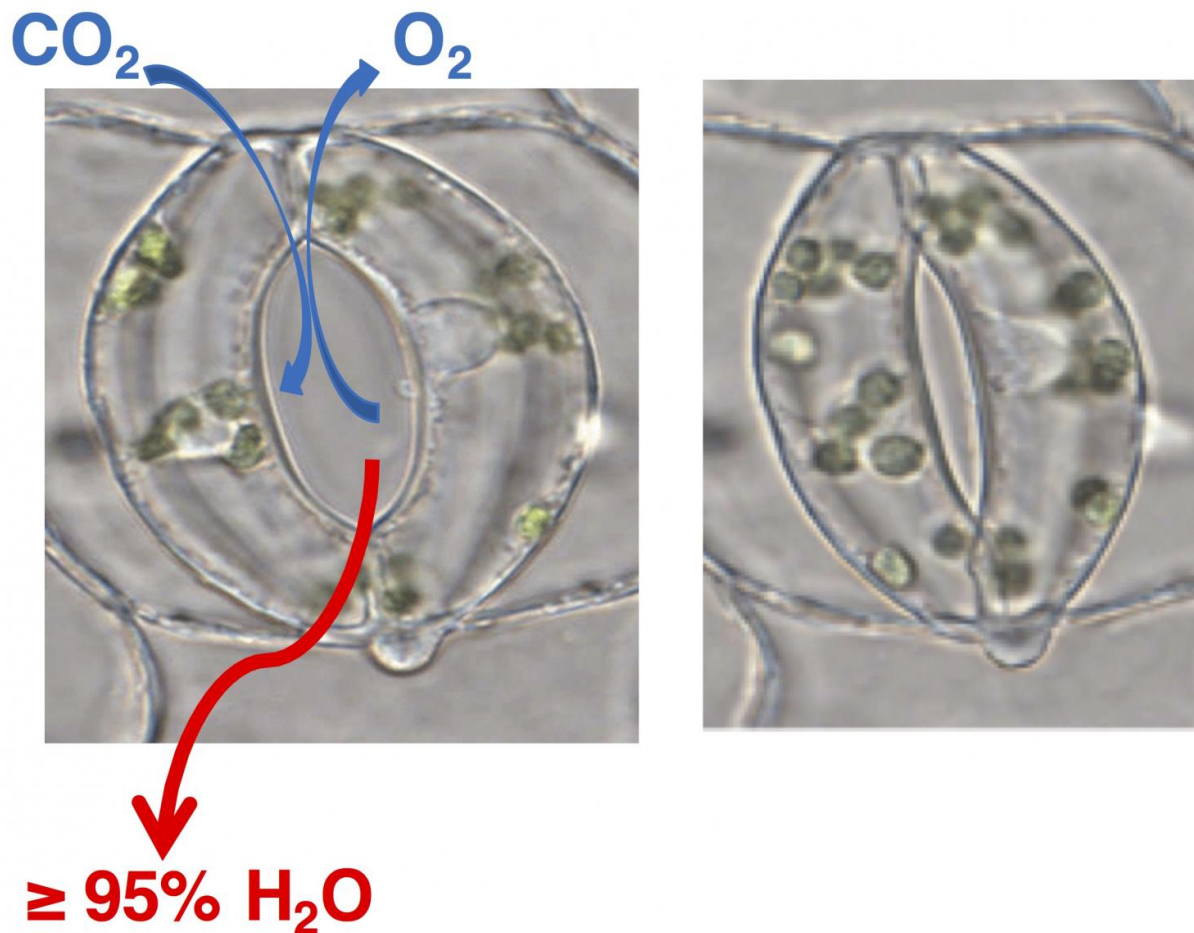


# An amino acid controls plants' breath

December 6 2016



Open (left) and closed (right) stomata found on the surface of plants' leaves, stems and other organs. Through the stomata, plants exchange oxygen and carbon dioxide, and lose more than 95 percent of their water as well. Therefore, correct regulation of stomata closure is important to adapt to drought conditions. Credit: IBS

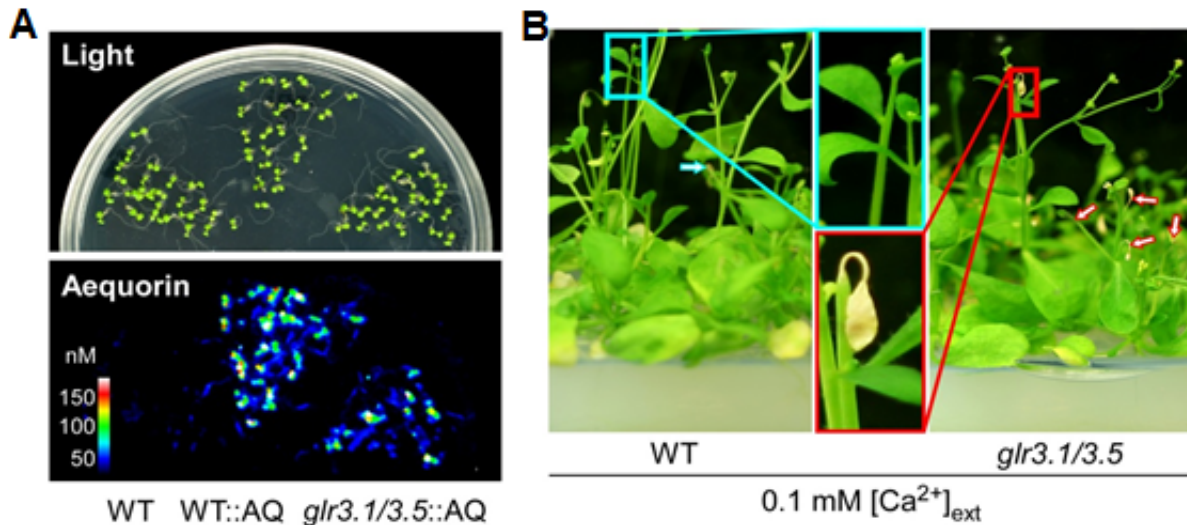
Plants breathe and "sweat" through stomata, microscopic pores found on leaves, stems and other plant organs. Through the stomata, plants take up carbon dioxide for photosynthesis and breathe out the products of this process, oxygen and water vapor. Group leader Dr. June M. KWAK and his colleagues at the Center for Plant Aging Research, within the Institute for Basic Science (IBS), identified a novel calcium channel and found out how it regulates stomatal closure. Published in *Cell Reports*, this discovery helps to clarify how plants maintain and adjust an adequate intracellular calcium level for their growth and breathing.

Calcium deficiency in humans can lead to weak hair, nails, osteoporosis and other serious symptoms, and [calcium](#) is essential for [plants](#) too. For example, blossom-end rot is a disorder found in tomatoes, watermelons, chili pepper, etc. caused by insufficient calcium in the soil. In plants, calcium contributes to several functions, including growth, differentiation and daily rhythms.

IBS scientists focused on the role of calcium in the opening and closing of stomata. They found that [calcium concentration](#) inside [guard cells](#), which surround stomata pores, is influenced by proteins called glutamate receptor homologs (GLRs), in particular GLR3.1 and GLR3.5. GLRs got their names because they are structurally similar to animal glutamate receptors, which are activated by the neurotransmitter glutamate and play a role in learning and memory. The team found that GLR3.1 and GLR3.5 form calcium channels in the plasma membrane of guard cells.

IBS researchers produced *Arabidopsis thaliana* plants lacking GLR3.1 and GLR3.5 proteins and found that when grown in a medium with a concentration of calcium 95% lower than the normal soil, these plants have yellow dying inflorescences, similar to the blossom-end rot disorder. "These tips of inflorescence have cells that are rapidly growing and dividing, so they need a lot of calcium. However, plant cells are quite selfish with calcium, and they do not share it with other cells if

they do not have enough," explains Kwak.



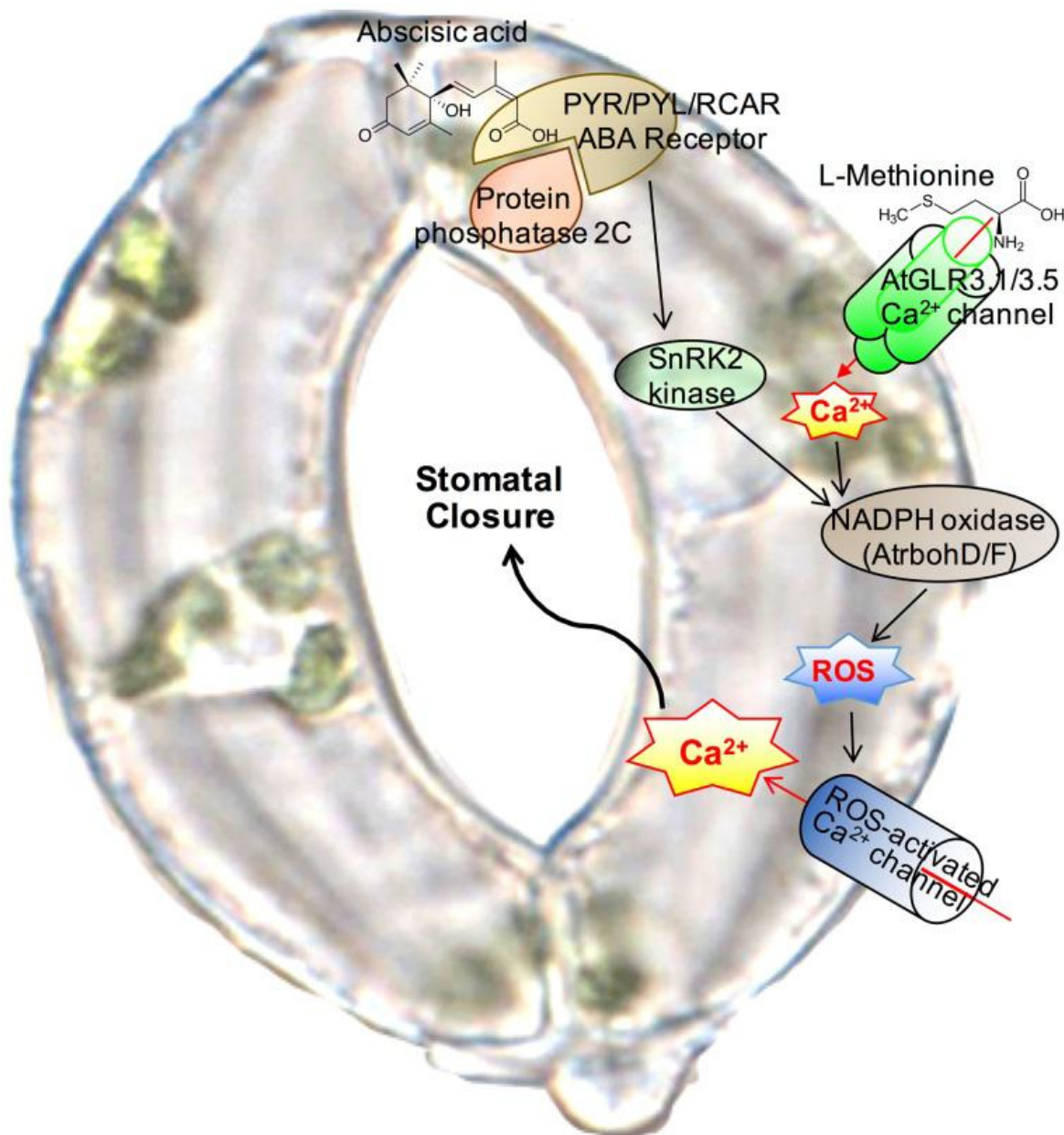
(left) Aequorin shows the level of intracellular calcium in plants. The plants lacking GLR3.1/GLR3.5 on the right of the dish have less calcium than the normal plants in the middle. (right) Mutant plants deficient in GLR3.1/GLR3.5 have dying flowers (yellow), which resembles the blossom-end rot disorder. Credit: IBS

Using aequorin, a protein that senses calcium and emits light of different color depending on the concentration of calcium, IBS scientists found that mutant plants deficient in GLR3.1/GLR3.5 have 25% lower concentrations of intracellular calcium. Then, they used electrophysiological techniques to verify that GLR3.1/GLR3.5 channels are activated by the amino acid L-methionine.

Results from a series of further experiments clarified that L-methionine opens the GLR 3.1/3.5 calcium channels and allows [calcium ions](#) to enter the cell. The elevation in intracellular calcium, in turn, activates

other membrane channels, like the reactive oxygen species (ROS)-activated [calcium channels](#), leading to stomatal closure.

"In the future, we would like to understand the source of L-methionine, which type of cells provide it for the guard cells and in response to what kind of stimuli," explains Kwak.



Stomatal closure involves the plant hormone abscisic acid, intracellular calcium and reactive oxygen species (ROS). IBS scientists discovered how L-methionine-activated calcium channels GLR3.1/3.5 (green) contribute to this mechanism.

Credit: IBS

**More information:** *Cell Reports*, [DOI: 10.1016/j.celrep.2016.11.015](https://doi.org/10.1016/j.celrep.2016.11.015)

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