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Trends in Plant Science

Opinion

Wandering between hot and cold: temperature dose-dependent responses

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Plants in most natural habitats are exposed to a continuously changing environment, including fluctuating temperatures. Temperature variations can trigger acclimation or tolerance responses, depending on the severity of the signal. To guarantee food security under a changing climate, we need to fully understand how temperature response and tolerance are triggered and regulated. Here, we put forward the concept that responsiveness to temperature should be viewed in the context of dose-dependency. We discuss physiological, developmental, and molecular examples, predominantly from the model plant *Arabidopsis thaliana*, illustrating monophasic signaling responses across the physiological temperature gradient.

Highlights

Temperature effects on plant growth, physiology, and development are conceptualized from a dose-response perspective.

Since plants are exposed to natural fluctuations in temperature, we draw attention to the temperature optimum of responses instead of the (study of) binary extreme temperatures.

Plant growth responsiveness to temperature dose often aligns with the monophasic response pattern, fitting an Arrhenius type model.

Temperature dose-modulated changes of two phytohormone levels, jasmonate (JA) and gibberellin (GA), follow the monophasic response pattern.

The thermosensory responses of phyB, ELF3, and the secondary structure of *PIF7* mRNA show, to the extent tested, a temperature threshold dose-dependent pattern and could fit a monophasic pattern.

Plant life under fluctuating temperature conditions

Plants experience continuously fluctuating temperatures in their natural habitat with small and large variations that, from a physical perspective, fall within a temperature gradient from freezing to severe heat [1,2]. Temperature variations can trigger specific acclimation or tolerance responses. In the context of this paper, we consider acclimation responses as those that are induced to adjust to suboptimal yet sublethal environmental conditions in order to sustain growth and performance. On the other hand, tolerance responses are induced to ensure survival when the physiological range is exceeded and usually comprises of cellular stress responses. Tolerance responses are typically exemplified by stalling of growth and development and involve the production of chaperones to protect cells and membranes [3]. In view of climate change, we urgently need to comprehensively understand the genetic, molecular, biochemical, and physiological basis of temperature responses. Here, we put forward the concept that responsiveness to temperature should be viewed from a context of dose-dependency instead of a binary process, as is common in research practice. The resulting molecular understanding of temperature dose-dependent signaling and response networks will be crucial to develop thermo-resilient crop varieties that can cope with diverse temperature scenarios associated with global climate change.

Dose-dependent responses drive growth and development

Plants respond to various internal and external cues in a dose-dependent manner. Internal signals, such as plant hormones {e.g., auxin [4,5] and brassinosteroids (BR) [6]}, transcriptional coactivators (e.g., ANGUSTIFOLIA3 [7]), and small RNAs [8,9], have been shown to generate a specific output depending on the concentration. External cues, such as the essential environmental factor temperature, exhibit small and large variations that can range from minutes, to a day/night (diurnal) cycle, to seasons [10–12]. On one side of the natural temperature spectrum within the biosphere is what is called the low temperature range (freezing to chilling cold) and on the other side of the spectrum is high temperature (ambient high and heat). In the suboptimal ambient high range, the interaction between genotype and environment determines phenotypic plasticity to coordinate an as optimal performance as possible. However, the environmental component is generally dominant over the genotype in the more stressful events of freezing

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and heat, where plants aim to tolerate or survive [13–15]. For example, the hypocotyl growth rate of the arabidopsis (*Arabidopsis thaliana*) *phyb* mutant is higher than the rate of wild type at the high ambient temperature of 30°C. At stressful 40°C, however, the growth stops in both the *phyb* mutant and wild type arabidopsis seedlings [15]. However, there are better adapted species (e.g., Arctic or desert plants) [16,17] that survive at extreme environmental conditions where other species cannot tolerate the extreme temperatures. In this case, the genotype is dominant over the environmental condition.

In between cold and warmth is a temperature window that is considered optimal for a given process. In contrast to a 'response maximum' (Figure 1A), 'optimal' should be defined as a temperature range where all the growth, developmental, physiological, and biochemical parameters are balanced in the context of their environment. The optimal temperature may vary between species, varieties, and phenotype considered and is often dependent on the natural habitat the species evolved in, making the genotype the dominant factor determining the

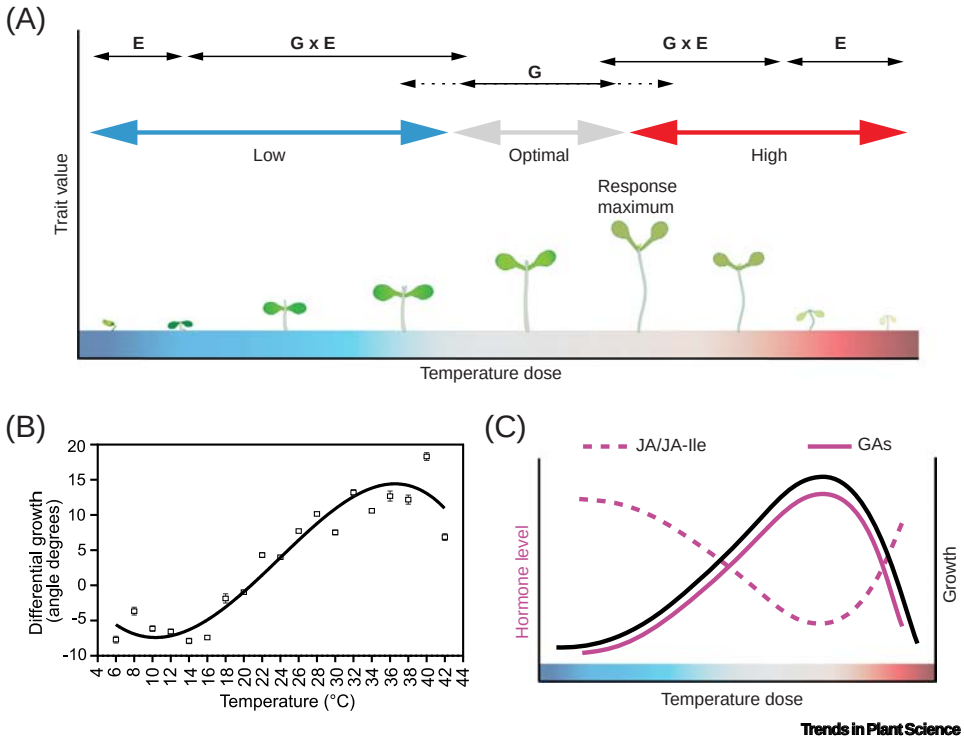

Figure 1. Temperature dose-dependent monophasic responses. (A) Schematic representation of plant growth responses across a physiological temperature range (*x*-axis) from cold (blue range) to heat (red range). Note the changes in hypocotyl/petiole elongation and leaf hyponasty. The temperature dose depicted here is considered the physiological range where acclimation is possible proportional to the signal strength, with tolerance responses occurring at the extreme ends (freezing and heat). The blue, gray, and red double-headed arrows indicate the low, optimal, and high temperature range, respectively. The horizontal span of each black double-headed arrow represents the relative size of the temperature response window and the dominant contributing factor: genotype (G), environment (E), or their interaction (G × E). Note that the warm temperature acclimation window is proportionally smaller, but with larger phenotypic differences than the cold acclimation range. (B) Temperature dose-dependent thermonasty of arabidopsis (Columbia-0) leaf petioles (angle degrees) of plants pregrown at 20°C and then shifted to the indicated temperature for 6 hours. The unbroken line is plotted by normalizing the angles to the corresponding value at 20°C, with a fourth polynomial trendline. Data obtained from [20]. (C) Conceptualization of temperature dose-mediated monophasic responses of jasmonate (JA)/ jasmonoyl-isoleucine (JA-Ile) levels (pink broken line), which is inversely correlated with plant elongation growth (black unbroken line), and bioactive gibberellins (GAs, pink unbroken line), which positively correlates to dose-regulated plant elongation growth.

Glossary

Evening complex (EC): a transcriptional repressor complex and a core component of the plant circadian clock.
Liquid droplets: a biological phenomenon that refers to the components of similar properties that form droplet condensates (also called speckles) in cells (also referred to as liquid–liquid phase separation).
Morphogen: a substance whose nonuniform distribution governs the pattern of tissue development in the process of morphogenesis or pattern formation.
Nuclear bodies: membraneless structures found in the cell nuclei of eukaryotic cells.
Q10: temperature coefficient that is a measure for changes in the rate of biological processes as a function of a 10°C increase in temperature.
Thermomorphogenesis: morphological and architectural changes, including hypocotyl/petiole elongation, leaf hyponasty, and accelerated flowering in higher plants, which is induced by high ambient (nonstressful) temperatures.
Thermonasty: a nondirectional organ movement response in plants to temperature, often leaf movement (hyponasty or epinasty).

optimum [10,13,18–20] (Figure 1A). So far, research efforts have largely focused on acclimation and/or tolerance responses (e.g., functionality, molecular regulation, and agronomic output parameters), but very little is known on what factors *de facto* determine the optimal temperature for a given species and process. Moreover, in experimental approaches in laboratory settings, temperature is often approached as a binary signal (i.e., the comparison of a set test temperature compared with an arbitrary control temperature) [19], precluding the detection of the temperature optimum per definition.

We here advocate for conceptualizing temperature as a dose-response signal as the way forward in the foreseeable future of plant temperature research, in contrast to studying temperature as a binary signal. We need to incorporate temperature gradients in experimental designs to determine temperature optima, identify (natural) variation therein, elucidate the underlying genetics and biochemistry, and translate this to crops in the development of climate-ready varieties that exhibit variation in temperature responsiveness.

Temperature dose-modulated monophasic responses

The temperature dose-dependent impact on plant growth, physiology, and development often follows a monophasic response pattern (Box 1). For example, plant organ elongation is directly or indirectly affected in a temperature dose-dependent manner. Cell elongation is, for instance, gradually promoted with increasing temperature until a maximum level is reached, after which elongation growth is rapidly inhibited [18,20–23] (Figure 1A,B).

Box 1. The monophasic response

A monophasic model is a dose-response pattern with an initial gradual increase (or decrease) followed by a subsequent decrease (or increase), with only a single phase in the total response window across the input signal gradient. This is in the context of temperature reflected in two main types of curves, namely a bell-shaped curve [a symmetrical graph depicting the normal (response) distribution that is concentrated around a central peak (the median) and with decreasing values on either side of the median proportional to the independent factor] and Johnson and Lewis master reaction model [a model (graph) with a long tail towards the response optimum and a sharp decline when the optimum is exceeded] (Figure I). The term 'monophasic' is often used in pharmacology [77] and associates to a 'blue–gray–red' temperature dose model (see Figure 1A in main text) similar to the 'French flag model' that was put forward by Lewis Wolpert when proposing **morphogen**-related patterning events in animals [78,79].

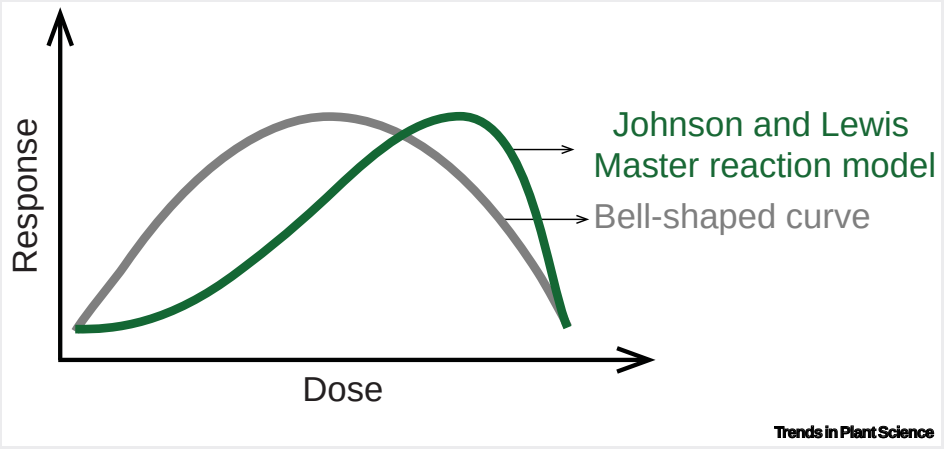

Figure I. Concept of monophasic response visualized as bell-shaped and Johnson and Lewis master reaction model.

A typical phenotypic output of temperature-controlled cell elongation at the leaf petiole abaxial side relative to the adaxial side is leaf **thermonasty** (see Glossary) (Figure 1B) [20]. This directional response coincides with nondirectional elongation growth of the petioles. Together with other traits, such as elongation of the hypocotyl or stems, this is called **thermomorphogenesis**. Thermomorphogenesis is considered an adaptive acclimation strategy that allows avoidance of heat interception by direct sun light to avoid photo-inhibition, promotion of leaf cooling capacity by triggering a more open plant architecture, and moving thermolabile meristematic tissues away from heated soil [24]. Mathematical modeling suggested that both leaf thermonasty and petiole elongation combined are required for effective leaf cooling in arabidopsis [25]. On the contrary, thermomorphogenesis may contribute to avoiding shade conditions in an effort to outcompete neighboring vegetation if (light) resources are limited [24]. Altogether, thermomorphogenesis traits thus allow for optimal performance in suboptimal environmental conditions [24,25]. It should be noted that unlike thermonasty, not all phenotypic output is reversible. For instance, once hypocotyls and the petiole have elongated in reaction to ambient warmth, a subsequent exposure to higher or lower temperatures cannot fully reverse to a short stature, despite that the (elongated) phenotype may be suboptimal in the new situation.

By contrast, to survive very low/high temperature stresses beyond the physiological range, plants conserve energy by suppressing or inhibiting cell elongation to tolerate the stressful environment [26,27]. Indeed, temperature-mediated differential petiole growth is repressed when plants experience heat stress or cold [20] (Figure 1B). Plant elongation responses thus typically follow a monophasic response pattern (well-aligned with the temperature dose; Figure 1A,B).

Typically, temperature dose-response curves fit the Johnson-Lewin master reaction model [28] (Box 1), also referred to as 'Arrhenius-type' response curve [18]), which is characterized by a gradual increase at the suboptimal temperature range, followed by a relatively sharp decrease in response strength shortly after exceeding the optimal temperature. This model relates to the perhaps more familiar bell-shaped curve [13,18] (Figure 1A,B) in which the tails on each side of the optimum are mirrored.

Temperature dose-modulated plant growth responses to two phytohormones, jasmonates (JAs) [22,29–33] and gibberellins (GAs) [34–37], fit the concept of a monophasic response in plant elongation (Figure 1C) and are here highlighted as case examples. The bioactive JA (jasmonoyl-isoleucine, JA-Ile) and its CORONATINE INSENSITIVE 1 (COI1)/JASMONATE-ZIM DOMAIN (JAZ) coreceptor are central to JA responsiveness [38]. The JA signaling pathway plays a role in plant growth inhibition [22] and at the low end of temperature spectrum the endogenous JA/JA-Ile levels are high compared with optimal temperatures (Figure 1C) [30,31,39]. For example, low temperature (8°C) significantly induces JA-Ile accumulation in rice seedlings. This triggers acclimation and tolerance to low temperature by growth inhibition [30,31]. Similarly, arabidopsis plants with hampered growth contain a much higher JA level during chilling (4°C) [39,40]. At moderately high temperature, activation of JA catabolism leads to decreased endogenous JA/JA-Ile levels, which correlates with promotion of elongation growth in different species, including arabidopsis [22,41] and wheat [22]. In tomato, warm temperature leads to downregulation of the expression of JA-biosynthetic enzymes, such as *FATTY ACID DESATURASE 2/3* (*FAD2/3*), resulting in reduced endogenous JA/JA-Ile levels in tomato stamens. This allows for longer stamens due to the increased stamen cell size (width and length) in a temperature treatment duration-dependent manner [42]. When experiencing heat stress, endogenous JA/JA-Ile or JA precursors levels increase, resulting in inhibition of plant growth and energy conservation needed to survive the supra-optimal temperature (Figure 1C) [32,33,43]. For example, *Marchantia polymorpha* plants grown at

30°C are small and contain higher levels of JA precursors compared with those grown at lower optimal temperature [32].

In contrast to the growth inhibitory effect of temperature concentration-modulated JA/JA-Ile levels, the change in endogenous GA levels follows the growth promotion curve (Figure 1C). When plants are subjected to cold or heat (both ends of the temperature spectrum), the endogenous bioactive GA levels are much lower than at optimal temperatures (Figure 1C) [34,37,44–47]. This leads to a shorter stem/hypocotyl of pea/soybean seedlings or reduced plant height of ryegrass at moderately low temperature or heat [34,44–46] and plant growth cessation at chilling temperatures [37,48,49]. Endogenous bioactive GA levels accumulate at moderately high temperature, which promotes growth [35,44,50]. Overall, GA levels are thus positively and closely correlated with the monophasic elongation growth response within the temperature gradient (Figure 1C).

Different to the apparent monophasic patterns of jasmonates and gibberellins, the levels of auxin and BR do not strictly follow the monophasic pattern across the temperature range [51–53]. Moderately low temperature causes a reduction of endogenous auxin levels and plant growth limitation [47,54], for example, a shorter hypocotyl or primary root in soybean [44] and arabidopsis [55], respectively. Conversely, a relatively small increase in endogenous auxin [42,44,56] at warm temperature promotes plant elongation growth. Heat, however, induces a much higher level of auxin [34,46] and plant growth is inhibited [34,51]. Partially different to auxin, castasterone, one of the endogenously bioactive BRs, is highly accumulated at chilling stress, where plant growth is suppressed [49]. Warm temperature induces a slight increase in BR level [49], resulting in promotion of elongation growth. However, a much higher level of BR accumulates under heat [46], which results in inhibition of plant growth [34,51]. It is interesting to note that while auxin and BR responses do not follow a monophasic pattern, these phytohormones underpin monophasic, irreversible responses, such as temperature-driven hypocotyl growth.

Molecular factors involved in perception of (subtle) changes in temperature

Plants sense temperature via biomolecules and interpret and actively convey the information to downstream response mechanisms. Different temperature sensors have now been described in plants [e.g., phytochrome B (phyB) [23,57], EARLY FLOWERING 3 (ELF3) [58], phototropin (PHOT) [59], and RNA thermo-switches [60]]. Of these, at least the **evening complex (EC)** component ELF3, phyB, and PHYTOCHROME INTERACTING FACTOR 7 (PIF7) mRNA secondary structure have the intrinsic potential to respond proportionally to the signal strength (i.e., temperature input dose) [23,58,60] (Figure 2).

As a first example, phyB occurs in two photo- and temperature interconvertible conformations: the biologically inactive Pr cytosolic state and the nuclear-localized active Pfr state [61,62]. PhyB Pfr spontaneously reverts back to Pr in a light-independent reaction [15,23]. The stability of phyB Pfr-Pfr dimer is positively related with the number of phyB **nuclear bodies** [23,57] and temperature progressively reduces the number of phyB nuclear bodies (Figure 2A) in a not well-understood manner [15,57], which correlates with hypocotyl elongation (Figure 2B). At cold temperatures, the thermal inactivation is slow, hence phyB retains active for a relatively long time, thereby suppressing elongation growth. When temperature increases, thermal inactivation speeds up, along with phyB, alleviating the repression of elongation. Hence, *phyb* mutants are elongated at control temperature conditions [15,63]. Altogether, while the molecular changes were not tested at temperatures higher than 30°C, plant growth shows a monophasic response. At stressful 40°C, however, the growth stops in both the *phyb* mutant and wild type arabidopsis

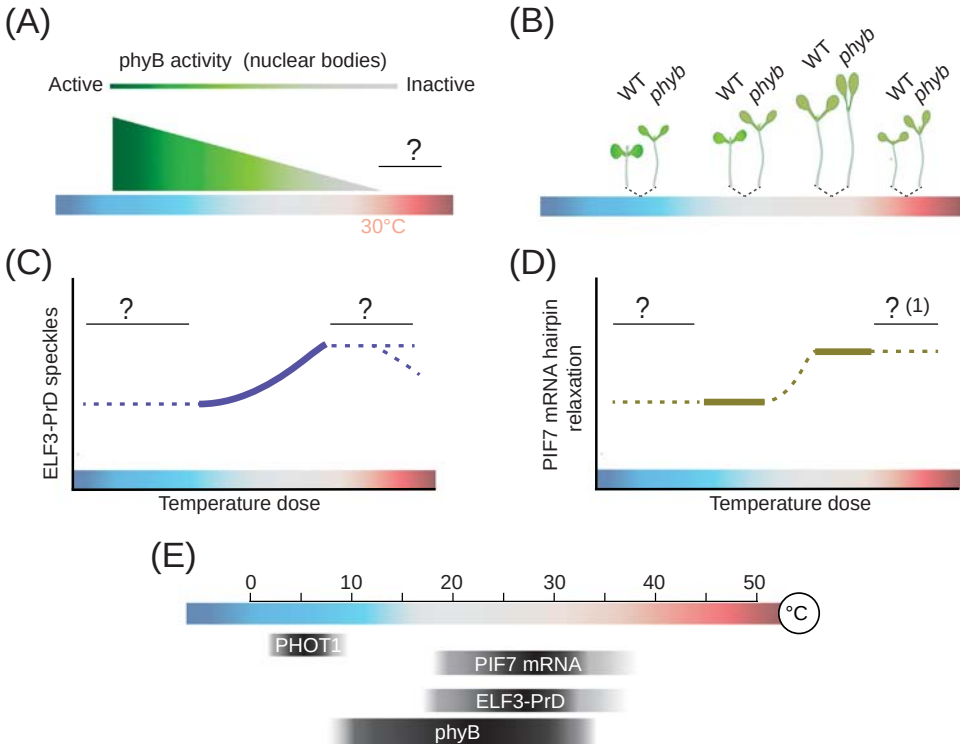

Figure 2. Interpretation of temperature dose at the thermosensory level. (A,B) Phytochrome B (phyB) activity decreases as temperature increases. The formation of nuclear bodies correlates with the biologically active Pfr state of phyB [15] (A) and growth of wild type and *phyb* mutant plants at different temperatures is differentially affected along the temperature gradient (B). (C) ELF3-PrD liquid droplets increase with temperature, represented by the unbroken purple line, based on [58]. (D) The conformational changes of the secondary structure of 5′-untranslated region (UTR) of the *PIF7* mRNA with temperature. The unbroken line presents the relaxed extent of the structure with the temperature range, based on data from [60]. In (C) and (D), broken lines represent different hypothetical extrapolations of activity across the temperature gradient. The question marks in (A), (C), and (D) indicate uncertainty of the response in this range. In addition, (1) indicates likely complete disruption of the secondary mRNA structure in (D). (E) Distribution of temperature sensor activity [EARLY FLOWERING 3 (ELF3), phyB, PHYTOCHROME INTERACTING FACTOR 7 (PIF7) mRNA, and PHOT1] across the tested temperature dose. Those thermosensors might also respond to temperature outside the indicated range, but data are lacking at the moment.

seedlings [15]. This might suggest that beyond the ‘response maximum’, plants stop growing upon further increasing temperatures, possibly independent of phyB activity.

ELF3 forms **liquid droplets** proportionally to the temperature dose in a prion domain (PrD)-dependent manner [58]. High ambient temperature-induced localization of ELF3-PrD in liquid droplets attenuates the ELF3 activity, and these liquid droplets disaggregate under low ambient temperatures [58,64]. However, a complete quantification of ELF3-PrD liquid droplets in cells along a broad temperature gradient is lacking, making it difficult to fully assess dose-dependency of the resulting responses [58,64] (Figure 2C). Furthermore, another study observed that, in contrast to the earlier work, increasing temperature reduces the localization of ELF3 to foci, leading to a reduction in ELF3 activity [65]. It remains to be investigated what causes these apparent discrepancies. It nevertheless seems plausible that ELF3 can interpret the temperature according to its cellular localization (and thus activity).

The secondary structure within the 5′-untranslated region (UTR) of *PIF7* mRNA can change in response to a temperature cue [60]. The conformational changes result in roughly two secondary structure states (unrelaxed and relaxed) at low temperatures and warm temperatures, respectively [60] (Figure 2D). The unrelaxed structure of *PIF7* mRNA is relatively stable at 17°C and 22°C, but a more relaxed state is observed at 27°C and 32°C. This relaxed structure enhances *PIF7* translation and this is necessary for growth to occur with increasing temperature [60]. The changes in *PIF7* mRNA secondary structure occur partially in a temperature threshold dose-dependent pattern; however, this was not yet tested along a complete temperature gradient (Figure 2D).

PHOT1 contains light/oxygen/voltage (LOV) domains at the N-terminal region [59,62], which have active and inactive states to mediate temperature perception [59]. For example, the lifetime of active LOV (specially LOV2) at 5°C is fourfold higher than that at 22°C and this is essential for PHOT-mediated temperature perception in *Marchantia* [59]. However, how PHOT1 precisely responds to different temperatures remains to be explored, but the lifetime of its photoactivated state may play an important role in dose-dependent responses.

Interestingly, despite a large extent of overlap in the temperature window, ELF3 [58,65], *PIF7* mRNA [60], and phyB [15,23,63] sense and interpret the temperature dose in partly different temperature ranges (17–35°C, 17–32°C, and 10–30°C, respectively) (Figure 2E). Plants thus may rely on different temperature sensors when exposed to a certain temperature dose. As each sensor has its own specific signaling output, diversification of sensing mechanisms across the temperature gradient may have evolved from a benefit of appropriate responsiveness in (slightly) different temperature windows (Figure 2). Knowledge on how specific windows for temperature sensors (and downstream components alike) originated in plants and how this relates to the functional outputs (i.e., orchestrating acclimation and tolerance mechanisms) is needed. In this regard, using the concept of a temperature coefficient (**Q10**), which is a measure for changes in the rate of a biological processes as a function of a 10°C increase in temperature, could be helpful. While the Q10 concept has been explored for several plant physiological traits (i.e., photosynthesis and respiration) [66,67], but also transcription rates [62], this concept can also be applied to temperature sensors in a temperature dose-dependent way. A typical biochemical reaction has a Q10 of about 2–3. Human thermosensors, transient receptor potential (TRP) channels, have been identified based on their unusual high Q10 values and can have a Q10 of >100 [68]. The genome of chlorophyte algae seems to contain several types of putative *TRP*-like genes [69]. However, no *bona fide TRP*-encoding genes have been identified in land plants so far [69]. TRP channels might have been lost after their divergence from the chlorophyte algae, but other channels might have similar biochemical functions in land plants.

Interestingly, diversification of TRP channels occurred in animals, with each TRP having partly different and overlapping response windows in respect to others [70,71]. However, the responses of plant thermosensors have not yet been accurately determined along a complete temperature gradient (Figure 2). For example, measuring the rate of mRNA hairpin relaxing or refolding with a 10°C increase or decrease, respectively, could be assessed for *PIF7* mRNA.

Concluding remarks and future perspectives

Generally, plant growth is inhibited at temperature ‘extremes’ (freezing and heat stress), which indicates that the environment is dominant over the genotype in these conditions and plants aim to tolerate/survive by stalling growth instead of inducing acclimation strategies (Figure 1A,B). Between extreme and optimal temperatures (i.e., the suboptimal range), interaction between the genotype (i.e., the genetic variation determining plasticity) and the temperature cue (environment)

Outstanding questions

How conserved are temperature dose-modulated monophasic responses in the green lineage?

To what extent does natural (genetic) variation occur in temperature-response optima within germplasms of natural plants and crops and what can we learn from this to improve crop thermotolerance?

How can temperature responses of developmental traits be altered in crops while precluding declines in overall performance due to developmental miscoordinations?

What are the differences and extent of overlap in response windows of diverse temperature-sensing molecules and how is this related to functional outputs in terms of acclimation and tolerance?

Are the putative TRP-like genes in chlorophyte algae functionally conserved thermosensors and what are their counterparts in land plants?

How can the concept of Q10 be utilized to detect novel thermosensing molecules?

drives the graded responses in growth, physiology, and development. Not surprisingly, in this 'mild' range, the genetic component is seemingly dominant over the environmental influence [13,15,18,19,22,72] (Figure 1A).

Plants are facing the challenge to synchronize responses of several processes (e.g., photosynthesis and photorespiration) within a natural, often supra-optimal and highly dynamic, temperature range. Seen from this viewpoint, we argue that research efforts should focus more on identifying genetic factors determining the temperature optimum, instead of on genetic factors driving acclimation/tolerance *per se*. This requires that including temperature ranges should become an intrinsic part of experimental designs. In this regard, temperature fluctuations need to be considered as well (e.g., semi-controlled field studies), as most plant species face temperature fluctuations in different timescales (minutes to days to seasons) throughout their lifetime. This has already been done in the context of, for example, the vernalization response [73,74]. So far, however, most studies, often for very understandable practical reasons, focused on fixed single and relatively extreme temperature conditions (see Outstanding questions).

Commercial crops have largely been bred against phenotypic plasticity for agronomic reasons, for example, to advance uniformness and easiness of harvest and optimize yield per hectare, by breeding compact varieties, and by suppressing traits like shade-avoidance capacity [18,75,76]. Past selection and breeding did not majorly affect the response of development to temperature, including the temperature optimum across crop varieties [18]. Parent and Tardieu argue that it is difficult to breed for changes in temperature optima as it would require a synchronic shift in responses of several processes simultaneously because it is likely that a change in the temperature responses of a single developmental trait leads to a miscoordination between developmental processes, resulting in suboptimal performance (see Outstanding questions). Overall, this may very well be a reason that breeding and selection likely occurred against genetic variation in temperature response traits, with the result that genetic variation for temperature responses of developmental traits has been largely eliminated from the germplasm pools of crops [18,76]. Classical genetics approaches starting from the domesticated variant directly therefore might not be always a fruitful way forward. We rather advocate focusing on existing natural genetic variation in wild/ancestral populations as a more desired approach to mine for alleles determining temperature responsiveness, including the temperature optimum.

We propose that determining Q10 values could be used to search in a systematic manner for novel thermosensory molecules across the temperature spectrum and to detect their temperature optima, by measuring (biochemical) reaction rates. Although Q10 values have been used to describe properties of thermosensitive development and growth traits [19], redefining the Q10 concept to such aggregate (organismal) levels would probably require a redefinition of the concept, as high Q10 values, for example, >100 as for TRP channels, are unlikely reached for such traits.

Overall, the information on the mechanisms governing plant temperature dose interpretation is still fragmented and further systematic studies are required to better understand the regulation of plant responses and signaling networks in a temperature-graded manner and to appreciate its genetic complexity.

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Declaration of interests

No interests are declared.

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