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Meeting abstract

Signals from reactive oxygen species

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Reactive oxygen species (ROS) can arise from normal metabolic activity such as organelle-based electron transport or be intermediates in signal transduction pathways activated by plant respiratory burst oxidase homologue (*Rboh*). UV-B exposure induces a pathogenesis-like response in leaves that can be abrogated by ROS scavengers [1]. A local signal is propagated by hydrogen peroxide and is sensitive to the application of catalase [2]. Similarly, local ROS production initiated by elicitors or pathogens can arise from stimulation of superoxide producing Rboh activity [3,4]. In this case as well, propagation of a ROS signal to adjoining cells is sensitive to catalase. Antisense of Rboh homologues in tomato lead to reduced ROS production [5]. The tomato plants show compromised wound-dependent responses. In addition, the antisense plants have a highly branched phenotype and fasciated-like reproductive organs. Transcriptome analysis of these plants revealed ectopic expression of homeotic MADS box genes that are normally expressed only in the reproductive organs. In addition, various applications of hormones were found to regulate Rboh levels. Thus, regulated ROS bursts and the general effect of Rboh activity on the steady state cellular redox milieu control short term physiological reactions and plant development. Divergent stress including temperature, drought and UV-B exposure yield overlapping transcriptome response profiles whose origin can be traced to the use of reactive oxygen signaling intermediates. Cellular scavenging systems and local production of NO are likely to temper these signalling properties by interacting with ROS and thus help to contribute to the specificity of particular responses.

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