

**P007** A central role for S-nitrosothiols in plant disease resistance  
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The emerging evidence suggests that nitric oxide (NO), which acts as a signal in the immune, nervous and vascular systems of animals, also has pivotal functions in plant biology. During the establishment of plant disease resistance, for example, NO has been shown to reprogramme plant transcription and in combination with reactive oxygen intermediates, to potentiate hypersensitive cell death. In animals, a variety of mechanisms are thought to transduce NO signals, in addition to production of the key second messenger, cGMP. Among these is protein S-nitrosylation, a prototypic redox-based post-translational modification.

While three distinct plant enzymes have been uncovered that generate NO, little is currently known regarding NO turnover. We have identified gain- and loss-of-function mutations in a plant S-nitrosoglutathione (GSNO) reductase. While GSNO is the only substrate S-nitrosothiol (SNO) recognised by this enzyme, loss-of-function mutations resulted in greater increases in SNO-proteins than in GSNO itself. While elevated levels of SNOs accelerated the kinetics of hypersensitive cell death, cued by avirulent pathogens, decreased concentrations of SNOs delayed this process. Paradoxically, increased SNO levels strikingly compromised basal resistance against virulent pathogens. In contrast, decreased SNO concentrations resulted in the establishment of resistance against such pathogens. Our results argue for a central role of SNOs in plant disease resistance.