## IN BRIEF

**Twist of Fate: Ribosomal Stress Reprogramms Root Hair Patterning**

The root epidermis presents an elegant model to study cell differentiation. Based on positional cues, Arabidopsis (*Arabidopsis thaliana*) distinguishes two epidermal cell types. Cells in the H position, adjacent to the junction of two cortex cells, have the capacity of developing root hair identity, whereas the non-hair cells occupy the N positions and are in contact with a single cortical cell. The fate of root epidermal cells lies in the hands of a complex of three transcription factors (TFs): WEREWOLF (WER), GLABRA3/ENHANCER OF GLABRA3 (GL3/EGL3), and TRANSPARENT TESTA GLABRA1 (TTG1). In N position cells, this trio activates GL2 expression and the CPC protein is exported to an H position cell, where it counteracts WER function and subsequently represses GL2 (see figure). Thus, in the N position, cells with high GL2 expression inhibit root hair identity, while low GL2 expression in the H position cells activates root hair identity (Salazar-Henao et al., 2016).

In the wer-1 mutant, N position cells gain root-hair fate and most epidermal cells form a root hair. The cpc-1 mutant produces ectopic non-hair cells, but nevertheless approximately one-third of its epidermis cells forms root hairs, suggesting additional regulators of root epidermis specification are yet to be discovered (reviewed by Salazar-Henao et al., 2016). Wang et al. (2020) addressed this issue with a cpc-1 enhancer screen. By analyzing an ethyl methanesulfonate-mutagenized population of the cpc-1 mutant, the authors identified a double mutant completely deprived of root hairs. The additional mutation was mapped to *ARABIDOPSIS PUMILIO23* (*APUM23*), which encodes a ribosome biogenesis factor. This suggests that a ribosomal defect can switch hair cell fate to a non-hair cell fate.

To dissect the mechanism of defective root patterning caused by ribosomal perturbation, Wang et al. (2020) examined expression of GL2 in the *apum23-4* epidermis. Although GL2 expression in wild-type plants is restricted to N position cells, the lack of functional *ARABIDOPSIS PUMILIO23* resulted in GL2 transcription in some H cells as well. Next, the group examined GL2 expression in a series of crosses of *apum23-4* plants with mutants disrupted in GL2 regulators. Mutations in GL3/EGL3 or TTG1 abolished GL2 transcription and the *gl2-1 apum23-4* mutant developed no root hairs, whereas in the *wer-1 apum23-4* double mutant, GL2 was expressed in some epidermal cells and root hairs were formed in both N and H positions. These experiments provided the genetic evidence that a switch from hair- to non-hair cell fate in the *apum23-4* mutant results from ectopic GL2 expression, which requires both GL3/EGL3 and TTG1, but is independent from WER. This finding opens up an avenue for elucidation of previously unidentified regulators of root epidermal cell patterning.

Because WER contribution to GL2 up-regulation was excluded, the authors further searched for the TF that would act together with GL3/EGL3 and TTG1. MYB23, known previously to function redundantly to WER in N cells, appeared a likely suspect (Kang et al., 2009). Concomitant disruption of MYB23 and WER in *apum23-4* mutant resulted in no GL2 expression. In wild-type plants, MYB23 expression is confined to N position cells, but in *apum23-4* roots, was also observed in H position cells. MYB23 is also activated by WER-GL3/EGL3-TTG1 complex (Kang et al., 2009), yet the *wer-1* mutation did not affect MYB23 expression in the *apum23-4* line. In searching for a potential MYB23 activator, the authors used the
anac082-1 mutant, previously described to reverse the developmental defects caused by ribosomal perturbations (Ohbayashi et al., 2017). Indeed, knocking out ANAC082 in the apum23-4 mutant restored wild-type root hair patterning, indicating that ANAC082 drives the MYB23-dependent GL2 expression (Wang et al., 2020).

Wang et al. (2020) investigated how stress, expressed as altering ribosome biogenesis, plays a role in root epidermis specification. Ultimately the team demonstrated that ribosomal defects activate, independently from positional cues, an ANAC082-MYB23 signaling module, which by upregulation of GL2 leads to an ectopic, non-hair cell fate (see figure). Further studies may explain whether modulation of root hair development by environmental stresses such as salinity or nutrient deficiencies, and by ribosomal stress, share any of these regulators. Identification of the apum23-4 mutant created an excellent system of perturbed root epidermis cell fate to elucidate the integration of stress responses and developmental regulation.

**REFERENCES**


