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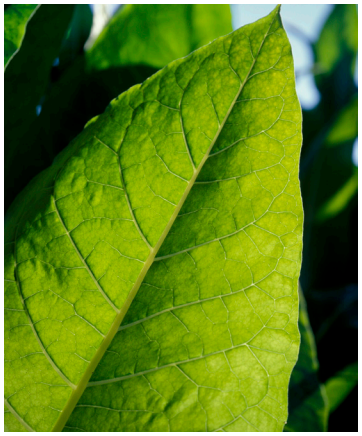
Identifying Mechanisms for Regulating Gas Exchange in Plants

Superfund Research Program (SRP) researchers at the University of California, San Diego (UCSD) are identifying important mechanisms that plants use to respond to changes in the environment. They recently discovered molecular mechanisms that enhance the activity of proteins essential to closing stomata, or pores found on the surface of leaves, in response to environmental stressors.



Stomata facilitate gas exchange between plants and the atmosphere, and control water loss during drought.

When high concentrations of carbon dioxide or ozone are present in the atmosphere, or during drought conditions, the stomata on the surface of leaves close to control the plant's water loss, ozone sensitivity, and carbon dioxide supply.

Revealing Mechanisms of Stomatal Closure

The new study, led by Julian Schroeder, Ph.D., shows that elevated levels of bicarbonate in guard cells, which is produced with an influx of carbon dioxide, can enhance the activity of the slow anion channel-associated 1 (SLAC1) plasma membrane protein. SLAC1 is essential for closure of stomata in response to high carbon dioxide concentration or stressors. The researchers also found that an enzyme that boosts production of bicarbonate also interacts with PIP2;1 aquaporin, which are small proteins that form pores in cell membranes.

Based on their findings, the researchers suggest that when carbon dioxide concentration in leaves is elevated, carbon dioxide influx across the plasma membrane is enhanced through PIP2;1 aquaporins. PIP2;1 aquaporins then interact with enzymes that enhance the production of bicarbonate, boosting the activity of SLAC1. The increase in SLAC1 activity then contributes to the closure of stomatal pores in response to elevated levels of carbon dioxide.

Identifying Other Pathways for Further Research

The researchers also found that enhancement of SLAC1 activity requires the presence of specific protein kinases, which are enzymes that modify other proteins and are key regulators of cell function. Although they found that bicarbonate increases the activity of SLAC1, significant SLAC1 activity still occurs without bicarbonate, suggesting that a second pathway activates protein kinases to increase SLAC1.

The study also provided insight on how carbon dioxide and water move through plants. Carbon dioxide and water are transported across membranes in plant cells via aquaporins. However, it isn't known whether water and carbon dioxide are transported by the same channel pores or whether there are different mechanisms. Because a mutation in the PIP2;1 aquaporin disrupted both water and carbon dioxide transport, the researchers propose that both are transported through common pores, but more research is needed to confirm this finding. This research is identifying mechanisms by which plants can use water more efficiently, which is of relevance when using plants to remove toxicants from soils in regions of limited water availability.

Investigating Environmental Toxicant Accumulation in Plants

The UCSD SRP work to identify molecular and biological systems in plant cells has also led to the parallel discoveries of genes encoding the central heavy metal detoxification enzymes in plants. Based on this ongoing work, UCSD SRP researchers are determining the molecular mechanisms by which plants accumulate toxicants. These advances provide key tools for avoiding toxic heavy metal and arsenic accumulation in edible plant tissues and can contribute to engineering plants for environmental remediation. By understanding how plants respond to different environmental

stressors, Schroeder and his research team are providing important information about how plants grow and upload nutrients and toxicants in the environment.

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To learn more about this research, please refer to the following source:

Wang C, Hu H, Qin X, Zeise B, Xu D, Rappel WJ, Boron WF, Schroeder JI. 2016. Reconstitution of CO2 regulation of SLAC1 anion channel and function of CO2-permeable PIP2;1 aquaporin as CARBONIC ANHYDRASE4 interactor. Plant Cell 28(2):568-582. doi: 10.1105/tpc.15.00637 PMID: 26764375 PMCID: PMC4790870