

mRNA is abolished. Thus, ABA causes accumulation of full-length *FCA* mRNA. Razem *et al.* show that ABA causes a dramatic increase in *FLC* mRNA, which in turn would delay the transition to flowering. Consistent with this model, the authors report that ABA causes a delay in flowering in *Arabidopsis*. As *Arabidopsis* plants can flower early in response to drought, which increases ABA production, the ABA–*FCA* response may be overridden during this response¹³. Possible modulation mechanisms during drought stress could be investigated by analysing the newly revealed direct ABA regulation of *FCA* mRNA (full-length versus truncated) and the strong ABA-induced increase in levels of *FLC* mRNA.

Interestingly, the RNA-recognition motif in *FCA* is absent in the barley *ABAP1* protein⁵. Indeed, ABA-binding studies of *Arabidopsis FCA* in which the protein lacked specific structural regions show that ABA-binding activity lies in the carboxy-terminal half of *FCA*, which does share homology with *ABAP1* (ref. 1).

Razem *et al.*¹ went on to show that in plants with a loss-of-function mutation in *FCA*, the ABA-induced closing of stomatal pores and inhibition of seed germination — two classical ABA responses — were not impaired. Furthermore, ABA inhibition of flowering was not affected in two dominant ABA-insensitive mutants, *abi1-1* and *abi2-1*, in which most of the stress-related ABA responses are impaired. Thus, other ABA receptors are needed to explain the classical ABA signalling responses to stress. The hunt could be on to characterize

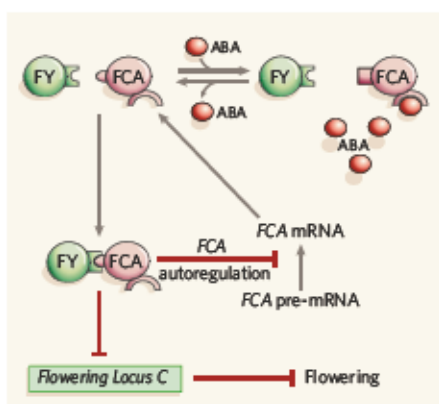


Figure 1 | Abscisic acid, RNA metabolism and control of flowering in plants. Binding of two proteins, *FCA* and *FY*, to one another results in a decrease in expression levels of *Flowering Locus C (FLC)*, causing a transition from vegetative growth to flowering. The *FCA*–*FY* complex also causes synthesis of a truncated, non-functional *FCA* messenger RNA in a negative feedback loop that results in fewer full-length *FCA* mRNA transcripts and less *FCA* protein^{6,7}. Razem *et al.*¹ report that binding of abscisic acid (ABA) to *FCA* abolishes the interaction of *FCA* with *FY*, leading to an increase in full-length *FCA* transcripts and — through increased *FLC* activity — a delay in flowering. Red lines depict negative regulation. (Diagram modified from a figure provided by R. Hill)

homologues to the ABA-binding carboxy terminus of *FCA*¹ and barley *ABAP1*. A simple search of protein databases reveals only one distant *FCA* homologue in the *Arabidopsis* genome. Alternatively, the *FCA* and *ABAP1* proteins provide an opportunity to elucidate

the structure of an ABA-binding pocket, which may reveal important sub-domains and structural constraints for ABA binding.

A door to understanding ABA perception has been opened. The binding of ABA to *FCA* and *ABAP1* is apparently a further example of newly emerging mechanisms by which plant growth regulators mediate their responses. Further questions arise with each advance. Plant scientists will need to keep on trekking to illuminate how their immobile lab subjects perceive abscisic acid when faced with drought, cold and salinity.

Julian I. Schroeder and Josef M. Kuhn are at the Division of Biological Sciences, Cell and Developmental Biology Section, University of California, San Diego, 9500 Gilman Drive, La Jolla, California 92093-0116, USA. e-mail: julian@biomail.ucsd.edu

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CHEMICAL ECOLOGY

In defence of maize

They can't run and they can't hide. But with those reactions denied to them, plants have evolved an extensive and varied repertoire for responding to threats to their well-being. Apart from the tolerance mechanisms discussed above by Schroeder and Kuhn, such responses can include calling upon insect allies to deal with pests that would otherwise damage or destroy the plant by eating it.

Christiane Schnee and colleagues have started to dissect one such signal system — that used by maize seedlings when they are attacked by caterpillars (*Proc. Natl. Acad. Sci. USA* doi:10.1073/pnas.0508027103; 2005). The plant signals consist of volatile chemicals, which in the case investigated by Schnee *et al.* attract female wasps of the species *Cotesia marginiventris*. The wasps lay their eggs

in the caterpillars, with predictably unhappy results for the latter.

Maize emits a cocktail of volatile defence signals in response to an attack by herbivores, and the general difficulty in studying the process is identifying which constituent of these complex blends has which effect. From investigations of maize biosynthetic pathways, Schnee *et al.* first isolated an enzyme, a terpene synthase dubbed TPS10, that is responsible for producing most of the herbivore-induced volatiles.

But that was only an initial step. To look into the biological effects of these terpenes, the authors used genetic engineering to insert the gene that encodes TPS10 into *Arabidopsis*, the standard lab plant for biologists. The transgenic *Arabidopsis* plants were then used



in experiments in which female *C. marginiventris* could choose between the options offered in an 'olfactometer' (fresh air and untransformed *Arabidopsis* being the other choices).

The main result to emerge was that the wasps indeed showed a strong preference for the plant that produced the TPS-mediated terpene — but only after they had learned the association between the defence signal and the host by having previously

laid eggs in the caterpillar host.

This approach, say Schnee *et al.*, is an example of the value of using transgenic technology to study the effects of complex volatile compounds. Even when the compounds themselves or their constituents are not available, if the genes concerned have been identified, the ecological influences of these signals can nonetheless be investigated in genetically transformed plants.

Tim Lincoln