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The role of reactive oxygen species in plant immunity and susceptibility

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Reactive Oxygen Species (ROS) and especially hydrogen peroxide (H_2O_2) are formed during normal cell metabolism in plants. High levels of ROS that cause oxidative damage are commonly linked with opposing environmental conditions (Mittler, 2002; Noctor et al., 2002). The progression of aerobic metabolic processes for instance respiration and photosynthesis, lead to the continuous production of reactive oxygen species (ROS) in mitochondria, chloroplasts and peroxisomes, endoplasmic reticulum and in the cytosol (Gill and Tuteja, 2010). Therefore, ground state oxygen is changed to different ROS either by energy or electron transfer reactions. The former leads to the creation of singlet oxygen (O_2), whereas the latter results in the serial reduction to superoxide anion radical (O_2•−), hydrogen peroxide (H_2O_2) and hydroxyl radicals (OH•) (Foyer and Noctor, 2000). Different ROS have a common feature which is their ability to react with wide range of biomolecules such as lipids, proteins, and nucleic acids that are necessary for cells activity and integrity. ROS is scavenging different antioxidant defence mechanisms under unstable conditions. The balance between ROS production and scavenging may be disturbed by different abiotic and biotic stress conditions, leading to a quick and transitory increase of the intracellular level of ROS (Gill and Tuteja, 2010).

ROS is known as signalling and regulatory molecules rather than harmful products of metabolic imbalance (Apel and Hirt, 2004; Mittler et al., 2004; Pastori and Foyer, 2002). Also ROS is controlling the regulation of defence responses and cell death (Alvarez et al., 1998; Zhang et al., 2003), stomatal aperture (Kwak et al., 2003; McAinsh et al., 1996; Murata et al., 2001; Pei et al., 2000), cell expansion and polar growth (Coelho et al., 2002; Foreman et al., 2003; Liszkay et al., 2004; Rodrıguez et al., 2002, 2004; Schopfer et al., 2002) and leaf and flower development (Sagi et al., 2004). In addition, ROS produced in response to biotic and abiotic stresses regulate signal change and gene expression (Baxter-Burrell et al., 2002; Desikan et al., 2001; Mittler et al., 2004; Pastori and Foyer, 2002; Shin and Schachtman, 2004; Shin et al., 2005). Accumulation of ROS occurs in different cells in response to pathogen attack (Trujilo et al., 2004). In addition to the reverse effects in a single cell type, for example, hydrogen peroxide inhibits hair growth of roots while hydroxyl radical stimulates root hair growth (Jones et al., 1998; Foreman et al., 2003).

The mechanism mediating such distinct responses rely in part on the complement of enzymes for productions and scavenging of ROS in a given cell or organelle (Mittler et al., 2004) plus the proteins and lipids lying upstream or downstream of the ROS, for example phospholipase D and phosphatidic acid (Zhang et al., 2003), ROP GTPases (Baxter-Burrell et al., 2002) and MAP kinases (Kovtun et al., 2000; Rentel et al., 2004). In the control of stomata opening, cell expansion and polar growth, plasma membrane (PM) Ca^{2+} channels appear to be the downstream of ROS production (Coelho et al., 2002; Foreman et al., 2003; Kwak et al., 2003; Murata et al., 2001; Pei et al., 2000). The resultant elevation of cytosolic Ca^{2+} could act as a second messenger or regulator of exocytosis and the cytoskeleton. ROS activation of Ca^{2+} channels probably forms the basis of a regulatory network in which specificity of 'output' is determined by the input combination of an individual ROS (superoxide anion, hydroxyl radical or H_2O_2) and a target Ca^{2+} channel in any given cell type. This would permit cell specificity and spatio-temporal heterogeneity in ROS/Ca^{2+} mediated signalling reactions.