

UCSD biologists identify gene in plants that conserves water

October 8, 1998

HOLD FOR SCIENCE EMBARGO: 1 p.m. PDT, October 8, 1998

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UCSD BIOLOGISTS IDENTIFY GENE IN PLANTS THAT CONSERVES WATER

Biologists at the University of California, San Diego haven taken a step forward in creating plants that better withstand adverse conditions brought on by drought.

In an article in the October 9 issue of the journal *Science*, the biologists describe a process in which a gene plays a key role in opening and closing microscopic holes in the leaves of the plant *Arabidopsis*. After nearly two weeks of drought treatment in the study, control plants yellowed and wilted, while plants with a "knocked out" gene preserved water and subsequently remained green and healthy.

"It's too early to say whether we can call this 'drought tolerant' or not, but the data we have compiled suggest that this gene mutation definitely helps slow down desiccation of plants during drought," said UCSD biologist Julian Schroeder, who conducted the study with first author Zhen-Ming Pei, a UCSD post-doctoral researcher, and co-author Peter McCourt of the University of Toronto. "This information might be good for plants that experience transitory periods of drought, for example, during the summer when the next rain might be several weeks away."

Plants lose more than 90 percent of their water through leaf holes, called stomatal pores, by evaporation. A plant hormone called abscisic acid, or ABA, helps reduce this water loss by triggering closure of the stomatal pores during drought periods. Stomatal pores are surrounded by specialized cells called guard cells, which open and close the holes. The better a plant achieves stomatal closure during drought, the more water it conserves.

In their study, the biologists analyzed a mutant of *Arabidopsis* with a deleted gene called farnesyltransferase, a gene that helps transduce signals throughout the plant.

"The guard cells of the mutant were hypersensitive to the ABA hormone and therefore showed an enhanced ability for ABA to close the stomatal pores in leaves," said Schroeder. "In other words, the mechanism by which the stomatal pores close was potentiated when the (farnesyltransferase) gene was knocked out."

Previous research in Schroeder's laboratory had identified that "anion channels" in the guard cells are essential for closing stomatal pores. In the new study, Pei and Schroeder further showed an enhanced ability of the ABA hormone to activate anion channels in guard cells.

In the study, normal and mutated plants were grown and watered for approximately 21 days. Researchers then took normal and mutated plants that exhibited similar developmental characteristics and subjected both to drought treatment. To ensure that evaporation would take place virtually exclusively through the leaves, the researchers covered the soil and pots of the plants with aluminum foil.

After 12 days of drought treatment, normal plants showed severe wilting and chlorosis, a yellowing or blanching condition. The mutated plants, on the contrary, showed reduced water loss. They grew outward robustly and their leaves remained green.

Importantly, however, the researchers also observed that the mutants preserved their ability to open the stomatal pores under normal circumstances.

"Interestingly, when the plants are watered, the stomatal pores in the mutants open up well," added Schroeder. "That's important because plants have to take in carbon dioxide through stomatal pores, which is essential for plant growth."

Pei and Schroeder further found that in addition to deleting the farnesyltransferase gene, a second method of eliciting ABA hypersensitivity of stomatal closing could be produced by chemically inhibiting farnesyltransferases.

The mutated gene, labeled *eral* (Enhanced Response to ABA), was initially described in research analyzing seed germination by McCourt. In addition to water preservation, the biologists observed that the mutated plants grew differently and visually exhibited different characteristics than their unmutated counterparts. Schroeder believes that difference is due to the fact that the gene was knocked out of the entire plant. Future studies will investigate what happens when the gene is suppressed specifically in the guard cells that operate the opening and closing of the stomatal pores.

As a final point to the study, Schroeder's group analyzed crosses of the desiccation resistant mutant *eral* with two other known mutants: ABA Insensitive 1 and 2. These mutants have the opposite effect of *eral* by enhancing stomatal opening and causing plant wilting. The result of the crosses was that *eral* "won" and the plants recovered their normal response.

Research for this study was supported by grants from the National Science Foundation and the Department of Energy.

(October 8, 1998)