Journal Club

A hairy tale: Glabrous meets Werewolf

Two transcription factors of the MYB family, GLABROUS1 (GL1) and WEREWOLF (WER) are important players in epidermal cell type determination in *Arabidopsis*. GL1 specifies the production of trichomes (single-cell hairs) on the surface of leaves and stems, whereas WER establishes the development of hairless cells in root epidermis and of non-stomatal cells in hypocotyl epidermis. Their names describe the phenotypes of plants in which the corresponding genes are mutated: leaves of the *glabrous1* (*gl1*) mutants are as bold as some old Romans (Latin: glaber), and the *werewolf* (*wer*) mutants have scary, extra-hairy roots.

GL1 and WER are 57% identical at the amino acid sequence level, and 91% identical at this level in the functionally important MYB domain. However, their promoters control non-overlapping expression patterns and both factors determine distinct epidermal cell types in different organs. The *gl1 wer* double mutant, bold on leaves and hairy on roots as expected, has no additional phenotype that would indicate any synergism between the two factors. This made Myeong Min Lee and John Schiefelbein[1] ask whether their functional specificity originates from the protein coding sequence of the factors or from divergence of their regulatory elements.

Transformation of the *wer* mutant with the WER promoter fused either to the WER or the GL1 coding sequence results in complete complementation in both cases. In the mirror image set up, the GL1 promoter controlling either the GL1 or the WER coding sequence could fully restore epidermal determination in the *gl1* mutant. Therefore, both factors can functionally substitute for each other as long as they are expressed at the right time and in the right place, and different functions for a protein can originate solely from specific regulatory sequences.

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The presumptive target gene of both MYB factors, *GLABRA2* (*GL2*), is correctly expressed in the reciprocal complementation lines (*WER::GL1* in *wer* and *GL1::WER* in *gl1*) – additional and perceptible evidence for the functional equivalence between GL1 and WER. By contrast, the transcription factor MYB2, more distantly related to both GL1 and WER, substitutes for neither WER nor GL1, regardless of the promoter.

The authors' work shows that distinct functions of *MYB* gene family members in *Arabidopsis* might be as much inscribed in specific regulatory elements as in their coding sequences. A third MYB gene, among >100, codes for a close relative of WER and GL1, and it is tempting to speculate on its functional relationship with these equivalent factors and its possible involvement in epidermal cell type determination. The data described to date imply that, during evolution, proteins could have achieved new roles by acquiring new regulators, while maintaining their well established and time-honoured protein structures. MYB genes have undergone an extensive amplification during plant evolution, compared with the relatively small number of family members in other kingdoms. It will be exciting to learn from a comprehensive analysis of plant MYB genes how much the variability of *cis*-regulatory elements contributed to the origin of novel gene functions.

1 Lee, M.M. and Schiefelbein, J. (2001) Developmentally distinct MYB genes encode functionally equivalent proteins in *Arabidopsis*. *Development* 128, 1539–1546

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A hydrophobin aids water-mediated dispersal of *Cladosporium* conidia

Fungal hydrophobins are a class of low-molecular weight (~8 kDa), cysteine-rich proteins. They can be excreted from the fungus and self-assemble on the cell wall, or excreted into the extracellular fluid. Multimeric assemblies of Class I hydrophobins are insoluble in hot solutions of sodium dodecyl sulfate (SDS), whereas Class II hydrophobins are SDS soluble. Hydrophobins are generally localized on the outer surfaces of conidia and of the hyphal wall, and are involved in mediating contact and communication between the fungus and its environment. Hydrophobins are essential for the development of aerial structures, for example, the SC3 hydrophobin from *Schizophyllum commune* coats the outer surface of aerial hyphae with an insoluble, non-wettable protein rodlet film, and rodlet-deficient mutants are impaired in their ability to disperse aerial spores. Among the fungal pathogens, hydrophobins facilitate adhesion of plant pathogenic fungi to hydrophobic surfaces.

Now, James Whiteford and Pietro Spanu[1] have shown that the HCf-1 gene of *Cladosporium fulvum* codes for a hydrophobin that facilitates efficient water-mediated conidium dispersal. Six

'The HCf-1 hydrophobin appears to be a principal component of rodlets on the conidium surface, probably the main structure in the outer conidial wall that contributes to surface hydrophobicity and the ability of conidia to be collected by water.'

hydrophobin genes have been described in *C. fulvum*; the authors report the generation of two new deletion mutants, one lacking HCf-2, and a second lacking both HCf-1 and HCf-2, in addition to an HCf-1 mutant generated previously. The absence of HCf-1

