How plants halt hair growth: A developmentally timed molecular pathway controls cell size in Arabidopsis

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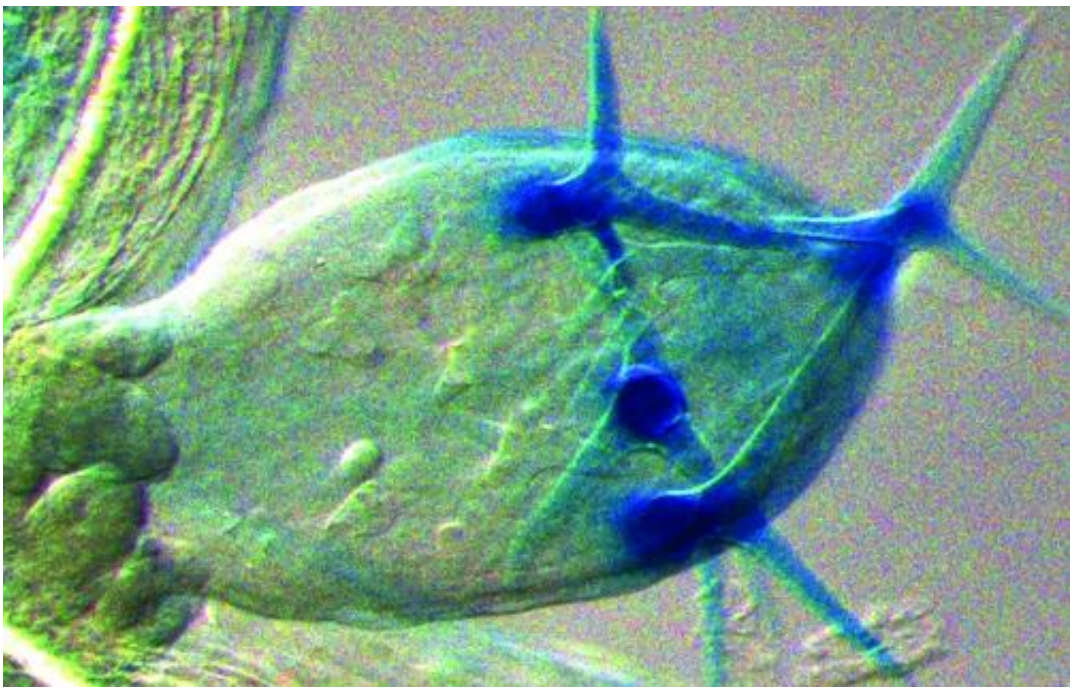

A developing Arabidopsis leaf showing regions of GTL1 activity. Although GTL1 activity is absent in leaf hairs at early stages of development (closer to the stem), it becomes strong in mature leaf hairs (stained blue). Credit: 2012 Keiko Sugimoto et al., RIKEN Plant Science Center

Normal development and function in multicellular organisms relies on tight control of cell growth, yet surprisingly little is known about how such control is achieved. Although some promoters of growth have been

identified, very few growth suppressors are known.

Keiko Sugimoto and colleagues from the RIKEN Plant Science Center have now uncovered new evidence that the endocycle—a growth mechanism particularly prominent in large cells such as neurons and hairs—is actively terminated through a developmentally timed pathway.

New cells in multicellular organisms are generated by mitosis—cell division preceded by the duplication of the cell's genetic material. Endocycling is a mechanism by which cells grow in size through the same genetic duplication, increasing their nuclear gene content or 'ploidy', without triggering cell division. Ploidy-dependent growth is in fact so prevalent, in animals as well as plants, that it is estimated to account for up to half of all biomass on Earth.

Sugimoto's team studied the gene GTL1 in the leaf hair cells of the model plant Arabidopsis. GTL1 is expressed only in fully grown hair cells and codes for the protein GTL1, a transcription factor, which modifies the action of other genes. Using a genome-wide survey, they identified around 3,900 genes bound by GTL1. Microarray analysis of thousands of genome fragments also located genes responsive to GTL1. Cross-referencing the two lists gave 182 potential GTL1 targets, of which two were already implicated in the control of cell growth. The team focused on one of these, CCS52A1, which is directly bound and repressed by GTL1. CCS52A1 is known to activate an enzyme complex known as APC/C, which is central to the endocycle.

The researchers demonstrated the importance of CCS52A1 using mutant plants defective in CCS52A1, which produced smaller leaf hairs with lower ploidy. Mutants with increased CCS52A1 activity, on the other hand, bore larger, higher-ploidy hairs. The results prove that GTL1 is both necessary and sufficient to stop the endocycle.

Sugimoto's study also demonstrates the importance of CCS52A1, APC/C and the endocycle itself in plant cell growth. In late leaf hair development, GTL1 down-regulates CCS52A1, reducing APC/C activity, halting the endocycle and stopping cell growth.

"The endocycle is often referred to as an aberrant form of mitosis," says Sugimoto. "We should instead start considering it an equally important alternative. Now we know that plants have a brake, we want to investigate how they use it in development and in response to environmental change."

More information: Breuer, C. et al. Transcriptional repression of the APC/C activator CCS52A1 promotes active termination of cell growth. *The EMBO Journal* 31,4488–4501 (2012). dx.doi.org/10.1038/emboj.2012.294

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