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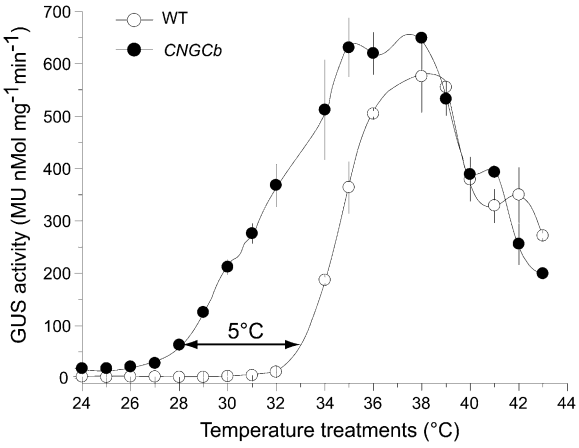
**IN BRIEF**

# Calcium Channels and Acquired Thermotolerance: Here Comes the Sun and It's All Right

When the sun comes up on a clear summer morning, it starts to get hot and you know it's going to get hotter. What's remarkable is that plants know it too. However, rather than making plans to hit the swimming pool, plants have to make other arrangements. These arrangements result in transcriptional activation of genes encoding heat shock proteins (Hsps); this dramatic mobilization can involve more than 1% of the genome (reviewed in Saidi et al., 2011) and therefore represents a substantial commitment of resources. Many Hsps prevent protein misfolding and aggregation, stabilize membrane structure, and are transcribed after activation of heat shock transcription factors. Upstream of Hsp transcription, the triggering of the heat tolerance response involves the cell membrane in two ways. First, the lipid composition and fluidity of the membrane condition the response to heat, as treatments that increase membrane fluidity cause a stronger response. Second, a transient influx of $Ca^{2+}$ activates heat shock transcription factors, through mechanisms involving calmodulin, kinase activity, histone modification, and other pathways.

The thermotolerance response allows the plant to survive a short exposure to a high temperature that would otherwise be lethal. However, what is the basis for activation of the plant heat tolerance response? For activation, the plant must accurately measure temperature, to produce a response in sufficient time, but not waste resources by activating an unnecessary response. How does a plant sense temperature? Previous work in moss had established that the primary thermosensor involves a heat-sensitive plasma membrane $Ca^{2+}$ channel, but the identity of the key genes remained unknown. To supply this missing link in the chain of events leading to thermotolerance, **Finka et al. (pages 3333–3348)** examine the role of cyclic nucleotide gated $Ca^{2+}$ channels in thermosensing in land plants. The authors characterize the orthologous *Physcomitrella patens* and *Arabidopsis thaliana* loci encoding the cyclic nucleotide gated calcium channel (CNGC), Pp *CNGCb* and At *CNGC2*, respectively. They found that both moss and *Arabidopsis* mutants with a disruption of CNGC grew more slowly and acquired futile thermotolerance by induction of Hsp production at temperatures about five degrees lower than the wild type (see figure). Further examining the moss mutant to determine the basis of this hyperthermosensitive phenotype, they used the $Ca^{2+}$ sensing protein aqueorin to show that the mutant had a stronger $Ca^{2+}$ flux that was induced at a lower temperature. Patch-clamp analysis showed that there were three types of $Ca^{2+}$ channels in the membranes of moss protoplasts, but one type was missing in the mutants. Moreover, deletion of Pp *CNGCb* also increased the open probability, or percentage of time that the channels are in the open configuration, of the remaining two types of channels, thereby increasing the $Ca^{2+}$ flux.

These results improve our understanding of the mechanisms of thermotolerance and may prove useful to produce crops with improved survival and yield under high-heat conditions. For example, examination of $Ca^{2+}$ flux may allow the selection of heat-resistant varieties. Moreover, identification of the key players may allow targeting of the heat shock response to particularly important or sensitive tissues, such as reproductive structures.




Hyperthermosensitive phenotype of moss *CNGCb* mutants. Induction of Hsps is measured at different temperatures by induction of a heat shock reporter HSP–β-glucuronidase (GUS) fusion construct and quantification of GUS enzyme activity. (*Reprinted from Finka et al. [2012]*, *Figure 3*.)

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