temperature information into the spliceosome regulation.
2. How does HLS1-PIF4 module regulate alternative splicing? One possibility is that HLS1 and/or PIF4 recruits spliceosome component or regulatory proteins to modulate the spliceosome assembly or activity. It is deserved to test the binary protein–protein interactions of spliceosome components with HLS1-PIF4.
3. The coordination between transcriptional and post-transcriptional regulation under high temperature is not clear. In fact, there are few overlaps between differentially alternatively spliced genes and differentially expressed genes during thermomorphogenesis (Jin et al. 2020). Hence, it is interesting to pursue the underlying regulatory mechanisms and biological consequences, especially looking for the specificity on the selection of alternative splicing genes.

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**Author contributions** JL and ZZ wrote the manuscript.

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## Compliance with ethical standards

**Conflict of interest** None of the authors have any conflict of interest.

**Ethical approval** This article does not contain any studies with human participants or animals performed by any of the authors.

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