

# Safeguarding chloroplasts from sunburn

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Intense sunlight damages the chloroplasts that are essential for photosynthesis, and generates toxic products that can lead to cell death. LMU biologists have now identified a signaling pathway which mitigates the effects of light stress.

Biological photosynthesis—the process by which solar radiation is converted into chemical energy—is carried out by cyanobacteria, algae and [plants](#), and is vital for the maintenance of animal life on Earth. In plants, photosynthesis takes place in specialized organelles called chloroplasts, which are found in the leaves. Chloroplasts actually evolved from photosynthetic bacteria, which were incorporated into nucleated [cells](#) as symbionts. Over the course of evolution, most of the genes they brought with them were transferred to the nucleus of the host, but modern-day chloroplasts still retain a small set of genes that are essential for photosynthesis. As a result, photosynthesis in plants requires an extensive exchange of information between the nucleus and chloroplasts. The network involved is very complex, and many of its components remain to be identified. Using thale cress (*Arabidopsis thaliana*) as a model system, LMU biologists led by Privatdozentin Dr. Tatjana Kleine have now characterized a previously unknown [signaling pathway](#) that makes a significant contribution to stress tolerance and suppresses light-induced programmed cell death. This pathway could potentially be exploited to improve stress resistance in crop plants. The study appears in the journal PNAS.

Exposure of plants to excessively high light levels inevitably generate what '[reactive oxygen species](#)' (ROS), such as '[singlet oxygen](#)' ( $^1\text{O}_2$ ) in leaf chloroplasts. In high concentrations ROS are toxic to cells, but they also serve as alarm signals that activate countermeasures. "Previous studies had shown that [singlet oxygen](#) serves as a signal molecule, and triggers a stress response that can lead to a cessation of growth or even to programmed cell death," Kleine says. "The obvious next step was to identify the proteins involved in the  $^1\text{O}_2$ -induced signaling relay."

In their experiments, Kleine and colleagues made use of a double mutant (*flu ex1*) of *Arabidopsis thaliana*. The *flu* mutation permits the production of singlet oxygen to be activated in a controlled manner by exposure to light, while the *ex1* mutation inactivates a gene called

EXECUTER1. The EXECUTER1 protein is known to be required to induce programmed cell death. Hence, the ex1 mutation allows the concentration of singlet oxygen to rise without triggering cell death.

To identify other genes involved in  $^1\text{O}_2$ -dependent signaling, the team mutated the flu ex1 strain once more, and screened for mutants in which the cell-death response to  $^1\text{O}_2$  was restored. This [experimental design](#) revealed that a protein called SAFEGUARD1 protects the double mutant from the deleterious consequences of the build-up of singlet oxygen. "Inactivation of the SAFE1 gene in the double mutant again results in cell death. Crucially, this is not due to the 'repair' of EXECUTER1. Therefore, the SAFE1 gene must code for a component of an independent and previously unknown signaling pathway, which is itself induced by singlet oxygen," explains Liangsheng Wang, lead author of the new paper. Notably, loss of SAFE1 is associated with damage to the margins of the membrane stacks (known as 'grana') in which the light-driven reactions occur. The authors therefore propose that  $^1\text{O}_2$  produced at the grana margins activates an EXECUTER1-independent signaling relay, and that SAFE1 normally suppresses this pathway by protecting the grana margins from damage.

Of all the types of ROS generated by exposure to high light levels, singlet oxygen is the most deleterious to photosynthesis. As an effective inhibitor of the stress reaction induced by the build-up of singlet oxygen, SAFE1 represents a promising starting point for efforts to enhance stress resistance in crop plants. "Perhaps overexpression of the protein would make plants more tolerant to the levels of singlet oxygen that are produced under intense sunlight," says Kleine.

**More information:** Liangsheng Wang et al. The Arabidopsis SAFEGUARD1 suppresses singlet oxygen-induced stress responses by protecting grana margins, *Proceedings of the National Academy of Sciences* (2020). [DOI: 10.1073/pnas.1918640117](https://doi.org/10.1073/pnas.1918640117)

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