CORRESPONDENCE

Mutually reinforcing patterning mechanisms

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In a recent Opinion article published in *Nature Reviews Molecular Cell Biology* (Computational morphodynamics of plants: integrating development over space and time. *Nature Rev. Mol. Cell Biol.* **12**, 265–273 (2011))[1], Roeder and collaborators integrate some of the recent work in the emerging field of plant computational morphodynamics, focusing on models based on cellular live-imaging data. One of the examples these authors review is that of *Arabidopsis thaliana* root epidermal patterning, for which they discuss two mathematical models, namely the WEREWOLF (WER) self-activation model, which we authored[2], and the mutual support model[3]. They rule out the WER self-activation model, based on experimental data showing that expression of the MYB transcription factor *WER* is the same in wild-type cells and *wer* loss-of-function mutant cells. Moreover, Roeder *et al.* did not consider the results and discussions presented in a more recent version of the WER self-activation model[4] and in a further theoretical paper[5].

We argue that the WER self-activation model[4] — which assumes that *WER* can positively regulate itself, either directly or through the action of other genes[2,4] — cannot be ruled out based only on the expression of *WER* in *wer* mutants. A similar situation was encountered for the *AGAMOUS* self-regulatory loop in flower morphogenesis[6]. In the case of *A. thaliana* root epidermal patterning, recent experimental data show that *WER* expression is downregulated by the Leu-rich repeat receptor-like kinase *SCRAMBLED* (*SCM*)[7–9] and that *SCM* expression is, in turn, downregulated by *WER*[8,9], giving rise to a positive feedback loop. This results in the indirect and local self-activation of *WER* and stabilizes *SCM*-mediated position signalling. The presence of *WER* in *wer* mutants, which was used to discount our model, indeed seems to contradict these data. However, both pieces of evidence can be reconciled if *WER* is also regulated by other factors, and thus its promoter can still be activated in *wer* mutants. Furthermore, additional evidence in support of *WER* being self-activated is that the closely related *MYB* gene *MYB23* — which can substitute for the function of *WER*[10] — participates in a positive feedback loop with itself. This suggests that the observed cell pattern can still occur in *wer* mutants, owing to the self-maintained expression levels of *MYB23* (REF. 10).

This correspondence may be important for the interpretation of experimental data and modelling studies in other cases, given that the coexistence of various apparently redundant circuits seems to be relevant for the robustness of morphogenetic systems[5]. In the particular case of *A. thaliana* epidermal cell patterning, we argue that the WER self-activation model cannot be ruled out on the basis of the evidence currently available, as published data may still support it. We believe that other circuits, such as the mutual support model, are also consistent with the available data and are possibly reinforcing each other, conferring robustness to the overall spatiotemporal cellular patterns[5,11].

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Competing interests statement
The authors declare no competing financial interests.