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Review

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Thermal adaptation and plasticity of the plant circadian clock

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Summary

Plant growth and development is widely affected by diverse temperature conditions. Although studies have been focused mainly on the effects of stressful temperature extremes in recent decades, nonstressful ambient temperatures also influence an array of plant growth and morphogenic aspects, a process termed thermomorphogenesis. Notably, accumulating evidence indicates that both stressful and nonstressful temperatures modulate the functional process of the circadian clock, a molecular timer of biological rhythms in higher eukaryotes and photosynthetic prokaryotes. The circadian clock can sustain robust and precise timing over a range of physiological temperatures. Genes and molecular mechanisms governing the temperature compensation process have been explored in different plant species. In addition, a ZEITLUPE/HSP90-mediated protein quality control mechanism helps plants maintain the thermal stability of the clock under heat stress. The thermal adaptation capability and plasticity of the clock are of particular interest in view of the growing concern about global climate changes. Considering these circumstances in the field, we believe that it is timely to provide a provoking discussion on the current knowledge of temperature regulation of the clock function. The review also will discuss stimulating ideas on this topic along with ecosystem management and future agricultural innovation.

I. Introduction

The circadian clock is a molecular timing device that generates the biological rhythms of numerous physiological and developmental processes with a period of *c.* 24 h in most living organisms. In many plant species, correct matching of the clock with the environment contributes to improved fitness, ensuring survival and optimal growth and performance under fluctuating environmental conditions (Michael *et al.*, 2003b; Dodd *et al.*, 2005a). Accordingly, a large fraction of plant genomes (> 30%) is estimated to be clock-controlled (Harmer *et al.*, 2000; Michael *et al.*, 2003b; Hayes *et al.*, 2010; Filichkin *et al.*, 2011 Marcolino-Gomes *et al.*, 2014). It is

now evident that the circadian clock serves as a master regulator of plant life and, thus, is linked with diverse signaling and metabolic pathways that facilitate developmental and environmental adaptive responses (Covington & Harmer, 2007; Covington *et al.*, 2008).

The plant circadian clock consists of three major components: the self-supporting central oscillator that generates rhythmic output signals, the input or entrainment pathways that entrain the central oscillator to environmental timing cues, and the output pathways that regulate diverse molecular and physiological events (Harmer, 2009). Large-scale molecular approaches, such as genomics, transcriptomics and proteomics studies, and mathematical and computational modeling have established the wiring structure of the plant circadian clock (Onai *et al.*, 2004; Fogelmark & Troein, 2014). More than 20 genes encoding clock components and signaling mediators have been identified through these approaches in *Arabidopsis*, and orthologs of the *Arabidopsis* clock genes have begun to be functionally characterized in other plant species (Song *et al.*, 2010). Similar to what is known in yeast and animals (Zhang & Kay, 2010), the plant central oscillator is composed of several interlocked transcription–translation feedback loops (Fogelmark & Troein, 2014). These feedback control mechanisms and associated multilayered signaling events constitute a complex network that confers versatile roles on the circadian clock, far exceeding the simple measurement of time.

The clock output pathways regulate a wide array of plant adaptation processes, including stem growth, flowering induction, photosynthesis and sugar metabolism, nutrient homeostasis, growth hormone signaling, and responses to biotic and abiotic stresses (Dodd *et al.*, 2005b; Covington & Harmer, 2007). Especially, global gene expression studies have shown that circadian datasets largely overlap with those triggered by environmental stimuli (Harmer *et al.*, 2000; Covington *et al.*, 2008), supporting an intimate linkage between the clock function and environmental adaptation.

Of particular interest are the thermal adaptive responses of the plant circadian clock, such as temperature compensation and temperature entrainment, which are mediated by a physiological range of ambient temperatures, and responses to stressful temperatures. Although diel temperature variations are known to act as a daily timing cue that entrains the clock (Heintzen *et al.*, 1994; Michael & McClung, 2002), the clock period is largely unchanged over a physiologically relevant range of temperatures (Harmer *et al.*, 2001), a process termed temperature compensation. Several signaling molecules and photoreceptors are identified as being essential for the temperature compensation response (Edwards *et al.*, 2005; Portolés & Más, 2010; Salomé *et al.*, 2010). Environmental temperatures also modify the alternative splicing of many clock genes (James *et al.*, 2012; Seo *et al.*, 2012). One of the best characterized is the cold-responsive alternative splicing of *CIRCADIAN CLOCK-ASSOCIATED 1* (*CCA1*) gene (Seo *et al.*, 2012). Meanwhile, it has been reported recently that the E3 ubiquitin ligase ZEITLUPE (ZTL) and its interacting partner, heat shock protein 90 (HSP90), constitute a distinct protein quality-control mechanism that stabilizes the plant circadian clock under high-temperature conditions (Gil *et al.*, 2017). The wide repertoire of temperature effects on the clock function illustrates the thermal plasticity of the plant circadian clock and certainly sheds light on how global warming would be managed with innovative engineering of future crop agriculture.

Understanding gene function and molecular mechanisms underlying the clock functioning is a prerequisite for developing means of enhancing plant environmental fitness. It is notable that many clock-manipulated plants exhibit normal growth with minimal side effects, which is a desired trait for future crop biotechnology (de Montaigu *et al.*, 2010). Along with the accumulating data supporting the critical influence of environmental temperatures on the clock function, we will provide an overview of recent progress in understanding the thermal adaptation of the plant circadian clock. We will also discuss ways of engineering clock components and their working schemes with an aim of improving plant growth and performance under changing temperature conditions.

II. Molecular organization of the plant circadian clock

1. Plant circadian rhythms and behaviors

Circadian rhythms are biological cycles generated from the self-sustaining central oscillators. The first experimental evidence supporting the presence of an endogenous timer was described by the French astronomer de Mairan in 1727. He observed that plant leaves move rhythmically even in the absence of any environmental light stimuli (Más, 2005). Following the experimental demonstration of the plant circadian clock, diverse studies have investigated the prevailing roles of the clock in molecular and physiological control of growth and behaviors, using mainly the dicot *Arabidopsis thaliana* and the green algae *Ostreococcus tauri* and *Chlamydomonas reinhardtii* (Linde *et al.*, 2017). In recent decades, great progress has been made in understanding the molecular nature and gene regulatory networks governing the clock function in plants.

In higher plants, the circadian rhythms of biological activities and behaviors are actively adjusted under changing environments to allow plants to anticipate regular diel and seasonal changes (Troein *et al.*, 2009). The molecular mechanisms of the cellular and physiological processes and behaviors controlled by the clock have been comprehensively studied in leaf movement, stomatal opening and closing, hypocotyl growth, subcellular localization of organelles, and photoperiodic control of flowering induction (Somers, 1999; Harmer, 2009). The clock-driven oscillations in gene expression and downstream biochemical events, such as protein production, cellular translocation, and ubiquitination and controlled turn over, underlie the circadian behaviors (Giraud *et al.*, 2010).

It is notable that disruption of the clock function often incurs fitness costs for plants. For example, although clock-defective plants exhibit enhanced performance under certain stressful conditions, they are outcompeted by plants possessing functional clock systems under normal growth conditions (Dodd *et al.*, 2005a; Seo *et al.*, 2012; Kim *et al.*, 2013). This characteristic supports the adaptive value of the circadian clock in coordinating plant behaviors and performance with the environment. Consequently, sustaining robust circadian rhythms and behaviors is undoubtedly

necessary for optimal plant growth and performance (Green *et al.*, 2002; Michael *et al.*, 2003b; Dodd *et al.*, 2005a).

2. Molecular modeling of the circadian feedback loops

The plant clock system consists of gene circuits containing multiple negative feedback loops (Shalit-Kaneh *et al.*, 2018). Although the central oscillators of angiosperm clocks are modeled as a complicated network of interlocked transcription–translation feedback loops, those of green algal clocks are described as a simple loop consisting of only a few genes (Matsuo *et al.*, 2008; Corellou *et al.*, 2009).

In *Arabidopsis*, the constituents of the central oscillator are classified according to the timing of their occurrence during the clock signaling (Harmer, 2009; McClung & Gutiérrez, 2010; Pruneda-Paz & Kay, 2010). The biological oscillator consists of cascades of repressive transcription factors (Fig. 1). The main feedback loop contains CCA1, its functional ortholog LATE ELONGATED HYPOCOTYL (LHY), TIMING OF CAB EXPRESSION 1 (TOC1, also known as PSEUDO-RESPONSE REGULATOR1 (PRR1)) and some additional clock components, such as the evening complex (EC; Hazen *et al.*, 2005; Kikis *et al.*, 2005; Nusinow *et al.*, 2011). The EC complex harbors the myeloblastosis (MYB) transcription factor LUX ARRHYTHMO (LUX) and two additional transcriptional regulators, EARLY FLOWERING 3 (ELF3) and ELF4. In the feedback loop, CCA1 and LHY repress the transcription of the *TOC1* gene in the morning, whereas TOC1 represses the transcription of the *CCA1* and *LHY* genes in the evening (Alabadí *et al.*, 2001, 2002; Gendron *et al.*, 2012; Huang *et al.*, 2012). By contrast, CCA1 and LHY activate the *PRR7* and *PRR9* genes (Harmer & Kay, 2005; Mizuno & Nakamichi, 2005), whose gene products in turn repress the *CCA1* gene transcription (Nakamichi *et al.*, 2010). Meanwhile, TOC1 represses the transcription of the EC components, *PRR* and *GI* genes (Huang *et al.*, 2012; Nagel & Kay, 2013). The repression of the *PRR9* gene by the EC complex in the morning adds further complexity to the gene networks (Dixon *et al.*, 2011), and thus the boundaries of individual feedback loops are mutually overlapped.

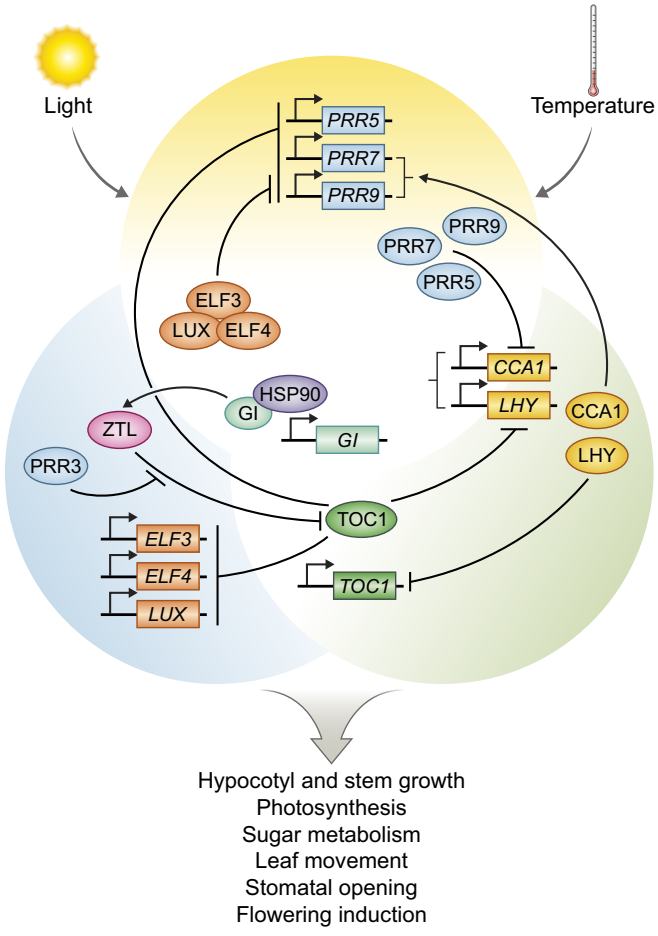

Fig. 1 Molecular organization of the plant circadian clock. Light and temperature information is integrated into the central oscillator via the entrainment or input pathways. Circadian rhythms generated from the central oscillator regulate diverse plant growth and developmental processes and environmental adaptation through the output pathways. The central oscillator consists of several interlocked transcription–translation feedback loops. Genes are shaped as rectangles, and proteins are shaped as ellipses. Lines with arrowheads indicate positive effects. Lines with bars indicate negative effects. PRR, pseudo-response regulator; CCA1, circadian clock-associated 1; LHY, late elongated hypocotyl; TOC1, timing of cab expression 1; GI, gigantea; HSP90, heat shock protein 90; ZTL, zeitlupe; ELF, early flowering; LUX, lux arrhythmo.

Other major constituents of the central oscillator include GIGANTEA (GI), ZTL and PRR3 (Kim *et al.*, 2007; Martin-Tryon *et al.*, 2007; Ito *et al.*, 2008). GI is conserved among various plant species (Izawa *et al.*, 2011; Ke *et al.*, 2017). It is interesting that although the protein domains of GI have not been functionally characterized, it has pivotal roles in a wide range of plant developmental and physiological processes (Mishra & Panigrahi, 2015). GI-defective *Arabidopsis* mutants exhibit delayed flowering under long days (Fowler *et al.*, 1999; Sawa *et al.*, 2007). They also have an altered circadian period (Kim *et al.*, 2007). The transcription of the *GI* gene is regulated by the circadian clock (Fowler *et al.*, 1999). LHY and CCA1 repress *GI* transcription in the early morning, whereas *GI* transcription reaches a peak in the middle of the day (Lu *et al.*, 2012). GI also is known to mediate sugar metabolic pathways in a clock-dependent manner (Dalchau *et al.*, 2011).

ZTL is an F-box protein that assembles a Skp, Cullin, F-box containing complex (Han *et al.*, 2004). The ZTL-mediated degradation of TOC1 establishes a critical circadian regulatory system (Kim *et al.*, 2007). PRR3 inhibits TOC1 degradation by sequestering it from ZTL (Para *et al.*, 2007). It is notable that although *ZTL* transcription is constant across the diel cycles (Somers *et al.*, 2000, 2004), ZTL protein accumulation is rhythmic, peaking at dusk (Kim *et al.*, 2003). GI interacts with ZTL via the LOV (light, oxygen, voltage) domain (Kim *et al.*, 2007). This protein–protein interaction stabilizes the ZTL protein in blue light, leading to the rhythmic accumulation of ZTL. Moreover, it was recently demonstrated that GI, in conjunction with the chaperone activity of HSP90, enhances the maturation of ZTL during the day (Cha *et al.*, 2017). Thus, it is evident that ZTL function is modulated primarily at the protein level during circadian clock functioning.

Through the concerted actions of the clock components, the circadian clock enables plants to shape their molecular genetic, physiological, metabolic, developmental and behavioral processes according to the most appropriate time of the day. In addition, the circadian clock also controls cell cycle progression by modulating elements of the DNA pre-replicative machinery (Fung-Uceda *et al.*, 2018), further supporting that the synchronization of the clock with the environment is crucial for sustaining optimal plant growth (Dodd *et al.*, 2005a; Yerushalmi & Green, 2009).

3. Entrainment of the plant circadian clock

In the laboratory, most chronobiological studies on the clock are conducted under constant conditions to examine free-running circadian rhythms. However, external signals oscillate continuously over daily cycles under natural conditions, as observed with diel variations in temperature, humidity, wind, and light quality and quantity. These oscillating environmental cues coordinately entrain the clock to the surroundings. Light and temperature are the two strongest entraining stimuli for the plant biological clocks. Consequently, plants can orchestrate the timing of their biological processes with natural light and temperature cycles.

Light entrainment Light is not only the sole energy source for plant growth, but also a prevailing environmental signal that triggers a broad spectrum of plant developmental and adaptation processes. Light also serves as a dominant timing cue that resets the circadian clock. Accordingly, there is substantial evidence proving that light-sensing photoreceptors are closely linked with the clock function (Millar, 2003).

Most early studies on light entrainment were focused mainly on single light stimuli, such as a pulse of light, under laboratory conditions. A single pulse of light is sufficient to rapidly change the phase of the clock, thus altering the expression of the clock components (Johnson, 1999; Ohara *et al.*, 2015). Therefore, the phase of the clock is readily shifted by light treatments in a manner that achieves useful entrainment in terms of the opposing effects of a light pulse relative to the existing phase of the circadian clock. This type of light entrainment of the clock, termed nonparametric entrainment, is analyzed systematically by deriving phase response curves (PRCs; Granada *et al.*, 2009), a useful method for obtaining mechanistic information on the clock responses to resetting stimuli, such as light. Through PRCs, it was found that light exposure before dawn typically triggers a phase-advanced response, whereas before dusk it produces a delayed phase in circadian rhythms (Salomé & McClung, 2005a). This indicates that the modes of phase shifts are determined by the timing of the onset of a light pulse.

Continuous or prolonged illumination affects the period of circadian rhythms, called parametric entrainment. With parametric entrainment, light fluence rates influence the clock period: increasing fluence rates lead to faster clock rhythms, resulting in a shorter clock period; decreasing fluence rates, meanwhile, produce slower clock rhythms, resulting in a longer period (Oakenfull & Davis, 2017). In nature, light intensity and duration fluctuate constantly throughout the day and year, highlighting a requirement for both parametric and nonparametric entrainment mechanisms for successful synchronization of the endogenous timers with the environment. This would explain, at least in part, why plants have evolved complicated light-sensing mechanisms.

Both the red/far-red light-sensing phytochrome (phy) and the blue light-sensing cryptochrome (cry) photoreceptors modulate the clock period by conveying light input signals to the circadian clock (Somers *et al.*, 1998). In addition, the transcription of the photoreceptor genes is rhythmic (Tóth *et al.*, 2001). There is evidence that the photosensitivity and expression of the photoreceptors are under the control of the clock gating (Tóth *et al.*, 2001). Mutations in either the phyA or phyB phytochrome alter the clock period in a light intensity-dependent manner (Somers *et al.*, 1998). Moreover, mutations in phyA have been found to lengthen the clock period by up to 3 h under low fluence red and blue light regimes. Furthermore, sensitivity to high fluence rates under red light is disrupted in phyB-defective mutants. It is apparent that plants detect the wavelengths and fluence rates of light by adjusting the recruitment of different phytochromes for the light entrainment of the clock.

The cry photoreceptors are known to convey low-intensity red light and high-intensity blue light signals to the circadian clock (Devlin & Kay, 2000). As cry1 has no peak of absorption in the red light spectrum, the functional role of cry1 in conveying light signals to the circadian clock is likely to be exerted downstream of phyA. Meanwhile, alterations to the clock period in ZTL-defective mutants were found to depend on fluence rates, suggesting that the ZTL photoreceptor functions in combination with other photoreceptors in modulating the clock period, at least in part (Somers *et al.*, 2000). Further molecular genetic studies on the potential interactions between different photoreceptors are required to elucidate the interdependency of photoreceptors in mediating the light entrainment of the clock.

Temperature entrainment Recent studies have emphasized the significance of temperature cues in entraining the plant circadian clock. However, although molecular mechanisms underlying light entrainment of the clock are relatively well characterized, those mediating temperature entrainment are only poorly understood. One explanation for the lack of a full understanding of the thermal adaptation process may be that variation in environmental temperatures alters virtually all biochemical and morphological properties in plants (Hatfield & Prueger, 2015). Overall, it is extremely difficult to identify critical thermosensors that specifically mediate the entrainment event and to define the direct effects of temperature. Nevertheless, circadian rhythms are now recognized as being efficiently entrained by warm/cold temperature cycles that differ by 4°C or less in *Arabidopsis* (Thines & Harmon, 2010), showing that temperature information acts as a strong stimulus for the entrainment of the plant circadian clock.

Although no clock-specific thermosensors have been identified in plants to date, several molecules have been proposed to integrate temperature information to the clock, including phyB, which fulfills the role of a thermosensor, at least partly (Jung *et al.*, 2016; Legris *et al.*, 2016). Although phyB is not a clock component, it may be able to convey temperature information to the clock in

conjunction with its associated clock-related components. Nucleosomes harboring an alternative histone H2A.Z have been reported as being important for perceiving ambient temperatures through DNA–nucleosome fluctuations (Kumar & Wigge, 2010); temperature-responsive accumulation of H2A.Z in nucleosomes alters chromatin architecture, conveying temperature information to gene transcriptional responses. Phytochrome B-mediated temperature sensing and temperature-induced alterations in DNA–nucleosome dynamics are suspected of being associated with the temperature-mediated entrainment of the clock. Meanwhile, it has been reported that *Arabidopsis* plants lacking functional PRR7 and PRR9 are unable to entrain the clock in response to warm–cold cycles (Salomé & McClung, 2005b), suggesting that these PRR proteins also are important for the temperature entrainment of the clock.

It is notable that many – if not all – clock genes undergo alternative splicing. The alternative splicing patterns of the clock genes are influenced by environmental conditions, especially temperature (Filichkin & Mockler, 2012; James *et al.*, 2012; Kwon *et al.*, 2014). The protein splice variants, which are either N-terminally or C-terminally truncated and thus lack one or two protein domains, act as negative regulators of the *bona fide* clock components, as seminally illustrated by CCA1 alternative splicing (Seo *et al.*, 2012). It is becoming clear that splicing components, such as Protein Arginine Methyltransferase 5 (Sanchez *et al.*, 2010), SNW/SKI-INTERACTING PROTEIN (SKIP; Wang *et al.*, 2012), SICKLE (SIC; Marshall *et al.*, 2016) and GEM NUCLEAR ORGANELLE ASSOCIATED PROTEIN 2 (Schlaen *et al.*, 2015), are important for temperature control of the clock function. These observations indicate that the alternative splicing of the clock genes also may function in delivering temperature information to the clock entrainment process.

III. Temperature compensation

Maintaining accurate timing is essential for plant survival and optimal growth. It also is important for anticipating daily and seasonal cues, such as light duration and temperature variation. These requirements necessitate that the circadian clock adapts to changing temperatures, but without disturbing its biological timing. The plant circadian clock is known to possess a molecular buffering system that enables plants to sustain robust circadian rhythms and thus render the clock output pathways largely insensitive to a physiological range of temperature fluctuations (Gould *et al.*, 2006). This phenomenon is referred to as temperature compensation (Harmer *et al.*, 2001). The temperature compensation mechanism is highly conserved and considered a critical property of the circadian clock in living organisms.

The molecular systems regulating temperature compensation have been explored intensively in *Neurospora* fungi, which are exposed to a wide range of ambient temperatures under natural conditions (Ruoff *et al.*, 2005; Dunlap *et al.*, 2007). The FREQUENCY (FRQ) protein is a key component of the central oscillator in fungal cells. Alternative splicing of *frq* pre-mRNA produces two protein isoforms, a long-form FRQ (l-FRQ) and a short-form FRQ (s-FRQ; Liu *et al.*, 1997). The l-FRQ isoforms accumulate at high temperatures, whereas the s-FRQ isoforms accumulate at low temperatures (Fig. 2a). Phosphorylation of the FRQ proteins by casein kinase 2 is known to modulate the

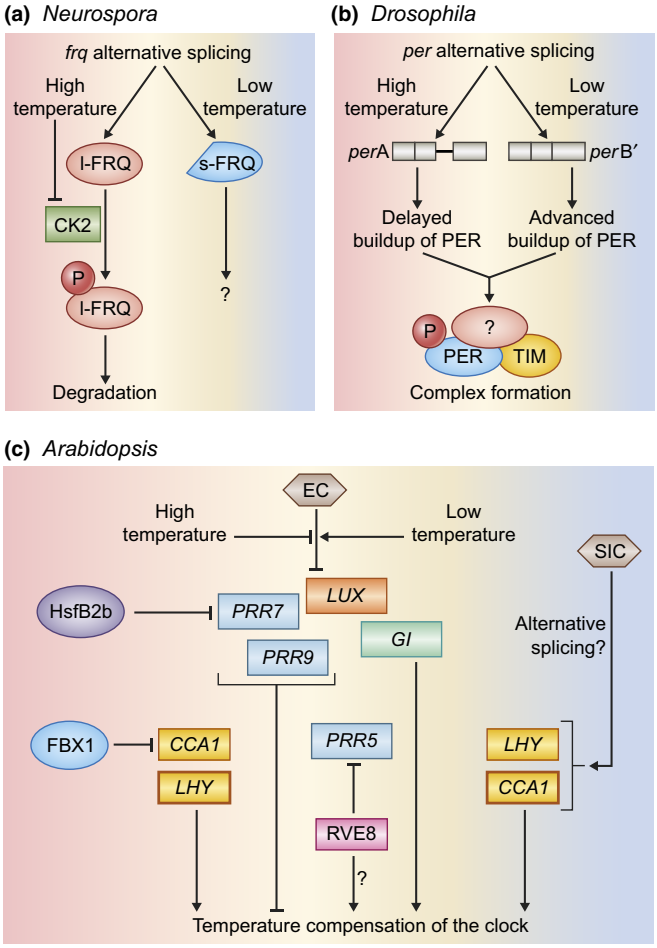

Fig. 2 Schematic models of temperature compensation in animals and plants. The red-to-blue color gradient represents a high-to-low ambient temperature range. Clock components are arranged in the appropriate positions in the color gradient. (a) Temperature compensation in *Neurospora*. High ambient temperatures stimulate the production of long-form FREQUENCY (l-FRQ). Casein kinase (CK)2-mediated phosphorylation of l-FRQ, which triggers its degradation, is inhibited under high-temperature conditions. It is unclear whether the short-form FRQ (s-FRQ) form plays any role in thermal adaptation. (b) Temperature compensation in *Drosophila*. Low ambient temperatures stimulate the splicing of the 3′-intron, producing period (*per*)B′ mRNA. High ambient temperature-induced alternative splicing results in accumulation of a splice variant *per*A mRNA and its gene product. The temperature-mediated formation of protein complexes containing PER and Timeless (TIM) or phosphorylated PER is a critical constituent of the proposed models for temperature compensation in *Drosophila*. (c) Temperature compensation in *Arabidopsis*. A group of clock components, with associated regulatory mechanisms, is arranged in the signaling pathways. It is likely that multiple signaling networks function coordinately to achieve temperature compensation. EC, evening complex; HsfB2b, heat shock factor B2b; LUX, lux arrhythmo; PRR, pseudo-response regulator; GI, gigantea; FBX1, flowering basic helix-loop-helix 1; CCA1, circadian clock-associated 1; LHY, late elongated hypocotyl; RVE8, reveille 8; SIC, sickle. For details, see Section III.

alternating turnover of the FRQ isoforms during the temperature compensation process (Mehra *et al.*, 2009).

In mammals and *Drosophila*, the Period (PER) and Timeless (TIM) proteins are central components of the circadian pacemakers (Lee *et al.*, 1999). Differential splicing of *per* pre-mRNA and formation of PER-PER or PER-TIM dimers are known to be temperature-sensitive (Huang *et al.*, 1995; Edery, 1999). At high temperatures, intron retention leads to the *per*A RNA variant being produced predominantly, resulting in a delayed buildup of overall PER concentrations. At low temperatures, however, the *per*B RNA splice variant is produced, which results in an advanced accumulation of PER proteins (Fig. 2b). Although several models have been proposed to explain the alternative regulatory steps (Hong *et al.*, 2007; Hatakeyama & Kaneko, 2012), none have been experimentally confirmed, due primarily to the complexity of the clock signaling.

In higher plants, accumulating evidence support that a group of central clock components, including CCA1, LHY, GI, PRR7, PRR9 and the EC complex, is involved in the temperature compensation process (Gould *et al.*, 2006; Salomé *et al.*, 2010). It is notable that CCA1 and its functional ortholog LHY have differential roles during temperature compensation (Gould *et al.*, 2006). Although CCA1 functions primarily in a range of low ambient temperatures, LHY function is more prominent in a range of high ambient temperatures (Fig. 2c). Notably, the alternative splicing of several clock genes is temperature-responsive. A recent study has shown that SIC, which regulates some microRNA biogenesis and degradation of spliced intron RNA (Zhan *et al.*, 2012), is required to produce CCA1 and LHY splice variants under low ambient temperature conditions (Marshall *et al.*, 2016). SIC and associated biochemical processes are thought to affect the temperature compensation process. GI in particular is critical for the CCA1/LHY-mediated buffering of robust circadian rhythmicity (Gould *et al.*, 2006), supporting a role for GI as a potential modulator of temperature compensation. Furthermore, *Arabidopsis* mutants defective in both PRR7 and PRR9 exhibit a temperature overcompensation phenotype (Salomé *et al.*, 2010). It is currently unclear whether the clock components function independently or cooperatively via a unified signaling network during the temperature compensation process.

Recent studies indicate that FLOWERING BASIC HELIX-LOOP-HELIX 1 (FBH1) and a group of heat shock factor (HSF) proteins also are important for the responses of the plant circadian clock to warm temperatures. On the one hand, overproduction of FBH1 has been shown to alter the pace of the clock by modulating CCA1 responses to temperature changes (Nagel *et al.*, 2014). The heat-responsive HsfB2b transcription factor also is known to repress *PRR7* transcription by binding to its promoter under elevated temperature conditions (Kolmos *et al.*, 2014). On the other hand, lack of HsfB2b leads to a shortened circadian period under the same temperature conditions, highlighting the harmonized integration of temperature cues and clock information for optimal plant growth and fitness.

IV. Temperature regulation of circadian behaviors

In nature, plants frequently encounter drastic shifts in diverse temperature regimes (Fig. 3). Consequently, they are equipped with versatile defense mechanisms to trigger distinct adaptive responses to various temperature stimuli. *Arabidopsis* plants exhibit optimal growth and performance at *c.* 22°C. However, temperatures below 16°C and above 29°C are stressful to this plant species, causing a severe reduction in growth, distortion of plant architecture and morphology, and reduced seed production. Although a temperature range of 16–28°C is not stressful to *Arabidopsis*, it induces distinct thermomorphogenic adjustments. For example, plants exhibit hypocotyl elongation, leaf petiole extension, increased leaf hyponasty and formation of small, thin leaves in

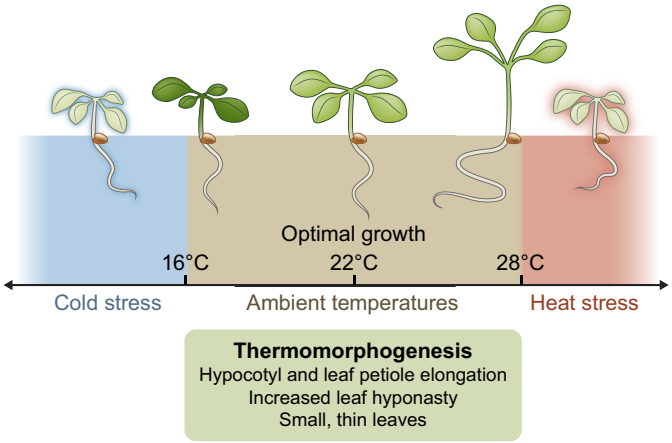

Fig. 3 Effects of different temperature regimes on plant growth and adaptation. *Arabidopsis* plants achieve optimal growth at 22°C. However, at temperatures lower than 16°C or higher than 28°C, they exhibit stress responses, such as reduced stem and leaf growth, chlorophyll biosynthesis and seed production. Under a physiological range of high ambient temperatures (23–28°C), whereas overall plant growth is normal without discernible stress symptoms, *Arabidopsis* exhibit distinct thermomorphogenic adaptation responses, such as hypocotyl elongation, leaf petiole extension, increased leaf hyponasty and small, thin leaves. These thermomorphogenic traits help plants to distance the heat-labile shoot apical meristemic tissues from the warm soil surface and enhance evaporative leaf cooling, ensuring optimal growth and performance. Note that circadian rhythms are sustained in this temperature range via the temperature compensation mechanism. It is possible that temperature-mediated leaf hyponasty might be associated with the clock-controlled leaf movement.

response to warm temperatures. These thermomorphogenic traits help plants maintain the heat-labile shoot apical meristemic tissues away from the hot soil surface and facilitate evaporative cooling of the leaves (Quint *et al.*, 2016). Distinct sets of genes and signaling mechanisms are known to mediate plant responses to heat and cold stress and thermomorphogenic stimuli.

Environmental temperatures also affect the plant clock function profoundly. As described above, temperature compensation is the most prominent property of the circadian clock. Through the nonstressful ambient temperature adaptive strategy, plants can maintain biological activity at optimal levels even under fluctuating temperature conditions (Gould *et al.*, 2006).

The plant circadian clock also responds actively to stress-inducing high and low temperatures, although genetic components and underlying signaling schemes are not fully understood at the molecular level. In recent years, global warming is emerging as a critical ecological concern for vegetation and crop agriculture worldwide (Willis *et al.*, 2008). Moreover, the global rise in temperatures frequently results in stressful local temperature extremes. It has been investigated how stressful high or low temperatures negatively affect the plant throughout its life cycle, such as disturbance of plant morphology, cytoskeleton stability and cellular mechanisms (Wahid *et al.*, 2007; Miura & Furumoto, 2013). We also will provide an overview of the current knowledge of how plants adapt their clock systems to stressful temperatures.

1. Circadian behaviors under cold stress

Plants activate diverse cold adaptation mechanisms under cold-temperature stress. A small group of cold-responsive transcription factors, the C-repeat binding factors (CBFs), plays a central role by activating downstream target genes, such as *cold-regulated* (*COR*) genes, constituting the pivotal CBF-COR regulon (Wahid *et al.*, 2007; Miura & Furumoto, 2013). The inducer of CBF expression 1 (ICE1) transcription factor acts as the master regulator of the CBF-mediated transcription and the resulting acquisition of freezing tolerance (Gilmour *et al.*, 1998).

Notably, the acquisition of cold tolerance is gated by the time of the day (Fowler *et al.*, 2005). In other words, the induction of cold tolerance is differentiated, depending on the time of the day at which plants are exposed to cold temperatures, indicating that cold stress responses are functionally interconnected with the circadian clock. In addition, the cold-temperature-mediated induction of *CBF* genes exhibits circadian rhythms, reaching a peak at noon and a low point at night (Fowler *et al.*, 2005), indicating that the clock output pathways apparently function upstream of the CBF-mediated cold response pathway.

Several clock components act as potential upstream regulators of *CBF* expression under cold-temperature conditions (Fig. 4a,b). The PRR5, 7 and 9 proteins repress the expression of *CBF* genes to shape its circadian rhythms (Nakamichi *et al.*, 2009), as do TOC1 and EC (Chow *et al.*, 2014). Likewise, phytochrome interacting factor 4 (PIF4) and PIF7 bind to the G-box sequence element within the *CBF* promoters to repress their expression in a clock-dependent manner (Lee & Thomashow, 2012). It also is known that CCA1 and LHY activate *CBF* expression by binding directly

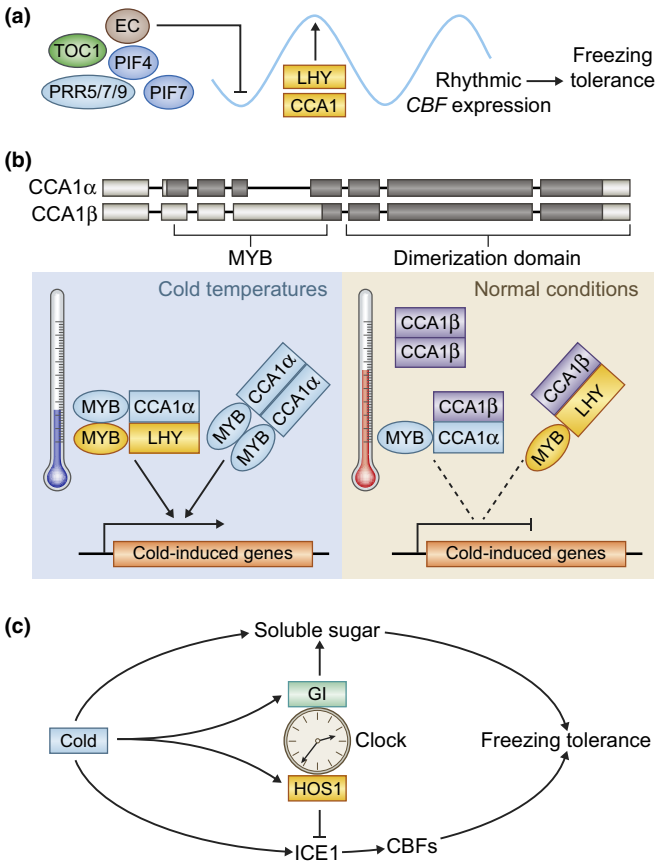

Fig. 4 Molecular mechanisms governing circadian behaviors under cold-temperature conditions. (a) Clock regulation of the expression of the *C-repeat binding factor* (*CBF*) genes. Coordinated actions of clock components and clock-related components (phytochrome interacting factor 4 (PIF4) and PIF7) generate a rhythmic expression of *CBF* genes, leading to the induction of freezing tolerance. Lines with arrowheads indicate induction of gene expression. Those with bars indicate repression of gene expression. (b) Functional linkage between the clock and cold adaptation via *CIRCADIAN CLOCK-ASSOCIATED 1* (*CCA1*) alternative splicing. Both the CCA1α and CCA1β isoforms are required for proper clock functioning. The splice variant CCA1β lacks the myeloblastosis (MYB) DNA-binding domain and forms nonfunctional heterodimers with CCA1α or LATE ELONGATED HYPOCOTYL (LHY). Cold temperatures suppress the alternative splicing reaction, leading to the overproduction of CCA1α that binds directly to the *CBF* promoters. Thus, under cold-temperature conditions, plants obtain cold resistance by sacrificing the clock function. (c) Putative signaling events in clock-mediated regulation of cold adaptation via GIGANTEA (GI) and HOS1. Cold temperatures induce the expression of two clock genes, *GI* and *HOS1*. GI activates the production of soluble sugars, which contribute to the induction of freezing tolerance. HOS1 acts as a cold signaling attenuator, preventing excessive cold responses that are harmful to plant survival.

to the promoters (Dong *et al.*, 2011; Fig. 4a). Interestingly, CBF1 was observed to directly regulate the expression of the *LUX* gene, thereby integrating cold-temperature information to the clock (Chow *et al.*, 2014). All of these data imply that the signaling link between cold responses and the clock functioning could be more complex than previously thought.

Alternative splicing of the *CCA1* primary transcripts produces two CCA1 isoforms, the transcriptionally active CCA1α form and the N-terminally truncated CCA1β form (Fig. 4b). It has been

proven that a balance between the two CCA1 isoforms was necessary for proper clock functioning (Seo *et al.*, 2012). Interestingly, alternative splicing of the *CCA1* is suppressed at cold temperatures, resulting in a high level of CCA1α accumulation. CCA1α directly activates the *CBF* genes (Dong *et al.*, 2011; Fig. 4b). Although CCA1β contains a protein domain required for heterodimerization with CCA1α, it lacks the MYB DNA-binding domain; consequently, the CCA1α-CCA1β heterodimers are not able to efficiently bind to the *CBF* promoters. Overall, the working scenario is that under normal temperature conditions, the CCA1α and CCA1β proteins are balanced to sustain robust circadian rhythms. By contrast, the CCA1α isoform accumulates to a high concentration in response to cold temperatures (Seo *et al.*, 2012; Kwon *et al.*, 2014), leading to the induction of *CBF* genes and freezing tolerance. Perturbation of the balance between the CCA1 isoforms disrupts the clock function at cold temperatures but triggers the acquisition of freezing tolerance. Therefore, *CCA1* alternative splicing is a molecular device that links the clock with cold stress responses, providing a distinct strategy for cold adaptation.

Clock control of GI-mediated sugar metabolism also is interconnected with plant responses to cold stress (Cao *et al.*, 2005, 2007). It was observed that cold temperatures induce *GI* expression, leading to the stimulation of soluble sugar production, which in turn contributes to the establishment of freezing tolerance (Cao *et al.*, 2005; Fig. 4c). In addition, the HOS1 E3 ubiquitin ligase, a negative regulator of cold responses through the degradation of ICE1 proteins (Dong *et al.*, 2006), is a component of the plant clock system (MacGregor *et al.*, 2013). HOS1 likely conveys the clock information to the CBF-COR regulon to adjust cold responses appropriately (Fig. 4c). Whether HOS1-mediated clock signaling is linked with GI-mediated modulation of freezing tolerance deserves further investigation.

The intimate link between the clock functioning and cold-temperature responses would serve as a molecular knob that helps plants anticipate cold temperatures, ensuring a timely induction of the cold adaptive responses required for plant survival. It will be interesting to investigate how metabolites and cellular energy are reallocated during the coordination between freezing tolerance responses and the clock function in response to cold stress.

2. Circadian behaviors under heat stress

Recent reports strongly support that the circadian clock also is associated with plant responses to high-temperature stress (Karayekov *et al.*, 2013; Grundy *et al.*, 2015). However, although the functional linkage between the clock and cold stress responses at the molecular level has mostly been elucidated, the molecular mechanisms underlying high-temperature control of the clock function have not been explored in detail. The genes and signaling pathways governing heat stress adaptation have been extensively studied in plants (Iba, 2002; Wahid *et al.*, 2007). Plant clock genes and timing mechanisms have been appropriately investigated. Therefore, a potential way of elucidating the genetic link between the clock function and heat stress responses would be to integrate the two gene regulatory networks and identify the molecular mechanisms that mediate the signaling crosstalks. We will briefly discuss two potential molecular mechanisms governing the high temperature control of the clock function.

Alternative splicing of clock genes under heat stress Although distinct sets of genes are involved in cold and heat stress responses, similar molecular and biochemical mechanisms are shared by the two thermal adaptation processes. For example, as in cold adaptation, alternative splicing is a critical molecular event mediating plant responses to high temperatures. Core clock genes, such as *CCA1*, *LKP2*, *PRR7*, *TOC1* and *ELF3*, undergo heat-responsive alternative splicing (Filichkin & Mockler, 2012; Kwon *et al.*, 2014), supporting that alternative splicing links the clock function with high-temperature responses.

It is currently unknown how alternative splicing of the clock genes is modulated by high temperatures. One potential mechanism would involve the splicing factor SKIP, a component of the spliceosome (Lim *et al.*, 2009; Fig. 5a). It has been reported that SKIP-deficient *Arabidopsis* plants exhibit a long-period circadian phenotype, most likely due to the accumulation of unproductive PRR7 and PRR9 splice variants (Wang *et al.*, 2012). Notably, SKIP activity is heat-sensitive (Wang *et al.*, 2012), and SKIP-

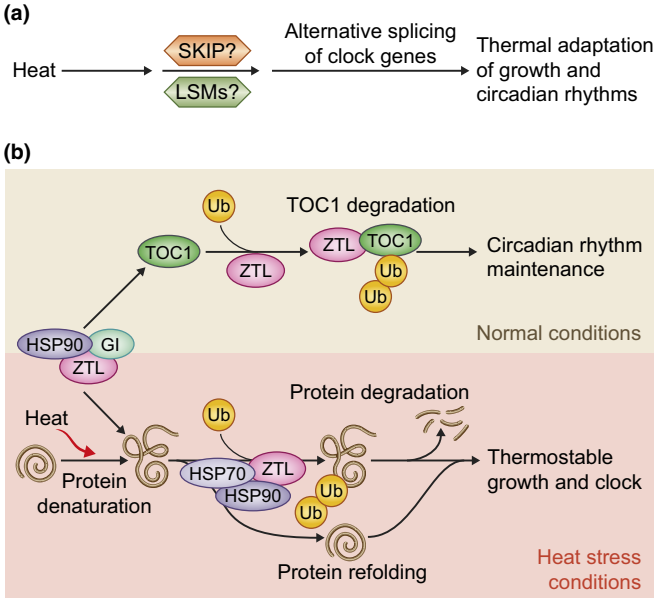

Fig. 5 Heat stress adaptation of circadian behaviors. (a) Schematic alternative splicing of clock genes under heat stress. Splicing factors, SNW/SKI-interacting protein (SKIP) and SM-like (LSM) would mediate the heat stress-induced alternative splicing of key clock genes, such as circadian clock-associated 1 (*CCA1*), pseudo-response regulators, and early flowering 3. This scheme is conceptually similar to the alternative splicing event of *CCA1* under cold conditions. (b) Working model for the ZEITLUPE (ZTL)-mediated thermostabilization of the circadian clock. heat shock protein 90 (HSP90) and gigantea (GI) stabilize ZTL maturation, leading to rhythmic accumulation of ZTL in the daytime. Under normal conditions, targeted degradation of timing of cab expression 1 (TOC1) by the ZTL-mediated ubiquitin-proteasome pathways maintains circadian rhythms. Upon exposure to heat stress, the ZTL–HSP complex clears heat-induced protein aggregates, ensuring robust clock function and thermostable growth.

mediated alternative splicing of the clock genes is thought to be altered by high temperatures. The heat-induced alternative splicing of the clock genes also would be mediated by SM-LIKE (LSM) RNA-binding proteins, which constitute the spliceosome U6 small nuclear ribonucleoprotein complex (Tharun, 2009). Recent studies have shown that LSM proteins play a role in maintaining circadian rhythms by modulating alternative splicing and RNA stability of the clock genes in both plants and mammals (Perea-Resa *et al.*, 2012; Pizarro *et al.*, 2013; Perez-Santángelo *et al.*, 2014). LSM proteins also are involved in plant responses to abiotic stresses (Xiong *et al.*, 2001; Zhang *et al.*, 2011). Together, these observations suggest that the LSM splicing factors mediate alternative splicing of the clock genes under heat stress conditions.

ZTL-mediated protein quality control under heat stress A large set of HSP family members acts as molecular chaperones during heat stress adaptation in eukaryotes. HSP family members are widely conserved in both plants and animals. Under unfavorable high-temperature conditions, they stabilize client proteins by facilitating the refolding process of denatured proteins (Finka & Goloubinoff, 2013). They also collaborate with E3 ubiquitin ligases to clear irreversibly denatured proteins via the ubiquitin–proteasome pathways (Lee *et al.*, 2009; Samant *et al.*, 2014). Consistent with the roles in heat stress adaptation, HSP transcription and protein concentrations are elevated under high-temperature conditions.

Among the HSP family members, HSP90 has been the most extensively studied in diverse thermal adaptive responses, including the thermal control of the clock function. In animals, HSP90 plays a role in sustaining the clock function (Schneider *et al.*, 2014). In plants, HSP90 is essential for the maturation and stabilization of ZTL (Kim *et al.*, 2011), an E3 ubiquitin ligase that constitutes a fundamental component of the central oscillator. A recent study reported that ZTL and its interacting molecular chaperone, HSP90, establish a heat stress-inducible protein quality control system, which contributes to maintaining thermostable growth and circadian behaviors at high temperatures (Gil *et al.*, 2017). In response to heat stress, ZTL and HSP90 are colocalized to insoluble protein aggregates and direct their degradation via the ubiquitin–proteasome pathways (Fig. 5b).

It is interesting that ZTL itself is a client of the HSP90 molecular chaperone under normal temperature conditions (Kim *et al.*, 2011). ZTL in turn targets the clock components TOC1 and PRR5 in the dark, establishing the diel rhythmicity of the clock (Más *et al.*, 2003; Kiba *et al.*, 2007). In this view, it is unexpected that whereas ZTL does not affect the stability and accumulation of TOC1 and PRR5 under heat stress, heat-induced global protein aggregates are cleared by ZTL. The most probable explanation would be that under heat stress, HSP90 and other HSP members, including HSP70, recognize insoluble protein aggregates, to which ZTL is recruited through the interactions with the HSP family members. In support of this notion, the E3 ubiquitin ligase-HSP modules are highly conserved in eukaryotes (McClellan *et al.*, 2005; Lee *et al.*, 2009; Samant *et al.*, 2014). For example, HSP family members and their interacting E3 ubiquitin ligase C-terminus of HSP70 Interacting Protein (CHIP) are known to mediate thermotolerance in yeast and plants (Dickey *et al.*, 2007; Lee *et al.*, 2009), further supporting the physiological relevance of the ZTL-HSP module in thermostabilizing plant growth and the clock function.

V. Thermal adaptation of the clock: evolutionary considerations

The advent of the ZTL-mediated protein quality control machinery may have spurred the evolution of land plants from aquatic life. It is believed that the earliest land plants encountered a completely new environment as they evolved from green algae *c.* 470 Myr ago (Kenrick & Crane, 1997). The transition from an aquatic to a terrestrial environment likely brought both advantages and challenges for the earliest land plants. On the one hand, there are higher amounts of sunlight, carbon dioxide and mineral nutrients on land. By taking advantage of the metabolic richness, land plants would have grown faster and bigger. On the other hand, living on land would have posed serious challenges, such as uncontrollable water loss from the leaves and stems, the need for mechanical support, and severe environmental fluctuations. To deal with these challenges, land plants developed versatile architectural and morphological devices, such as a vascular system, a cuticle layer on the leaf surface and biochemical gadgets, including more efficient light-harvesting machinery (Graham *et al.*, 2000; Ligrone *et al.*, 2012).

One of the greatest challenges facing the earliest land plants is likely to have been the harsh fluctuations in environmental temperatures. Soil temperatures would have been even higher than air temperatures due to the absorption of sunlight by the soil surface. Therefore, land plants, including higher plants, ferns and spike mosses, would have needed to cope with these stressful temperatures. In addition, daily and seasonal temperature extremes would have required plants to develop robust time-keeping systems to precisely anticipate upcoming temperature extremes. It is tempting to speculate that, during the colonization of terrestrial environments, the ZTL/HSP90 module may have evolved in the earliest land plants to ensure thermostable growth as well as a thermostable clock system. Accordingly, ZTL, and perhaps HSP90, may have originated as early as land plant speciation.

A database search revealed that ZTL orthologs are readily identified in the genomes of both ancient nonvascular and vascular land plants (Fig. 6). *Marchantia polymorpha* (Bryophyta), one of the most ancient land plants, and *Selaginella moellendorffii* (Lycophyta) also harbor ZTL proteins. In addition, it has been reported that thermostable growth and thermal adaptation of photosynthesis in *M. polymorpha* resemble those of higher plants (Kubota *et al.*, 2014), further supporting the ancient origin of the ZTL/HSP90 module.

The genome of *Physcomitrella patens* (Bryophyta), considered one of the earliest plants to colonize the land, harbors several clock genes, including *PpCCA1*, *ELF3*, *ELF4*, *LUX-like* and *PpPRR* genes (Holm *et al.*, 2010). However, orthologs of ZTL, TOC1 and GI are not identified in the *P. patens* genome. Because these clock components also are absent in green algae (Matsuo *et al.*, 2008), their appearance is likely to have been closely associated with the

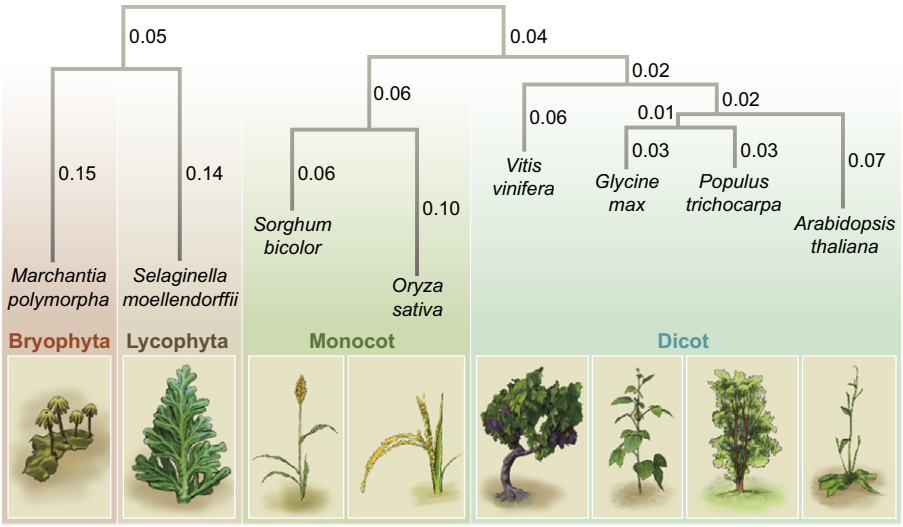

Fig. 6 Evolutionary conservation of ZEITLUPE (ZTL) proteins in land plants. ZTL homologs were identified from the genomes of representative land plant species using bioinformatics tools available in the Phytozome database (https://phytozome.jgi.doe.gov/pz/portal.html). Amino acid sequences of the ZTL proteins were inferred from the genome sequences of *Marchantia polymorpha*, *Selaginella moellendorffii*, *Sorghum bicolor*, *Oryza sativa*, *Vitis vinifera*, *Glycine max*, *Populus trichocarpa* and *Arabidopsis thaliana*. A phylogenetic tree was generated using the neighbor-joining method of the MEGA7 software (https://www.megasoftware.net/).

evolution of land plants. *Physcomitrella patens* is typically found in habitats near water edges, likely due to the lack of vascular tissues and thus would not have encountered harsh temperature fluctuations, partly explaining the absence of the thermal adaptation system consisting of ZTL and GI.

The E3 ubiquitin ligase ZTL and HSP molecular chaperones, such as HSP90 and HSP70, provide a thermo-adaptive clearing mechanism, which removes denatured cellular proteins via the ubiquitin–proteasome pathways. Through ZTL/HSP-mediated protein quality control, plants are able to thermostabilize their growth and circadian behaviors. We believe that the ZTL-mediated thermostabilization system provides a typical evolutionary developmental approach to understand how plants evolved specific developmental traits to adapt to changing environments.

VI. Light and temperature information for the clock function – synergic or individual?

Light and temperature are two major environmental cues that fluctuate throughout the day and seasons. Accordingly, light and temperature signals are interconnected during adaptation responses in eukaryotes. It is widely documented that light and temperature information also is integrated into the clock system to generate fine-tuned transcriptional profiles and biological rhythms (Michael *et al.*, 2003a, 2008). For example, it is known that light and temperature cycles exert cooperative effects on the clock entrainment in *Drosophila* and *Arabidopsis* (Yoshii *et al.*, 2009; Arana *et al.*, 2017). In *Arabidopsis*, various clock elements mediate the integration of temperature and light cycles into the clock for optimal growth and seed germination (Arana *et al.*, 2017). However, temperature and light cues also exert their roles through independent signaling pathways in some cases (Franklin *et al.*, 2014), obscuring the functional relationship between the two signaling cues, especially in modulating the clock function.

Thermomorphogenesis, represented by morphogenic and developmental adaptation to changes in ambient temperatures (Quint *et al.*, 2016), is emerging as an important topic in the field, primarily because of its physiological link with global warming and crosstalk with clock and light signaling. The first signaling mediator that integrates light and temperature signals during thermomorphogenesis is phyB (Legris *et al.*, 2016; Song *et al.*, 2017). The rhythmic expression of the *PIF4* gene, a downstream target of phyB, is tightly regulated by the clock via the EC (Nusinow *et al.*, 2011). In addition, transcription of core clock genes is affected by temperatures during extended dark periods (Tóth *et al.*, 2001). These observations support extensive signaling integrations of light and temperature in the clock functioning.

Plant responses to stressful temperatures also are extensively associated with light signaling. Under natural conditions, light energy is transmitted to the soil surface during the day, where plants encounter high temperatures. It has been demonstrated recently that light-derived signals gate plant responses to heat stress, assigning the diel patterns of basal thermotolerance (Dickinson *et al.*, 2018). Heat and light signals also impose synergic effects on hypocotyl elongation, and thus *Arabidopsis* mutants defective in the clock genes exhibit disrupted hypocotyl elongation under heat stress (Karayekov *et al.*, 2013).

Although most experimental evidence supports the synergic effects of light and temperature on the clock function, the molecular mechanisms mediating signaling interactions remain elusive. Although phyB is widely believed to be one of the key integrators of light and temperature signals (Legris *et al.*, 2016), the molecules that integrate clock information and temperature and/or light signals have not been explored. The activities of many clock components are regulated in a light-dependent manner, with ZTL being a pivotal example. Interestingly, following light-induced conformational changes, ZTL acts as a blue light receptor (Somers *et al.*, 2000). Strikingly, ZTL interacts with phyB in yeast cells

(Jarillo *et al.*, 2001). It is worth investigating whether ZTL–phyB interactions occur *in planta* and play a role in integrating light and temperature signals to the clock.

VII. Concluding remarks and future prospects

Changes in the average global temperature and local extreme weather events could markedly affect global crop production (Ali *et al.*, 2017). It is notable that plant temperature responses are often modulated by the clock information (Franklin & Whitelam, 2007; Box *et al.*, 2015; Zhu *et al.*, 2016; Dickinson *et al.*, 2018). Versatile molecular mechanisms mediate the temperature control of the clock function. Temperature influences the alternative splicing of many clock genes (Filichkin & Mockler, 2012; James *et al.*, 2012; Seo *et al.*, 2012; Kwon *et al.*, 2014). The best characterized is the cold-repressible alternative splicing of *CCA1* gene, which links the clock with cold responses (Seo *et al.*, 2012). Meanwhile, the E3 ubiquitin ligase ZTL and associated molecular chaperones thermostabilize the clock system through a protein quality control mechanism (Gil *et al.*, 2017). The diverse effects of temperature support the thermal plasticity of the clock and provide some indication of future crop biotechnology in view of climate change.

The agronomic importance of the circadian clock is becoming evident in diverse agricultural traits. A pivotal example is the photoperiodic control of flowering induction (Mockler *et al.*, 2003). For example, a barley variety harboring a mutation in the homolog of the *ELF3* gene exhibits accelerated flowering, a trait that could enable the variety to be cultivated in short growing seasons. Transgenic approaches that manipulate clock genes also have been proven to be successful in many plant species (Preuss *et al.*, 2012). The circadian clock regulates >30% of the transcriptomes in *Arabidopsis* (Harmer *et al.*, 2000; Michael *et al.*, 2003b), rice (Filichkin *et al.*, 2011), maize (Hayes *et al.*, 2010) and soybean (Marcolino-Gomes *et al.*, 2014), supporting the functional significance of the clock system for plant growth and performance (Green *et al.*, 2002; Michael *et al.*, 2003b; Dodd *et al.*, 2005a; Izawa, 2012). Further elucidation of signaling networks mediating the clock functioning will significantly contribute to our understanding of how plants flourish in a predictably changing environment, and these insights will have important implications for the improvement of crop productivity. It also is envisaged that we would be able to improve crop production not only by manipulating the clock genes, but also by modifying the shoot : root ratio, a distinct characteristic of individual crop species, and sugar metabolism, which is greatly affected by light and temperature conditions.

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