Chapter 15

Integration of Signaling in Antioxidant Defenses

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Summary

In the last few years, it has become apparent that reactive oxygen species (ROS) have important roles as signaling intermediaries in a large number of cellular processes, especially in relation to plants' interactions with their environment. A complex network of low molecular weight antioxidants, ROS scavenging enzymes, and enzymes that maintain antioxidant pools are required to control the levels of ROS in all subcellular compartments. The coordinated regulation of this network by ROS themselves and stress-associated hormones such as salicylic acid, abscisic acid, and jasmonic acid reveals that antioxidant metabolism is central to considerations of how signaling networks are regulated. Furthermore, it is becoming apparent that key antioxidants such as glutathione and ascorbate are involved in the regulation of stress hormone-directed signaling pathways without any interaction with ROS. Therefore ROS and antioxidants may be key points at which the coordination of different signaling pathways is achieved. These issues are considered in this chapter.

I. Introduction

Plant growth, development, and reproduction all require the continual involvement of reactive oxygen species (ROS; Pennel and Lamb, 1998). ROS is a collective term for the reduction intermediates or electronically excited forms of chemically relatively unreactive triplet state molecular oxygen ($^3O_2$). These are described in more detail in the chapters by Foyer et al. and Endo and Asada (this volume), but include both stable compounds (such as hydrogen peroxide [H_2O_2] and fatty acid hydroperoxides), free radicals of differing stabilities and reactivity (e.g. superoxide anion, the hydroxyl radical, lipid peroxy radicals), and singlet oxygen ($^1O_2$), a more reactive state of O_2. There is an extensive literature on how these various ROS can cause oxidative stress in plant cells, as in all aerobes, by the oxidation of cellular macromolecules and structures. However, it is now recognized that ROS in plants perform essential functions in diverse processes such as growth and development, acclimation to the environment, and establishing resistance to pathogens. The positive roles for ROS can be divided into either direct involvement in metabolic reactions or engagement in signaling mechanisms.

The involvement of ROS in metabolism is well established. Good examples are the numerous oxidases or peroxidases that are important in the interconversions of phenolic compounds, (poly)amines, ascorbate, and oxalate (Hiraga et al., 2001; Jansen et al., 2001; Sebala et al., 2001). Oxidative cross-linking of cells walls during normal growth and when challenged with disease agents is an important process in the life of plants (Bradley et al., 1992; Pennel and Lamb, 1998). However, it is the involvement of ROS as important components of signaling pathways that has attracted considerable attention in recent years. This is especially the case where such changes are associated with or initiate programmed cell death (PCD) and/or where other signaling molecules or phytohormones are involved.

This chapter focuses on those molecules and their associated signaling pathways that are affected by or influence ROS levels and antioxidant metabolism.

Abbreviations: ABA – abscisic acid; *ABI* – ABA insensitive gene (*ABI1*, *ABI2*, etc); APX – ascorbate peroxidase isoform (APX1, APX2, etc); *APX* – ascorbate peroxidase gene (*APX1*, *APX2*, etc); CAT1 – catalase, peroxisomal isoform; *CAT* – catalase gene (*CAT1*, *CAT2*, *CAT3*, etc); CBF1 – CRT/DRE binding factor 1; CHS – chalcone synthase; *CHS* – chalcone synthase gene; DHAR – dehydroascorbate reductase; γ-ECS – gamma-glutamylcysteine synthetase; *EDS1* – enhanced disease susceptibility 1 gene; *ELIP2* – early light induced protein 2 gene; ETR1 – ethylene resistant 1 protein; GR – glutathione reductase; GSH – reduced glutathione; GSSG – oxidised glutathione (glutathione disulphide); GST – glutathione-S-transferase (GST1, GST2, etc); *GST* – glutathione-S-transferase gene (*GST1*, *GST2*, etc); HR – hypersensitive response; JA – jasmonic acid; LSD1 – lesion simulating disease 1 protein; *LSD1* – lesion simulating disease 1 gene; *lsd1* – mutant form of *LSD1*; MAPK – mitogen-activated protein kinase; MDHR – monodehydroascorbate reductase; NADPH – nicotinamide adenine dinucleotide phosphate, reduced form; NPR1 – non-expressor of PR-1 1 protein; NO – nitric oxide; $^1O_2$ – singlet oxygen, reactive form of molecular oxygen (O_2); $^3O_2$ – triplet molecular oxygen, which is the form normally referred to as O_2; *PAD4* – phytoalexin deficient 4 gene; PAL – phenyl(alanine)ammonia lyase; *PAL* – phenyl(alanine)ammonia lyase gene; PCD – programmed cell death; PP2C – protein phosphatase 2C; *PR-1* – pathogenesis related protein-1 gene; RCD1 – runaway cell death protein (wild type); *RCD1* – runaway cell death gene (wild type); *rcd1* – mutant form of *RCD1*; RNS – reactive nitrogen species; ROS – reactive oxygen species; RuBisCo – ribulose bisphosphate carboxylase/oxygenase; SA – salicylic acid; SAR – systemic acquired resistance; SOD – superoxide dismutase; *SOD* – superoxide dismutase gene (*SOD1*, *SOD2*, etc); TGA-B-Zip – TGA class basic leucine zipper transcription factor; *vtc1* – vitamin C deficient 1 mutant;