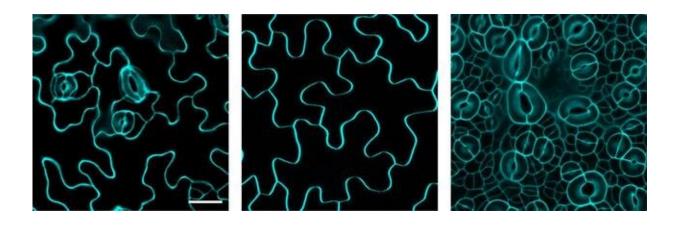


SPEECHLESS, SCREAM and stomata development in plant leaves

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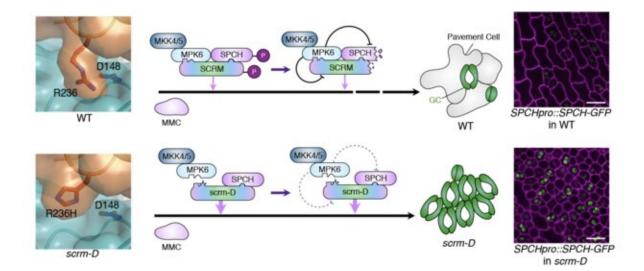
From left to right: Wild type stomata; SPEECHLESS (no stomata mutant); and SCREAM-D mutant (stomata-only leaf epidermis) in leaves of Arabidopsis (rockcress), a small flowering plant related to cabbage and mustard. Credit: Aarthi Putarjunan (U. Washington)/Keiko Torii (Nagoya University /U. Washington).

Plants constantly make trade-offs in their decisions: more light means more opportunity for photosynthesis, but then hot temperatures and dry air makes wilting more likely. Stomata—microscopic valves on the surface of a leaf's epidermis—are at the forefront of these trade-offs: stomata open to acquire fresh air (and the carbon dioxide in it) for photosynthesis, but water loss through stomatal pores causes plants to become dehydrated, and eventually to wilt.



The proper number and distribution of stomata on leaves is critical for plant productivity. Using the model plant Arabidopsis thaliana, a group of scientists including Keiko Torii (Howard Hughes Medical Institute/University of Washington and Institute of Transformative Biomolecules [ITbM] at Nagoya University), have identified key genes that make stomata and enforce proper stomatal patterning.

When a leaf starts to make <u>epidermal cells</u>, whether the initial cell becomes a stoma or non-stomatal epidermal cell is not yet decided. Differentiation of stomata starts when the master-regulatory proteins, SPEECHLESS and SCREAM, regulate gene expression. On the other hand, SPEECHLESS is inhibited by cellular signals involving MAP kinases MPK3 and MPK6, which transmit environmental signals to a cell. Depending on whether the master-regulatory proteins or the MAP kinases win, the initial cell becomes a stoma or non-stomatal epidermal cell. Nonetheless, the exact mechanism of how MPK3 and 6 inhibit SPEECHLESS was unclear until recently.



Mechanism enforcing the initiation of stomatal cell lineages via SCREAM's



unique anchoring module that recruits MAP kinases. Credit: Nagoya University

In the paper published in *Nature Plants*, Dr. Aarthi Putarjunan, Prof. Keiko Torii and colleagues discovered that the SCREAM protein possesses a pair of anchoring motifs that directly bridges MAP kinases (MPK3 and MPK6) to SPEECHLESS. A single amino-acid mutation in this SCREAM-anchoring motif causes an inability to recruit inhibitory MAP kinases, which results in a striking leaf epidermis covered solely by an enormous number of stomata (see Figure 1).

To understand how SCREAM anchors the MAP kinases, Putarjunan and Torii initiated a collaboration with Prof. Ning Zheng, a structural biologist who crystalizes proteins, and Prof. Florence Tama, a theoretical chemist who simulates and models protein-protein interactions. Together with these two protein-structure experts, the group solved the structure of the plant MAP kinase MPK6 and revealed the mechanism, at the <u>atomic level</u>, of how SCREAM brings about inhibitory MAP kinases.

Interestingly, MAP kinases play critical roles in cell proliferation and differentiation between human beings; moreover, dysfunction of MAP kinases is directly associated with cancers. While the structure of the plant MAP kinase MPK6, which was solved in this study, is nearly identical to that of human MAP kinases, the way the plant MAP kinase binds with SCREAM is different from how human MAP kinases bind with their 'client' proteins. This implies that plants may have evolved a unique motif to control a cell fate decision via the recruitment of the highly conserved signaling protein, the MAP kinases.

The atomic insight revealed in this study may be harnessed to design optimal stomatal development that allows plants to cope with the changing climate, and may also provide a tool for tweaking cellular



processes mediated by MAP kinases in animals (e.g. humans) for basic biomedical research.

More information: Aarthi Putarjunan et al, Bipartite anchoring of SCREAM enforces stomatal initiation by coupling MAP kinases to SPEECHLESS, *Nature Plants* (2019). DOI: 10.1038/s41477-019-0440-x

Provided by Nagoya University

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