

Unlocking the secrets of a plant's light sensitivity

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(PhysOrg.com) -- Plants are very sensitive to light conditions because light is their source of energy and also a signal that activates the special photoreceptors that regulate growth, metabolism, and physiological development. Scientists believe that these light signals control plant growth and development by activating or inhibiting plant hormones. New research from Carnegie plant biologists has altered the prevailing theory on how light signals and hormones interact. Their findings could have implications for food crop production.

It was previously known that a <u>plant hormone</u> called brassinosteroid is essential for plant's responses to <u>light</u> signals. This crucial steroid-type hormone is found throughout the plant kingdom and regulates many



aspects of growth and development. Surprising new research from a team led by Carnegie plant biologist Zhi-Yong Wang shows that light does not control the level of brassinosteroid found in plants as was expected. Instead brassinosteroid dictates the light-sensitivity of the plant. It does this by controlling the production of a key light-responsive protein.

The team's findings on interactions between brassinosteroid and light in sprouting seedlings have changed the prevailing model for understanding the relationship between light conditions and hormone signals in regulating photosynthesis and growth. Their results are published in *Developmental Cell* on December 14.

While under the soil's surface, in the dark, plant seedlings grow in a special way that speeds the process of pushing the budding stem out into the air, while simultaneously protecting it from damage. This type of growth is called skotomorphogenesis. Once exposed to light, seedlings switch to a different, more regular, type of growth, called photomorphogenesis, during which the lengthening of the stem is inhibited and the leaves expand and turn green.

Many components are involved in this developmental switch, including brassinosteroid. Previous studies showed that mutant plants created to be deficient in brassinosteroid grew as if they were in the light, even when in the dark. Research also showed that many genes responded to stimulation from light and brassinosteroid in opposite ways. But scientists were unsure how this antagonistic process worked, especially after they found the levels of brassinosteroid in plant cells were not significantly different between plants grown in the dark or in the light.

The Carnegie team's new research identifies a protein called GATA2 as a missing link in this communications system. This protein tells developing seedlings which type of growth to pursue.



GATA2 is part of the GATA factor class of proteins, which are found in plants, fungi and many animals. GATA factors promote the construction of a variety of new proteins, the recipes for which are encoded in DNA. It does this by switching on and off different genes. In Arabidopsis, the experimental mustard plant used in this study, there are 29 genes for different members of the GATA factor family. Some of these have been demonstrated to play a role in flower development, the metabolism of carbon and nitrogen, and the production of the green pigment chlorophyll.

Wang's team found that GATA2 switches on many genes that are turned on by light but turned off by brassinosteroid. It then showed that brassinosteroid inhibits the production of GATA2 and light stabilizes the presence of GATA2 protein in plant cells.

First, the team showed that GATA2 functions to turn on select plant growth genes in the presence of light. The scientists genetically manipulated Arabidopsis plants to cause the GATA2 protein to be overproduced. As a result, the plants started to show patterns of growing in light, even when they were in the dark. This manipulation demonstrates that GATA2 is a major promoter of light-type growth.

What's more, this is the same reaction that was produced when plants were genetically manipulated to be brassinosteroid-deficient. This means that the over abundance of GATA2 had the same result as the scarcity of brassinosteroids. These results show that GATA2 proteins and brassinosteroid hormones have antagonistic effects on developing plants.

Next, the Carnegie team showed that brassinosteroid is actually involved in inhibiting the actions of GATA2. Brassinosteroids turn on a protein that prevents GATA2 from working when the seedling is in the dark. This inhibition of GATA2 is stopped by exposure to light. This likely happens due to the involvement of yet another protein—one that is



widely involved in light-signaling— although further study is needed to be sure.

Together all these results show that GATA2 is an important factor in signaling light-type growth. It also serves as a communications junction between internal plant systems that are turned on by light and those that are turned on by brassinosteroids.

"Brassinosteroids and light antagonistically regulate the level of GATA2 activity, and thus the creation of proteins stimulated by GATA2," says Wang. "As a result, GATA2 represents a key junction of crosstalk between brassinosteroid and light signaling pathways."

The framework created by this research leaves plenty of avenues for further study of the various components of light signaling in <u>plants</u>. Some other members of the GATA class of proteins may be involved, as well as other light-responsive compounds.

Provided by Carnegie Institution

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