CURRENT PERSPECTIVE ESSAY

Redox Homeostasis and Antioxidant Signaling: A Metabolic Interface between Stress Perception and Physiological Responses

Low molecular weight antioxidants, such as ascorbate, glutathione, and tocopherol, are information-rich redox buffers that interact with numerous cellular components. In addition to crucial roles in defense and as enzyme cofactors, cellular antioxidants influence plant growth and development by modulating processes from mitosis and cell elongation to senescence and death (De Pinto and De Gara, 2004; Potters et al., 2004; Tokunaga et al., 2005). Most importantly, antioxidants provide essential information on cellular redox state, and they influence gene expression associated with biotic and abiotic stress responses to maximize defense. Growing evidence suggests a model for redox homeostasis in which the reactive oxygen species (ROS)–antioxidant interaction acts as a metabolic interface for signals derived from metabolism and from the environment. This interface modulates the appropriate induction of acclimation processes or, alternatively, execution of cell death programs.

THE CONCEPT OF REDOX HOMEOSTASIS IN PLANTS

Efficient flux through plant electron transport cascades requires the simultaneous presence of both oxidized and reduced forms of electron carriers. This requirement, known as redox poising, involves a continuous flux of electrons to molecular oxygen from multiple sites in the photosynthetic and respiratory electron transport chains. Apart from the specialized water-producing reactions catalyzed by specific oxidases, the initial product of this flux is superoxide, from which other ROS are subsequently produced (Table 1). Singlet oxygen is also formed during light capture and photochemistry. Numerous enzyme systems produce superoxide or $\text{H}_2\text{O}_2$.

The reactive nature of these intermediates means not only that their accumulation must be controlled but also that they are able to act as signaling molecules. The extent to which ROS accumulate is determined by the antioxidative system, which enables organisms to maintain proteins and other cellular components in an active state for metabolism (Figure 1). Like all other aerobic organisms, plants maintain most cytoplasmic thiols in the reduced ($\text{–SH}$) state because of the low thiol-disulfide redox potential imposed by millimolar amounts of the thiol buffer, glutathione. Unlike many animal cells, however, plant cells synthesize high concentrations of ascorbate (vitamin C), an additional hydrophilic redox buffer that provides robust protection against oxidative challenge. Redox homeostasis is governed by the presence of large pools of these antioxidants that absorb and buffer reductants and oxidants (Figure 1). Plants also make tocopherols (vitamin E) that act as important liposoluble redox buffers. Although tocopherol is considered to be a major singlet oxygen scavenger, it is also an effective scavenger of other ROS, and, in this case the reduced scavenging form may be regenerated by ascorbate (Foyer et al., 2005). Moreover, because the tocopherol redox couple has a more positive midpoint potential than that of the ascorbate pool, it increases even further the range of effective superoxide scavenging. The ability of the ascorbate, glutathione, and tocopherol pools to act as redox buffers in plant cells is one of their most important attributes.

Pathways of ROS signaling are made possible by homeostatic regulation achieved by antioxidant redox buffering. Because antioxidants continuously process ROS, they determine the lifetime and the specificity of the ROS signal. Plant cells generally cope very well with high rates of generation of superoxide, $\text{H}_2\text{O}_2$, and even singlet oxygen. Although cellular oxidation is important in all abiotic and biotic stress responses, the extent and physiological significance of oxidative damage is debatable. For example, plants with low activities of both catalase and cytosolic ascorbate peroxidase show less severe stress symptoms than plants that lack either one of these enzymes (Rizhsky et al., 2002). It now appears that singlet oxygen-mediated cell death is not a direct result of damage per se but rather is genetically programmed via the EXECUTOR1 pathway (Wagner et al., 2004). Moreover, plants adapt very well to depletion of antioxidants by signaled induction of other defense systems. For example, tocopherol-deficient Arabidopsis vte mutant seedlings have high amounts of lipid peroxides, but the mature plants display only a slightly abnormal phenotype (Kanwischer et al., 2005). One presumes that rapid protein turnover or DNA repair is increased to compensate for increased oxidation or loss of antioxidants in these circumstances.

REACTIVE OXYGEN-ANTIOXIDANT INTERACTIONS IN REDOX SIGNALING

The last 5 years have seen a radical change in the appreciation of the pivotal importance of antioxidant status. In addition to its roles in removing ROS, antioxidant status appears to set the threshold for general plant defense responses, particularly those provoked by biotic stresses and wounding. Indeed, modulation of the ROS-antioxidant interaction plays a part in many stresses, as well as other responses to the environment, and in the regulation of plant development. Symbiotic associations between organisms also involve ROS-antioxidant interactions, and this can lead to an enhancement of antioxidant status such that the
The ultimate electron acceptor in the apoplast is either oxygen or 3,4-dihydroxyphenolic compounds such as chlorogenic acid, caffeic acid, quercetin, and catechin that influence cell wall composition. These reactions, acting together with ascorbate oxidase, are part of a futile cycle that regulates production of both reduced ascorbate and oxidized forms (monodehydroascorbate and dehydroascorbate) in the apoplastic environment for functions in metabolism and growth. Such a cycle may be crucial in controlling cell expansion and in facilitating ROS-mediated signal transmission that occurs in response to atmospheric pollutants, pathogens, or hormones (Pignocchi et al., 2003). It is intriguing that another, as yet uncharacterized, apoplastic protein, encoded by VTC2, is involved in ascorbate accumulation in leaves.

Our current knowledge of redox controls in the apoplast and cytoplasm predicts that (1) the plasma membrane is an important site for perception and transduction of environmental change through redox signals; (2) apoplastic redox changes facilitate interactions between receptor proteins containing oxidizable thiols that are sited in or near the membrane surface; (3) because the apoplastic redox buffering capacity is low, a steep redox gradient is present across the plasma membrane; (4) membrane electron transport could be important in driving plasma membrane and tonoplast electron transport chains. Both membranes contain an apoplastic-dependent cytochrome b561, which is reduced on one face by ascorbate and oxidized on the other by monodehydroascorbate or phenolics that can act as substrates for monodehydroascorbate reductase (Preger et al., 2004).

Most attention has focused on the apoplast as a site where oxidants are produced and perceived. A key point, therefore, concerns the antioxidant status of the apoplast. Despite the presence of many antioxidants, such as flavonoids and polyamines, the redox buffering capacity of the apoplast is much weaker than inside the cell (Horemans et al., 2000; Pignocchi et al., 2003). Unlike the cytoplasm, the apoplast is deficient in NAD(P)H and glutathione and contains a very active and regulated ascorbate oxidase. Thus, apoplastic ascorbate is markedly more oxidized than cytoplasmic ascorbate (Pignocchi et al., 2003). Moreover, the pathway for ascorbate degradation has recently been shown to be located in the apoplast (Green and Fry, 2003).

Membrane ascorbate and dehydroascorbate transport systems fail to maintain a highly reduced apoplastic ascorbate pool. Nonetheless, an ascorbate-based system could be important in driving plasma membrane and tonoplast electron transport chains. Both membranes contain an apoplastic-dependent cytochrome b561, which is reduced on one face by ascorbate and oxidized on the other by monodehydroascorbate or phenolics that can act as substrates for monodehydroascorbate reductase (Preger et al., 2004).

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triggered by secondary oxidant-induced signaling events in the cell wall, such as release of small oligosaccharides generated during the breakdown of pectins.

In addition to the apoplast, the thylakoid lumen is another compartment in which low antioxidant buffering may heighten or facilitate redox signaling. The thylakoid membrane transports ascorbate by diffusion alone (Foyer and Lelandais, 1996; Horemans et al., 2000), despite the location of key ascorbate-requiring reactions such as violaxanthin de-epoxidation on the internal face of the membrane. Low antioxidant buffering could allow oxidative signals to accumulate within the lumen, and this could be important in redox signal transduction leading to programmed cell death (PCD).

ANTIOXIDANTS AND REDOX SENSING MECHANISMS

Signaling mediated by ROS involves heterotrimeric G-proteins (Joo et al., 2005) and protein phosphorylation regulated by specific MAP kinases and protein Tyr phosphatases (Kovtun et al., 2000; Gupta and Luan, 2003; Rentel et al., 2004). The biochemical and structural basis of kinase pathway activation by ROS remains to be established in plants, but thiol oxidation likely plays a key role. The best-characterized redox signal transduction system in plants is the stromal ferredoxin-thioredoxin system, which functions in the regulation of photosynthetic carbon metabolism. Signal transmission involves disulfide-thiol conversion in target enzymes and is probably achieved by a light-induced decrease in the thioredoxin redox potential from approximately $-0.26$ V (dark: 20% reduced thioredoxin, pH 7) to approximately $-0.36$ V (light: 90% reduced thioredoxin, pH 8) (Setterdal et al., 2003). Thiol groups are likely important in other types of redox signal transduction, including ROS sensing by receptor kinases, such as ETR1 (Desikan
Thiol-based regulation may be important in plant acclimation to environmental change, particularly where redox interactions play a key role in the orchestration of the abiotic stress response. The heat shock response, for example, can be completely inhibited by effective removal of H₂O₂ because the expression of genes such as those encoding HSF21 and HSF5 and cytosolic APX1 is modulated by ROS signals (Davletova et al., 2005).

In plants, as in other organisms, thiol-containing domains are oxidized by ROS, either directly or indirectly, to give relatively stable oxidation products with modified physical conformations or biochemical activities. Both the bacterial oxyR and the yeast yAP-1 transcription factors are activated through protein thiol oxidation by peroxides (Bauer et al., 1999; Delauney et al., 2002). Similarly, mammalian heat shock factor 1, which is a key player in the response to H₂O₂ and other stresses, is shock factor 1, which is a key player in the heat shock response, for example, can be completely inhibited by effective removal of H₂O₂ because the expression of genes such as those encoding HSF21 and HSF5 and cytosolic APX1 is modulated by ROS signals (Davletova et al., 2005).

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Thermodynamics (redox potential of oxidizable thiols) and kinetics (ability to compete with the antioxidative system) are key considerations in assessing the functional importance of putative thiol-based ROS sensors in plants. The H₂O₂-reactive thiols in oxyR have a midpoint redox potential of approximately −0.18 V and a rate constant for reaction with H₂O₂ that is comparable to peroxidases. These properties mean that the protein generally will be in its reduced, inactive form under optimal conditions, when the redox potential of the glutathione pool is approximately −0.24 V, but that increases in H₂O₂ availability or changes in glutathione redox potential (or both) can readily cause oxidative activation of the sensor. Because of the low redox buffering in the apoplast, sensors located in this compartment may have different properties compared with those operating intracellularly. Sensor oxidation may also be facilitated by programmed withdrawal of the antioxidative system or could be catalyzed by specific peroxidases, as shown for the yAP-1 system in yeast (Delauney et al., 2002).

Whereas redox adjustments are central to most stress responses, the extent to which intracellular ROS concentrations increase as a result of stress is highly variable. Many observations suggest that changes in glutathione status may be as important as enhanced ROS pools in redox signaling (Creissen et al., 1999; Vanacker et al., 2000; Mou et al., 2003; Ball et al., 2004; Gomez et al., 2004; Evans et al., 2005). The glutathione pool is oxidized and enhanced in catalase-deficient plants (Noctor et al., 2002; Rizhsky et al., 2002) and in incompatible plant–pathogen interactions (Vanacker et al., 2000). Glutathione is essential for the turnover of the cell cycle and root and nodule meristem activity. The Arabidopsis mtl1 mutant, in which roots fail to develop, was shown to be glutathione deficient because of low activity of γ-glutamylcysteine synthetase (Vernoux et al., 2000), a key enzyme in

![Figure 2. Sensitive Control of Cellular Redox Responses by Protein Thiol Status.](image)
glutathione synthesis that has recently been shown to be located in plastids (Wachter et al., 2005). Moreover, accumulation of GSSG is often associated with tissue death or quiescence.

A key question concerns how glutathione status might be perceived by the cell. Although several mechanisms are possible, current knowledge suggests a potentially important role for protein glutathionylation (i.e., the formation of a mixed disulfide bond between glutathione and specific Cysteine residues) (Figure 2). This post-translational modification can modulate enzyme activity by modification of catalytic site Cys residues or affect biological activity by competing with other thiol modifications. Increased GSSG may be sufficient to trigger protein glutathionylation, although ROS-catalyzed generation of protein thyl radicals may allow glutathionylation in the absence of GSSG accumulation. Glutathionylation may also occur independently of enhanced ROS production or redox perturbation of the glutathione pool through induction or activation of enzymes that are able to catalyze the reaction, such as glutaredoxins (Figure 2). In plants, so far, two Calvin cycle enzymes (aldolase and triose phosphate isomerase) have been found to be targets for glutathionylation (Ito et al., 2003). In some cases, glutathionylation could act as a protective mechanism by preventing formation of sulfenic acid groups (Figure 2). Other roles could include buffering the cellular redox potential by preventing excessive accumulation of free GSSG and signal reinforcement during ROS sensing. One important function of glutathionylation in ROS signaling in animals could be through its antagonistic effects on distinct MAP kinase activities (Cross and Templeton, 2004). Further work is required to elucidate the importance of protein glutathionylation in redox signaling processes operating in plants.

In bacteria and yeast, oxidative activation of ROS sensors is reversed by glutaredoxin or thioredoxin (Figure 2). In plants, thioredoxins and glutaredoxins are encoded by multigene families, and targeted proteomic studies are extending the list of proteins that are potential thioredoxin targets in photosynthetic organisms (Balmer et al., 2004; Lemaire et al., 2004; Yamazaki et al., 2004). As well as their established roles in the regulation of the enzymes of carbon metabolism, thioredoxins may act to modulate the activity of key antioxidative enzymes, such as ascorbate peroxidase and catalase (Lemaire et al., 2004; Yamazaki et al., 2004). Such responses might be crucial to redox signal transduction in light- and pathogen-triggered PCD responses (Mateo et al., 2004). Certain glutaredoxins and thioredoxins play direct roles in the antioxidative system by regenerating peroxiredoxins oxidized by peroxides (Rouhier et al., 2001; Collin et al., 2004; Rey et al., 2005). In addition to their antioxidant function, peroxiredoxins could play roles in redox signaling through oxidant-induced oligomerization (König et al., 2002) or perhaps as catalysts of ROS sensor oxidation, a reaction that might be analogous to oxidation of vAP-1 by the thiol-based peroxidase Gpx3 (Orp1; Delauney et al., 2002).

Recent data suggest that ROS-triggered increases in cytosolic calcium could also be influenced by glutathione status (Gomez et al., 2004; Evans et al., 2005). There is an intimate connection between ROS production and intracellular increases in calcium, which are both early events during pathogen responses (Dangl and Jones, 2001; Lecourieux et al., 2002). Increases in cytosolic calcium are also triggered by ROS production during root hair formation and abscisic acid signaling (Foreman et al., 2003; Kwak et al., 2003).

ANTIOXIDANTS AND STRESS-INDUCED PCD

A current hypothesis is that all cells survive by suppression of a default death pathway that is a legacy from the host–endosymbiont interactions through which eukaryotic cells evolved (Ameisen, 2002). The default death pathway, PCD, is an integral part of many development and defense processes and can be triggered by ROS and reactive nitrogen species. The best-studied PCD process in plants is the hypersensitive response to incompatible pathogens (Dangl and Jones, 2001). Apoplastic ROS accumulation during the hypersensitive response follows activation or induction of ROS generating systems.

The pathogen-induced apoplastic oxidative burst alone may be insufficient to trigger PCD (Mur et al., 2005). However, ROS accumulation in organelles such as mitochondria may also play a role during PCD (Maxwell et al., 2002). Genetic alteration of the mitochondrial electron transport chain desensitizes the plant to stress-induced cell death (Dutilleul et al., 2003). Chloroplasts were shown to be the site of the earliest accumulation of ROS in the guard cells of ozone-fumigated Arabidopsis leaves, with apoplastic ROS accumulation occurring only later as a result of activation of the AtbbohD- and AtbbohF-encoded NADPH oxidases (Joo et al., 2005). The chloroplast response was extremely rapid and involved the participation of intracellular G-proteins and associated signaling pathways that participate in the stress-elicited oxidative burst (Joo et al., 2005). Other evidence also points to an important role for H2O2 generated during photosynthesis in chloroplasts and through photorespiration in PCD (Mateo et al., 2004).

Among numerous lesion mimic mutants that more readily undergo PCD than wild-type plants, lsd1 has been particularly useful in elucidating the role of superoxide (Jabs et al., 1996) and H2O2 (Mateo et al., 2004). It is possible that LSD1 binds to the C-terminal region of the transcription factor AtbZIP10, blocking its entry to the nucleus and retaining it in the cytosol. Such basic domain/leucine zipper transcription factors play key roles in the orchestration of many plant responses to the environment, particularly light and stress signaling. The absence of the LSD1 protein function results in the initiation and propagation of PCD under growth conditions that are optimal for wild-type Arabidopsis, such that exposure to even low light causes lesion formation on the lsd1 leaves. Thus, PCD initiation in lsd1 is dependent on environmental triggers, particularly light. Photosynthetic electron transport inhibitors block lesion formation in lsd1. Lesion development was favored by high light and low CO2, implicating photorespiratory
H$_2$O$_2$ production in the PCD response (Mateo et al., 2004). Moreover, a major leaf isoform of catalase (CAT1) is decreased when PCD is initiated in lsd1 (Mateo et al., 2004).

Interestingly, PCD is also observed in plants deficient in the major catalase isoforms (Rizhsky et al., 2002). Because very high rates of H$_2$O$_2$ production occur during photosynthesis, particularly in the peroxisomes and chloroplasts (Nocito et al., 2002), we conclude that ROS-triggered PCD can occur in two ways. Either ROS production can be activated in compartments where antioxidant buffering is low (e.g., the apoplast) or antioxidant capacity can be withdrawn from compartments in which ROS production is high (e.g., the chloroplast and peroxisome). The extent of interplay between these individual processes and the relationship of each to reactive nitrogen species accumulation remains to be established. However, the fact that several key antioxidative enzymes are dual targeted to chloroplasts and mitochondria (Chew et al., 2003) may be significant in PCD regulation.

Much attention has focused on the role of salicylic acid in the regulation of PCD and the induction of PR proteins associated with systemic acquired resistance. Both ROS and antioxidants have been strongly implicated in salicylic acid signaling. Glutathione is able to induce PR transcript induction (Gomez et al., 2004 and references cited therein), whereas localized cell death occurs in ascorbate-deficient plants (Pastori et al., 2003). These effects point to opposing functions for ascorbate and glutathione in redox signal transduction and are likely related to redox modulation of NPR1, a protein necessary for salicylic acid signaling. It has been shown that NPR1 exists as an oligomer formed by intermolecular disulfide bonds and that induction of systemic acquired resistance leads to a change in cellular reduction potential that reduces NPR1 to its monomeric form, which accumulates in the nucleus and activates target gene expression (Mou et al., 2003). Furthermore, the interaction of NPR1 with the transcription factor TGA1 and resulting induction of TGA1 DNA binding activity is dependent on the reduction of TGA1 Cys residues induced by SA (Després et al., 2003). In addition, ascorbate deficiency is associated with upregulation of extracellular defenses (nonspecific peroxidases) and constitutive activation of processes known to be controlled by apoplastic ROS production, notably abscisic acid signaling, pathogen resistance, and constitutive expression of PR proteins (Pastori et al., 2003; Barth et al., 2004). In the ascorbate-deficient vtc1 mutant, these effects are accompanied by enhanced glutathione and nuclear localization of NPR1 (our unpublished data).

Based on these and other recent data, the hypothetical scheme shown in Figure 3 illustrates some of the likely roles of cellular reductants and antioxidants in ROS signaling linked to PCD and salicylic acid signaling. Key points are, first, that redox signaling during an incompatible plant–pathogen interaction involves both oxidative and reductive steps and, second, that there is a relationship between salicylic acid, catalase (and probably also ascorbate peroxidase), and glutathione in this process (Vanacker et al., 2000; Mou et al., 2003). Reductive signaling triggered by an upstream oxidative signal parallels ROS-mediated signaling in mammalian cells through the transcription factors NF-$\kappa$B and AP-1. Binding of these transcription factors to DNA is favored by a reducing environment and is inhibited by GSSG or oxidized thioredoxin (Dröge, 2002). The extent to which the processes shown in Figure 3 occur in the same cells is unknown, but as noted above, ROS signaling can be initiated first in chloroplasts after exposure to ozone, and this contributes to the activation of membrane-associated NADPH oxidases that are involved in intercellular redox signal transduction (Joo et al., 2005). Redox signals can then be transmitted via H$_2$O$_2$ locally in the apoplast or long distance through the vascular system where this oxidant has a longer lifetime than in antioxidant-rich compartments or tissues.

Recent evidence strongly suggests that cell death triggered by either singlet oxygen (Wagner et al., 2004) or paraquat (Chen and Dickman, 2004) in the chloroplast does not result from oxidative physiochemical damage but is the consequence of activation of a genetic program. In animals, pro- and anti-apoptotic factors control mitochondria-dependent cell death pathways. Early work in this area showed that expression of a human anti-apoptotic factor in tobacco did not confer protection against pathogen-induced cell death (Mittler et al., 1996). More recently, however, several studies have shown that animal PCD regulators modulate plant PCD and stress responses. Expression of the mammalian pro-apoptotic protein Bax promoted death, whereas the anti-apoptotic protein Bcl2 suppressed PCD in rice cells challenged with elicitors (Matsumura et al., 2003). Interestingly, overexpression of glutathione peroxidase inhibited Bax- and oxidative stress-induced PCD in tomato (Chen et al., 2004). Bleaching of tobacco leaves induced by superoxide and singlet oxygen-generating herbicides was prevented by expression of either mammalian Bcl proteins or Caenorhabditis elegans CED-9 (Chen and Dickman, 2004). These results confirm that plant PCD can be regulated by pro- and anti-apoptotic factors that interact with ROS. Moreover, they suggest that the mechanisms through which ROS-generating chloroplast herbicides kill plants may involve PCD rather than physicochemical damage that overwhelms the antioxidative system.

CONCLUSIONS AND PERSPECTIVES

Redox signal transduction is a universal feature of aerobic life honed through evolution to balance information from metabolism and the environment. Here, we have presented a view of the plant cell as a series of interconnected compartments with different antioxidant buffering capacities determined by differences in synthesis, transport, and/or degradation. The result is a set of discrete locations where signaling is controlled (or buffered) independently. This permits redox-sensitive signal transduction to occur in locations such as the apoplast, the thylakoid, and, perhaps, the endoplasmic reticulum, whereas other highly buffered spaces have a much higher threshold for ROS signals. Both oxidants...
and antioxidants fulfill signaling roles to provide information on general plant health, particularly in terms of robustness for defense, using kinase-dependent and independent pathways that are initiated by redox-sensitive receptors modulated by thiol status. Antioxidants are not passive bystanders in this crosstalk, but rather function as key signaling compounds that constitute a dynamic metabolic interface between plant cell stress perception and physiological responses. Current data suggest that glutathione is a key arbiter of the intracellular redox potential, and ascorbate is particularly influential in setting thresholds for apoplastic and cytoplasmic signaling. Differential antioxidant concentrations between compartments permit antioxidant-driven vectorial signaling through processes such as ascorbate-driven electron transport or futile cycles. The future will
determine more precisely how ascorbate, glutathione, and tocopherol are involved in initiating and controlling redox signal transduction and how they trigger other related responses such as PR gene expression to optimize survival strategies.

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