

Meeting abstract

## Nitric oxide in cell damage and protection

Steven Neill\*, Jo Bright, Radhika Desikan, Judy Harrison, Tanja Schleicher and John Hancock

Address: Centre for Research in Plant Science, Genomics Research Institute, University of the West of England, Bristol, Coldharbour Lane, Bristol BS16 1QY, UK

Email: Steven Neill\* - Steven.Neill@uwe.ac.uk

\* Corresponding author

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Less than ten years ago, the impact on plant biology of the gaseous free radical gas nitric oxide (NO) related only to its toxic effects as a component of NO<sub>x</sub> released into the atmosphere as an air pollutant during the combustion of fossil fuels. It is now clear that NO is a multi-faceted and versatile endogenous signalling molecule with an importance in many if not all aspects of plant growth and development. At least three enzymatic sources of NO in plants have been characterised and mechanisms that serve to scavenge NO have also been identified. Downstream signalling responses to NO include generation and action of the second messenger molecules calcium, cyclic GMP and cyclic ADPR, protein phosphorylation, protein nitrosylation and specific effects on gene expression. NO also interacts directly with Reactive Oxygen Species (ROS) and with components of ROS-activated signalling pathways. NO and ROS play key roles in an orchestra of plant defence responses. Rapid generation of NO and ROS following pathogen or elicitor challenge mediates a multitude of metabolic and transcriptional alterations including Programmed Cell Death (PCD). However, it is important to note that in some cases the actions of NO can be cytoprotective rather than toxic, potentially via antioxidant effects of NO. Furthermore prevention of NO synthesis or action can delay or inhibit PCD. In addition to the roles of NO and ROS in biotic stress responses, NO and ROS generation also occurs in response to various abiotic stresses, including UV radiation which itself can induce PCD. Recent data suggest that NO mediates some UV responses and that UV radiation can also stimulate the release of NO<sub>x</sub> from leaves. Key research questions to be addressed must be directed to the effects of UV on NO generation and action in plants. Research programmes will require methods to assess accurately NO emissions from leaves

and other organs and to determine NO concentrations in cells and sub-cellular microdomains; the use of mutants and transgenic plants altered in NO synthetic and scavenging capacities; analyses of the molecular and biochemical events required for activation of PCD by NO and UV; and the development of techniques to monitor simultaneously cell death, NO and ROS generation in the field during exposure to UV.