

## Plant Biology **More Light, Less Disease**

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When plants are exposed to excess excitation energy (EEE), which occurs when the amount of energy exceeds that used for photosynthesis and is often caused by excess light, plants dissipate the energy as heat or by transferring electrons to oxygen, for example through photorespiration, which results in the production of reactive oxygen species (ROS). EEE also triggers programmed cell death, which is part of the acclimation response that occurs both in the leaves exposed to EEE directly and in unexposed leaves, in which the process is called systemic acquired acclimation (SAA). Mühlenbock *et al.* found that exposure of *Arabidopsis* to EEE, in the form of white light or 680-nm wavelength light, but not 700-nm wavelength light, triggered programmed cell death. Concomitant exposure to ethylene, a gaseous plant hormone, increased the area affected, and plants in which ethylene signaling was compromised had a very limited area of cell death. Chemicals that oxidize the plastoquinone pool inhibited the production of ethylene and peroxide and prevented the decrease in stomatal conductance that was associated with EEE. Chemicals that reduce the plastoquinone pool (increase the ratio of PQH<sub>2</sub> to PQ) triggered the production of ethylene and peroxide and stomatal closure even under low light conditions. Thus, the redox status of this electron carrier in photosynthesis appears to play a critical role in initiating EEE responses. Gene profiling experiments comparing genes regulated in leaves directly exposed to EEE (local response) and genes regulated in leaves undergoing SAA suggested that many of the genes regulated by EEE were also involved in plant defense responses. Indeed, exposing leaves to the plastoquinone-reducing agent or EEE limited growth of the virulent pathogen *Pseudomonas syringae*. *LESION SIMULATING DISEASE1 (Lsd1)* has been implicated in regulating the EEE acclimation response, and plants in which this gene is mutated exhibit excessive cell death under many conditions. Mühlenbock *et al.* show that, compared with wild-type plants, ethylene and peroxide production in *Lsd1* mutant plants is elevated to greater levels when the plants are exposed to the plastoquinone-reducing agent. This increase in ethylene and peroxide is blunted if the *Lsd1* mutation is combined with mutations in *PHYTOALEXIN DEFICIENT4 (pad4)* or *ENHANCED DISEASE SUSCEPTIBILITY1 (eds1)*. Furthermore, the runaway cell death associated with *Lsd1* mutant plants was decreased in plants with a mutation in *ein2*, which encodes an ethylene receptor. Thus, LSD1 appears to serve as a negative regulator of the EEE response upstream of PAD4 and EDS1, which then stimulate ethylene production that combines with ROS to trigger cell death and acclimation responses.

P. Mühlenbock, M. Szechyńska-Hebda, M. Płaszczycza, M. Baudo, P. M. Mullineaux, J. E. Parker, B. Karpińska, S. Karpiński, Chloroplast signaling and *LESION SIMULATING DISEASE1* regulate crosstalk between light acclimation and immunity in *Arabidopsis*. *Plant Cell* preview, published 12 September 2008 as 10.1105/tpc.108.059618. [[PubMed](#)]

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