Current Biology 20, R1086–R1092, December 21, 2010 ©2010 Elsevier Ltd All rights reserved DOI 10.1016/j.cub.2010.10.035

Ambient Thermometers in Plants: From Physiological Outputs towards Mechanisms of Thermal Sensing

Minireview

C. Robertson McClung[1] and Seth J. Davis[2,*]

Plants respond to ambient temperature changes over a series of timescales. Genetic and physiological studies over the last decades have revealed myriad thermally sensitive pathways in plants. A recent study provides a genetic and biochemical mechanistic description of how thermal changes can be transduced to influence gene expression. What remains to be revealed in this, and other thermally controlled responses, is a description of the primary temperature-sensing event. Cooling and warming alter membrane fluidity and elicit intracellular free-calcium elevations, a process that has been considered the primary event controlling plant responses to temperature. Such direct thermal sensors appear to process temperature information. Future efforts will be required to identify the effector proteins linking perception to response. This review considers the evidence for plant thermometers to date, provides a description of several notable physiological and developmental processes under ambient temperature control, and outlines major questions that remain to be addressed in the understanding of thermometers in plants.

Introduction

Plants grow and develop across a range of ambient temperatures (Figure 1). Species have adapted to persevere over a ~50°C range, including extremes of plant growth near 0°C at the coastlines of Antarctica and near 50°C in the desert of the hottest place on Earth: El 'Azizia, Libya. Even for one plant, leaf temperatures can vary by more than 20°C within minutes, for example, as a result of heating from solar irradiation followed by cooling from a sudden breeze [1]. Interestingly, local changes in mean growth temperature can elicit both developmental and physiological changes that range from subtle metabolic readjustments to dramatic effects on growth and reproduction. For example, in a recent common-garden experiment of *Arabidopsis thaliana* accessions across a latitudinal cline of Western Europe [2], temperature gradients of >30°C could be tolerated, presumably because of plasticity in developmental responses. These developmental and physiological changes confer enhanced fitness in anticipation of similar ambient environments in the future. For example, the transition process from mild temperatures to sub-extreme temperatures, which in themselves do not induce thermal damage, provide signals to 'prime' the plant for further, future stress conditions of freezing or heat-shock. The predicted consequences of global climate change include increased fluctuation and greater extremes superimposed on a trend to warmer mean temperatures [3]. There are likely to be profound consequences from global climate change in both agricultural and non-agricultural plant communities. Some estimates have suggested that, by 2080, up to 33% of European plant communities may go extinct, or become vulnerable or committed to extinction, in response to the projected relatively modest increases in mean ambient temperature [4]. It is thus critical to understand the mechanism(s) of thermal sensing in plants.

Most mechanistic studies on temperature sensing in plants have focused on the pathways leading to temperature stress tolerance. In particular, coolness acclimates a plant to resist freezing, and warmth induces heat tolerance [5]. It is notable that temperature also affects plants within a non-stress range, and on a timescale from milliseconds to the lifespan. Dominated by experiments relating to the effects of small changes in temperature on photosynthetic potential, decades of research have clearly shown biophysical effects of temperature on light capture, and thus, energy production ([6] and references within). Furthermore, non-stressful temperatures strongly influence developmental decisions, including flowering time [7]. It is thus clear that ambient temperature is a critical issue of consideration in interpreting plant performance.

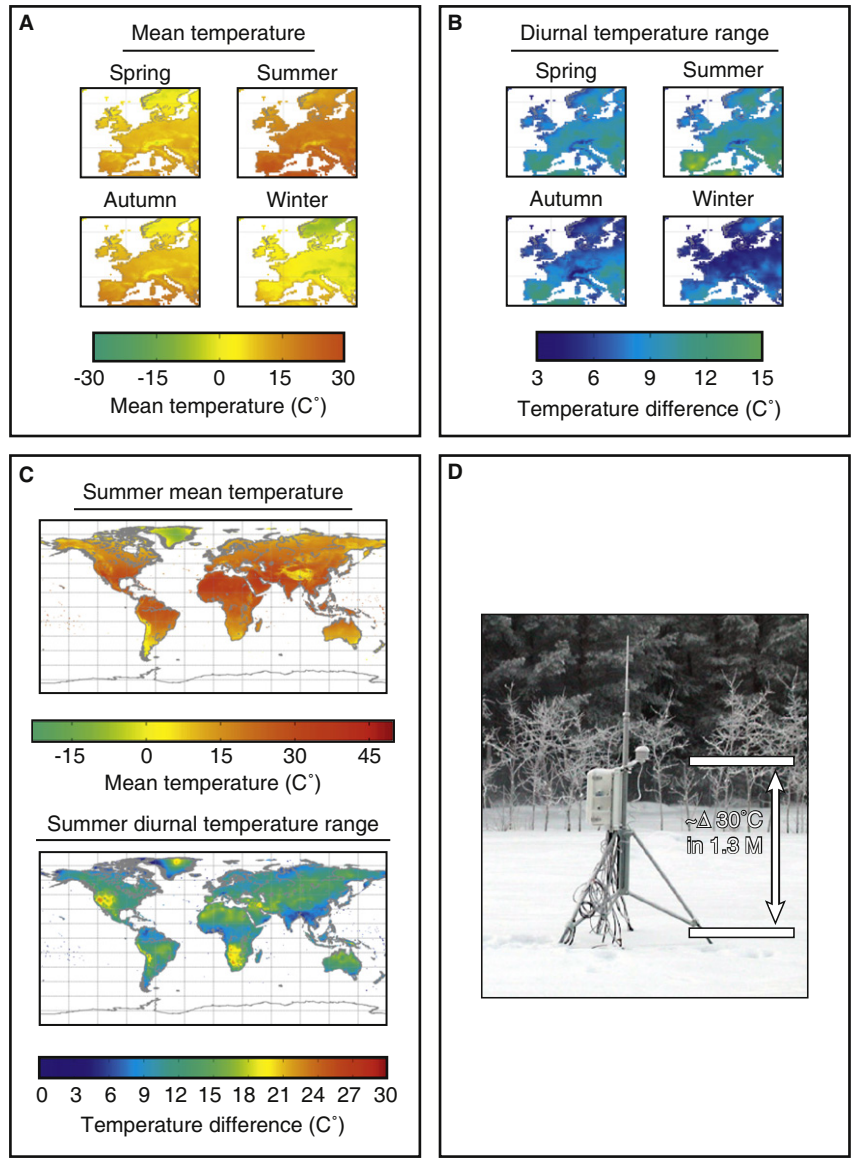
Over the last ten years, developmental studies, chiefly in *A. thaliana*, have demonstrated molecular–genetic paradigms for thermal responsiveness. These include the effects of temperature on hormone signaling, flowering time, the circadian clock, light-signal transduction, and cold and heat acclimation, which is the pre-priming of the acquisition of hardiness [5]. Taken together, it is clear that a wide range of processes in plants are controlled by thermal-perception systems, termed here 'thermometers'. In microbial and animal systems, various biophysical thermometers have been proposed, from the melting of RNA hairpins in bacteria [8], and RNA splicing and ribosome-loading in fungi [9], to ion-channel activation in insects [10,11]. However, in no instance has the nature of the primary perception of temperature in plants been fully described. Indeed, the identity of plant thermometers remains one of the great mysteries in the plant sciences. Here we discuss several physiological processes under temperature control, consider attributes for a plant thermometer (Box 1), and consider potential thermometer candidates.

The Role of Temperature in Control of Flowering Time

The developmental transition from vegetative to reproductive growth is affected by a number of endogenous and external cues, the most influential of the external cues being temperature and photoperiod [12]. A number of signaling pathways converge to control the expression of the floral integrator gene *FT* (*FLOWERING LOCUS T*) [13]. Here we focus on one aspect of flowering-time regulation involving temperature: warmth-induced flowering.

The phytochrome B (phyB) protein is a photoreceptor prominent in the detection of shade. Interestingly, the *phyB* mutant was found to be a temperature-dependent early-flowering mutant [14]. These physiological experiments revealed a connection between light perception and thermal sensing but did not reveal how this was possible. Plants

[1]Biological Sciences, Dartmouth College, Hanover, NH, USA. [2]Max Planck Institute for Plant Breeding Research, Cologne, Germany. *E-mail: davis@mpiz-koeln.mpg.de



Current Biology

Figure 1. Temperature varies in dynamic ways.
(A) Surface mean temperatures over the European landmass, as a function of season. (B) Diurnal temperature range (the difference between maxima and minima temperature) over the European land mass, as a function of season. (C) Global mean temperature and diurnal temperature range in the summer. For A–C, note that there is no strict correlation between mean temperature and diurnal variation in temperature. In general, whereas the equatorial lands are generally warmer, it is the drier, internal continental areas that show greater temperature differences over diurnal time. Furthermore, coastal areas are generally buffered from large diurnal temperature fluctuations regardless of the mean ambient temperature. A–C were assembled from data present at the Intergovernmental Panel on Climate Change (http://www.ipcc-data.org/) [65]. (D) Small changes in altitude can have profound effects on measured ambient temperature. In the inset is an image of a meteorological instrument that includes a thermometer at the typical 1.3 meters used worldwide in surface weather stations and a second ground-level thermometer. This device measured up to Δ30°C differences over this small change in altitude [2]. Image was captured by Tuomas Kauppila (University of Oulu, Finland).

express a small family of phytochromes (five in *A. thaliana*) and one possibility is that changes in ambient temperature modify functional relationships among family members [15]. The shade-avoidance response elicited through phytochromes in response to low red to far-red light ratios increases critical cold-responsive *CBF* gene expression (see below) to levels sufficient to elicit cold acclimation at higher temperatures than normally required [16]. In addition, there may be additional temperature-sensitive steps in or downstream of the phytochrome-signaling pathways [16].

One candidate in the signaling mechanism linking temperature sensing to the transition to flowering is a class of microRNAs (miRNAs) responsive to ambient temperature [17]. Changes in the ambient range lead to alterations in steady-state miRNA abundance. Furthermore, overexpression of miR172 led to increased expression of *FT* and early flowering, irrespective of temperature. This suggests that altered accumulation of a specific miRNA, as a reaction to changing ambient temperatures, is important in the generation of a thermal response [17]. It seems quite plausible that a second temperature-sensitive step could be the binding of the miRNA to targets, but this has not been investigated, underscoring the fact that much remains to be learned about the role of small RNAs in temperature responses and flowering time.

Auxin and Growth

Organ size in plants is exclusively controlled by cell division and cell expansion, and plant hormones play a major role in both processes. Auxin is a key hormonal factor potentiating both division and elongation [18]. Interestingly, the capacity of auxin to influence organ size is highly sensitive to subtle changes in ambient temperature. A small increase in temperature promotes growth, an effect that is diminished in auxin mutants with reduced sensitivity to, or levels/transport of, this phytohormone [19]. This warmth stimulation of elongation growth requires *TRANSPORT INHIBITOR RESPONSE2* (*TIR2*), which encodes an enzyme required for auxin production [20]. Loss of *tir2* blocks growth promotion at elevated temperature. *TIR2* mRNA accumulation increases at elevated temperature, supporting the earlier observation that auxin levels increase with temperature [19]. Consistent with this, expression of an auxin-induced marker increases at elevated temperature [20]. Thus, at least part of the stimulation of elongation growth at elevated temperature stems from increased auxin synthesis. This does not preclude temperature-dependent alterations in auxin transport or in auxin sensitivity, but as yet there is no mechanistic evidence that this occurs.

Box 1

Some necessary components for a thermometer.

Plant thermometers may require the following:

Primary considerations:

- Ability to detect absolute temperature or relative temperature changes
- To be biophysically plausible to mediate a temperature perception event
- To operate over the majority of the temperature range likely to be encountered
- To distinguish a *bona fide* temperature signal from transient noise, and thus, relay information that requires a response

Secondary considerations:

- Coupling differential thermometers could assist in broadening the range of minima and maxima that can be perceived
- The parameter that a given thermometer needs to convey will depend on the response to which it is coupled, *e.g.*, perception of average, relative, and maximal versus minimal temperatures are all likely to be required for different outputs. Longer-term responses appear less likely to require information about rapid, short-term temperature changes
- Memory of previous temperature information may need to be incorporated; this requires a mechanism for information storage and subsequent retrieval. This could occur via modulation of the thermometer output or memory through epigenetic mechanisms
- Different thermometers are conceptually plausible for ambient temperature versus stress-temperature perception. However, signal convergence is expected

A class of potent growth repressors in plants, termed DELLAs, directly represses transcription of genes that coordinate cell division and cell elongation [21]. The bHLH transcription factor PIF4 has been proposed to potentiate DELLA responses [22,23], including responses to warmer ambient temperature [24,25]. Interestingly, PIF4 can also function in warm responses independent of DELLA signaling [18], and PIF4 is also a component of a coolness response [26]. It remains controversial from various PIF4/DELLA experiments if this is an auxin-mediated process [25], or is more dominantly controlled by another class of plant hormones [22,23]. Either way, the observed changes in developmental architecture were attributed to elevated transcription of an auxin-responsive gene [24].

Circadian Clock: Compensation versus Entrainment

The plant circadian clock is required for plants to synchronize to predictable changes in the diurnal environment that occur each day [27]. The day–night cycle generates matched light/warmth and dark/coolness cycles. Predicting future changes in light and temperature provides a fitness benefit to plants, and the circadian clock is required for this process [28,29]. It perhaps is not surprising that, as plants are photoautotrophic organisms, the circadian clock plays a major role in the capacity of the organism to temporally couple light capture to carbon fixation, and thus basal metabolism, and to apportion these processes to appropriate times of day [27,30]. Circadian clock mutants grow poorly because of defects in these processes [31,32].

Temperature is a key environmental signal directing clock action. The role of temperature in the clock is exemplified by the regulatory dominance of the clock on global transcription. Current estimates are that the plant clock coordinates the steady state levels of about 10,000 transcripts [27], illustrating the clock's dominant role in physiology and growth. Elegant molecular–genetic studies have largely defined the core of the oscillator [27], and this allows a preliminary understanding of how environmental signals intersect with this predictive signaling system. Temperature has two competing actions on the plant circadian system. On one hand, changes in mean ambient temperature are resisted by the oscillator in a process termed 'temperature compensation'. This compensation buffers oscillator speed from changes in the ambient thermal environment, thus ensuring an about-a-day cycle for the clock despite the vagaries of weather. For this compensation mechanism, clock components are required [33], and components previously known for their role in the response to prolonged exposure to near-freezing temperatures that occur in the winter, termed vernalization, have also been shown to modify clock action [34]. This has led to the interesting discovery that this winter response can itself modify daily timing behaviors. Still, how the enzymatic machine that is the circadian oscillator resists ambient thermal changes remains poorly understood in plants.

A second effect of temperature is to act as a resetting cue in a process termed entrainment. Daily temperature oscillations as low as $\Delta 4^{\circ}C$ reset the plant circadian oscillator [35]. How such small differences in temperature reset the clock is not known. Furthermore, it is entirely unclear how a temperature-compensated clock can maintain constant period at different temperatures yet be reset by those same temperature changes in entrainment processes. Interestingly, and seemingly relevant to understanding these conflicting effects, several clock components are known to be preferentially required for the resetting signal provided by temperature cycles [36]. What neither the compensation nor the entrainment experiments have to date assessed is the nature of the temperature sensor that is relevant to the circadian oscillator.

Genetics and Fitness: A Role for Heat-Shock Proteins

A key distinction between plant and animal development is the extent of plasticity displayed by plants. As described above, numerous plant responses are altered dramatically by small changes in temperature. Micro-evolutionary studies have been used to test the assumption that such changes provide a fitness benefit. One candidate for resistance of extended ambient warmth is the heat-shock pathway. In a small survey of natural accessions of *A. thaliana*, transcript abundance of the protein chaperone *HSP101* was found to vary, and this correlated with the latitude from which accessions originated. Furthermore, mutations in *HSP101* rendered plants less fit than wild-type individuals [37]. In a separate study, the molecular basis of incompatible epistatic allele interactions was shown to be dependent on the ambient thermal environment. The defined genetic

interaction requires deregulated cell death programs typical of disease resistance [38]. Interestingly, the capacity of disease-resistance proteins to signal depends on the heat-shock protein (HSP) system, as manipulation of the chaperone HSP90 attenuates resistance-protein signaling [39]. Connecting to this, transcript accumulation of the chaperone gene *HSP70* was found to be induced in response to elevated, non-stressful temperatures, indicating that it is an output of the temperature-sensing pathway, and therefore probably downstream of the primary temperature-sensing event(s) [40]. Collectively, one can wonder if the thermosensitive interactions of protein chaperones with their targets offer a plausible mechanism for temperature sensing and whether, considered collectively, protein chaperones could be ambient thermometers in plants.

Towards an Understanding of Primary Temperature Sensing Events

The second messenger calcium (Ca^{2+}) is used in transduction of numerous signal stimuli across kingdoms [41]. An elevation of cytosolic free calcium levels ($[Ca^{2+}]_{cyt}$) is one of the earliest events in plants' responses to cooling [42], primarily due to influx of Ca^{2+} from the cell wall [43]. The initial response and subsequent acclimation to freezing is dependent upon Ca^{2+} influx [43,44]. Ca^{2+} influx occurs within milliseconds of stimulation; this indicates it is close to the primary sensing event. Indeed, plasma membrane TRP Ca^{2+} channels, such as mammalian CMR1, have been cited as primary thermal sensors [45].

Cold acclimation entails many biochemical and physiological changes, including alterations in membrane composition, increases in total soluble-protein content, and increases in levels of cryoprotectants such as proline and sugars [46]. Significant changes in gene expression are associated with cold acclimation and a class of cold-regulated (*COR*) genes lies downstream of primary perception and Ca^{2+} influx [47]. Key among these are the *CBF* (CRT/DRE binding factor)/*DREB1* (DRE-binding factor 1) genes, which are rapidly induced within 15 minutes, reaching peak expression within about 2 hours after onset of cold [48,49]. *CBF* genes encode transcriptional activators that bind to CRT/DRE elements present in the promoters of *COR* genes. One of these genes is activated by a calmodulin-binding transcription activator, CAMTA, offering a putative connection to calcium signaling and the very rapid Ca^{2+} influx following onset of coolness [48]. Other CBF-independent pathways are also induced in response to cold [49]. It is worth noting that the expression levels of *COR* and *CBF* genes do not correlate fully with freezing tolerance among 50 *A. thaliana* accessions, suggesting considerable complexity in the coolness priming of cold resistance [50].

As with coolness responses, cytosolic Ca^{2+} levels rise in response to heat [51]. Recent work in a lower plant demonstrates that the heat-shock response depends upon the activity of plasma membrane Ca^{2+} channels [52]. This leads to one of the longstanding mysteries associated with Ca^{2+} signaling: how are different responses elicited by the same change in $[Ca^{2+}]_{cyt}$? This dilemma has led to a hypothesis that Ca^{2+} transients are key, early events in temperature perception. In animal cells, the dynamics of Ca^{2+} transients — the calcium signature — is thought to encode information about the nature and strength of the stimulus, although this concept is not universally accepted in plant cell biology [53]. Nonetheless, the magnitude of Ca^{2+} transients in the plant cell correlates positively with the rate of temperature reduction and is also a function of the final temperature reached [54].

Cold-inducible gene expression as a measure of lowering temperature indicates that Ca^{2+} influx is preceded by, and dependent upon, alterations in membrane fluidity. Interestingly, microfilament destabilization occurs downstream of membrane rigidification [55]. This suggests that mechanical changes in the actin cytoskeleton are responsible for activating plasma membrane Ca^{2+} channels. The actin cytoskeleton and membrane fluidity are similarly implicated in the responses to Ca^{2+} influx. Activation of a cold-sensitive protein kinase, MAPK, requires membrane rigidification, whereas activation of a heat-responsive MAPK is dependent upon membrane fluidity [56]. Therefore, it is possible that cytoskeletal/membrane dynamics offer a supplement to Ca^{2+} transients in distinguishing coolness from warmth. Certainly these observations support the hypothesis that the plasma membrane is one primary site of ambient-temperature perception.

Chromatin as a Site of Temperature Sensing

The *A. thaliana* transcriptome undergoes major reconfiguration in response to cool versus warm temperatures, even at temperatures in which stress responses are not induced. The abundance of ~2,500 transcripts increased and another ~2,900 decreased at 27°C versus 12°C [40]. A genetic screen for plants whose gene expression profile in cool conditions resembled wild-type plants at warmer temperatures identified mutations in *Actin Related Protein 6* (*ARP6*) [40]. Among the known physiological responses of *A. thaliana* to elevated temperature is that of early flowering [7,57]. Indeed, *arp6* flowers earlier than wild type at cool temperatures [40], which is consistent with a role for ARP6 in delaying flowering at cool temperatures, and hence, in ambient-temperature sensing. *ARP6* encodes a component of the SWR1 complex necessary for inserting the alternative histone H2A.Z into nucleosomes, which implicates H2A.Z-containing nucleosomes in the response to ambient temperature. Histone H2A.Z deposition at the flowering-time repressor *FLC* locus was shown previously to be controlled by the SWR1 complex and to control *FLC* expression [58].

Chromatin immuno-precipitation (ChIP) assays revealed, in response to warmth, a decrease in H2A.Z histone occupancy at the +1 nucleosome position of the *HSP70* promoter, and this was concomitant with increased *HSP70* expression. Similar changes in occupancy were also seen in the promoter of the flowering-time integrator *FT*, possibly explaining the accelerated flowering in *arp6* [40]. However, there is no simple relationship between depletion of H2A.Z-containing nucleosomes and transcriptional induction because elevated temperature results in depletion of H2A.Z from nucleosomes at all promoters, independent of their transcriptional response (either activation or repression) to elevated temperature.

The Wigge group showed that H2A.Z-containing nucleosomes wrap DNA more tightly [40]. They proposed that, for genes upregulated at elevated temperatures, low-temperature inclusion of H2A.Z near the transcriptional start site either prevents the recruitment of a necessary activator or blocks elongation of a bound, but stalled, RNA polymerase II (RNA Pol II). At elevated temperatures, the depletion of H2A.Z allows either the recruitment of the necessary activator or relieves the block of RNA Pol II elongation. For genes

Box 2

A hypothetical list of plausible thermometers in plants based on physical capacities.

1) Cellular membranes
   a. Membrane fluidity and lipid rafts; capacity to modulate membrane–protein interactions
   b. Stretch/mechanical activation of ion channels, in particular, Ca^{2+} channels, in response to changing membrane dynamics
   c. Movement rates of redox-related metabolites within the photosynthetic and mitochondrial electron-transport chains, leading to temperature-dependent generation of NADPH/NADH and ATP
2) Chromatin state and thermal interactions of DNA with proteins
3) Partitioning of hormonal import and antiport channels
4) RNA
   a. Temperature-sensitive intramolecular folding of transcripts
   b. Melting kinetics of miRNAs with their binding targets
   c. mRNA: splicesomal association with pre-mRNAs, and the capacity to generate transcript variants
5) Proteins
   a. Protein: translation and polysome loading kinetics
   b. Temperature-sensitive protein stability
   c. Temperature-dependent enzyme activity through Q_{10}* (specific examples might include effects on metabolic and chromatin modifying enzymes)
   d. The protein chaperone system and temperature-dependent protein folding

Note: this is not an exclusive list.

*Q_{10} is the temperature coefficient. It is a measure of the rate of change of a system as a consequence of increasing the temperature by 10°C.

down-regulated at elevated temperatures, the presence of H2A.Z-containing nucleosomes would block the recruitment of a transcriptional repressor, which can only access the promoter upon depletion of H2A.Z at higher temperatures. Thus, a single temperature-sensitive response, the depletion of H2A.Z-containing nucleosomes at elevated temperatures, permits either upregulation or downregulation, dependent on the specific mechanism of expression of the promoter. H2A.Z-containing nucleosomes are not found at all promoters, and it will be interesting to see if targets include promoters of genes, such as the *COR* genes, that are upregulated at cool temperatures. This study by the Wigge group [40] leaves us with the intriguing question of how temperature is initially perceived by the plant, and how this information is translated into changes in histone occupancy. The authors argued that post-translational modifications, such as histone acetylation, might modify the tightness of the nucleosome cores and such modifications could themselves be directly thermally responsive. This could explain one thermometer in plants, but to date, the data are inadequate to resolve this unambiguously.

Concluding Comments

Over a global scale, terrestrial temperature varies in a great number of ways, depending on locality and season. This variation takes, for example, the form of differences in mean temperature as well as the diurnal range of temperature minima and maxima (Figure 1). Temperature modulates many processes in plants at a number of levels, suggesting a high likelihood that distinct thermometers exist within a plant cell to detect temperature changes. A recent study has concluded that a single, simple relationship between temperature and all of plant responses to it does not exist [59]. Notably, whereas growth could be empirically modeled as a temperature-dependent process, the correlative analyses of numerous enzyme activities and integrated metabolic processes have revealed a complex relationship between given parameters. The simplest explanation for these complications was reasoned to be the requirement of multiple thermometers, and the presented mathematical models incorporated this logic [59]. Some responses to temperature may be initiated by signaling events, others initiated by the direct physical effects of temperature on protein conformation and enzyme activities. Whether alterations in chromatin state, such as those used to control flowering time, are the result of a direct effect of temperature on protein function, or the result of signaling in response to temperature changes via membrane fluidity and Ca^{2+} transients, remains to be demonstrated. As such, the infant sub-discipline of ambient temperature sensing in plants has a great number of grand challenges (Box 2).

While it is likely that non-stressful ambient temperature changes are sensed and transduced differently compared to sensing of extreme temperature changes, it is interesting to note that a number of common components are in pathways previously thought to be unrelated, such as flowering time and cold-regulated gene expression. Notably, numerous proteins play a role in both pathways [60–64], indicating unsuspected mechanistic commonalities and perhaps suggesting an adaptive advantage to coordinating developmental and stress-tolerance responses to temperature changes over short and longer timescales. This may argue that common thermometers are used in some pathways that require coordinate regulation by ambient and stress temperature signals.

Global climate models forecast large-scale changes in ambient temperature over virtually all landmasses of the Earth ([3,4] and references within). As such, knowing how plants respond to temperature, over differing timescales, is more than simply an academic challenge. Breeding and the ongoing creation of food security will require a fundamental, mechanistic exploration of how plants detect and respond to ambient temperature. With this knowledge, crops can be tailored to faithfully match the projected climate.

Acknowledgments

We are ever grateful for primary writing support from Heather Knight. Work in the McClung lab is supported by the National Science

Foundation (IOS 0960803, IOS 0923752, and IOS 0605736) and from the United States–Israel Binational Science Foundation (2005223). Temperature work in the Davis lab is supported the Max Planck Society, the German Israeli Program (DIP: H 3.1), and the German Science Foundation (DFG: DA1061/4-1). We are grateful for the comments on climate by Tim Osborn, and his assistance in generating the figure.

References

1. Sharkey, T.D., and Singsaas, E.L. (1995). Why plants emit isoprene. Nature *374*, 769.
2. Wilczek, A.M., Roe, J.L., Knapp, M.C., Cooper, M.D., Lopez-Gallego, C., Martin, L.J., Muir, C.D., Sim, S., Walker, A., Anderson, J., *et al*. (2009). Effects of genetic perturbation on seasonal life history plasticity. Science *323*, 930–934.
3. IPCC (2007). Climate Change 2007: Climate Change Impacts, Adaptation and Vulnerability. Fourth Assessment Report, Intergovernmental Panel on Climate Change.
4. Thuiller, W., Lavorel, S., Araujo, M.B., Sykes, M.T., and Prentice, I.C. (2005). Climate change threats to plant diversity in Europe. Proc. Natl. Acad. Sci. USA *102*, 8245–8250.
5. Penfield, S. (2008). Temperature perception and signal transduction in plants. New Phytol. *179*, 615–628.
6. Bernacchi, C.J., Rosenthal, D.M., Pimentel, C., Long, S.P., and Farquhar, G.D. (2009). Modeling the temperature dependence of C3 photosynthesis. In Photosynthesis in Silico: Understanding Complexity from Molecules to Ecosystems (Advances in Photosynthesis and Respiration Volume 29), L.N. Agu Laisk and Govindjee, eds. (Dordrecht: Springer), pp. 231–246.
7. Blazquez, M.A., Ahn, J.H., and Weigel, D. (2003). A thermosensory pathway controlling flowering time in *Arabidopsis thaliana*. Nat. Genet. *33*, 168–171.
8. Johansson, J., Mandin, P., Renzoni, A., Chiaruttini, C., Springer, M., and Cossart, P. (2002). An RNA thermosensor controls expression of virulence genes in *Listeria monocytogenes*. Cell *110*, 551–561.
9. Diernfellner, A.C., Schafmeier, T., Merrow, M.W., and Brunner, M. (2005). Molecular mechanism of temperature sensing by the circadian clock of *Neurospora crassa*. Genes Dev. *19*, 1968–1973.
10. Hamada, F.N., Rosenzweig, M., Kang, K., Pulver, S.R., Ghezzi, A., Jegla, T.J., and Garrity, P.A. (2008). An internal thermal sensor controlling temperature preference in Drosophila. Nature *454*, 217–220.
11. Rosenzweig, M., Brennan, K.M., Tayler, T.D., Phelps, P.O., Patapoutian, A., and Garrity, P.A. (2005). The Drosophila ortholog of vertebrate TRPA1 regulates thermotaxis. Genes Dev. *19*, 419–424.
12. Ausin, I., Alonso-Blanco, C., and Martinez-Zapater, J.M. (2005). Environmental regulation of flowering. Int. J. Dev. Biol. *49*, 689–705.
13. Michaels, S.D. (2009). Flowering time regulation produces much fruit. Curr. Opin. Plant Biol. *12*, 75–80.
14. Halliday, K.J., Salter, M.G., Thingnaes, E., and Whitelam, G.C. (2003). Phytochrome control of flowering is temperature sensitive and correlates with expression of the floral integrator FT. Plant J. *33*, 875–885.
15. Halliday, K.J., and Whitelam, G.C. (2003). Changes in photoperiod or temperature alter the functional relationships between phytochromes and reveal roles for phyD and phyE. Plant Physiol. *131*, 1913–1920.
16. Franklin, K.A., and Whitelam, G.C. (2007). Light-quality regulation of freezing tolerance in *Arabidopsis thaliana*. Nat. Genet. *39*, 1410–1413.
17. Lee, H., Yoo, S.J., Lee, J.H., Kim, W., Yoo, S.K., Fitzgerald, H., Carrington, J.C., and Ahn, J.H. (2010). Genetic framework for flowering-time regulation by ambient temperature-responsive miRNAs in Arabidopsis. Nucleic Acids Res. *38*, 3081–3093.
18. Mockaitis, K., and Estelle, M. (2008). Auxin receptors and plant development: a new signaling paradigm. Annu. Rev. Cell Dev. Biol. *24*, 55–80.
19. Gray, W.M., Ostin, A., Sandberg, G., Romano, C.P., and Estelle, M. (1998). High temperature promotes auxin-mediated hypocotyl elongation in Arabidopsis. Proc. Natl. Acad. Sci. USA *95*, 7197–7202.
20. Yamada, M., Greenham, K., Prigge, M.J., Jensen, P.J., and Estelle, M. (2009). The TRANSPORT INHIBITOR RESPONSE2 gene is required for auxin synthesis and diverse aspects of plant development. Plant Physiol. *151*, 168–179.
21. Harberd, N.P., Belfield, E., and Yasumura, Y. (2009). The angiosperm gibberellin-GID1-DELLA growth regulatory mechanism: how an "inhibitor of an inhibitor" enables flexible response to fluctuating environments. Plant Cell *21*, 1328–1339.
22. de Lucas, M., Daviere, J.M., Rodriguez-Falcon, M., Pontin, M., Iglesias-Pedraz, J.M., Lorrain, S., Fankhauser, C., Blazquez, M.A., Titarenko, E., and Prat, S. (2008). A molecular framework for light and gibberellin control of cell elongation. Nature *451*, 480–484.
23. Feng, S., Martinez, C., Gusmaroli, G., Wang, Y., Zhou, J., Wang, F., Chen, L., Yu, L., Iglesias-Pedraz, J.M., Kircher, S., Schafer, E., *et al*. (2008). Coordinated regulation of *Arabidopsis thaliana* development by light and gibberellins. Nature *451*, 475–479.
24. Koini, M.A., Alvey, L., Allen, T., Tilley, C.A., Harberd, N.P., Whitelam, G.C., and Franklin, K.A. (2009). High temperature-mediated adaptations in plant architecture require the bHLH transcription factor PIF4. Curr. Biol. *19*, 408–413.
25. Nozue, K., Covington, M.F., Duek, P.D., Lorrain, S., Fankhauser, C., Harmer, S.L., and Maloof, J.N. (2007). Rhythmic growth explained by coincidence between internal and external cues. Nature *448*, 358–361.
26. Sidaway-Lee, K., Josse, E.M., Brown, A., Gan, Y., Halliday, K.J., Graham, I.A., and Penfield, S. (2010). SPATULA links daytime temperature and plant growth rate. Curr. Biol. *20*, 1493–1497.
27. Harmer, S.L. (2009). The circadian system in higher plants. Annu. Rev. Plant Biol. *60*, 357–377.
28. Resco, V., Hartwell, J., and Hall, A. (2009). Ecological implications of plants ability to tell the time. Ecol. Lett. *12*, 5835–5892.
29. Yerushalmi, S., and Green, R.M. (2009). Evidence for the adaptive significance of circadian rhythms. Ecol. Lett. *12*, 970–981.
30. Pruneda-Paz, J.L., and Kay, S.A. (2010). An expanding universe of circadian networks in higher plants. Trends Plant Sci. *15*, 259–265.
31. Dodd, A.N., Salathia, N., Hall, A., Kevei, E., Toth, R., Nagy, F., Hibberd, J.M., Millar, A.J., and Webb, A.A. (2005). Plant circadian clocks increase photosynthesis, growth, survival, and competitive advantage. Science *309*, 630–633.
32. Graf, A., Schlereth, A., Stitt, M., and Smith, A.M. (2010). Circadian control of carbohydrate availability for growth in Arabidopsis plants at night. Proc. Natl. Acad. Sci. USA *107*, 9458–9463.
33. Gould, P.D., Locke, J.C., Larue, C., Southern, M.M., Davis, S.J., Hanano, S., Moyle, R., Milich, R., Putterill, J., Millar, A.J., and Hall, A. (2006). The molecular basis of temperature compensation in the Arabidopsis circadian clock. Plant Cell *18*, 1177–1187.
34. Salathia, N., Davis, S.J., Lynn, J.R., Michaels, S.D., Amasino, R.M., and Millar, A.J. (2006). FLOWERING LOCUS C-dependent and -independent regulation of the circadian clock by the autonomous and vernalization pathways. BMC Plant Biol. *6*, 10.
35. Somers, D.E., Webb, A.A., Pearson, M., and Kay, S.A. (1998). The short-period mutant, *toc1-1*, alters circadian clock regulation of multiple outputs throughout development in Arabidopsis thaliana. Development *125*, 485–494.
36. Salomé, P.A., and McClung, C.R. (2005). PSEUDO-RESPONSE REGULATOR 7 and 9 are partially redundant genes essential for the temperature responsiveness of the Arabidopsis circadian clock. Plant Cell *17*, 791–803.
37. Tonsor, S.J., Scott, C., Boumaza, I., Liss, T.R., Brodsky, J.L., and Vierling, E. (2008). Heat shock protein 101 effects in *A. thaliana*: genetic variation, fitness and pleiotropy in controlled temperature conditions. Mol. Ecol. *17*, 1614–1626.
38. Alcazar, R., Garcia, A.V., Parker, J.E., and Reymond, M. (2009). Incremental steps toward incompatibility revealed by Arabidopsis epistatic interactions modulating salicylic acid pathway activation. Proc. Natl. Acad. Sci. USA *106*, 334–339.
39. Hubert, D.A., He, Y., McNulty, B.C., Tornero, P., and Dangl, J.L. (2009). Specific Arabidopsis HSP90.2 alleles recapitulate RAR1 cochaperone function in plant NB-LRR disease resistance protein regulation. Proc. Natl. Acad. Sci. USA *106*, 9556–9563.
40. Kumar, S.V., and Wigge, P.A. (2010). H2A.Z-containing nucleosomes mediate the thermosensory response in Arabidopsis. Cell *140*, 136–147.
41. Hepler, P.K. (2005). Calcium: a central regulator of plant growth and development. Plant Cell *17*, 2142–2155.
42. Knight, M.R., Campbell, A.K., Smith, S.M., and Trewavas, A.J. (1991). Transgenic plant aequorin reports the effects of touch and cold-shock and elicitors on cytoplasmic calcium. Nature *352*, 524–526.
43. Knight, H., Trewavas, A.J., and Knight, M.R. (1996). Cold calcium signaling in Arabidopsis involves two cellular pools and a change in calcium signature after acclimation. Plant Cell *8*, 489–503.
44. Tahtiharju, S., Sangwan, V., Monroy, A.F., Dhindsa, R.S., and Borg, M. (1997). The induction of *kin* genes in cold-acclimating *Arabidopsis thaliana*. Evidence of a role for calcium. Planta *203*, 442–447.
45. McKemy, D.D., Neuhausser, W.M., and Julius, D. (2002). Identification of a cold receptor reveals a general role for TRP channels in thermosensation. Nature *416*, 52–58.
46. Thomashow, M.F. (1999). Plant cold acclimation: freezing tolerance genes and regulatory mechanisms. Annu. Rev. Plant Physiol. Plant Mol. Biol. *50*, 571–599.
47. Van Buskirk, H.A., and Thomashow, M.F. (2006). Arabidopsis transcription factors regulating cold acclimation. Physiol. Plant *126*, 72–80.
48. Doherty, C.J., Van Buskirk, H.A., Myers, S.J., and Thomashow, M.F. (2009). Roles for Arabidopsis CAMTA transcription factors in cold-regulated gene expression and freezing tolerance. Plant Cell *21*, 972–984.
49. Hua, J. (2009). From freezing to scorching, transcriptional responses to temperature variations in plants. Curr. Opin. Plant Biol. *12*, 568–573.
50. McKhann, H., Gery, C., Berard, A., Leveque, S., Zuther, E., Hincha, D., De Mita, S., Brunel, D., and Teoule, E. (2008). Natural variation in *CBF* gene sequence, gene expression and freezing tolerance in the Versailles core collection of *Arabidopsis thaliana*. BMC Plant Biol. *8*, 105.

51. Gong, M., van der Luit, A.H., Knight, M.R., and Trewavas, A.J. (1998). Heat-shock-induced changes in intracellular Ca2+ level in tobacco seedlings in relation to thermotolerance. Plant Physiol. *116*, 429–437.

52. Saidi, Y., Finka, A., Muriset, M., Bromberg, Z., Weiss, Y.G., Maathuis, F.J., and Goloubinoff, P. (2009). The heat shock response in moss plants is regulated by specific calcium-permeable channels in the plasma membrane. Plant Cell *21*, 2829–2843.

53. Scrase-Field, S.A., and Knight, M.R. (2003). Calcium: just a chemical switch? Curr. Opin. Plant Biol. *6*, 500–506.

54. Plieth, C., Hansen, U.P., Knight, H., and Knight, M.R. (1999). Temperature sensing by plants: the primary characteristics of signal perception and calcium response. Plant J. *18*, 491–497.

55. Orvar, B.L., Sangwan, V., Omann, F., and Dhindsa, R.S. (2000). Early steps in cold sensing by plant cells: the role of actin cytoskeleton and membrane fluidity. Plant J. *23*, 785–794.

56. Sangwan, V., Orvar, B.L., Beyerly, J., Hirt, H., and Dhindsa, R.S. (2002). Opposite changes in membrane fluidity mimic cold and heat stress activation of distinct plant MAP kinase pathways. Plant J. *31*, 629–638.

57. Balasubramanian, S., Sureshkumar, S., Lempe, J., and Weigel, D. (2006). Potent induction of *Arabidopsis thaliana* flowering by elevated growth temperature. PLoS Genet. *2*, e106.

58. Deal, R.B., Topp, C.N., McKinney, E.C., and Meagher, R.B. (2007). Repression of flowering in Arabidopsis requires activation of FLOWERING LOCUS C expression by the histone variant H2A.Z. Plant Cell *19*, 74–83.

59. Parent, B., Turc, O., Gibon, Y., Stitt, M., and Tardieu, F. (2010). Modelling temperature-compensated physiological rates, based on the coordination of responses to temperature of developmental processes. J. Exp. Bot. *61*, 2057–2069.

60. Dong, C.H., Hu, X., Tang, W., Zheng, X., Kim, Y.S., Lee, B.H., and Zhu, J.K. (2006). A putative Arabidopsis nucleoporin, AtNUP160, is critical for RNA export and required for plant tolerance to cold stress. Mol. Cell Biol. *26*, 9533–9543.

61. Kim, H.J., Hyun, Y., Park, J.Y., Park, M.J., Park, M.K., Kim, M.D., Kim, H.J., Lee, M.H., Moon, J., Lee, I., and Kim, J. (2004). A genetic link between cold responses and flowering time through FVE in *Arabidopsis thaliana*. Nat. Genet. *36*, 167–171.

62. Lee, H., Xiong, L., Gong, Z., Ishitani, M., Stevenson, B., and Zhu, J.K. (2001). The Arabidopsis HOS1 gene negatively regulates cold signal transduction and encodes a RING finger protein that displays cold-regulated nucleo-cytoplasmic partitioning. Genes Dev. *15*, 912–924.

63. Seo, E., Lee, H., Jeon, J., Park, H., Kim, J., Noh, Y.S., and Lee, I. (2009). Crosstalk between cold response and flowering in *Arabidopsis* is mediated through the flowering-time gene *SOC1* and its upstream negative regulator *FLC*. Plant Cell *21*, 3185–3197.

64. Yoo, S.Y., Kim, Y., Kim, S.Y., Lee, J.S., and Ahn, J.H. (2007). Control of flowering time and cold response by a NAC-domain protein in Arabidopsis. PLoS ONE *2*, e642.

65. Mitchell, T.D., and Jones, P.D. (2005). An improved method of constructing a database of monthly climate observations and associated high-resolution grids. Int. J. Climat. *25*, 693–712.