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Comprehensive Invited Review

Redox Regulation in Photosynthetic Organisms: Signaling, Acclimation, and Practical Implications

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Abstract

Reactive oxygen species (ROS) have multifaceted roles in the orchestration of plant gene expression and gene-product regulation. Cellular redox homeostasis is considered to be an "integrator" of information from metabolism and the environment controlling plant growth and acclimation responses, as well as cell suicide events. The different ROS forms influence gene expression in specific and sometimes antagonistic ways. Low molecular antioxidants (*e.g.*, ascorbate, glutathione) serve not only to limit the lifetime of the ROS signals but also to participate in an extensive range of other redox signaling and regulatory functions. In contrast to the low molecular weight antioxidants, the "redox" states of components involved in photosynthesis such as plastoquinone show rapid and often transient shifts in response to changes in light and other environmental signals. Whereas both types of "redox regulation" are intimately linked through the thioredoxin, peroxiredoxin, and pyridine nucleotide pools, they also act independently of each other to achieve overall energy balance between energy-producing and energy-utilizing pathways. This review focuses on current knowledge of the pathways of redox regulation, with discussion of the somewhat juxtaposed hypotheses of "oxidative damage" *versus* "oxidative signaling," within the wider context of physiological function, from plant cell biology to potential applications. *Antioxid. Redox Signal.* 11, 861–905.

I. Introduction

Cellular redox metabolism in humans and plants is intimately linked through our consumption of the plant foods that we cultivate and through human effects on the natural environment and control of agricultural conditions. Plant organs are often rich in antioxidants, some of which, such as ascorbate and tocopherol, are essential vitamins in the human diet. For many years, people have been urged to take antioxidant supplements in the belief that this practice

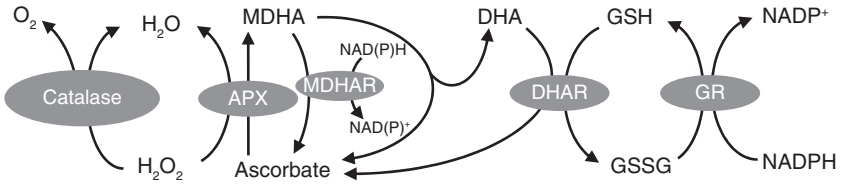

FIG. 1. Two of the major pathways for H_2O_2 metabolism in plants. Other enzymes such as thioredoxin or glutaredoxin-linked peroxiredoxins and peroxidatic glutathione transferases also contribute. In particular, it should be noted that 2-cys peroxiredoxins could be important in H_2O_2 metabolism in the chloroplast, as depicted in Fig. 2 of Dietz *et al.* (87). APX, ascorbate peroxidase; DHAI, dehydroascorbate (reductase); GR, glutathione reductase; GSH, glutathione; GSSG, glutathione disulfide; MDHAI, monodehydroascorbate (reductase).

improves health and prevents disease. Recently, the routine use of antioxidant supplements has become highly controversial, as a number of comprehensive studies have demonstrated that taking them is actually harmful because it increases all-cause mortality (41). The negative effect of antioxidants on human mortality can be explained by the fact that reactive oxygen species (ROS) are essential to the well-being of all organisms, including humans, and that an enhancement of their elimination interferes with the essential mechanisms for the creation and controlled eradication of cells that ensure human health (41). Of these mechanisms, animal cell apoptosis [or programmed cell death (PCD) in plants] is perhaps the best characterized in terms of the central role of cellular redox homeostasis.

Despite the fact that the antioxidant theory of health improvement has now been called into question, epidemiological studies show that consumption of vegetables and fruit is associated with improved vascular health, with decreased risk of cancer, heart disease, and stroke (395). The answer to the question of why the intake of plant foodstuffs promotes human health appears to reside in the ability of plant metabolites to regulate human gene transcription and to induce endogenous defenses that counter carcinogenesis and the development of high cholesterol and lipids. One way in which dietary bioactive compounds can induce protective gene expression is by the activation of transcription factor NF-E2-related factor-2 (Nrf2; 395). Nrf2 binds to the "antioxidant response element" (ARE) in the promoters of genes encoding a battery of metabolic and defense enzymes (95) such as γ-glutamylcysteine synthetase (γ-ECS), glutathione reductase (GR), aldo-keto reductase, and glutathione transferases (GTs, formerly known as glutathione *S*-transferases) and enzymes of the pentose phosphate pathway, such as transketolase and transaldolase, as well as components of the ubiquitin-independent 20S proteasome that degrades damaged protein (231, 379, 400).

In the absence of an appropriate signal, Nrf2 is held in the cytosol in a complex with Kelch-like ECH-associated protein 1

A

Name	Symbol	Radical	Ion	ROS	Primary sources in plants
Triplet oxygen	$^3O_2$	Yes	No	No	Photosystem II
Singlet oxygen***	$^1O_2$	No	No	Yes	Photodynamic transfer (excited chlorophylls, etc)
Superoxide**	$O_2^{\cdot-}$	Yes	Yes	Yes	Electron transport chains, oxidases
Hydrogen peroxide*	H_2O_2	No	No	Yes	Reduction/disumutation of superoxide, oxidases
Hydroxyl radical****	$OH^{\cdot}$	Yes	No	Yes	Reductive cleavage of H_2O_2
Water	H_2O	No	No	No	Absorption, various reactions
Hydroxide	OH^-	No	Yes	No	Various reactions, ionization of water

B

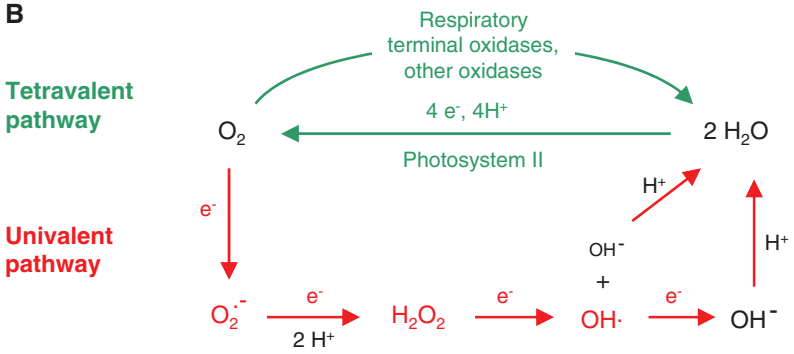

FIG. 2. Simple summary of principal chemical species of oxygen (A) and the two main paths of oxygen reduction (B). The scheme shows only the main forms thought to exist under physiological conditions. Protonated forms of superoxide and deprotonated forms of H_2O_2 also exist. The relative reactivities of the different ROS are denoted by *asterisks* in (**A**). The term 'ROS' refers to any triplet oxygen species that is more reactive than O_2; a radical is defined as a molecule carrying an unpaired electron; an ion refers to a compound that carries a charge (*i.e.*, its total number of electrons is not equal to its total number of protons).

(Keap1). Keap1 is a substrate adaptor protein for the Cullin-3 (Cul3)-dependent E2 ubiquitin ligase complex, directing Nrf2 for proteasomal degradation (132). Phosphorylation of Keap1 or oxidation of Keap1 Cys residues diminishes its affinity for Nrf2 which is then released (395). Once released, Nrf2 is translocated to the nucleus where it binds to AREs and increases protective gene expression. Both the Nrf2-Keap1 and Keap1-Cul3 interaction have been observed in several cell types (59, 207, 339). The elicitation of Nrf2-dependent orchestration of defense metabolism by plant compounds provides a much wider range of protection than that offered by increased antioxidant capacity alone. The protection offered by enzyme-dependent detoxification processes is not only more powerful than that potentially offered by antioxidant supplement, but is also more durable, as metabolite accumulation is limited by degradation and excretion. A wide range of secondary metabolites in fruit and vegetables act as "elicitors" that activate Nrf2. These include oxidized omega-3 fatty acids, carotenoids, glucosinolate-derived isothiocyanates and indoles, polyphenols, and allyl sulfides. Regardless of how the plant signals are perceived, for example, as pro-oxidants or xenobiotics, and the mechanisms through which they enhance cellular resistance responses that underpin health benefits, one is drawn to the conclusion that it is crucial not only to understand the redox processes that drive plant primary and secondary metabolism to produce these metabolites in abundance but also the holistic relationships between cellular redox homeostasis and plant growth and development.

Significant drivers for research on redox processes in plants have been the aims of achieving foods that are enriched in classical low molecular weight antioxidants (tocopherol, ascorbate, glutathione) and improving plant sustainability and yield through enhanced stress tolerance. A key scientific question concerns why plants accumulate such high amounts of low-molecular-weight antioxidants, particularly ascorbate. The answer lies in the complex web of processes in which these metabolites participate, spanning growth and development as well as defense. Historically, ascorbate and glutathione have been described largely as the crucial players in a high-capacity plant enzymatic recycling system that functions, together with catalases (CAT), to metabolize H_2O_2 (Fig. 1). For basic information on these processes, we invite the reader to consult our earlier reviews (114, 116–119, 121, 125, 127, 291, 292) and those of others (14–16, 22, 33, 86, 265, 273, 275, 381, 382). Here, we discuss some of the pertinent advances and essential features of the ROS/antioxidant relationship in plants, with references where appropriate to other recent authoritative reviews on particular aspects. The discussion is presented against the backdrop of the ongoing debate over the relative importance of the concepts of "damage" and "signaling."

II. The Plant Paradigm of Redox Control and Signaling: At the Heart of Plant Physiology

A. Oxygen chemistry: The basics

1. Univalent reduction of oxygen. The global wheel of oxidation–reduction (redox) systems by which present-day living organisms store and release energy was set in motion by the evolution of oxygenic photosynthesis (7). Photosynthetic organisms created the oxygen-rich atmosphere of the earth, and then tackled the problems of living with oxygen, finding a use for what was originally oxidative "damage" to proteins, lipids, and DNA in signaling, in the form of redox cues, the impact of a changing environment on metabolism. It is estimated that prokaryotic oxygen-evolving ancestors of the plant chloroplast first appeared between 3.4 and 2.3 billion years ago (7) and initiated the oxygen-rich atmosphere of the earth today, unique among the planets of our solar system. As a result, molecular oxygen became intimately involved with the essential energy exchange reactions on which life is based, allowing the routine and widespread use of the high electrochemical potential ($E_{m7} = +815$ mV) of the O_2/H_2O redox couple as a terminal electron acceptor by respiratory oxidases. A key feature of these oxidases, and other enzymes such as ascorbate oxidase, is that they reduce O_2 to water through a tetravalent pathway that does not involve the release of reactive, partially reduced intermediates (Fig. 2). Likewise, the photosystem (PS) II water-splitting system of photosynthesis undertakes the concerted four-electron oxidation of water without the release of ROS. However, many processes in plants catalyze only partial reduction of oxygen and produce superoxide, H_2O_2, and hydroxyl radicals, all of which are more reactive than ground state triplet O_2 (Fig. 2). While superoxide production by autoxidation of PSII components has been discussed (15, 16), it is generally accepted that PSI is the major site of superoxide generation in the photosynthetic electron transport (PET) chain.

Superoxide production at PSI can be greatly enhanced by the pro-oxidant herbicide methyl viologen (paraquat), which acts as a cycling catalyzer of electron transfer from PSI FeS centers to O_2 (Fig. 3). Exposing leaves to paraquat leads to rapid shrinkage of the tissue associated with PCD, as illustrated in Fig. 3. It is important to note that the term PCD covers a wide range of processes in plants, but these are distinct from necrosis in that they involve cell shrinkage in a physiological process with hallmark characteristics, as discussed in relation to the role of ROS by Garmier *et al*. (136). In contrast to PCD, necrosis is a pathological process that occurs when cells are exposed to a physical or chemical wound or insult; it involves rapid loss of membrane integrity, the cytoplasm and mitochondria swell and cell lysis occurs.

2. Singlet oxygen. As well as being activated by reduction, ground-state triplet O_2 can be rendered much more reactive by photodynamic processes that modify the electron configuration of the O_2 molecule to produce singlet oxygen (150). Significant production of singlet oxygen can occur in all living organisms through various mechanisms but is particularly prominent in the chloroplast because of the routine formation of excited pigments in photosynthesis (127). Though less reactive than the hydroxyl radical, singlet oxygen is more reactive than both superoxide and H_2O_2, and was considered for many years as a highly toxic molecule with very limited diffusion. Recent evidence suggests, however, that singlet oxygen might diffuse significant distances from its site of production (109). Moreover, chloroplast lipid peroxidation has been shown to be almost exclusively mediated by singlet oxygen (407). Singlet oxygen, like H_2O_2, is involved in cell signaling, leading to the execution of a cell suicide program, although, as we discuss later, these two ROS may operate in an antagonistic fashion in the regulation of gene expression (14).

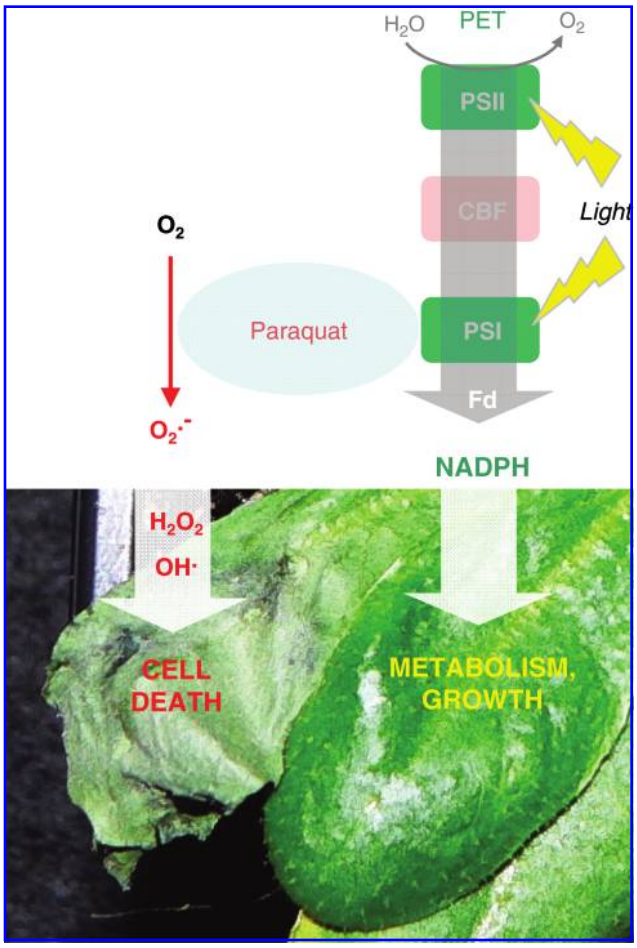

FIG. 3. Paraquat-induced tissue collapse in Arabidopsis leaves. Paraquat (methyl viologen) is a very efficient acceptor of electrons from the low potential iron–sulfur groups of photosystem I and can out-compete electron acceptors such as ferredoxin and $NADP^+$. This starves chloroplast metabolism of reductant. Even more seriously, in the short-term, the reduced paraquat radical promotes superoxide formation through auto-oxidation by molecular oxygen. Paraquat is thus a cycling catalyzer of superoxide formation. If the antioxidative enzymes are unable to keep pace, superoxide and derived species (Fig. 2) trigger cell death. CBF, cytochrome b_6f complex; Fd, ferredoxin; PET, photosynthetic electron transport; PSI, photosystem I; PSII, photosystem II.

B. Redox poising and signaling from electron transport chains

1. Redox turnover, homeostasis, and poising. In green plant cells, the PET and respiratory electron transport (RET) chains interact directly with the metabolic pathway of carbon assimilation in a producer–consumer relationship with regard to reducing power (reduced ferredoxin, NAD(P)H) and energy (ATP) (Fig. 4). The redox relationships of the chloroplast with the rest of the cell (129) have many parallels with those of the mitochondria. Together, the two major energy-producing organelles of the plant cell interact to influence and regulate cellular redox homeostasis.

A key theme of this review is that redox regulatory mechanisms in the membrane-bound and soluble phases are intimately linked at multiple levels. However, it is important to define the concepts underlying redox regulation and homeostasis, and to distinguish between processes occurring in electron transport chains and those that occur in the soluble phase. Key notions are thermodynamics and kinetics. The concept of "cellular redox homeostasis" applies to the soluble phase, where the major redox couples (NADPH, ascorbate, glutathione) turn over *relatively* slowly (seconds to minutes) and are often considered to be maintained close to thermodynamic equilibrium. A "cellular redox potential" of about −250 to −300 mV is often assumed, though it remains unclear whether soluble redox couples really are in equilibrium at these values. Furthermore, both NADPH and ascorbate functions are intimately associated with electron transport chains, and the concept of strict redox homeostasis in the soluble phase may be less applicable to photosynthetic organisms than to heterotrophic organisms that maintain a near constant cellular temperature. In plants, PET operation can affect the redox state of soluble components such as NADP(H), to drive the induction of assimilatory metabolism, and also that of stromal thioredoxins (372), to allow light signaling that is essentially thermodynamically driven (*i.e.*, the key factor is that certain thioredoxins are more reduced in the light than the dark). In contrast, the overall redox state of the ascorbate and glutathione pools is not significantly changed as a result of the immediate impact of light on PET. These "antioxidants" are generally maintained in a highly reduced state, though this can change as a result of intensive ROS production, for example, in the presence of a pro-oxidant such as methyl viologen. Indeed, such ROS-induced changes in ascorbate and/or glutathione status are often taken as indicative of "oxidative stress".

In contrast to the relatively slow turnover and so more slowly changing redox states of soluble redox couples, components in the PET system generally have sub-second turnover rates. Since electron flux between components is driven by large changes in redox potential, the concept of homeostasis is less applicable because redox states can change rapidly depending on input and output. The PET system is subject to fluctuations in light (input) and in temperature (which are an important determinant of the capacity for use of output, *i.e.*, ATP and NADPH). Nevertheless, PET redox status is not simply the outcome of thermodynamics. Numerous regulatory mechanisms operate to ensure that both oxidized and reduced forms of components are simultaneously present. This balancing act is known as "redox poising" and is necessary to avoid inhibition of flux by bottlenecks. Feedforward and feedback mechanisms that coordinate PET activity and metabolism have been studied for many years. These concepts include light activation of photosynthetic metabolism by thioredoxins (442), photosynthetic control over electron transport (120), and ΔpH-dependent regulation of light harvesting efficiency (78, 293). Recent work suggests that NADP(H) status could also feed back to regulate electron transport rates (155). The reduction state or redox poise of PET components such as the plastoquinone pool and the cytochrome b_6f complex is important not only in determining short-term acclimatory mechanisms such as "state transitions", and the amount of light energy that is dissipated as heat, but it is also instrumental in initiating signal cascades that regulate gene expression (174, 353).

In addition to the regulatory mechanisms mentioned above and discussed further in the next Section, oxygen is a key

player in preventing bottlenecks in the PET and RET chains. Over-reduction of electron transport components favors production of singlet oxygen and superoxide (which subsequently gives rise to H_2O_2 via reduction or dismutation). Superoxide production plays a crucial role in redox poising, as it acts as an electron overflow system, and was described several decades ago as electron "leakage" to oxygen. This term, which suggests malfunction or imperfect function, has unfortunately become embedded in the literature. Moreover, in the context of cell survival signaling, superoxide production on the reducing side of PSI may serve to induce defense gene expression and so offset the influence of singlet oxygen-induced cell death signals (14). As discussed in detail below, different ROS signals produced at various sites in the PET chain have an inverse "balancing action," on gene expression.

The above discussion emphasizes that although electron transport chains and soluble redox couples in plants can be distinguished on the basis of turnover rates, homeostasis, and poising, they are nevertheless inextricably entwined. The redox state of electron transport components is not determined simply by thermodynamics (because of the influence of homeostatic regulatory mechanisms) while that of the soluble phase is not immune from thermodynamics (because the impact of environmental changes can feed downstream from electron transport chains). A recurring theme of the discussion in several of the following Sections is to what extent light energy balance (through its effect on PET and, less directly, on RET) can influence "cellular redox homeostasis," for example, by modifying ROS production and associated metabolism or by otherwise driving changes in the redox state of components within the soluble phase.

2. Monitoring the motor: Signals from electron transport chains. In harnessing light energy to drive metabolism, plants had to master the art of redox control and, since neither uncontrolled oxidation nor over-reduction are compatible with efficient cellular energy utilization, the early photosynthetic cells had to adapt to oxygen generation. Hence, survival demanded and continues to demand redox controls of energy metabolism and ultimately the expression of genes that are the essential drivers, modulators, and protectors of the energy-exchange processes. It is the poising function of oxygen that marks out ROS as primary candidates as diffusible and reactive mediators of signaling linked to electron transport status. Besides ROS, other signals of electron transport status clearly exist (Fig. 4) and exert a major influence on gene function and post-transcriptional modification of proteins. Control mechanisms operate at multiple levels of gene expression (summarized in 323), from transcription (324–327) and translation (406, 438), and involve retrograde signaling from the chloroplast to the nucleus (109, 300), as well as thylakoid membrane protein phosphorylation (465). Similarly, thiol–disulfide exchange reactions modulate the activities of a wide range of chloroplast enzymes (51, 370), including those of starch synthesis (164, 215) and starch degradation (385). The redox state of PET components, such as the plastoquinone pool, regulates many plastid and nuclear genes (108, 202–205, 359, 455, 456).

Several thylakoid protein kinases, including the plastid transcription kinase (23) and the STN7 and STN8 kinases, have been linked to signaling cascades that orchestrate the readjustments in the expression of genes encoding thylakoid proteins and so achieve an appropriate composition of light harvesting complexes (LHC) and PS stoichiometries consistent with the light environment (201). The STN7 and STN8 kinases interact in the signaling network that coordinates nuclear and chloroplast gene expression and that induces the expression of stress response genes, particularly heat shock proteins (353). To date, relatively few components of this pathway have been identified. This is surprising given that the pathway is widely considered to be central to the light acclimation processes and the prevention of over-reduction of PSI electron acceptors that would enhance ROS production. More recently, a histidine sensor kinase (chloroplast sensor kinase, CSK) has been implicated in the redox control of chloroplast gene expression (338). Signals derived from RET chain components also modulate nuclear and mitochondrial genes involved in primary metabolism (photosynthesis and respiration) and influence other processes such as stress tolerance (99, 456). The persistence of redox control of gene expression within chloroplasts and mitochondria is considered to provide evidence that redox signaling is a primary function of their small, specialized but vital genomes (4, 6).

C. Metabolic and regulated production of reactive oxygen species

1. The richness of ROS metabolism in photosynthetic tissues. Concepts of the roles of ROS in heterotrophic plant cells have partly been inspired by animal models and other systems such as yeast and nonphotosynthetic bacteria. However, the overwhelming focus on the roles of ROS in plant responses to stress has been on photosynthetic tissues. It is often stated that the oxygen content of chloroplasts is significantly higher than the rest of the photosynthetic cell (15, 16). It is worth noting, however, that although chloroplasts generate oxygen on the interior face of the thylakoid membrane, there is as yet little supporting evidence that either the thylakoid or chloroplast envelope membranes present a significant barrier to oxygen diffusion. Long-standing experimental data suggest that the evolution of O_2 in photosynthesis causes only a modest increase in already high chloroplast O_2 concentrations [$\sim$20% during photosynthesis in air (386)]. Nevertheless, photosynthetic cells indisputably have several unique ROS-producing pathways and a multiplicity of ROS-metabolizing systems.

Singlet oxygen can be produced in plants by several reactions, including lipid peroxidation and photodynamic energy transfer to O_2 from excited triplet-state chlorophyll molecules in both photosystems. Although singlet oxygen is formed by reaction of O_2 with both reaction centers and accessory light harvesting chlorophylls (226), it is thought that the most significant source of singlet oxygen is probably in the PSII reaction center where quenching by carotenoids is less effective (127). Superoxide and H_2O_2 can be produced by numerous pathways in photosynthetic cells. In addition to production linked to PET and RET, a pathway called photorespiration is a major producer of H_2O_2 (117, 295). Photorespiration is due to the oxygenase activity of ribulose-1, 5-*bis*phosphate carboxylase/oxygenase (Rubisco), which produces 2-phosphoglycolate: This small molecule is metabolized through a sequence of reactions that includes H_2O_2 production by glycolate oxidase (Fig. 5). The plasmalemma and cell wall/apoplast are also rich in ROS-producing

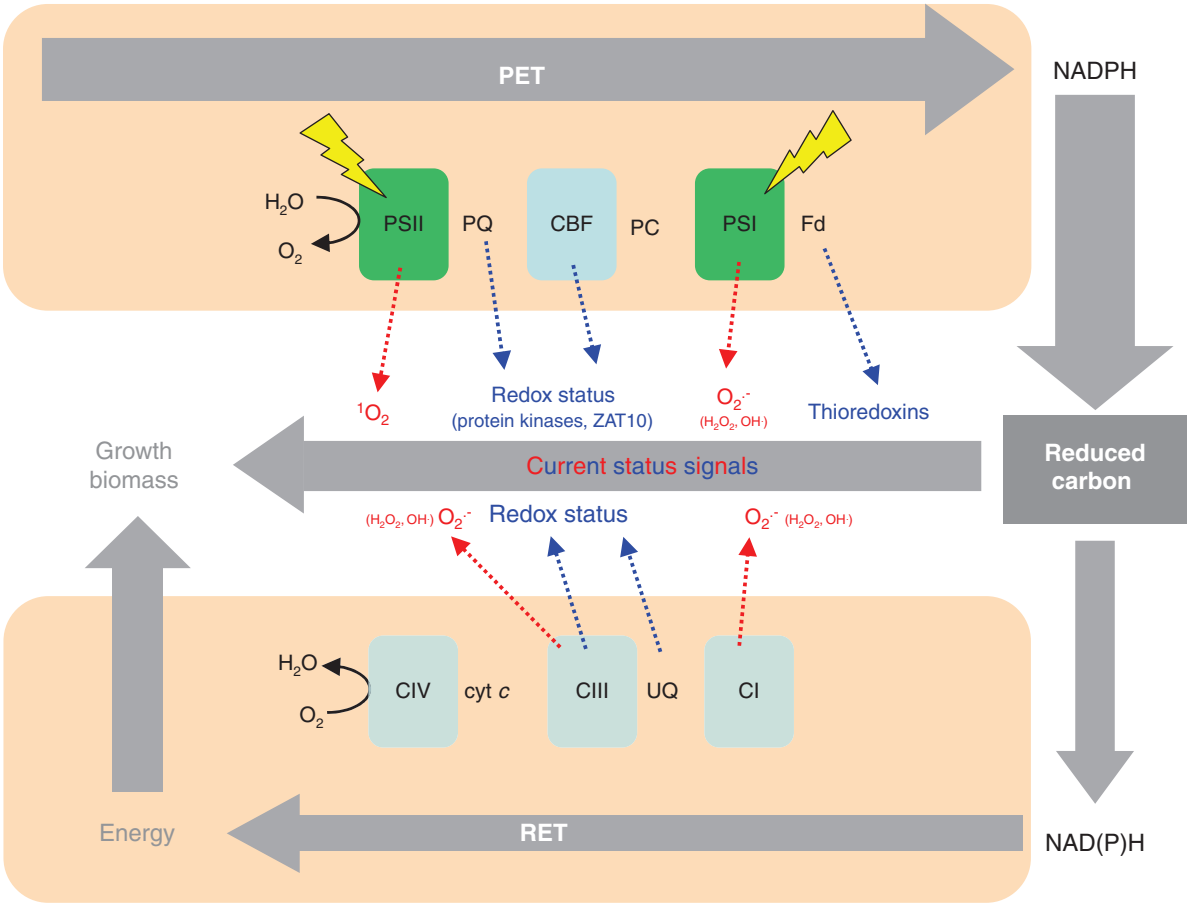

FIG. 4. The role of the photosynthetic and respiratory electron transport chains (PET and RET, respectively) in the production of redox signals, including ROS that provide information on current energy status for plant growth and acclimation responses to developmental and environmental cues. The scheme does not imply that ROS readily diffuse across the membranes, and it is most probable that localized ROS signals are transduced by other components. CI, CIII, CIV, complexes I, III and IV; cyt *c*, cytochrome *c*; PC, plastocyanin; PQ, plastoquinone; UQ, ubiquinone. Other abbreviations are as in Fig. 3.

enzymes like NADPH oxidases (also sometimes called Respiratory Burst Oxidase Homologs; RBOH), peroxidases, amine oxidases, and oxalate oxidases (44, 362).

Current concepts distinguish between two types of ROS accumulation in plant cells: (a) metabolic accumulation and (b) regulated burst accumulation. Though we emphasize in this review that this distinction is somewhat simplistic and that there may be considerable interplay, the former category would include PET and RET chains as well as glycolate oxidase. As in animals, the best characterized systems in the second category in plants are NADPH oxidases that act as ROS-producing systems that are triggered in response to specific environmental or metabolic cues. These enzymes are considered to produce ROS at appropriate moments of plant development and in response to environmental challenge such as salt stress and pathogen attack.

2. ROS accumulation and compartmentation. The increasingly refined identification of marker transcripts induced by different ROS is providing useful tools for distinguishing between the operation of different ROS signaling pathways in various stress conditions (133). In addition to the information derived from transcript abundance *per se*, marker transcript promoters can be used to drive reporter genes. Use of such tools is particularly important, given that accurate quantification of ROS, either *in vivo* or in extracts, remains problematic in plants (342, 422). Significant problems are related to low stability and abundance (caused by reactivity and the presence of a highly active antioxidative system), assay specificity, and artifactual interference during both extraction and assay. Among the different ROS, H_2O_2 is the least reactive (Fig. 2). However, there is still no consensus on likely leaf contents or concentrations in different intracellular compartments, and the quantification of even this relatively stable ROS is problematic because of a range of potential artifacts, for example, linked to ascorbate (342, 422).

The concept that inter-compartmental gradients in H_2O_2 are important response determinants is widely accepted. It has been suggested that cytosolic peroxidases are important in metabolizing H_2O_2 of chloroplastic origin (73), though it remains unclear how such diffusion can be driven without negative effects on H_2O_2-sensitive chloroplast reactions. However, recent data show that H_2O_2 can be transported within vesicles (240), clearly suggesting that membranes can restrict H_2O_2 movement sufficiently to generate gradients and unidirectional signaling, for example, from cytoplasm to

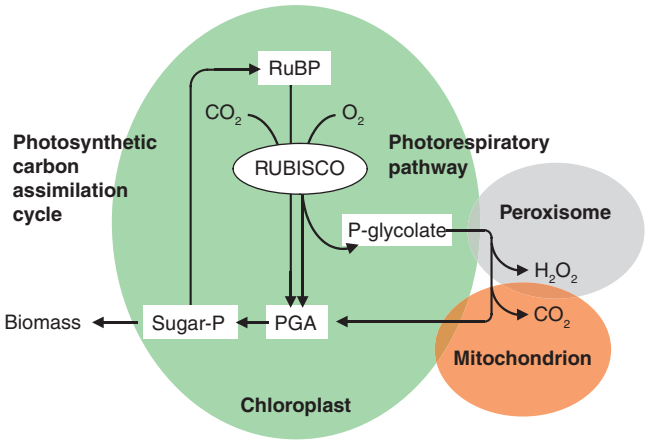

FIG. 5. The dual function of earth's most abundant protein (Rubisco). Rubisco is the catalyst for the autocatalytic fixation of CO_2, entry point of most of the carbon in the biosphere. A second catalytic function of the enzyme is oxygenation of its substrate, RuBP, setting in motion the photorespiratory pathway that results in evolution (and so potential loss) of CO_2 but also high rates of H_2O_2 production via the glycolate oxidase reaction. PGA, 3-phosphoglycerate; RuBP, ribulose 1,5-*bis*phosphate. Rubisco, ribulose 1,5-*bis*phosphate carboxylase/oxygenase; P-glycolate, 2-phosphoglycolate; sugar-P, sugar-phosphate.

external compartments (cell wall/apoplast) or to one focal point on a specific membrane, as occurs in papilla formation or in stomatal closure, as illustrated in Fig. 6. This type of vesicle trafficking involves integral membrane proteins called "vesicle associated membrane proteins (VAMP)," that form the major component of the SNARE (soluble *N*-ethylmaleimide-sensitive factor attachment protein receptor) complexes that function in facilitating vesicle fusion with target membranes (240). Trafficking of membrane-bound solutes like H_2O_2 is essential not only for signaling but also to accommodate the cellular volume changes associated with opening and closing of the stomatal aperture. Stomatal functioning requires cooperation between plasma and vacuolar membrane vesicles, with which the AtVAMP7C complex is associated. The regulated operation of H_2O_2-transporting aquaporins (38) is therefore very important in the control of H_2O_2 concentration gradients (165). The transport of H_2O_2 through the vesicular trafficking system likely allows very specific targeting of required responses, such as those involved in the positioning of root hair growth (111).

3. ROS movement in local and systemic signaling. H_2O_2 has been implicated in long-distance inter-organ signal in systemic acclimation to excess light (131, 204), but to date corroborative evidence is lacking (359). More likely explanations are that ROS are continuously generated along the signaling pathway or that there is a mobile signal that is induced by H_2O_2 at the point of departure and that generates H_2O_2 at the point of arrival. The latter mechanism could be partly analogous to mechanisms occurring during establishment of systemic acquired resistance of plants to pathogens (345, 388). An attractive candidate for the long distance mobile signal in the case of excess light acclimation is abscisic acid (ABA; see Section VI, A, 3) which is an important inducer of H_2O_2 production in response to abiotic stimuli (177, 466).

Although superoxide has been suggested as a signal independent of H_2O_2 (185), it is considered unlikely to diffuse over significant distances, given its rapid rate of nonenzymic dismutation in aqueous solution and the presence of superoxide dismutase (SOD) in most cell compartments. Equally, the multiplicity of components with which the hydroxyl radical can interact means that this ROS essentially cannot move from the site of its production. Thus, while its formation is an important part of plant cell wall metabolism, it is generally produced in the immediate vicinity of the required reaction (130). The hydroxyl radical has also been implicated in oxidative signaling necessary for cellular elongation in root hair growth (111), but again its production site must be tightly controlled in order for an appropriate targeted reaction to take place. An important part of the control of root cell growth by the gibberellic acid (GA)-mediated control of DELLA proteins is regulation of the extent of ROS accumulation through effects on the expression of antioxidative enzymes (1).

Singlet oxygen is even more difficult to measure than H_2O_2, though there is some evidence that it can also move between compartments (109). While recent evidence suggests that singlet oxygen can signal independently, it can be converted to other ROS such as H_2O_2, for example, by reaction with ascorbate (222). The extent to which signaling is singlet oxygen-specific (rather than secondarily transmitted via H_2O_2) presumably depends on how quickly specific secondary singlet oxygen signals are formed. Candidate second messengers for signal transmission to the nucleus include jasmonic acid (JA) and other derivatives of chloroplast fatty acid hydroperoxides whose production can be elicited by singlet oxygen (see Section V, E).

III. Control of ROS Accumulation in Plants

A. Cellular specificity of ROS control and signaling

ROS accumulation is controlled both by the rate of production and the rate of elimination by the antioxidative system, linked to components in a "reactive oxygen gene network" (275). Attempts to define such a network are complicated by the fact that ROS, as indicators of cellular redox state and reductant availability, are likely entwined with multiple cellular processes [*e.g.*, signaling through phytohormones and compounds such as JA, salicylic acid (SA), and ABA]. It may be difficult to establish whether a gene should be placed within or outside such a network. A further complication arising from this type of analysis of whole tissues is that they contain different cell types. This may mask many subtleties in the interactions between ROS signaling pathways, antioxidant genes, and their outcomes at the cellular level. In the animal stem cell, redox functions influence the balance between multiple processes related to self-renewal and differentiation (384). In plants, differentiated cells such as those of the leaf mesophyll are maintained at the G0 or G1 phase of the cell cycle and respond more rapidly to PCD triggers than dividing or meristematic cells. PCD was readily induced in response to the addition of the bacterial cell suicide-elicitor cryptogein at the S1 and G1 phases of the cell cycle in cultured tobacco BY-2 cells but suppressed in G2 and M phases (198). While little information is available on the genetic suppression of PCD in meristematic or dividing cells, it is clear that

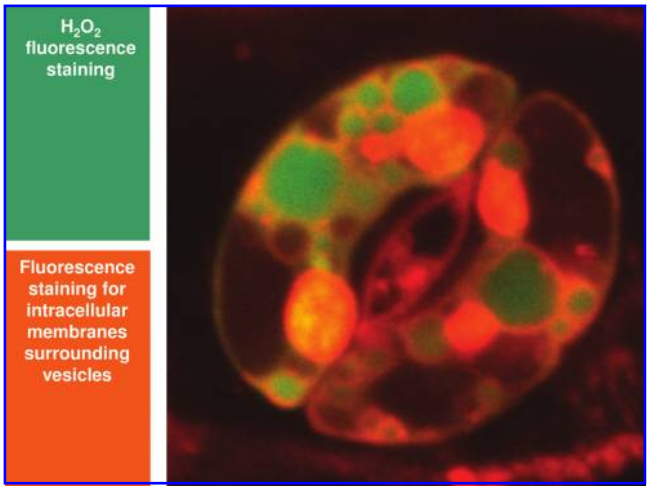

FIG. 6. H_2O_2 accumulation in the intracellular vesicles of guard cells when stomatal closure is triggered by ABA. The confocal image shows guard cells on the epidermis of a transformed Arabidopsis leaf with decreased expression of the intracellular vesicle protein (AtVAMP711), a manipulation that alters the size and number of visible vacuoles. The leaf had been treated with 20 μM ABA 2 h prior to the image analysis. The induction of H_2O_2 which is one of the earliest cellular responses to ABA in the guard cells, is shown using the fluorescent dye: 2′, 7′ - dichlorodihydrofluorecein diacetate (H2DCFDA; *green color*), which is a cell-permeant ROS indicator that is nonfluorescent until removal of the acetate groups by intracellular esterases in the presence of H_2O_2. The image demonstrates that H_2O_2 is localized in intracellular vesicles (endosomes) whose bounding membranes are visualised here using the potential-independent membrane dye, MitoFluorTM Red 589 (*orange-yellow*).

cell identity not only has a very strong effect on cellular redox state but it also determines the outcomes of ROS signaling pathways in different cell types (190), as well as their responses to abiotic stress (88).

The importance of cell identity in redox biology has been most studied in the root rather than the leaf, as root cell layers are more easily differentiated for comparisons between stem cells, which are capable of unlimited proliferation, self maintenance, and self renewal, and those in the root quiescent center (QC) and surrounding tissues (88, 189, 190). The root QC is established and maintained as a consequence of auxin that is transported in a polar fashion to the root tip, where it can accumulate to relatively high levels, leading to the oxidized state in the QC (188–190). Even though the root QC mitochondria are maintained in a highly oxidizing environment, they are indistinguishable from those in adjacent, actively dividing cells. Crucially, even in a highly oxidizing environment, the QC cells and their mitochondria do not enter PCD or senescence. Hence, there must be a mechanism for attenuating the ROS signaling pathways that would otherwise lead to PCD at the G1/G0 phase of the cell cycle, and such mechanisms may explain survival and enhanced stress resistance in mutant tobacco plants that lack mitochondrial complex I (101). QC cells may avoid entering cell suicide programs by actively modulating ROS signaling pathways. Moreover, the different cell layers in roots show specific gene expression responses to defense hormones such as ABA (88). Given the action of the growth-regulating DELLA proteins in the regulation of antioxidant gene expression (1), the inevitable conclusion is that the ROS signaling pathways of a given cell type and their consequences with regard to either PCD or enhanced defense are set not only by environmental stimuli but also by the genes that determine cell identity.

B. Multilevel control of ROS signal strength

For signaling to occur, depletion or withdrawal of certain antioxidative components may be necessary. Implicit within this concept is the notion that the signaling role of ROS extends beyond inducing antioxidative enzymes in a simple feedback loop. These loops may be useful models for ROS function in simple organisms such as prokaryotes, but recent data in plants overwhelmingly show that the roles of ROS are more subtle and complex (Fig. 7). As noted above, oxidized states may be crucial for certain cell functions, and for ROS to play a role in stress signaling the antioxidative system must be tightly controlled. Examples are microRNA-dependent downregulation of CuZnSOD expression (98, 394) and coordinated control of growth and stress tolerance through DELLAs (1). Other mechanisms involve stress and light-mediated effects on turnover or post-translational CAT regulation (367, 375, 425), post-transcriptional regulation or

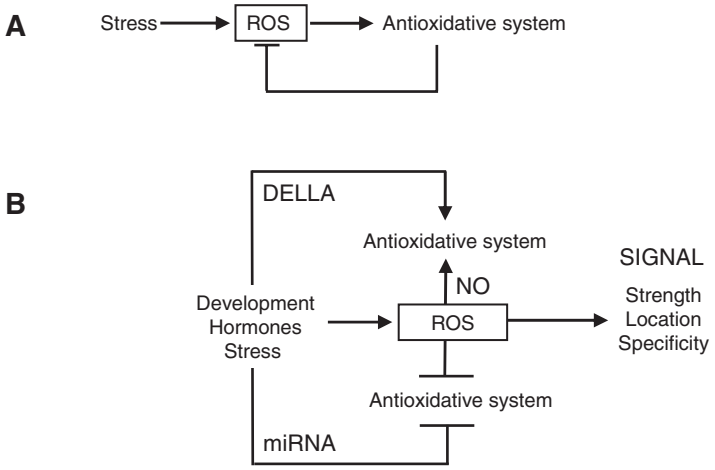

FIG. 7. ROS signaling in plants: subtle and complex. (**A**) Control of ROS concentration and antioxidant expression by simple regulatory circuits. This model may apply to prokaryotes and other simple unicellular organisms but is of limited validity for complex organisms, particularly plants. (**B**) In plants, ROS are at the heart of developmental and stress signaling, and the effects of different ROS are controlled at multiple levels, including rates of production, removal, and location. Recently described mechanisms affecting the activity of the antioxidative system include DELLA proteins, miRNAs, and NO (for further discussion, see text).

inactivation of ascorbate peroxidase (81, 274, 377), and overoxidation or nitrosylation of peroxiredoxins (87).

Four factors can be considered to be most important in determining ROS signaling strength: rates of production, compartmentation of ROS production (*e.g.*, in vesicles), rates of removal, and presence of receptors or detection systems. Sequestration of ROS in vesicles with weak antioxidant activity would partially overcome the requirement for downregulation of the antioxidative systems. Alternatively, selective downregulation of the antioxidant system may be crucial for signaling, for example, in PCD responses (136, 358). Another important aspect could be conditional failure to induce defensive patterns of gene expression in response to ROS, as observed in CAT-deficient plants growing in long-day conditions (341). While a transient dramatic decline in the activities of a cytosolic ascorbate peroxidase isoform (APX1) and peroxisomal CAT2 at the point of flowering has been suggested to lead to a temporary increase in leaf H_2O_2 that is important in the orchestration of senescence (471, 472), there are relatively few studies in the literature where changes in tissue antioxidant transcripts/activities and oxidants have been discussed in terms of signaling requirements.

C. Complexity of the plant antioxidative system

The ubiquity of ROS in plant metabolism no doubt explains the complexity and apparent partial redundancy of the antioxidative system in plants, which includes numerous classes of reducing metabolites as well as enzymes (275). Low-molecular-weight antioxidants are often described as molecules that are able to reduce oxidants without themselves having significant pro-oxidant action. Numerous classes of compounds found in plants have been discussed as antioxidants (*e.g.*, ascorbate, glutathione, tocopherols, carotenoids, flavonoids, and related phenylpropanoid derivatives, polyamines) and many other common molecules can be considered to have this property (*e.g.*, sugars and sugar alcohols). However, some of these compounds can also potentially be pro-oxidants, depending on the biochemical context and presence of enzymes such as oxidases. For example, signaling roles of apoplastic polyamine oxidase and polyamine-derived H_2O_2 have been described in defense and PCD responses to abiotic stress (279).

While singlet oxygen and the hydroxyl radical are thought to be essentially controlled by nonenzymatic detoxification systems, several enzymes play important roles in H_2O_2 removal. Enzymes that participate in antioxidative metabolism and related redox reactions can be distinguished as follows: (a) the protein acts as a primary antioxidative enzyme (*i.e.*, it uses superoxide, H_2O_2 or organic peroxide as substrates); (b) it is involved in maintenance of redox state (*e.g.*, regeneration of reduced forms of reductants); (c) it functions to control secondarily released metabolite signals (*e.g.*, conjugases). Within the first category, the best-studied primary antioxidative enzymes in plants are superoxide dismutases, CAT, and APX (Fig. 1). While the first two catalyze dismutation reactions, the third requires reductant in the form of ascorbate. Many other types of peroxidases exist in plants (44, 86, 182) as well as in other organisms (110). Among these, peroxiredoxins may have an important function alongside APX in H_2O_2 removal (87). Compared to the well-characterized APX, less information is available on the enzymatic properties of peroxiredoxins. Available data suggest that 2-cys peroxiredoxins could play a significant role in the chloroplast, where peroxiredoxin capacity to reduce H_2O_2 was reported to be about half that of soluble APX (87). Many other peroxidases may be more important in metabolism of organic peroxides (some thiol-based peroxidases), oxidation of organic compounds with possibly incidental H_2O_2 removal or, indeed, production rather than removal of H_2O_2 (other heme-based peroxidases). Attention has also been drawn to the possible roles of thiol-based peroxidase in signaling, rather than simple H_2O_2 removal (113).

The second category of antioxidative enzymes—proteins that supply or maintain reductant—includes enzymes such as dehydroascorbate reductase (DHAR), glutathione reductase (GR), and NADPH-generating dehydrogenases, as well as some glutaredoxins and thioredoxins, while the third category consists of enzymes such as glyoxylases, aldo/keto reductase, cytochrome P450s (CYPs), conjugase-type GTs, and glycosyl transferases.

D. The "induced by ROS" paradigm

Strictly, only enzymes that catalyze ROS removal or associated reductive reactions should be considered "antioxidative." Other criteria have been used, however, notably activities that diminish the probability of ROS production such as the mitochondrial alternative oxidase or genes that are induced by oxidizing conditions, though more general terms such as "cell rescue/defense" are often used to class all these enzymes together (*e.g.*, in transcriptomics studies). A concept that was established early in the development of the field is that plant antioxidative enzymes are generally induced by ROS (or stress) and that genes induced by ROS can be considered to have defense functions. As previously noted (292), this concept should be applied with caution. Many genes of the core antioxidant system are either not induced or only moderately induced by stress, and this may even be the case for enzymes that have a stress-specific function. Indeed, a recent meta-analysis of transcriptomics responses across a range of organisms showed that while external H_2O_2 induced antioxidative genes in unicellular organisms, responses were less clear or absent in multicellular organisms (418). This is consistent with the far greater subtlety of ROS functions in complex organisms (Fig. 7).

The lack of strong ROS induction of many antioxidant genes in plants may also reflect the importance of regulation of the antioxidant system at the post-transcriptional level (264, 316, 366). Many true antioxidant genes are quite strongly expressed even in nonstress conditions, either to deal with the high rates of constant ROS production that occur during photosynthesis, photorespiration, and respiration (Fig. 8), or in readiness for impending environmental fluctuations that are likely to quickly ramp up these rates (*e.g.*, changes in light intensity) (413).

Genes strongly induced by H_2O_2 include UDP-glucosyl transferases, CYPs, and glutathione transferases (341, 419). Many of these stress-specific genes have very low expression in optimal conditions and their functions in many cases remain to be established. These may include roles in secondary pathways producing signaling compounds or control of such compounds by modification and conjugation (235,

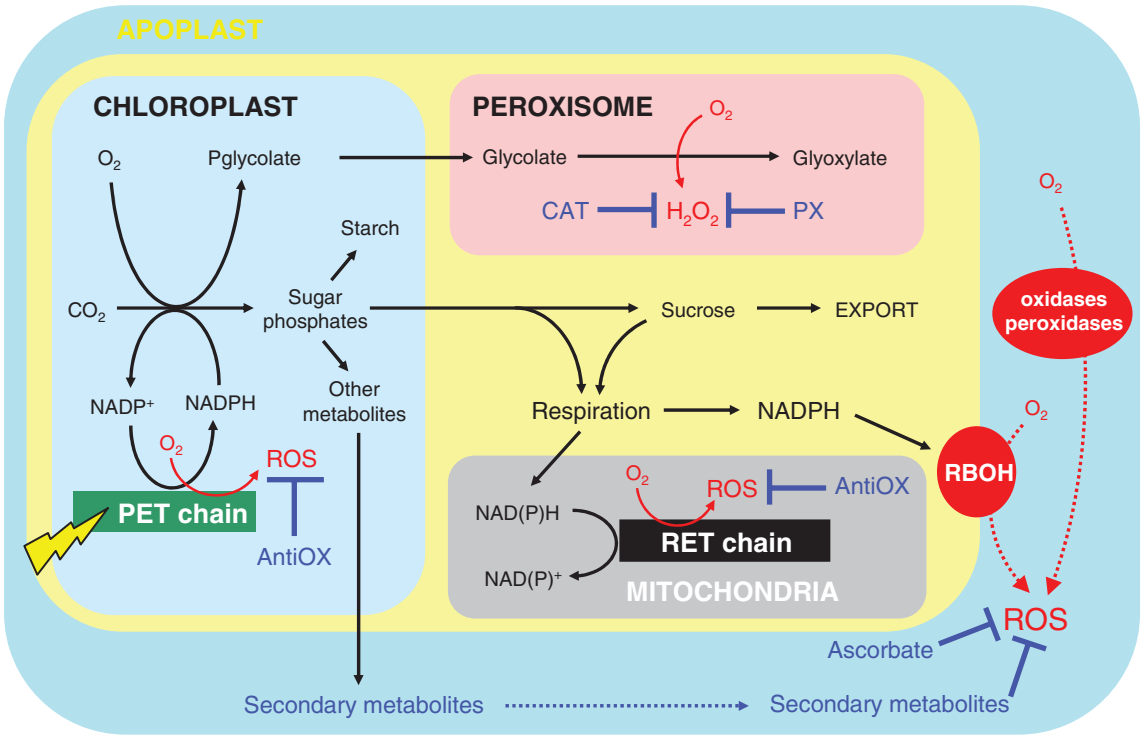

FIG. 8. Location of major paths of ROS production in photosynthetic cells. The scheme focuses on the most studied or quantitatively most important pathways, and situates these reactions within the major photosynthetic, photorespiratory, and respiratory functions of plant cells. AntiOX, antioxidative system; CAT, catalase; PET/RET, photosynthetic/respiratory electron transport; PX, peroxidases; RBOH, respiratory burst oxidase homolog.

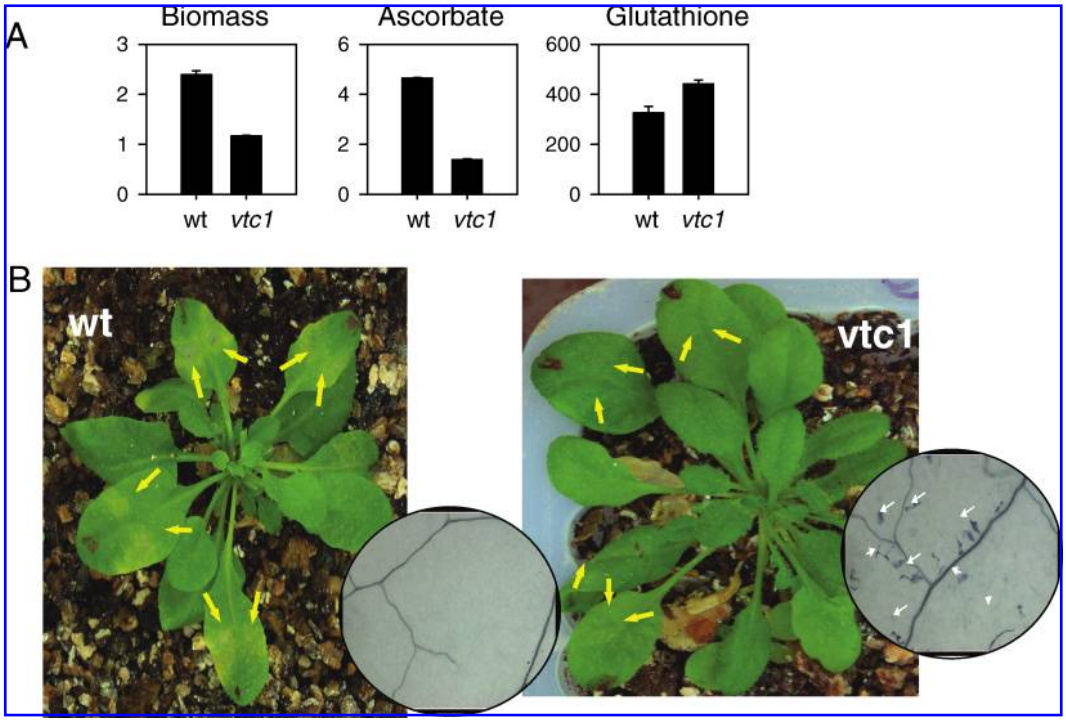

FIG. 9. The influence of ascorbate on growth and defense in Arabidopsis. Low ascorbate in the Arabidopsis *vtc1* mutant (**A**) is accompanied by a modest increase in leaf glutathione and is associated with slow growth and biomass production as compared to the wild type. The vtc1 mutant is more resistant to biotrophic pathogens that, upon inoculation, lead to spreading chlorotic patches on the leaves as illustrated by the *yellow arrows* in (**B**). The enhanced resistance to biotrophic pathogens is associated with the enhanced cell death phenotype that is illustrated in the *inserts* showing sections of the noninoculated control leaves stained for dead cells, which are indicated by the *white arrows*.

459). Glutathione transferases are further discussed in Section III,F,2.

E. Ascorbate and ascorbate peroxidases

In terms of cellular homeostasis and signaling, one important consideration is whether antioxidant enzymes require cellular reductants, as this property potentially allows ROS processing to exert secondary localized or more "bulk-phase" effects on antioxidant and reductant pools that could be sensed by the cell (291). In plants, ascorbate is by far the most abundant low molecular antioxidant. The interactions between ascorbate, superoxide and hydrogen peroxide in chloroplasts at the level of PSI (5), the enzymes of photosynthetic carbon assimilation (115) and the water–water cycle via APX and the reduction of monodehydroascorbate (MDHA) and dehydroascorbate (DHA; 15, 16, 115) have been extensively studied for many years. As well as a substrate for APX, ascorbate has numerous functions in plants, and this is reflected in its accumulation in many plant tissues to concentrations well above those required for APX activity. Ascorbate is a cofactor in various biosynthetic pathways and in the xanthophyll cycle which helps protects plants against the harmful effects of excess excitation energy (EEE) (114, 115, 292).

1. Ascorbate in plant development and signaling. Ascorbate is ubiquitous in eukaryotic organisms but is highly abundant in plants where it is clearly essential for development and growth regulation. Arabidopsis mutants with partly decreased ascorbate show slow growth and late flowering phenotypes (Fig. 9) while total depletion of ascorbate results in nonviable plants (94). The lethality of abolishing ascorbate synthesis in plants is explained by the multiple functions of ascorbate in plant growth and development as well as in metabolism and defense (292, 382).

Both the abundance of ascorbate and its redox state are regulated in plants in relation to the control of growth and development (28, 292, 318). Whereas dry seeds retain glutathione, they are depleted in ascorbate (223). Low levels of leaf ascorbate are associated with premature senescence (28) and enhanced susceptibility to PCD (318). Moreover, oxidation of ascorbate to DHA has been linked to decreased cell cycle activity (74, 79, 80, 243). Ascorbate and glutathione act in independent pathways to regulate the plant cell cycle (331, 332) and ascorbate cannot restore root growth to the *Arabidopsis thaliana rml1* mutant, which is deficient in glutathione (423).

Given the multifunctionality of ascorbate in plant biology, it is perhaps not surprising that it acts as a signal that modulates gene expression. Ascorbate deficiency leads to specific leaf transcriptome signatures (208, 317). Similarly, ascorbate has extensive interactions with phytohormone metabolism and signaling (317). For example, ascorbate interacts directly with the pathways of ABA synthesis and signaling, and also modulates downstream processes such as stomatal opening (60, 248).

2. Ascorbate synthesis and turnover. Animals and plants employ different pathways of ascorbate synthesis. The best studied pathway in plants is the L-galactose pathway (433), which is the only pathway in Arabidopsis leaves (94). The other pathways of ascorbate production in plants often involve carbon skeleton recycling networks (72, 382, 414). Ascorbate produced in leaves is not only accumulated in these organs but it is also loaded into the phloem through apoplastic and symplastic loading mechanisms. Ascorbate transported through the phloem may not only protect the vital systemic plant transport systems but also be important in plant–insect interactions. For example, plant ascorbate oxidase has been viewed as a defense protein that protects against herbivory as it decreases ascorbate that is essential for the efficient functioning of the herbivore's digestive system (27). Insects, like plants, have dedicated APX (261) and related enzymes (27). An intriguing and unexplored possibility is that leaf and phloem ascorbate contents might be an important determinant in the feeding choices of insects such as aphids.

Our knowledge of the complex network of environmental and metabolic factors that regulate ascorbate homeostasis in different tissues remains incomplete (30, 31). A key feature of plant ascorbate synthesis is the localization of the last enzyme of the pathway, galactonolactone dehydrogenase (GalLDH), in the mitochondria. This enzyme is physically associated with mitochondrial complex I and, since it uses oxidized cytochrome *c* as an electron acceptor, is also functionally linked to complexes III and IV (272). The capacity of plants to produce ascorbate is thus linked to the activity of the respiratory electron transport (RET) chain (272). The PET chain also exerts an influence on ascorbate synthesis and accumulation (453) and light quantity and quality are important determinants of leaf ascorbate contents (31, 245, 381). Leaf ascorbate contents decrease rapidly in darkness (31, 313). The accumulation of ascorbate in leaves during the light period regulates not only the rate of ascorbate degradation in darkness but also the amount transported to the cell wall/apoplast (421).

The factors that control ascorbate accumulation in fruit are different from those that operate in leaves. For example, while ascorbate synthesis decreases as leaves enter senescence, ascorbate production does not greatly decrease as fruit matures (194). Maintaining a high antioxidant status throughout fruit development is important as oxidative processes are often involved in the ripening process. The capacity to recycle reduced ascorbate from its oxidized forms (MDHA and DHA) is as vital to maintaining high tissue ascorbate contents as the capacity for synthesis (30, 61). Relatively few signaling molecules have been shown to influence the pathway of ascorbate synthesis. These include phytochrome (authors' unpublished observations), while JA has also been shown to alter the ascorbate synthesis through the regulation of the expression of genes encoding biosynthetic enzymes (443).

3. Ascorbate in the apoplast. Ascorbate is the only significant redox buffer in the apoplast, where it is very important in regulating cell wall synthesis and cross-linking, as well as defense against pathogens and atmospheric pollutants such as ozone. Like the thylakoid membranes (122) and the inner mitochondrial membrane (396), which has high affinity ascorbate transporter systems with K_M values ranging from 40 to 139 μM, the plant cell plasma membrane transports both ascorbate and DHA. However, to date no homologs of the animal-type sodium-dependent ascorbate transporters have been identified in plants. Ascorbate oxidase is uniquely localized in the apoplast/cell wall compartment, where it controls the redox state of the extracellular ascorbate pool (328,

329). Hence, the regulation of ascorbate homeostasis in this compartment is vitally important, not only to extracellular metabolism but also to sensing environmental perturbations and modulating the redox gradient across the plasma membrane. The ascorbate degradation pathway involves several novel intermediates including 4-O-oxalyl-L-threonate. Unlike the ascorbate synthesis pathway which is located inside the cell, degradation reactions have to date only been described in the extracellular compartment or surface (147).

4. Ascorbate peroxidases. While ascorbate oxidase catalyzes the oxidative production of MDHA using molecular oxygen, APXs catalyze the H_2O_2-dependent oxidation of ascorbate to MDHA (15, 16). Chloroplasts contain two APX isoforms, one localized in the stroma and the other bound to the stromal side of the thylakoid membrane by a C-terminal hydrophobic tail (15). The chloroplast APXs are also targeted to the mitochondria (62). Other APX isoforms, which are similar in sequence and structure to the chloroplast stromal form, are localized in the cytosol and peroxisomes (62, 73, 192, 274, 467). With few exceptions (405), the importance of the chloroplast and cytosolic APX forms to cellular redox homeostasis has been demonstrated. Impaired APX function enhances the susceptibility of plants to stress-induced oxidation while overexpression favors stress protection (73, 216, 217, 286, 319, 453).

Chloroplast APXs have a catalytic turnover number that is several-fold higher than that of the cytosolic forms (210). However, unlike the cytosolic forms, the chloroplast APXs are extremely susceptible to inactivation by H_2O_2, possibly because of the formation of a crosslink between heme and Trp35, which is located on the distal side of heme (211). When APX reacts with one molecule of H_2O_2, the ferric (FeIII) atom of heme is oxidized to the oxyferryl (FeIV = O) species, and a porphyrin-based cation radical intermediate is formed (compound I). The porphyrin-based radical is then reduced by an ascorbate molecule to form another intermediate (compound II). Its oxyferryl species is then reduced by a second ascorbate molecule to the ferric resting state. If ascorbate is absent and compound I is not reduced, the porphyrin-based radical is reduced by an amino acid residue of the apoprotein. As a result, the cation radical is transferred from porphyrin to the amino acid residues. APX inactivation is thought to be due to an attack on the reaction intermediates by H_2O_2 (210, 211).

F. Glutathione in plants

A high concentration of intracellular nonprotein thiols is closely related to aerobic metabolism (106) and in almost all eukaryotes, the major thiol is glutathione. In plants this metabolite has many roles that are also found in mammalian cells (thiol-disulfide buffering, peroxidase substrate, GT substrate, glutaredoxin substrate) but also others (phytochelatin synthesis, sulfur assimilation, and storage). Like ascorbate, glutathione is found predominantly in the reduced form in many compartments. Tissue GSSG contents often correlate with dormancy and cell death (224, 225), though GSSG can accumulate to high concentration in leaves without causing cell death though this is associated with much decreased growth (341).

1. Glutathione in peroxide metabolism in plants. There are important differences between the peroxidase function of glutathione in plants and in many animals. Plants lack a selenium-based glutathione peroxidase and thiol-dependent enzymes previously annotated as glutathione peroxidase in plants are now thought to use thioredoxins (168, 182). However, glutathione can also directly participate in peroxide metabolism as a substrate for glutaredoxin-linked peroxiredoxins (360) and peroxidatic GTs (427).

2. Glutathione transferases (GT). The GTs are a large group of enzymes found in both eukaryotes and prokaryotes which have evolved to fulfil diverse functions. They catalyze the *S*-conjugation of reactive compounds with GSH. Plants have many genes encoding GTs, and the major focus has been on their role in detoxification of xenobiotics driven by interest in herbicide action and the production of safeners. The major classes, both in terms of the sizes of their respective gene families and relative abundance of the encoded proteins, are the plant-specific tau (GTU) and phi (GTF) proteins. *Arabidopsis thaliana* (At) encodes 28 AtGTUs, 13 AtGTFs, as well as smaller gene families of the zeta AtGTZs and the theta AtGTTs. Moreover, the DHARs are now considered as a specific GT class, differing in their active site chemistries from other family members (89). Within the GT superfamily in plants, several transcripts are induced by H_2O_2, but the most strongly induced (on a fold-change basis) are not those that are predicted to have a direct antioxidative (peroxidative) function. These GTs (GTFs) are less strongly induced than certain GTUs, which are thought to have a conjugase function (427).

3. The ascorbate–glutathione link. Other than direct peroxidation catalyzed by the above enzymes, glutathione pools are also linked to H_2O_2 via the ascorbate–glutathione pathway in which ascorbate pools are maintained by glutathione-dependent reduction of DHA, as shown in Fig. 1 (115). This pathway exists in several cell compartments, including mitochondria and peroxisomes (62, 192), and has also been proposed to occur in animals. As discussed above, APX is found in at least four intracellular compartments and is encoded by a small gene family in Arabidopsis. Despite the fact that ascorbate, and not glutathione, is thought to be the major metabolite reductant for peroxidases in plants, a striking response to increased cellular H_2O_2 availability is often a dramatic increase in the total glutathione pool caused largely by the accumulation of GSSG (341, 351, 383, 436).

4. Glutathione synthesis and compartmentation. The first dedicated enzyme of glutathione synthesis, γ-glutamylcysteine synthetase (γ-ECS), is located in plastids in Arabidopsis (426). The enzyme is encoded by a single gene and knocking out its expression results in an embryo-lethal phenotype (55). Phenotypes and thiol perturbations in knockout mutants for the second enzyme, glutathione synthetase, which is encoded by a gene that produces proteins located in both plastid and cytosol, can be rescued by transformation with glutathione synthetase directed only to the cytosolic compartment (315). This implies that γ-EC and glutathione can be exchanged across the chloroplast envelope at rates sufficient to ensure normal plant function. Glutathione uptake by purified wheat chloroplast has been reported (296) and clues regarding the identity of the proteins responsible come from studies on the Arabidopsis mutant,

chloroquine-resistance-like TRANSPORTER1 (*clt1*), which was selected via resistance to the glutathione biosynthesis inhibitor L-buthionine-(*SR*)-sulfoximine (BSO). The CLT family in Arabidopsis consists of three members (CLT1, CLT2, and CLT3) and all these proteins reside in the chloroplast envelope. Triple mutant plants lacking functional copies of all three genes have glutathione levels similar to the control plants. However, the glutathione pool in the leaves of the triple mutant is restricted to chloroplasts with no GSH detectable in the cytosol. The expression of CLT1 in *Xenopus* oocytes confirmed that it is a γ-glutamylcysteine and GSH transporter (authors' unpublished observations). Other plant peptide transporters have also been identified that are able to transport glutathione (54).

There is no convincing evidence that glutathione is synthesized within plant mitochondria, yet recent work using immunolocalization reported high concentrations of glutathione in this compartment (464). Whereas mitochondrial uptake systems remain to be characterized in plants, it is interesting to note that plants have Bcl-2-like proteins, which regulate GSH transport into animal mitochondria and hence influence the essential pool of mitochondrial GSH that is involved in apoptosis in animal cells (470).

Tonoplast GSH uptake systems remain to be fully characterized, as do transporters capable of taking up glutathione conjugates (257, 401). Although neither GSH nor GSSG was detected in the large central vacuole of plant cells (464), it remains to be seen whether significant glutathione can accumulate in this compartment under certain conditions (*e.g.*, when GSSG formation is favored), as is particularly evident in plants exposed to ROS such as ozone or enhanced endogenous H_2O_2 (37, 341, 371) and which also occurs during plant–pathogen interactions and chilling (144, 280, 296, 415).

Synthesis of glutathione is controlled at multiple levels. Tissue glutathione contents can be increased by Cys supplementation (292) and overexpression of serine acetyl transferase, a key enzyme involved in Cys synthesis, increases glutathione as well as Cys contents (160). Downstream of Cys, much attention has focused on γ-ECS, as overexpression studies show that increases in this enzyme are sufficient to enhance glutathione contents (69, 294, 448) and control of the enzyme activity occurs at transcriptional and post-transcriptional levels (144, 264, 446). Post-transcriptional mechanisms include feedback regulation and thiol-disulfide regulation of the γ-ECS protein (169, 175) while mechanisms that regulate translation of γ-ECS transcripts have also been reported (447).

5. Glutathione degradation. Turnover of glutathione can occur either at the plasmalemma or in the vacuole (390). Turnover of the glutathione tripeptide in the vacuole is thought to be particularly important in the response to xenobiotics and other GT substrates. The first step may involve cleavage of glycine (carboxypeptidase) or glutamate (γ-glutamyltranspeptidase) and both activities have been reported in plant vacuoles (42, 153, 440). Transpeptidase activity shows differential tissue expression in corn (258) and genetic analysis is uncovering the tissue and subcellular specificity of the products of the four genes encoding these enzymes in Arabidopsis (153, 256, 303, 304).

6. GSSG concentrations and glutathione redox potentials in plants. As well as the key influence of the GSH/GSSG ratio, the glutathione redox potential is modulated by total glutathione concentration. Genetic and other evidence shows that glutathione concentration is important in meristem function, light signaling, and pathogen responses (25, 96, 128, 228, 263, 314, 346, 415, 423). Within the context of plant responses to pathogens it has been shown that both the cytosolic regulatory protein, NPR1, and the specific TGA-type transcription factors with which NPR1 interacts, are controlled by disulfide reduction (84, 280). While NPR1 is the only redox active protein of its type described to date (see Section IV, E, 2), it would be rather surprising if there were not other GSH or redox-modulated cytosolic proteins and transcription factors that mediate gene expression in a reversible manner through thiol–disulphide exchange reactions or glutathionylation. Moreover, while net GSH movement from the cytosol to the nucleus has not been described to date, it is possible that plant cells, like their animal counterparts, regulate GSH movement between these compartments to regulate gene expression. GSH movement from the cytosol to the nucleus is important in the control of progression through the mammalian cell cycle (253). GSH was located mainly in the nucleus of proliferating fibroblasts but only in the cytosol when the cells reached confluence (253).

The $NADP^+$/NADPH and GSH/GSSG midpoint potentials are separated by $\sim$90–100 mV. If these couples are indeed at thermodynamic equilibrium and glutathione is in the 2–5 m*M* range, a typical $NADP^+$/NADPH ratio of 1 (340) should co-exist with a $[GSH]^2$/GSSG ratio of $\sim$1,000, equivalent to GSSG in the low nanomolar range. Redox-sensitive GFP proteins have been introduced into plants by transformation (191, 268) and suggested to faithfully report on the cytosolic glutathione redox potential *in vivo*, yielding redox potential values below -300 rather than close to -200 mV. Such values imply that cytosolic GSSG concentrations are indeed in the nanomolar range or lower. Since the total glutathione concentration is in the millimolar range (265, 291), this means GSH/GSSG equal or greater to 10^6. A decrease in this ratio to 10^3 would represent an increase in absolute GSSG concentrations of $\sim$1,000-fold, a change extremely difficult to measure in homogenized extracts with commonly used techniques. Since the glutathione redox potential becomes $\sim$30 mV more positive for each 10-fold decrease in GSH/GSSG, the potential would increase $\sim$90 mV (Fig. 10), potentially promoting oxidation of thiol groups with midpoint potentials between the two values. A redox potential change of $\sim$90 mV is within the range of that required to achieve protein thiol/disulfide exchange in the well-known light–dark regulation of stromal enzymes by thioredoxins (372). Therefore, one enticing argument in favor of very low *in vivo* concentrations of GSSG in some compartments is that they would enable high signaling sensitivity to be achieved by relatively small absolute changes in GSSG concentration. However, it remains to be established whether glutathione-linked redox signaling necessarily involves thermodynamically-driven changes, or is rather more influenced by other processes (*e.g.*, glutaredoxin expression or activity, NO production).

In yeast, the cytosolic GSH:GSSG ratio is considered to be $\sim$100 (408), while numerous measurements in plant extracts indicate that even in the absence of stress, $\sim$5% of tissue glutathione is found in the GSSG form. At physiological glutathione concentrations, these ratios would predict a

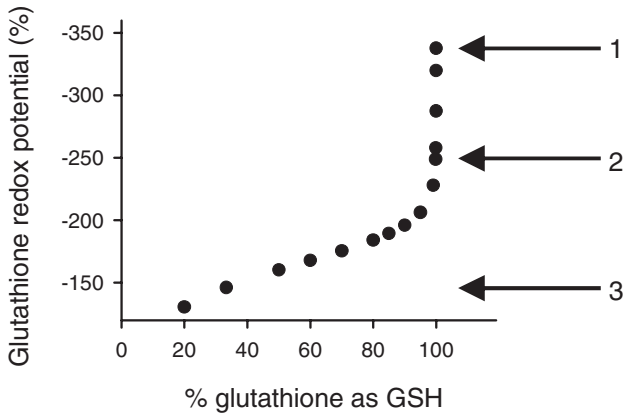

FIG. 10. Relation between glutathione redox potential and the fraction of glutathione as GSH. Total glutathione concentration is assumed to be 2 m*M*. *Numbered arrows* in (**A**) indicate 1. GSH/GSSG = 10^6. 2. GSH/GSSG = 10^3. 3. GSH/GSSG = 1. Changes in total glutathione do not appreciably change the shape of the curve, which is shifted to more negative values as concentration increases [for example, see Fig. 3 in Noctor, 2006 (291)]. If 99% of the total pool is GSH, the calculated redox potentials for 1, 2, and 5 m*M* glutathione are −219, −228, and −240 mV, the last corresponding to the midpoint potential at this concentration.

"global" redox potential close to −200 mV rather than −300 mV. However, even when used carefully, extraction techniques can cause some artifactual formation of GSSG. A key factor is careful control of the pH during sample preparation. In addition to possible artifactual effects, the GSSG that is detected *in vitro* may be preferentially sequestered in specific compartments *in vivo* (*e.g.*, vascular tissues or the vacuole). Certain tonoplast ABC transporters perform import of GS-conjugates into the plant cell vacuole (124). As glutathione disulfide is a GS–SG conjugate, these proteins may also be able to transport this form of glutathione (401), either exclusively or in preference to GSH. Such processes could play a significant role in avoiding excessive GSSG accumulation in the cytosol, yet concrete evidence is lacking.

G. The ascorbate–glutathione cycle, thiol–disulfide exchange, and oxidative protein folding

Many plant metabolic pathways are regulated by the enzymatic reduction of disulfide bonds in redox-regulated proteins (51, 370). Thiol/disulfide exchange reactions are particularly important in the regulation of metabolism in the chloroplasts, where over 90 thioredoxin targets have been identified (239). The preference of the disulfide-containing proteins for reduction by dithiol reductants, such as thioredoxin, provides a mechanistic basis for resisting reduction by GSH and similar compounds in the chloroplast environment. The different kinetics of monothiol *versus* dithiol reductants may be responsible for the stability of chloroplast disulfides. While some chloroplast proteins may have particular thiols that readily react with GSSG, as discussed above, the high preferential reduction of regulatory disulfides by thioredoxin rather than glutathione might be a mechanism allowing these bonds to be formed and regulated independently of ascorbate and glutathione pools. Since enzymes are required to transfer the electrons from target proteins to O_2 in the cellular disulfide-forming systems, one would predict that thiol oxidation would also be enzyme-regulated in the chloroplast. While the oxidizing components remain to be established, peroxiredoxin and glutathione peroxidase are probably not alone in being able to accept electrons from thioredoxin in the dark (370). Little is known about how the processes that form intermolecular disulfide bonds in chloroplast proteins in the light. However, in response to sucrose, cytosolic trehalose 6-phosphate stimulates dimerization of the ADP-glucose pyrophosphorylase protein via the formation of an intermolecular disulfide bond in the light (164, 215), that inhibits activity in a similar mechanism to that described for acetyl CoA carboxylase (221). Such reactions may be catalyzed by thiol oxidases, oxidative-type thioredoxins, or chloroplast protein disulfide isomerase-like proteins, as described in other organisms (349).

IV. Redox Compartmentation, Exchange, and Signaling

With ferredoxin, NADPH is the major driver of chloroplast assimilatory metabolism. These reductants are produced by the chloroplast PET chain and, together with ATP, used to

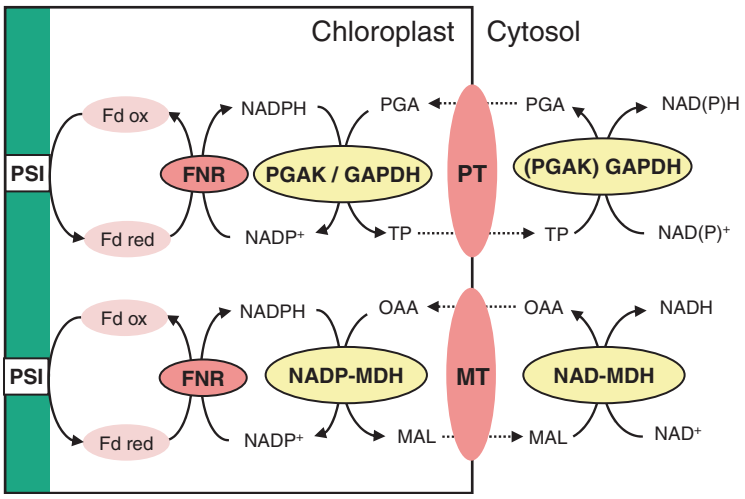

FIG. 11. Key chloroplast envelope redox shuttles. Fdox/Fdred, oxidized/reduced ferredoxin; FNR, ferredoxin-NADP$^+$ reductase; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; MAL, malate; MDH, malate dehydrogenase; MT, malate transporter; OAA, oxaloacetate.PGA, 3-phosphoglycerate; PGAK, PGA kinase; PSI, photosystem I; PT, phosphate translocator; TP, triose phosphate.

generate sugar-phosphates, amino acids, and many other metabolites that are then supplied to the rest of the cell (Fig. 8). As the main factory of the photosynthetic cell, the chloroplast drives much of its redox changes and mechanisms are known through which the influence of the PET chain could extend far beyond the redox state of the chloroplast stroma. These include potentially high-capacity redox shuttles across the chloroplast envelope.

A. NADPH compartmentation and shuttles

In addition to its function in biosyntheses, NADPH is a key factor underlying redox signaling and maintenance of cell homeostasis. This coenzyme is required for regeneration of both ascorbate and glutathione pools (Fig. 1), as well as NADPH oxidase activity, reduction of thioredoxins, and many other reactions of primary and secondary metabolism (291). Perhaps because of the primary focus on the chloroplast, less attention has focused on cytosolic NADP status in plant stress responses than in redox signaling in animal cells (441). In plants, redox gradients in NAD(P) status exist between chloroplasts, mitochondria, and cytosol (180). However, the redox states of compartments are not fixed and are likely to change with environmental fluctuations, in particular the balance between light availability and the capacity of metabolism to use light energy. Controls within the chloroplast include feed-forward mechanisms such as activation of metabolism by thioredoxins reduced in a light-dependent fashion by the PET chain (Fig. 4; 369, 442). Mechanisms exist to decrease light capture efficacy and constrain electron transport when light is in excess of metabolic capacity. Stromal ATP utilization is linked to light capture efficiency via ΔpH-dependent non-photochemical quenching (78, 173, 289, 293) and it has recently been proposed that NADP redox status could also feed back to regulate PET activity (155). Although the primary impact of changes in light availability is in the chloroplast, effects on NAD(P) status in extra-chloroplastic compartments are also possible (180). This could be important in relaying light signals to both the cytosol and could also affect mitochondrial redox state and regulation. A number of mitochondrial thoredoxin targets have been identified (26) but the factors that determine their redox state remain to be elucidated.

1. High-throughput redox exchange from the chloroplast. As well as possible direct movement of ROS or secondarily produced signals from the chloroplast, stress-induced changes in chloroplast metabolism could be transmitted by enhanced redox shuttling or other transporter activity. If this leads to promotion of increased substrate for the RET chain or NADPH oxidases, it would allow light-driven chloroplast processes to contribute to H_2O_2 production elsewhere in the cell. Attention has been drawn to interactions between apoplastic/plasmamembrane and chloroplastic events, for instance during ozone stress or plant–pathogen interactions (33, 136, 197). Examples of extra-chloroplastic NADPH-linked enzymes important in redox homeostasis and signaling are GR, NADPH-thioredoxin reductase, and plasmamembrane-bound or vesicular NADPH oxidases. While pyridine nucleotides do not cross the inner chloroplast envelope membrane at rates that are comparable to chloroplast NADP redox cycling, this membrane has shuttle mechanisms to allow high-flux exchange of reducing equivalents (Fig. 11), potentially linking changes in stromal and extra-chloroplastic NADP redox states. Key enzymes are malate dehydrogenases (MDH) and glyceraldehyde-3-phosphate dehydrogenases (GAPDH). The function of such "valves" may be double, as they could both relieve electron pressure in the chloroplast and signal overreduction to the cytosol/nucleus (171). Chloroplast NADP-MDH is known to be regulated by thioredoxins and NADP redox state (271,364) and recent data have shown that both chloroplast and cytosolic GAPDHs are subject to glutathionylation (171,462).

2. Production of NADPH in the cytosol. Whether the exchange reactions shown in Fig. 11 contribute directly to production of either NADH or NADPH depends on the specificity of the cytosolic dehydrogenase. In terms of NADPH production, one example of a cytosolic enzyme whose physiological role merits some attention is the nonphosphorylating glyceraldehyde-3-phosphate dehydrogenase (npGAPDH). This enzyme oxidizes glyceraldehyde-3-phosphate directly to 3-phosphoglycerate (without the 3-phosphoglycerate kinase reaction) and produces NADPH rather than NADH (206). An Arabidopsis knockout mutant for the single predicted cytosolic npGAPDH gene in this species showed higher leaf NADPH contents and enhanced staining for superoxide (350).

Cytosol NADP redox state could also be influenced by consumption by the mitochondrial electron transport chain, reactions made possible in plants by NADPH dehydrogenases localized in the inner mitochondrial membrane with catalytic orientation towards the cytosol (244, 344). Calcium signaling is known to be tightly intertwined with ROS responses in plants (105). At least some mitochondrial NADPH dehydrogenases are calcium-regulated (244), a property they share with enzymes such as NAD kinase and NADPH oxidases, as well as other enzymes potentially influential in determining cytosolic NADP(H) status (291).

While the cytosolic GR has been well characterized biochemically (387), much less information is available on NADPH oxidases. Neither their capacities nor their kinetic characteristics are well known in plants. It has been considered that calmodulin-regulated cytosolic NAD kinase is important in maintaining NADP pools for NADPH oxidase activity during plant–pathogen interactions (159). As well as NAD kinase, recent work has described kinases that can use NADH to produce NADPH directly (410). Although rates of NADP redox turnover in the cytosol (Fig. 12) are less easy to estimate than in the chloroplast (where approximate values can be inferred from photosynthetic rates), they are probably at least as fast as NADP synthesis and degradation reactions. There is likely to be some redundancy in NADPH generation given that several genes encoding cytosolic $NADP^+$-linked dehydrogenases may be simultaneously expressed (170, 351, 412, 429, 430). Recent work on pea nodules has provided some evidence for differential responses to oxidative conditions (252).

B. Photorespiration-linked redox exchange

Photorespiration is a chloroplast-initiated process that has significant effects on plant growth, yield, and ecological distribution. Flux through the pathway is light dependent, greatly increased by warm temperatures, and further favored

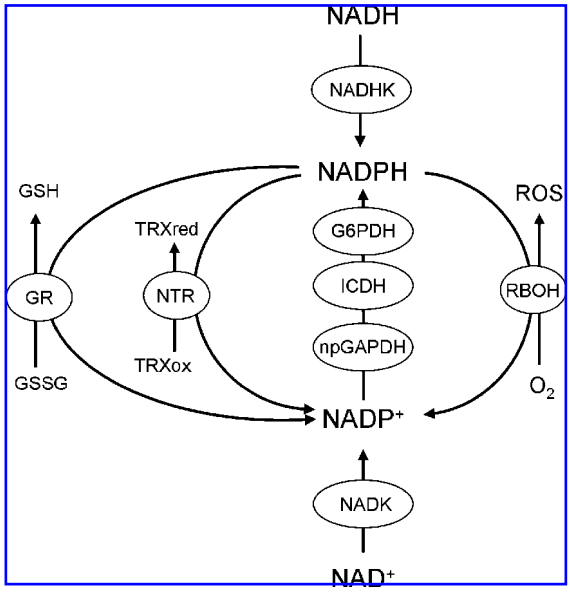

FIG. 12. Major NAD(P)H-producing and consuming reactions in the cytosol. G6PDH, glucose-6-phosphate dehydrogenase; GR, glutathione reductase; GSH, glutathione; GSSG, glutathione disulfide; ICDH, NADP-isocitrate dehydrogenase; NAD(H)K, NAD(H) kinase; NTR, NADPH-thioredoxin reductase; npGAPDH, nonphosphorylating glyceraldehyde-3-phosphate dehydrogenase; RBOH, respiratory burst oxidase homolog; TRXox/TRXred, oxidized/reduced thioredoxin.

by any condition that promotes stomatal closure (*e.g.*, drought). Although photorespiration is not very rapid in low light conditions or at low temperatures, it is in many other conditions a high-flux pathway whose complex nature means that it is likely to impact on redox status both inside and outside the chloroplast. The photorespiratory pathway essentially involves metabolism of glycolate to glycerate and CO_2 through reactions occurring in the peroxisomes, mitochondria, and chloroplasts. Metabolism of glycolate in the peroxisomes occurs through glycolate oxidase which produces H_2O_2, and this could be the major source of H_2O_2 in many types of photosynthetic cells in the light (117, 295). This H_2O_2 is mainly metabolized by a specific CAT isoform (CAT2 in Arabidopsis, CAT1 in tobacco), and knockdown and knockout lines for these enzymes in barley, tobacco, and Arabidopsis have clearly demonstrated that photorespiratory H_2O_2 is sufficient to produce characteristic changes in leaf glutathione pools and to induce oxidative stress transcripts and pathogen-linked reactions including, under some conditions, cell death (56, 341, 351, 383, 397, 419).

Glycine formed in the peroxisomes is subsequently metabolized to serine, CO_2, and NH_3 in the mitochondria, and this is linked to the production of NADH in the mitochondrial matrix at rates that in many conditions likely exceed those occurring through TCA cycle reactions. Glycine oxidation is thus an important process driving light-dependent changes in mitochondrial NAD redox state (180, 181). While energetically overshadowed by chloroplasts in photosynthetic cells, mitochondria play crucial roles in intracellular pyridine nucleotide redox shuttles, and thus metabolic integration, optimization of photosynthesis, and stress responses (100–102, 244, 297, 299, 301, 344, 348, 352).

C. Location and redox gradients in stress response specificity

The orchestration of appropriate acclimation responses requires a multilevel information cascade that starts with signal perception, through signal transduction and amplification, to induce primary and secondary responses in gene and protein function. Unlike many other signaling molecules, evidence has been found for ROS participation at all stages. However, questions remain regarding specificity and location. Different stress conditions are likely to produce different ROS or at least to produce them through different pathways, and this will also depend on cell or tissue type. However, while simple physiological considerations identify *a priori* favored pathways in different conditions, the picture is obscured by secondary, possibly signaled, effects. For example, ozone exposure not only causes ROS production on dissolving in the apoplast but also through activation of secondary reactions involving NADPH oxidases and organelles such as the chloroplast (197, 312), while recent work has shown that pathogen responses involve the mitochondria (101, 136, 352).

Whether ROS accumulation occurs through metabolic accumulation or through regulated bursts linked to activation of appropriate enzymes, the location of ROS accumulation is crucial in determining the secondary biochemical events and the physiological outcome. For example, lipid peroxidation is a primary event following ROS accumulation in the chloroplasts, whereas stomatal closure is the primary event following ROS accumulation in the guard cells as a result of ABA action (230). The ABA-dependent formation of superoxide and H_2O_2 is dependent on the production of superoxide via the same RbohD and RbohF NADPH oxidases that are involved in the pathogen response (230, 402) but no one to date has suggested that stomatal closure results from oxidative damage or that ABA signaling causes oxidative stress. These observations emphasize the gross oversimplification of the concept that suggests that low ROS concentrations lead to signal transduction and acclimation/defense responses while high levels lead to oxidative damage to lipids, DNA, and protein, and, ultimately, cell death. Location and timing are likely the key factors. The extent of synchronicity with parallel or synergistic events such as calcium signatures or circadian gene expression may be an important determinant of the outcome.

D. ROS signal perception and transduction

Signal perception includes physical and chemical interactions between the stimulus and a sensor or receptor. For example, surface receptor-like protein kinases (RLKs) are considered to play a fundamental role in sensing the external environment and initiating the signal transduction process, with as yet largely unidentified members of the plant RLK super family classified as leucine-rich repeats (LRR) initiating abiotic stress and ABA responses (311). It is currently considered that ROS produced at the cell surface signal through oxidative modification of proteins such as sensor kinases or gated Ca^{2+} channels and/or lipids and phospholipases that may be proximally sited to the producing enzymes. Kinases that are both induced by ROS and necessary for oxidative signal transduction have been identified (12, 13, 347) and oxidative signals are transmitted in plants as in animals by MAP kinase cascades (14, 220, 466).

No "ROS receptor/sensor" has as yet been clearly identified in plants. Given their reactive power, ROS could exert effects on numerous components through the effects discussed below in Section V,C, though transcriptional "master switches" likely control expression of multiple genes in response to given ROS signals. A recently characterized transcription factor controls nuclear expression of some chloroplast antioxidative enzymes through thiol/disulfide exchange (374), while another transcription factor, bZIP10, shuttles between the nucleus and cytosol, where it interacts with the cell death suppressor, LSD1, and thus influences cell death in response to pathogens (200; see also Sections VII,B and VIII,B). This latter system shares functional similarity with regulation through the NFκB-B/IκB and Keap1-Nrf2 systems (374), as does the action of plant NPR1 (280).

E. Oxidative and reductive signaling, oxidative and reductive stress?

1. GSSG as an oxidative signal. We have previously emphasized that ROS production, pyridine nucleotides, and antioxidants are intimately connected (118, 291), and glutathione status in particular has been proposed to be important in relaying oxidative signals originating from H_2O_2 or other ROS (125, 265). Either GSSG concentration or the glutathione redox potential could be sensed via protein thiol/disulfide exchange or glutathionylation of proteins. It is clear that GSSG can accumulate to high levels in plants. This has been observed in stress conditions such as pathogen attack, ozone, and cold (37, 144, 263, 371, 415). The intimate relationship between H_2O_2 and glutathione is evident from the spectacular effects on the glutathione pool observed in plants deficient in CAT (275, 292, 341, 351, 383, 436). In these systems, GSSG can accumulate to values as high as 1 μmol.g^{-1} leaf fresh mass, representing a global tissue concentration of $\sim$1 m*M* or up to 90% of the detectable glutathione pool (341). In Arabidopsis at least, this huge accumulation can occur without detectable tissue death (341). The role of APX is usually considered, like CAT, to be limited to H_2O_2 removal (Fig. 1). However, APX deficiency partly suppresses the phenotype of CAT-deficient plants (351).

GSSG is unlikely to accumulate to levels that are measurable in extracts simply because of thermodynamic factors or limitation by NADPH supply. The K_M for NADPH of glutathione reductase is $<$10 μM (103, 156), whereas free NADPH levels are probably 100 μM or greater (180). Current concepts suggest that GSSG accumulation occurs because the capacity of GR is insufficient to keep pace with other enzymes involved in H_2O_2 detoxification, either because these enzymes have higher capacities (total extractable leaf activity of APX is typically $\sim$10-fold higher than that of GR) or because other reactions are catalyzed by several enzymes with partial redundancy. Strong limitation by GR under conditions of increased peroxidatic removal of H_2O_2 may be important to allow rapid and sensitive changes in GSH/GSSG, as discussed above in Section III,F,5. Such changes could then be signalled via glutaredoxin modification of sensor proteins.

2. Reductive signaling in plants. Regulation of chloroplast enzymes by dynamic changes in redox potential has been known in plants for several decades (10, 97, 442), and involves the thioredoxin-mediated regulation of enzyme activity in response to light/darkness (369, 370). Work over the last decade has identified an ever-growing list of potential thioredoxin targets (370). Thioredoxin regulation is now known also to occur in other plant cell compartments such as the cytosol and mitochondria (26, 233, 269, 346), and is mediated by a family of proteins that can be classed into different groups (239, 269). Certain plastidial types of thioredoxins play a role in peroxiredoxin-linked peroxide metabolism (48, 66, 424). The physiological and environmental factors that determine thioredoxin function in extra-chloroplastic compartments remain to be fully characterized, though in photosynthetic cells these functions may be influenced by chloroplast and apoplastic events (*e.g.*, by redox shuttles, as described in Section IV,A). At least one cytosolic thioredoxin is implicated in plant responses to pathogens (234), and reductive signaling has been described for the NPR1/TGA transcription factor interaction in the regulation of the expression of pathogenesis-related (PR) genes, which are induced in plants subsequent to certain types of pathogen challenge (84, 280).

Like the mammalian Nrf2-binding protein, KEAP1, NPR1 contains the BTB/POZ domain that is associated with dimerization and interaction with Cullin-3 proteins, and this domain contains one of the two Cys responsible for NPR1 redox sensitivity (166, 280). The other Cys is situated in the link region between the BTB/POZ domain and the ankyrin repeat domain necessary for interaction with specific TGA-type bZIP transcription factors (166). In addition to NPR1, five NPR1-like genes exist in Arabidopsis. Recent data suggest that NPR3 and NPR4 also function in pathogen responses, though in an antagonistic fashion to NPR1 (469). Though NPR3 and NPR4 have BTB/POZ and ankyrin repeat motifs, they lack the Cys residue shown to be essential for retention of NPR1 in the cytosol (280, 469), suggesting that they may be redox-insensitive. Two other members of the NPR1 family, BOP1 and BOP2 (for BLADE-ON-PETIOLE), contain both conserved Cys residues found in NPR1 but they interact most strongly with a TGA transcription factor that does not interact with NPR1 (166). Double homozygous *bop1 bop2* knockouts show defects in floral patterning. Although redox-dependent movement of BOP1 and BOP2 has not yet been described, it is tempting to suggest that thiol/disulfide regulation could be a very important in the regulation of plant development as well as in stress responses. This hypothesis is entirely consistent with data obtained for mutants in glutathione synthesis and NADPH-thioredoxin reductase (346, 423). Further evidence of a role for thiol/disulfide exchange in plant development comes from the recent demonstration that ROXY1 and ROXY2, two glutaredoxins with active site motifs found only in terrestrial plants (238), play overlapping but essential roles in flower formation (451). Together with observations on the bZIP10-LSD1 interaction (200), the evidence from studies of NPR1 suggests the existence of central cytoplasm-nuclear shuttling mechanism for redox-sensitive proteins that operates in both oxidative and reductive signaling cascades.

Though some thioredoxin-regulated chloroplast enzymes are activated by thiol oxidation (10), most are activated by disulfide reduction. This involves increased reduction state of a pre-existing pool of thioredoxins mediated by light-driven reduction of ferredoxin (372), contributing to the regulation of stromal metabolic pathways but also to the transfer of reducing power from the stroma to the rest of the cell via the

NADP-dependent malate dehydrogenase (Fig. 11; 18, 171, 271, 364). The mechanisms that drive the cytosolic reductive signaling described above remain to be elucidated. If activation of NPR1 and similar proteins occurred solely through induction or recruitment of thioredoxins or glutaredoxins with appropriate affinities for specific protein targets, this kind of redox regulation could occur independently of changes in the redox potential of soluble reductant pools (NADPH, glutathione). In this case, only a threshold concentration of reductant would be necessary. A distinct (or additional) possibility is that certain stresses induce dynamic changes in cytosolic NADP or glutathione pools. This would be analogous to light effects on stromal thioredoxin pools, and could in fact require light-driven reductant export from the chloroplast, explaining some of the light dependence for certain stress responses. Although improved intra- and intercellular spatial resolution is necessary to fully understand the key events, there is some evidence that dynamic changes in thiol status occur during pathogen responses (280, 296, 415). It may also be significant that increasing glutathione, either by exploiting plant transformation or by chemical intervention, is sufficient to mimic induction of target genes by SA (69, 119).

3. Reductive stress in plants? The effects of dithiothreitol (DTT) on yeast cells have been described as "reductive stress" and some yeast genes such as thioredoxin2 can be induced by both H_2O_2 and DTT (408). Insufficiently oxidizing conditions can interfere with protein folding in the endoplasmic reticulum, mediated in plants, fungi, and animals by protein disulfide isomerases oxidized by homologous *e*ndoplasmic *r*eticulum *o*xyreductins such as Ero1 (90, 373). However, the importance of reductive stress in compartments such as the cytosol, chloroplast stroma, and mitochondrial matrix, which maintain a highly reducing environment, remains to be determined. Studies of knockout mice have reported that enhanced GSH:GSSG ratios driven by upregulation of both G6PDH and GR are associated with defects in cardiac muscle (343).

Two concepts of reductive stress can perhaps be distinguished. In the first, over-reduction of redox-active compounds would favor production of ROS. Such effects are well described in plants at the level of electron transport chains and reflect increased oxidative burden (ROS production rates) caused by over-reduction of autooxidizable compounds (Fig. 4). The second type of reductive stress could involve modifications of protein function through a drop in the redox potential of pyridine nucleotides, thioredoxins, or glutathione. Interestingly, engineering-enhanced contents of glutathione in the tobacco chloroplast led to a marked drop in the GSH/GSSG ratio accompanied by other symptoms commonly considered to be "oxidative stress" (69), though other data suggest that this effect of increased chloroplastic glutathione synthesis is not general to all plants (294). Nevertheless, "over-reduction" in specific compartments could play a vital role in gene regulation through components such as NPR1 and may partly explain some results in the literature. Knockout Arabidopsis lines for leaf-expressed NADPH oxidases show compromised ability to limit cell death in response to pathogens (404). This may reflect the absence of a ROS "survival" signal produced by NADPH oxidases at the cell surface (see also Section VI,F). Alternatively or in addition, it could partly result from over-reduction of cytosolic NADP pools. According to this second view, NADPH oxidases would under some conditions function as alternative respiratory pathways, much like oxygen reduction by the chloroplast PET chain, and their physiological "functions" would include both relieving reductant pressure and producing ROS (Fig. 13). Further information is required to resolve this issue (*e.g.*, regarding the capacities of plant NADPH oxidases). Another key issue is to what extent ROS produced at the cell surface or in the apoplast interact with intracellular antioxidative systems (Fig. 8). However, it should be noted that even if these ROS are metabolized exclusively at the cell exterior, this process is likely to involve ascorbate and thus affect intracellular events because DHA produced outside the cell has to be reduced to ascorbate by intracellular systems.

V. The ROS–Antioxidant Interaction as a Stress Convergence Regulator

The notion of uniform "oxidative stress responses" in plant cells is a highly simplistic generalization because, as noted in

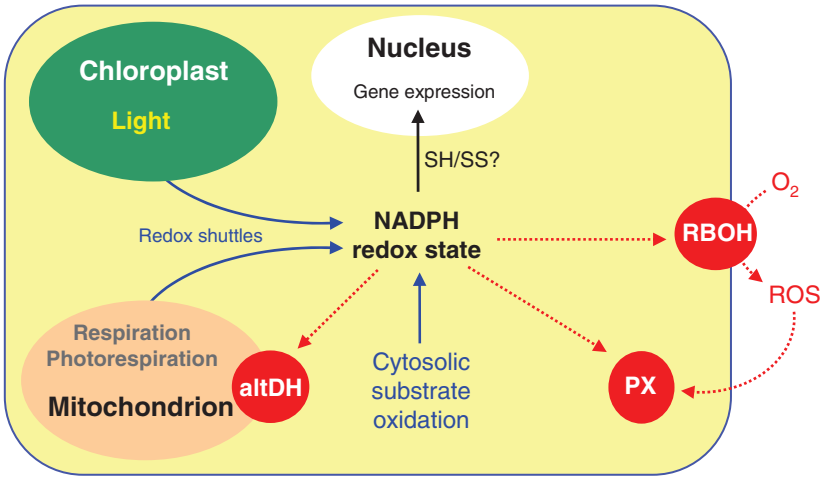

FIG. 13. Cytosolic NADP redox state: At the hub of reductive signaling? NADPH can be produced in the cytosol (*blue arrows*) by substrate oxidation (Fig. 12) or by export of reductant from the chloroplast and mitochondria through shuttles that are particularly active during photosynthesis (Fig. 11). As well as biosynthetic processes, NADPH is required for respiratory burst oxidase homolog (RBOH) activity located at the plasmalemma and peroxidase-linked H_2O_2 reduction. Among other components, alternative mitochondrial NADPH dehydrogenases (altDH: 244, 344) may also be important in influencing cytosolic NADP redox state through oxidation (*red arrows*). Although not shown in this figure, ROS produced in the chloroplasts, mitochondria, and peroxisomes could also impact cytosolic NADP redox state (Fig. 8). PX, peroxidase; RBOH, respiratory burst oxidase homolog. SH/SS, thiol/disulfide.

Section III,A, different cell types are primed to respond differently to an oxidizing environment. Moreover, oxidative stress responses also vary according to various biological clocks and endogenous circadian rhythms in plants that generate cycles of varying sensitivity to stress, as they do in other organisms (416). The varying susceptibility of plants to H_2O_2-mediated PCD is discussed below in more detail with regard to the influence of photoperiod (Section VI,B). These inherent features of the redox biology governing stress responses will be present even in plants grown in controlled environment conditions and their effects are probably exacerbated in natural or agricultural environments, where species become adapted to environmental variables. One notable example of a valuable adaptive response is that of shade species buried deep in forest canopies that can take advantage of the brief periods of exposure to high light known as "sun flecks," enabling them to greatly increase their carbon gain without incurring any inhibitory effects of high light exposure due to their rapid and effective engagement of thermal energy dissipation systems (227) that are often termed nonphotochemical energy quenching (NPQ) systems (174, 289).

Epigenetic controls are also increasingly acknowledged as being important components of plant stress responses (46). Stress memories from a single-generation exposure to stress can be inherited through mechanisms involving DNA methylation, small mRNAs, and histone modifications that cannot be explained by Mendelian genetics (46). The influence of redox processes on these systems has not yet been characterized. In contrast, our understanding the robust and complex signal transduction pathways by which plants respond to the redox signals generated by photosynthesis, particularly with respect to light, has greatly increased over the past decade.

A. Excess excitation energy

Light is the driver of photoautotrophic growth and the photosynthetic light-harvesting systems are adept at light capture. However, the efficiency of light harvesting can become a problem when it occurs in excess of the capacity that light energy can be used to drive the PET chain. This can occur frequently if transiently in natural environments where plants are constantly exposed to fluctuating light conditions. Energy that cannot be used to drive metabolism has been termed "Excess Excitation Energy" (EEE; 131, 203–205, 289, 290).

Information from the environment and metabolism is integrated in order to optimize plant growth and development appropriately in relation to prevailing external and internal conditions. Light has a major influence over plant stress responses, and this is possibly because the degree of stress, as determined by the degree of oxidative burden, is exacerbated many-fold in the light compared to darkness. Plants cannot attenuate light energy absorption in the short term, though they can dissipate a portion of the absorbed light energy as heat. Despite this, the amount of absorbed light energy is often in excess of that immediately useable by photosynthetic metabolism. The balance between light capture and light use in photosynthesis is influenced by most if not all environmental fluctuations. It is probably a key driver of the integrating "convergence regulator" mechanism described for ROS and related redox systems in biotic and abiotic plant/environment interactions. The regulation of photosynthesis is geared to minimising EEE and avoiding excessive ROS formation through the photosynthetic control of electron transport and thermal energy dissipation (120).

B. Acclimation strategies

The light-harvesting antenna systems and PET chain are highly efficient at low light intensities. However, too much light can damage PSII and enhance the probability of singlet oxygen generation and subsequent lipid peroxidation (407). Plants have thus evolved both rapid short-term and long-term acclimation strategies to deal with the changes in the conditions of their growth environment. The photosynthetic state transition, which is a short-term acclimatory response to low light involving LHCII phosphorylation following activation of the STN7 kinase, balances excitation energy distribution between the photosystems (36, 174, 201, 353). The NPQ mechanisms, which respond to light intensity, not only provide a safety valve that reduces excitation energy pressure in PSII but they are also considered to act as a "light intensity counter," providing the photosynthetic membrane with a "memory" of the light-exposure history of the leaf (174). The extent of NPQ is governed by at least three factors: the extent of the ΔpH that is generated across the thylakoid membranes, the de-epoxidation state of the xanthophyll carotenoids, and the PsbS protein. In addition to the acclimation processes such as state transitions which balance the excitation energy between PSI and PSII in response to short-term fluctuations in light intensity and quality, longer-term changes in the environmental light conditions provoke readjustments in PS stoichiometry and PET composition (64). This process is achieved through a signaling network involving signaling components such as plastoquinone and the cytochrome b6/f complex that regulate the activities of the thylakoid protein kinases such as STN7, STN8, and CSK in order to achieve the coordinated control of genes in the chloroplast and nucleus (353). Moreover, systemic as well as local signals rapidly transmit information concerning light intensity from exposed to distal shaded leaves (359). This photoprotective signaling system, known as systemic acquired acclimation (SAA), involves the expression of the ZAT10 zinc finger transcription factor and produces similar changes in global gene expression in shade leaves to that observed in exposed leaves. SAA enhances tolerance to oxidative stress and thereby allows nonexposed leaves to pre-acclimate to potential exposures to a high light environment. Constitutive ZAT10 overexpression increased the expression of antioxidative genes and enhances tolerance to exogenous H_2O_2 as well as high light (359).

Such acclimation responses maintain or restore photosynthetic electron flux under adverse environmental conditions and help keep net energy conversion as high as possible. Thus, they often have consequences far beyond primary metabolism because chloroplasts are not only the site of metabolic energy production and carbon fixation but also house key parts of most biosynthetic pathways, including reactions of nitrogen and sulfur assimilation, synthesis of tetrapyrroles, production of secondary metabolites, as well as several major plant hormones. The abundance of these compounds intricately depends on the appropriate functioning of the photosynthetic apparatus. Whereas efficient photosynthesis under light-limiting conditions is achieved by optimal distribution of excitation energy be-

tween the photosystems, exposure to high light necessitates the safe dissipation of excess excitation energy as heat to avoid over-reduction of PET carriers and decrease the probability of singlet oxygen formation at PSII. Moreover, optimal energy balance between PSII and PSI also depends on other environmental cues and stress factors.

Short-term acclimation mechanisms to a changing light environment such as state transitions, energy-dependent quenching, and other nonphotochemical mechanisms serve to modify the efficiency of the light reactions (78, 173, 289, 290). Other pathways such as cyclic electron flow, export of reductant from the chloroplast, and delivery of electrons directly to molecular oxygen in pseudocyclic electron flow, also serve to protect the PET system and decrease the probability of singlet oxygen formation at PSII (15, 364). More long-term acclimation to high light requires adjustments in the composition of the reaction center and light-harvesting pigment protein complexes. Through a complex interplay of environmental, phytohormone and nutritional/metabolic signals, a large number of such adjustments alter plant growth and development through what have been called "stress-induced morphogenic" responses (333).

C. Protein modifications

Proteins can be oxidatively modified in many different ways, involving diverse amino acid residues. A key distinction is whether oxidative modifications are reversible (140).

1. Protein thiols. The reversible redox modulation of Cys residues is perhaps the simplest mechanism for the ROS-mediated activation of mitogen-activated protein kinase (MAPK) pathways. It is entirely possible that MAPKs are activated by oxidation, while specific protein phosphatases are inhibited by oxidation (154). Oxidative modifications of protein Cys thiol groups can involve formation of disulfides with other protein thiol groups or soluble thiols such as glutathione, as well as production of more oxidized sulfur states (sulfenic, sulfinic, and sulfonic groups). Thiol modification is a canonical mechanism of oxidative signaling, for instance, in the bacterial oxyR and the yeast Yap1 systems (17, 75). Formation of highly oxidized Cys residues (sulfinic and sulfonic groups) was once classed as irreversible "damage" but sulfinic acids have been shown to be part of the catalytic cycle of certain peroxiredoxins (40, 57).

2. Glutathionylation in plants. There is growing interest in the potential importance of protein glutathionylation in mammalian cells (70), and recent work in plants has identified proteins that may be regulated by this process *in vivo* (91, 171, 184, 270, 462), though the mechanisms remain unclear. It is considered that glutathionylation is unlikely to occur by uncatalyzed exchange with GSSG in animal cells (70, 112). As well as roles in assembly of iron–sulfur clusters (186), glutaredoxins may play several roles in catalyzing protein thiol–disulfide exchange, including promoting either glutathionylation or the reverse reaction (35). Like thioredoxins, glutaredoxins are encoded in plants by a large gene family of several types, including plant-specific glutaredoxins (238, 269).

3. Protein methionine oxidation. Another possible redox mechanism by which ROS could regulate signaling pathways involves the reversible oxidation of Met residues. Methionine residues are readily oxidized by mild stress to sulfoxide forms (MetSO), and this can be reversed by peptide methionine sulfoxide reductases (PMSR). Methionine oxidation produces two MetSO stereoisomers, each of which is reduced by a specific enzyme. PMSRA reduces the *S* stereoisomer, while PMSRB reduces the *R* form. Cytosolic PMSRA2 and chloroplastic PMSRA4 have both been implicated in oxidative stress tolerance (32, 356).

4. Carbonyl group formation and other protein modifications. One consequence of excessive oxidation is irreversible oxidative damage to proteins. Oxidative damage occurs primarily on side chains of amino acids such as Pro, His, Arg, Lys, and Thr, and produces ketone or aldehyde derivatives (protein carbonyls) that are reactive with 2, 4-dinitrophenylhydrazine (DNPH). Other forms of protein oxidative damage can occur by reaction with lipid peroxidation products, or by conjugation with sugars (glycation) or their oxidation products (glycoxidation). Highly oxidized proteins, which are found in all cellular compartments, are generally assumed to lose their catalytic activity (73, 277, 334). Although "damage" may be a useful term for oxidation-induced loss of function at the protein level, it is misleading when applied at levels of greater complexity (*e.g.*, whole cells, tissues, or organisms). This is because protein oxidation is controlled by development in Arabidopsis (196) and maize (Fig. 14), as well by the nature of the environmental stress (209,229). In seeds, carbonyl formation on specific proteins has been implicated in the control of germination (308), while the extent of leaf protein carbonylation increases progressively during the vegetative growth of Arabidopsis rosettes and then decreases dramatically just prior to bolting and reproductive development (196). Similarly, the extent of leaf protein carbonylation is greatest in the youngest source leaves of maize plants at the flowering stage and least in the oldest source leaves (Fig. 14). The large subunit of Rubisco and the regulatory protein Rubisco activase are among the major oxidized proteins in Arabidopsis leaves (73, 196). Oxidative cleavage of chloroplast proteins such as chloroplastic

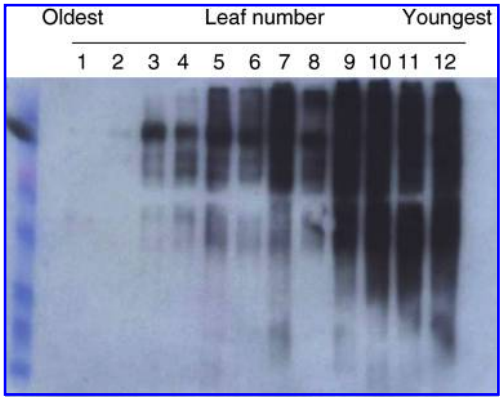

FIG. 14. Development-dependent accumulation of protein carbonyls in maize source leaves. The abundance of protein carbonyl groups, measured using the OxyBlot™ Oxidized Protein Detection Kit, is shown for all leaves on the stem from the oldest source leaf (leaf rank 1) to the youngest source leaf (leaf rank 12).

glutamine synthetase caused by oxidative carbonylation of His residues (183) has been well characterized (135).

5. Processing of oxidized proteins. In the case of Rubisco, enhanced cellular oxidation caused, for example, by exposure to high light, can lead to loss of activity related to redox modulation of Cys residues, rather than protein carbonylation (278). Within the chloroplast, oxidation of critical Cys residues enhances the binding of the Rubisco protein to the chloroplast envelope membranes, marking the protein for degradation (251, 278). The degradation route for irreversibly oxidized proteins remains somewhat controversial but the current evidence points to a ubiquitin-dependent degradation system involving the 20S proteasome (20SP) (187). Disruption of cytokinin metabolism has been shown to trigger the degradation of carbonylated proteins in detached Arabidopsis leaves (187). The degradation of carbonylated proteins was blocked in Arabidopsis mutants when the ubiquitin-proteasome system was inhibited (187). Similarly, an increased capacity for ubiquitin-independent proteolysis enhanced removal of oxidized proteins and increased tolerance to oxidative stress (229). While the 20SP appears to be much more resistant to oxidation than other components of the ubiquitin–proteasome system, exposure to oxidative stress also induces nonspecific autophagocytic pathways of protein degradation (452).

Oxidized Rubisco protein may exit the chloroplast through the stromules, which appear to interact with cytoplasmic vacuole-type compartments through the vesicular trafficking system (63, 336). Thereafter, Rubisco degradation products appear in the vacuoles (178). This process is important in young (336) as well as senescing leaves (172, 247, 306). However, if Rubisco breakdown is blocked by the constitutive ectopic expression of a Cys proteinase inhibitor, then the protein accumulates and activity is increased during stress and natural senescence, suggesting that the Rubisco turnover process has feedback mechanisms related to the reversible redox modulation of Cys residues (336).

D. Revising the "damage" concept

The use of terms such as "oxidative or photo-oxidative damage" continues in the plant research community. This concept holds that ROS exert their effects through indiscriminate widespread inactivation of cellular functions. However, the last decade has witnessed an explosion of interest in ROS and their roles in signaling in plants. Among the many advances in this field, the following key developments can be identified. The first is the identification in plants of specific enzyme systems important in induced ROS generation (230, 362). The second is that ROS such as H_2O_2 are modulators of gene expression through signaling components such as kinase cascades (12, 13, 220, 347). Third, ROS-activated kinase-dependent pathways also play a key role in hormonal signalling and development (134, 347). Thus, as in animals (389), H_2O_2 is considered to be an important signaling molecule in plants. Perhaps an even more striking conceptual shift has concerned singlet oxygen, until recently considered a very reactive (and, therefore, potentially toxic) molecule. Recent advances have shown that tissue death in response to singlet oxygen is largely under genetic control (237, 428). Similarly, as discussed further below in Section VII,B, H_2O_2-driven cell death in CAT-deficient plants is not simply caused by the extent of oxidative stress but is under the control of day length (341).

These and other results have led to a paradigm shift in which ROS are no longer considered to be damaging molecules that cause cell death by indiscriminate oxidation, but rather molecules that exert their effects through specific signaling pathways (119). With regard to ROS-triggered protein degradation, the term oxidative damage is understandable, as oxidized proteins can form aggregates or bind to membranes and induce PCD. However, even this process is likely to require active participation of dedicated cellular machinery (*i.e.*, is not simply an unavoidable deleterious process). Although the terms "damage" and "signaling" are ultimately interpretative terms that do not affect the underlying mechanisms, their implications can be distinguished. Key distinctions are specificity and inevitability. Whereas oxidative damage would be largely nonspecific, leading to the accumulation of irreparable oxidations of nucleic acids and proteins, oxidative signaling should be reversible, with regulatory circuits. If oxidative modifications operate primarily or exclusively through signaling components, the outcomes are likely to be more amenable to modification through plant breeding or transgenic manipulation (see Section IX). In the following Sections, we will discuss specific examples of ROS-mediated processes within the context of the ongoing debate of "damage" *versus* "signaling" paradigms. The choice of paradigm has a crucial influence on the interpretation of the physiological importance of experimental data and on avenues of future inquiry. Ultimately, it will influence attempts to understand and/or manipulate living cells, whether they are pathogenic bacteria, mammalian cancer cells, or plant cells susceptible to disease.

E. Singlet oxygen signaling

Singlet oxygen, like superoxide and H_2O_2, has long been regarded as potentially toxic to the chloroplast, not least because of the danger of the oxidation of chloroplast lipids that contain high contents of polyunsaturated fatty acids (PUFAs). Hence, there is a long history of papers considering such chloroplast redox processes in terms of "oxidative or photo-oxidative damage" with nonenzymatic lipid peroxidation as a primary event in photo-induced oxidative stress in chloroplasts.

Much of our current understanding of how singlet oxygen induces PCD has come from studies on the conditional *flu* Arabidopsis mutant, which accumulates photosensitizing chlorophyll precursors in the dark and therefore generates singlet oxygen upon subsequent illumination. While a relatively large number of genes have been identified as singlet oxygen-inducible (11, 236, 307), the mechanism of signal transduction is unknown. Singlet oxygen is generally considered to have a relatively short life-time of 200 ns (146), but it has recently been suggested that it may be able to diffuse further from the site of production than first thought, particularly during stress (109). Nevertheless, it is probable that singlet oxygen reacts with molecules in the chloroplasts that act as sensors. Attractive candidates for the sensing function are PUFAs (337), chlorophyll, and lipid-soluble antioxidants such as carotenoids and tocopherol. The oxidation of PUFAs leads to a wide range of metabolites called "oxylipins," some

of which are involved in signaling (107), as observed in plant–pathogen interactions and wounding (250, 266, 432). Similarly, the signaling functions of intermediates of tetrapyrrole and chlorophyll biosynthesis have been characterized in chloroplast to nucleus retrograde signaling (391).

Exposure of the conditional *flu* mutant to conditions that allow singlet oxygen formation causes leaf bleaching and accumulation of oxylipins (OPDA and JA) (337). Both responses are abrogated by introducing the *executor1* mutation into the *flu* background. However, genetically blocking oxylipin synthesis is not sufficient to prevent the phenotypic response, though it does modify the expression of subsets of singlet oxygen-induced genes (337). In etiolated *flu* seedlings, in which the singlet oxygen sensitizer accumulates to very high levels, transfer to light causes tissue collapse that cannot be prevented by the *executor1* mutation and which is accompanied by accumulation of fatty acid hydroperoxides produced through nonenzymatic reactions (337). Rather than signaled PCD, this *executor1*-independent response was interpreted as the result of indiscriminate damage both due to very high levels of singlet oxygen and lack of scavenging carotenoids and tocopherols in pigment-deficient etioplasts (337). Regardless of the mechanisms, singlet oxygen signaling pathways appear to be different and even in opposition to those of other ROS such as superoxide (133). This specificity is allowing the identification of genes that are regulated by different ROS. Singlet oxygen-mediated gene expression was found to account for the largest fraction of ROS-inducible genes under various abiotic stresses (133). Thus, both H_2O_2 and singlet oxygen are important in plant acclimation responses as well as the PCD responses (236, 285).

F. What and where are oxidative bursts?

The plasma membrane oxidative burst catalyzed notably by NADPH oxidases and activating calcium-dependent protein kinases (214) plays a central role in the plant innate immune response (24, 439). This oxidative burst consists of either mono- or biphasic ROS accumulation and is perhaps one of the best characterized plant responses to biotic and abiotic triggers (24, 93, 249, 368, 378, 415, 439). The initial burst or accumulation of ROS is mainly attributed to the rapid activation of plasma membrane-bound NADPH oxidases (82, 402–404). Although the identification of NADPH oxidases and their subsequent genetic analysis, as well as their established roles in animal systems, has led to them receiving most attention, it is noteworthy that the plant apoplast contains numerous other types of enzymes that could be responsible for ROS production. These include peroxidases and several types of oxidases, including oxalate oxidases and amine oxidases (Fig. 8; 39, 43, 279). The superoxide or H_2O_2 produced has been considered to diffuse through the cell wall or permeate the cell membrane to alter cellular redox homeostasis. However, given the powerful intracellular antioxidative system, interactions with vicinal redox-sensitive proteins or ion channels may be more important. Redox gradients across the plasma membrane could be also be a significant signal-transducing mechanism in terms of secondary events such as calcium release, as previously discussed (118).

The plasma membrane oxidative burst associated with plant pathogen responses has often been discussed in terms of induction of cell death, but this has recently been questioned (136, 404). In fact ROS produced during the oxidative burst could play a central role in the survival signaling process that induces the expression of defense genes in the cells surrounding those undergoing PCD. Moreover, regulated oxidative burst phenomena play indispensable roles in key physiological and developmental processes that do not involve a PCD response such as stomatal closure and root hair growth (111, 134, 320, 431). Such studies clearly indicate that it is not just the amount of ROS that matters in initiating an appropriate response but the precise location of the burst leading to ROS accumulation. Moreover, an increase in ROS invariably leads to an increase in NO and *vice versa* (466), and the ROS/NO ratio in a given cellular compartment could be more important than the ROS level alone (358). Interestingly, carbon monoxide, which can be endogenously produced by degradation of heme, has also been found to interact with ROS and the glutathione system in plants in stress conditions (158). Although PCD can be accomplished by high concentrations of H_2O_2 adding externally to cells, it is worth noting that lesions in mutants that accumulate singlet oxygen (307) or that are deficient in CAT (56, 341, 397, 419) are caused by intracellular triggers, and that genetic analysis of NADPH oxidase function pointed to a possible function of ROS in counteracting the death response (404). While dose effects have traditionally been considered a useful model for explaining differential effects of high and low concentrations of ROS, the proximity of receptors or sensing mechanisms is likely a key factor (112). Similarly, the site of the burst, whether intracellular or extracellular, can be targeted very precisely when vesicle trafficking is involved and H_2O_2 containing vesicles are drawn to a small focal point on a membrane where a receptor is localized.

The finding that double antisense plants deficient in both *CAT* or *APX* have a less severe phenotype than single antisense plants (351) also suggests that overall cellular tolerance to oxidative burden is not governed simply by the balance between ROS and antioxidant enzymes. This view is further supported by the generation of ROS as secondary messengers during growth and movement responses initiated by plant hormones such as auxin and ABA. These hormones use common elements of signal transduction and/or amplification, particularly the activation of enzymes such as NADPH oxidases that affects enzymes involved in other hormone metabolism and signaling pathways as well as that regulating protein turnover (14, 43, 143, 163). Further evidence comes from the finding that H_2O_2-driven cell death in CAT knockouts is not simply a question of the degree of oxidative stress, but is dependent on photoperiod (341; see Section VII,B).

VI. Redox Crosstalk with Other Pathways

Stress-induced oxidative bursts and associated redox signaling are highly complex and often involve separate bursts of ROS production originating from different sources within the plant cell, some local and some remote from the initial stimulus, followed by waves of amplified signals (119). Exposure to ozone, for example, triggers a local time-dependent, plasma membrane NADPH oxidase-catalyzed biphasic oxidative burst, involving an initial rapid transient burst of ROS production, followed by a second prolonged phase of ROS accumulation (249, 368). However, often only the second prolonged phase of ROS accumulation is observed upon

exposure to stresses such as xenobiotics or heavy metals such as cadmium (305, 357). The accumulation of ROS in chloroplasts has also been observed in response to certain stress situations or specific triggers such as the induction of PCD by the bacterial elicitor, harpin (136). Similarly, the stress-dependent accumulation of ROS in mitochondria has long been linked to PCD death processes in animals (299). In the following Sections we discuss how redox signaling including bursts of ROS accumulation, interact with other plant signaling systems such as phytohormones, sugars, and light acclimation pathways.

A. Redox–phytohormone interactions

It has become impossible to discuss ROS and redox signaling in plants without considering plant hormones and related signal molecules as it is now apparent that these compounds act together with redox-modulated signaling pathways to process and transmit environmental inputs in order to produce appropriate responses. Compounds interacting strongly with redox processes include classical hormones such as auxins, ethylene, and ABA, as well as defense-related signals such as SA and JA (58, 131, 259, 260, 283, 284, 312). The following discussion briefly focuses on the three most studied compounds in defense and stress reactions, though it is now clear that redox state also interacts with other hormones that are key factors in plant development.

1. Salicylic acid. This signal molecule can be produced by two pathways, one through isochorismate (435) and the other through phenylalanine ammonia lyase, the first enzyme of the phenylpropanoid pathway that also produces structural polymers such as lignin and multiple families of phenolic compounds with antimicrobial, antioxidant, and other biological activities (92). Salicylic acid is thought to amplify ROS signals in a feedback reinforcement loop (378). Indeed, induction of PR genes in CAT-deficient tobacco can be prevented by engineering low SA levels (56, 397). H_2O_2 and SA interact significantly with photoperiod signaling, as discussed further in Section VII,B.

2. Jasmonic acid. The octadecanoid pathway involves the key regulatory enzyme, lipoxygenase (LOX), that uses linolenic and linoleic acid as substrates (232, 409) and leads to the synthesis of oxylipins such as JA with antipathogenic and regulatory activities (176). While SA has been intensively studied in connection with its role in plant responses to pathogenic microorganisms (388), the octadecanoid pathway has been largely considered to be a key signaling pathway involved in direct defense against insects (266). However, the specificity of a stress response is given by "crosstalk" between different pathways that can suppress or enhance activation of other pathways through impinging on common signaling nodes (409, 463). An early response to singlet oxygen accumulation in the *flu* mutant is production of 13-hydroxyoctadecatrienoic acid (13-HOT), an intermediate in JA synthesis (307). Singlet oxygen, like the hydroxyl radical, can cause nonenzymatic peroxidation of fatty acid chains to produce molecules such as phytoprostanes (282). The resulting hydroperoxides are distinct from those produced enzymatically and current evidence from metabolite profiling suggests that the enzymatic path is the most important, at least in singlet oxygen signaling from the chloroplast (337).

3. Abscisic acid. This phytohormone has important functions in plant development, metabolism, and physiology, particularly in the control of stomatal closure, and also in biotic and abiotic stress responses (199, 241, 354). Intriguingly, human granulocytes both produce and respond to ABA (50). In plants, ABA-dependent bursts of H_2O_2 regulate stomatal closure (320, 431). The application of ABA leads to ROS accumulation with the calcium/calmodulin system acting upstream and downstream of H_2O_2 (177). Phospholipase D and its lipid product phosphatidic acid, can also be induced as part of the H_2O_2 response in functions as diverse as ABA signaling, root hair patterning, and various stress responses. Interactions with NO have also been demonstrated: ABA-induced H_2O_2 production has been shown to mediate nitric oxide (NO) generation in leaves (466) and stomata (47). Emerging data suggest that ABA is an integral part of some aspects of long-distance H_2O_2 signaling (Fig. 15).

B. Redox and sugars: The hormone link

Additional complexity arises from the integration of redox and other metabolic signals such as sugars, particularly in response to phytohormone triggers (68). The regulation of starch synthesis, for example, involves thioredoxin-dependent modulation of ADP-glucose pyrophosphorylase activity that is responsive not only to trehalose metabolism (215) but also the SnRK cascade, which is a major regulator of plant stress and energy signaling (20). Sugars are the dominant metabolic currency of many cells, from microorganisms to plants and mammals. Sucrose is the major end-product of photosynthesis and, with some exceptions, is the major transport form between plant cells, in contrast to many other groups of organ-

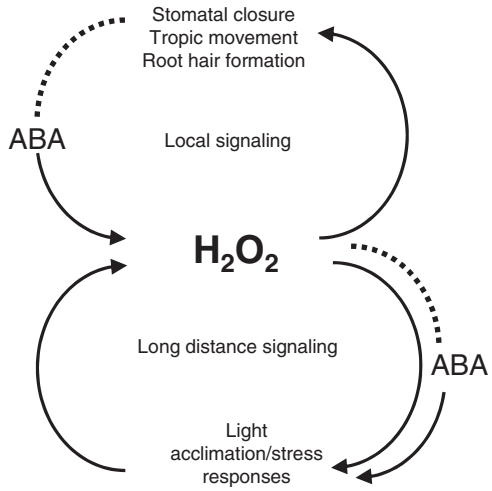

FIG. 15. H_2O_2 and abscisic acid (ABA) interactions in the regulatory circuits of local and long-distance signaling in plants. This simple model illustrates that phytohormone-dependent H_2O_2 signaling can not only act locally in the orchestration of cell growth and movement responses but also in long-distance signaling functions. We postulate that local H_2O_2 accumulation may stimulate the movement of the phytohormone signal to cells far removed from those experiencing the original stimulus and lead to enhanced H_2O_2 production and signaling in these remote cells that is important in the orchestration of acclimation responses.

isms (104). ROS generated in response to hormone action often influence metabolic processes that are also regulated or affected by sugars (68, 246, 335). Sugars can repress certain metabolic processes that generate ROS such as fatty acid mobilization and peroxisomal β-oxidation but they can also favor ROS production. Sucrose induces miR398, a microRNA which post-transcriptionally regulates specific mRNA targets including two Cu/Zn SODs, which are repressed when miR398 is abundant. Hence, miR398 is considered to act as a sucrose-modulated translational regulator of SOD.

The orchestration of gene expression in response to sugars is modified in plants exposed to environmental stresses (246, 335, 399). Sugar starvation can also enhance ROS accumulation and increases the expression of antioxidant genes such as CAT (67). Like ROS, sugars activate plant defense pathways (14, 273, 310), influence the expression of PR proteins (19, 167, 399), and play a central role in plant stress responses (246, 288, 392).

Cross talk between sugar and hormone signaling pathways has been demonstrated for several phytohormones, including ABA, gibberellins (141, 321), ethylene (137), auxins, and cytokinins (161). Accumulating evidence of the extensive dialogue between the sugar and ABA signaling pathways suggests that they have a pivotal relationship in plant growth and defense responses with multiple sites of reciprocal control. The germination and seedling establishment phases of plant growth are sensitive to both ABA and sugars, which together control developmental events (355, 376, 449). Moreover, ABA synthesis and signaling are important in the control of lateral root growth in response to sugar and nitrogen signals (83, 380). The similar cellular and molecular responses to ABA observed in human and plant cells suggest the possibility of common perception and signaling mechanisms (50).

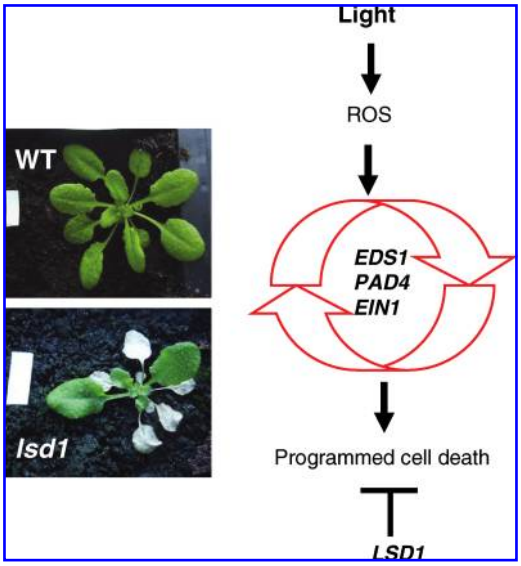

FIG. 16. ROS are important signaling hubs in the interacting pathways that govern light -and pathogen-mediated cell death responses in Arabidopsis. LSD is a negative regulator of plant programmed cell death, and hence its absence in the *lsd1* mutant leads to a light-induced cell death phenotype, as illustrated on the *left*, that is associated with extensive ROS accumulation. The LSD pathway interacts with plant defense components such as EDS1, PAD4, and EIN 1, as illustrated on the *right.*

VII. Light Sensing in Plants

A. Plant photoreceptors

The plant cell is equipped with sophisticated light-sensing mechanisms that are localized in the plasma membrane, cytosol, chloroplasts, and nucleus. Light responses are mediated by at least three types of photoreceptors: phytochrome, cryptochrome, and phototropin (65, 205, 242, 363). The best-characterized light-dependent effects on plant development and architecture are mediated by phytochrome (363). Phototropin appears to control light-induced movement responses such as phototropisms, chloroplast relocation, and stomatal opening. Phytochrome is active at long wavelengths of the visible spectrum, absorbing predominantly red light and far-red light. Conversely, cryptochromes and phototropins monitor blue light and ultraviolet-A, respectively (3).

B. Photoperiod and redox signaling

Redox homeostasis is influenced by photoperiod as well as light quantity (34) and recent work suggests that photoperiod-derived signals are a crucial orchestrator of the functional outcome of ROS signaling. Cryptochromes have been shown to participate in EEE responses (212) and in singlet oxygen signaling (71). The Arabidopsis line *lesion simulating disease 1* (*lsd1*) is one of many lesion mimic mutants that present spontaneous cell death on the leaves in the absence of pathogen challenge (Fig. 16). It was initially characterized by a superoxide-dependent spread of necrotic lesions that develop under long or continuous photoperiods or after infection with an avirulent pathogen (85, 185). Subsequently, it has been shown that cell death in this line is at least partly attenuated by high CO_2, implicating photorespiratory H_2O_2 as part of the triggering signal (259). Other Arabidopsis lines that show spontaneous lesions include mutants for a negative regulator of the phytochrome signaling pathway (138). The effects of both these mutations are linked to SA, and phytochrome knockout mutants are compromised in the establishment of pathogen responses (139, 151).

Recent work in Arabidopsis *cat2* mutants has established that day length context determines the response to H_2O_2 produced through the photorespiratory pathway (341). In short days, CAT deficiency causes strong upregulation of glutathione and defense genes, whereas H_2O_2 only leads to cell death in long days (341). Long-day-dependent H_2O_2-induced cell death involves SA (authors' unpublished observations). Thus, there is significant interplay between photorespiratory H_2O_2, SA, and photoperiod signaling in determining cell death and stress resistance in Arabidopsis. Like ascorbate (authors' unpublished observations), glutathione synthesis and contents may be under phytochrome control: a mutant identified in an arsenic resistance screen was defective in phytochrome A and had enhanced contents of glutathione and its thiol precursors (393).

C. Electron transport chains as light sensors and signal emitters

As well as the well-described systems of light sensing discussed above, for which genes are now annotated and an increasing number of physiological functions elucidated, the

PET chain is an important sensor of light quantity and quality through redox poise and reduction state of specific carriers such as the plastoquinone pool, coupled to proteins such as CSK and STN7 kinases (162, 202–205, 259, 260, 284, 289, 290, 324) and transcription factors such as ZAT 10 (359).

Singlet oxygen is one of many signals originating in chloroplasts that modulate nuclear gene expression in retrograde signaling. Other relevant redox-related retrograde signals are either derived from or are generated by (a) changes in basal antioxidant metabolism and organellar redox state (25); (b) the accumulation of other ROS; (c) metabolites in and flux through the tetrapyrrole biosynthetic pathway; and (d) the rates of organelle protein synthesis. Although the molecules that relay information through the cytosol to the nucleus remain to be identified, the tetrapyrrole Mg-protoporphyrin-IX (391) and the nucleic acid binding GUN1 protein (219) have been identified as putative signaling components. Others include ACD2 and red chlorophyll catabolite (459). The long-term acclimation responses to changes in the quantity and quality of available light, and the SAA signal/response system (359) involve the concerted regulation of nuclear and chloroplast gene expression. The role of PET redox potential changes, ROS, and selective protein turnover of light harvesting complexes and other proteins associated with the photosynthetic processes, is well documented (4, 21, 99, 325–327). As mentioned above, ROS have been discussed in terms of local and systemic signals that can influence and interact with other components systems to coordinate effective protection of chloroplasts in locations remote from those experiencing EEE (205, 284, 359).

VIII. Longevity, Senescence, Death, and Oxidative Stress in Plants

Genetic studies in aerobic organisms from *Caenorhabditis elegans* to plants to mammals have demonstrated that extended longevity is frequently associated with increased tolerance to oxidative stress (255). The analysis of different late-flowering *Arabidopsis* mutants has indicated that such tolerance is correlated with flowering time and longevity (228). For example, increased resistance to oxidative stress is observed in lines carrying mutations in the GIGANTEA protein, which is involved in red light signaling, central clock function, and flowering time regulation (254). Similarly, the flowering Arabidopsis mutants, *ore1*, *ore3,* and *ore9,* are also more tolerant to various types of stress (444, 445). These data add to the links between photoperiod and stress responses discussed above. From a physiological point of view, they might be understood in terms of resistance in short days but death/flowering programs in long days. If this is so, it would be interesting to examine these questions in other species that do not show the same light response as Arabidopsis, which is a quantitative long-day plant (*i.e.*, flowering is not absolutely dependent on exposure to light periods longer than a critical value but is gradually accelerated by increasing day length).

Factors that enhance tolerance to stress can modulate cold acclimation and also extend lifespan by retarding growth and development. Such factors include the C-repeat/dehydration-responsive element binding factor (CBF), that binds to promoter-region *cis*-acting C-repeat (CRT) dehydration responsive elements (DRE) (142). As temperature is one of the key environmental factors determining plant growth, and cold acclimation an important determinant of survival in low temperatures, it is interesting that the CBF pathway is universally present in those plant species studied, even those that do not show cold acclimation (*e.g.*, corn, tomato). *Arabidopsis* shows excellent cold and freezing tolerance, and constitutive CBF overexpression leads to growth retardation. Overexpression in species that do not have cold acclimation traits produces similar growth retardation but also induces tolerance to oxidative and other stresses (468). The interpretation of such observations is that common factors are used to limit growth and delay senescence while enhancing stress tolerance because this strategy enhances plant survival by delaying reproductive development until favorable growth conditions return. The genes involved in cold acclimation (COR genes) are regulated by ABA-dependent and -independent pathways that regulate a range of stress responses such as drought and salinity, in addition to cold (456). Downstream components of hormone signaling pathways such as ERF proteins that bind to GCC-box, DRE/CRE, CE1, and JERE elements, are involved in modulating plant tolerance to multiple stresses. They also regulate ROS signaling pathways through activation of genes involved in defense metabolism (398).

A. ROS and antioxidants in plant senescence

Plant senescence is a developmentally regulated process that can be induced prematurely by stress or by hormones such as ethylene, auxin, ABA, SA, and JA (52, 53). Leaf senescence begins with the loss of photosynthetic competence and degradation of photosynthetic proteins in the chloroplasts, with remobilization of the carbon and nitrogen skeletons to growing sink tissues. The development of reproductive structures governs the timing and onset of senescence in some plant species, and in others such as Arabidopsis the formation of reproductive structures at the "bolting" stage prevents the generation of new leaves. Sugar accumulation and sugar starvation induce senescence (49), as does a high leaf C/N ratio.

The antioxidative system undergoes significant changes with leaf age. The biosynthesis and content of ascorbate decreases (29, 340) and senescence is often accompanied by a decrease in the activities of some antioxidant enzymes and pyridine nucleotides (77, 193, 309, 340). Development-dependent and stress-dependent changes in the expression of APX and CAT genes have been described (454, 471, 472), as well as altered sensitivities of APX1 to H_2O_2 (471). The reduction in antioxidant capacity might be important in the activation of proteases such as Cys proteases, which tend to be inhibited by reductants such as GSH (152). As described in Section V.C.5, virtually no protein oxidation can be observed in leaves as they enter senescence, suggesting that any oxidized proteins are very rapidly degraded at this stage.

Studies on mutants provide direct evidence that redox factors and antioxidants play important roles in plant senescence. Novel roles for NAD in signaling and regulation of cell longevity are being elucidated in microorganisms, animals, and plants (179, 298, 420, 458, 461). One of a collection of *old* (*o*nset of *l*eaf *d*eath) Arabidopsis mutants that show early leaf senescence (195), *old5* was identified as disrupted in quinolinate synthase, the second enzyme of the bacterial and plant

de novo pathway of NAD synthesis (365). The timing of senescence is also altered in the ascorbate-deficient Arabidopsis mutant *vtc1* (28), but this may be due to the enhanced susceptibility of individual ascorbate-deficient cells to PCD (318). Transgenic inhibition of the protein degradation machinery by constitutive cystatin expression in tobacco favors delayed senescence and alters lifespan (336). Similarly, Arabidopsis mutants such as *sag101*, which is defective in an acyl hydrolase and *ore9*, which is lacking a functional F-box protein that is part of the ubiquitin-dependent proteasome system, show delayed senescence and extended longevity (444). Changed ROS abundance may also be important in the regulation of the WRKY6 and WRKY 53 transcription factors that are involved in the orchestration of gene expression (52, 53, 417).

While redox processes are important in the orchestration of senescence (287), this stage of development can in no way be viewed as a time of uncontrolled oxidation. Indeed, given the data on protein oxidation described above, the reverse appears to be correct. Oxidative stress in mutants deficient in antioxidative enzymes does not simply induce premature senescence (341). It should be noted also that leaf senescence is essentially a reversible process until the very final stage, which is PCD. Leaf senescence is crucial for overall plant fitness and it is essential for mobilization and recycling of nutrients from mature leaves to the developing reproductive structures. The reversibility of the first stages of the leaf senescence process is probably a prerequisite for survival that enables old leaves to re-green and become productive if the young leaves are eaten by herbivores or are destroyed by disease.

B. Life or death: Which side is ROS on?

PCD is a pivotal biological process by which eukaryotic cells die. Up to 70 billion human adult cells undergo apoptosis each day. PCD is critical for normal development of multicellular organisms, for example, in the development of the embryo and vital organs as well as in general cellular homeostasis. The controlled eradication of cells can be induced by toxic insult (*i.e.*, chemical damage) or physical disruption of cells, but it is now also recognized that PCD processes are under strict cellular control and mediated by complex signaling pathways, in which the energy-generating reactions in the mitochondrion (and in the chloroplasts in plants) are key regulators. For example, PCD-inducing and cell damage-controlled pathways feed back to the mitochondrion and induce membrane permeabilization. In animals this is, in part, under control of the Bcl-2 protein family, which contains members with either pro- (*e.g.*, Bax) or anti-apoptotic (*e.g.*, Bcl-2) functions. The mitochondrial inter-membrane space contains redox-active and thus potentially cytotoxic molecules such as cytochrome *c* that mediate the PCD process. On permeabilization of the membrane, cytochrome *c* is released from plant and animal mitochondria to act as a cell death effector. In animals, it is established that PCD pathways may be caspase-dependent or independent, while in plants it has been shown that PCD involves the activation of a suite of different Cys and aspartic proteases (148, 149). This ultimately leads to the well-recognized changes in cell morphology associated with PCD, including plasma membrane perturbations, condensation, and fragmentation of nuclear chromatin, and compaction of cytoplasmic organelles and cell volume (see, for example, 136).

A flavoprotein called "apoptosis inducing factor" (AIF), which is released from animal mitochondria, initiates caspase-independent pathways of cell death. AIF was the first flavoprotein shown to be involved in animal apoptosis and it has yet to be demonstrated in plants. AIFs are FAD- and NAD(H)-binding enzymes with a glutathione reductase-like fold that are released from the mitochondrion in response to cell death signaling, probably mediated by the nuclear enzyme PARP-1, which is itself activated by DNA damage (2). AIF then induces apoptosis by translocating to the nucleus and binding to DNA, causing chromatin condensation, and by recruiting nucleases to fragment DNA. A second flavoprotein AIF-M2, which participates in human apoptosis, is located in the cell cytosol, where it produces superoxide by NADPH-dependent reduction of oxygen. AIF-M2 binds to DNA or can bind NADPH but not both at the same time (145). Thus, ROS production by AIF-M2 is inhibited in the DNA-bound form. The steady state low level of superoxide/peroxide production by AIF-M2 in the absence of DNA binding is considered to be important for signalling cell viability. Its inhibition by the presence of foreign or "leaked" host DNA in the cytoplasm is a signal that cell death should be progressed. AIF or AIF-like proteins could therefore act in plants as superoxide generators that are necessary for cell survival signaling (145).

Over the last 10 years, our ability to generate mutants throughout the genome in the model plant Arabidopsis (9) has added considerably to insight from classical forward genetics in this species. These genetic approaches have greatly accelerated our understanding of how plants orchestrate cellular redox homeostasis in relation to environmental and metabolic cues. The analysis of a wide range of Arabidopsis mutants has established that redox changes in the glutathione pool and in components of the SA signaling pathway are recruited in the orchestration of gene expression in the shoot, particularly in response to factors such as the duration of illumination (day length), the quantity and quality of absorbed light, and EEE (25, 157, 185, 202–205, 260, 359). Moreover, PCD observed in the Arabidopsis *lsd1* mutant (Fig. 16) has been linked to reduced stomatal conductance and to enhanced photorespiratory H_2O_2 production as well as redox changes in the plastoquinone pool (259, 260). When the *lsd1* null mutant was crossed with the *chaos* (*cao*) mutant that has a reduced PSII antenna (213), the double mutants showed reduced lesion formation, illustrating the importance of photosynthesis to the PCD phenotype (259). While the pathways of systemic acquired acclimation to high light appear to be distinct from those orchestrating the pathogen response (359), there are nevertheless points of interaction through certain components as illustrated in Fig. 16. A number of pathogen defense signaling components such as *EDS1*, *PAD4*, and *EIN2* have been implicated in the light-induced PCD phenotype (259, 302). Mutations in *PAD4* and *EDS1* block the *lsd1*-dependent R-PCD phenotype that is triggered by light or other triggers such as SA (185, 361). Nonphotorespiratory conditions retarded the propagation of lesions in *lsd1*. These results demonstrate the important role of the LSD1 protein in mediating cellular responses to metabolic ROS, such as those generated through photorespiration following the closure of the stomata.

LSD1 has been described as a 'hub for the regulation of transcriptional mediators of responses to various sources of oxidative stress' (200). The LSD1 protein binds AtbZIP10,

which is a transcription factor that shuttles between the nucleus and cytoplasm and which in the *lsd1* mutant acts an uncontrolled mediator of PCD (200). Interaction with the LSD1 protein prevents AtbZIP10 movement into the nucleus and hence suppresses PCD. The interaction of the LSD1 protein with AtbZIP10 and possibly other transcription factors may also influence H_2O_2 scavenging capacity and interactions with plant hormones such as ethylene (259).

The SA pathway has been implicated in the processes that lead to acclimation to EEE (205, 259, 260). It is negatively controlled by MAP kinase 4 (322, 361) and it is interfaced with redox changes in the glutathione pool, leading to the expression of a suite of genes that are associated with pathogen resistance (202, 205, 259). EDS1 and PAD4 have been implicated in the amplification of ethylene and SA signals by processing ROS (361). This pathway also incorporates a flavin-dependent monooxygenase (FMO1) that positively regulates the EDS1 pathway and NUDT7, a member of the cytosolic nudix hydrolase family, which is a negative regulator of EDS1 signaling (Fig. 16). While the SAA signaling pathway that facilitates the pre-acclimation of photosynthetic tissues to high light is distinct from that of pathogen-related systemic acquired resistance (359), these studies show that PAD4, EDS1, and MPK4 interact with LSD1 in signal pathways, leading to PCD (434) and with some responses to EEE (259, 260).

IX. Practical Applications

A. Shoring up the ROS defenses

The last 20 years have witnessed a veritable revolution in our concepts regarding the functions of ROS and redox signaling in plants, particularly in relation to growth and cell division. Of necessity, this has led to a change in mindset with regard to the potential applications of this new knowledge and insight. Early findings that ROS are often increased in plants exposed to environmental stresses led plant scientists to attempt to improve the stress tolerance in model and crop species by transforming plants with genes for antioxidant enzymes targeted to the chloroplast or cytosol (for reviews, see 8, 121, 123). Over the years, such attempts have met with mixed results and this is perhaps not surprising given the complexity of ROS and antioxidant functions in plants (123). It is important to remain mindful of the fact that studies with negative results tend either not to get published or to obtain a lower publication profile. Similarly, the literature studies that report enhanced stress tolerance resulting from the overexpression of genes encoding antioxidative enzymes are often rather simplistic in design and execution, following a rather standard pattern of analysis. For example, most reports have only been undertaken in the laboratory on plants grown under controlled environment conditions and often they have tested only the responses of the transgenic plants to artificially-induced oxidative stress induced by pro-oxidant chemicals such as methyl viologen. For example, tobacco plants overexpressing a bacterial CAT targeted to chloroplasts showed enhanced methyl viologen tolerance with a significantly reduced capacity to inhibit bacterial growth (276, 330). From such studies, a substantial body of literature evidence has accrued demonstrating that constitutive overexpression of one or more antioxidative enzymes results in beneficial results on stress tolerance. In our own hands, for example, overexpression of GR in the chloroplast resulted in increased tolerance to cold-induced photoinhibition in poplar (126), and a lower rate constant of PSII photoinhibition in tobacco (411). Similarly, the constitutive expression of a late embryogenesis protein LEA, whose expression was enhanced by oxidants and ABA, was shown to confer enhanced tolerance to H_2O_2 in yeast and in Arabidopsis (281). The protective effect arising from overexpression of antioxidative enzymes in the chloroplast has been explained by effects on the PET, suggesting that enhanced antioxidant capacity is better able to maintain the photosynthetic apparatus in a more oxidized state (216, 217).

B. Modifying ROS signaling pathways: Shooting the messenger?

Reinforcement of antioxidative systems would seem to be the best strategy if the negative effects of oxidative stress are due to inevitable multiple molecular modifications that cause deleterious effects on growth, development, or yield. In this case, the only way to stop the damage is by improved policing of the culprits (ROS) that are accidentally overproduced in response to stress. More success than hitherto achieved may come by modifying expression of whole suites of genes, approaches that are likely to become more feasible as key transcription factors or other regulatory components are identified. Another approach recently reported is engineering multifunctional hybrid enzymes (457). Entirely different possibilities are opened up if links between stress conditions and their effects are mediated via ROS interacting with a limited number of signaling components. As we have emphasized in this review, ROS have multiple effects on plant function, some of which may be considered desirable or not, depending on the context. Intracellular oxidative stress often results in slower growth and/or cell death. Recent data obtained with Arabidopsis clearly suggest that the most physiologically relevant aspects of these effects are highly conditional and can be genetically modulated (341, 428). Although such findings are promising, further work is required to establish whether they are potentially applicable to improving crop performance in agricultural conditions.

C. From the lab to the field

The growing need to boost crop production for food, fiber, and bio-energy, coupled to the dual requirements for predictability of yield and agricultural sustainability is a key driver for fundamental and applied research on plants. Currently, mankind is faced with many problems such as the loss of fertile lands, caused in part by intensive conventional agriculture that depletes natural resources, the potential negative impacts of increasing environmental stresses (particularly drought) associated with climate change, and an increasing world population. The situation is particularly serious for developing countries such as in Africa, where the population has more than doubled between 1975 and 2005, rising from 335 to 751 million, but the rise in population has not been matched by concomitant increases in food production. Moreover, a severe drought cycle is experienced every 10 years in many countries in the eastern Africa region, while a moderate drought is experienced once in every 5 years. In 2006, for example, 27 sub-Saharan African regions experienced severe famine due to drought. In that year alone, 200 million people were left malnourished. According to the International Food Policy Research Institute (http://www.ifpri.org/December

2008), drought linked to poor rainfall was the major cause of this malnourishment. The International Federation's Disaster Relief Emergency Fund (DREF) estimated that in the short rainy season of 2006, 3.5 million people in Kenya were in need of food aid due to persistent droughts and delayed rains. The need for improved crops that will perform better and more reliably under stress conditions, particularly water deficit, is therefore real and, in fact, has never been more urgent. For these reasons, genetic modification of crop plants to introduce desirable traits such as stress tolerance and nutritional enhancement are increasingly becoming recognized as important weapons in the arsenal to improve and defend food security. Evidence in support of this trend also comes from the fact that 23 countries planted genetically-modified "biotech", crops in 2007, despite continued consumer resistance. Moreover, the increasing percentages of current crops such as maize (14%), soybean (60%) cotton (28%) and canola (18%) that are genetically modified reflects increasing recognition that promising technologies are essential to boost plant productivity and food production (http://www.isaaa.org, December 2008).

In contrast to the extensive literature reporting the effects of laboratory experiments, very few studies have been undertaken in the field, where so far increases in antioxidant enzyme capacity in transgenic plants has been found to have little beneficial effect in terms of stress tolerance (218). Hence it is crucial to address the question of the next approaches, given that field studies on transgenic plants are likely to be limited by the administrative complexities encountered in many countries in gaining permission for such tests. Recent years have seen an explosion in the numbers of new genes associated with stress tolerance traits, and these are being extensively tested as potential breeding markers, as is antioxidant defense strength. Characterization of gene function is destined to remain largely in the domain of the laboratory, even though increasingly large numbers of transgenic crops with valuable traits such as enhanced herbicide tolerance or insect resistance are now being grown in the field.

D. Crops and human health

The epidemiological evidence that consumption of fruit and vegetables decreases the frequency of the most important diseases of the developed world, together with general dietary (five-a-day) advice from governmental organizations, has led to increasing emphasis on the identification of plant dietary compounds that lead to human health benefits. This has led to intensive research efforts, not only to characterize the effects of different groups of plant secondary metabolites on animal cell viability and cellular detoxification systems, but also the extensive characterization of the pathways of plant secondary metabolism (see, for example, *The Plant Journal*, Special Issue on Harnessing Plant Biomass for Biofuels and Biomaterials, Vol. 54, 2008). While the simple concept of a few years ago of a clear causal relationship between oxidative damage and animal aging is giving way to a much more complex picture that views aging as a failure to recycle damaged cells and macromolecules, the notion that the antioxidant content of plant foods is largely beneficial in promoting health and well being, as well as maintaining youthfulness, remains fixed in the philosophy of human nutrition. Antioxidants remain a key component of cellular defenses against protein damage and detoxification systems for xenobiotics and other metabolites. For example, glyoxalase I, which has been shown to have anti-aging and related protective effects of in animals and plants, requires glutathione.

In many countries, the general consumption of fruit and vegetables falls well below the average recommended intake of 400 g per day. Hence, enhancement of micronutrient content in food crops by means of biotechnology and biofortification procedures is not only a major challenge in fighting deficiencies but also in ensuring that people are better protected against the major diseases of the developed world. Plants accumulate and maintain levels of ascorbate, glutathione, and tocopherol that are commensurate with their growth conditions, and environmental parameters such as light are now known to have a major effect on the accumulation of low-molecular-weight soluble antioxidants.

Whether the goal is to enhance plant stress resistance or nutritional content, approaches such as the identification and association of appropriate quantitative trait loci (QTL) offer an alternative to transgenic techniques. To date, very few QTLs that govern the abundance of antioxidants or potentially beneficial secondary metabolites have been described. However, the rapid increase in our knowledge of the underpinning environmental and developmental controls of the genes that govern the biosynthesis, metabolism, and accumulation of such compounds, dictates that plant breeding programs will soon be able to use appropriate genetic markers for appropriate enhancements, for example, of the vitamin C levels in fruits such as black currants.

Engineering plants to overproduce antioxidants such as ascorbate, glutathione, and tocopherol has also been moderately successful (see, for example, the *Physiologia Plantarum* Special Issue on Nutrigenomics: Vol.126, 2006). The biosynthesis of vitamins A and E or their precursors has been characterized in detail, leading to new approaches for enhancement by crop management as well over-expression of biosynthetic enzymes. The production of "golden rice" enriched in β-carotene (provitamin A) was a pioneering step in the field. Transgenic approaches have led to a $>$20-fold increase in β-carotene content of the rice grain compared to the original line (45). While this technology has the potential to pave a new way forward in controlling sight defects and other vitamin A-related disorders, it remains to be seen whether public acceptance of transgenic crops will increase to the extent required to realize the potential benefits of such advances in basic science capability.

X. Conclusions and Perspectives

The field of redox biology has recently witnessed a dramatic reappraisal of the function of reactive oxygen species (ROS) and antioxidants. For many years considered as "molecular hoodlums" to be suppressed or policed by the antioxidant system, ROS, like the low molecular weight antioxidants, are now considered to be dynamic information-rich signaling molecules. ROS and antioxidants govern cellular redox state, which in plants is a "convergence regulator" linking biotic and abiotic stress responses to the control of growth and programmed cell death. Low molecular weight antioxidants such as ascorbate, glutathione, and tocopherol fulfill functions that go far beyond their ROS-scavenging activities, particularly in cell signaling and the regulation of

gene expression. However, the concept persists that ROS exert their principal effects through chemical toxicity that causes oxidative damage to proteins, lipids, and DNA. While the biochemistry by which ROS oxidize cellular components can potentially be described in unambiguous terms, the complexity of living cells means "damage" verses "signaling" paradigms are less easily defined. However, the choice of paradigm governs our concepts, understanding, and appreciation of the significance of the underlying biochemical mechanisms and their physiological importance.

We have described here the concepts of cellular redox state and homeostasis in relation to plant fitness and responses to environmental challenges, with particular emphasis on oxidant–antioxidant relationships and signaling. The ROS–antioxidant interaction is viewed as a convergence hub for signals derived from metabolism and the environment. While we have not discussed NO synthesis and signaling in plants, as this topic has recently been reviewed (437), we remain mindful that the production of ROS and NO in response to hormonal and stress triggers is closely linked in plants (466), as may also be ROS and CO signaling in stress responses (450). Since there is no evidence that peroxynitrite is harmful in plants, even at millimolar concentrations (76), processes such as ABA-induced H_2O_2 production leading to NO generation in leaf mesophyll cells (466) and in stomata (47) must be viewed as signaling events involved in the orchestration of specific responses.

Such generic information is also important in the global context of the transformative bioeconomy. After several decades of apparently unlimited food supplies, at least in the most technologically advanced nations, the world is faced with the uncertainties of climate change, with rising global temperatures and energy prices, and an ever-increasing population. The need for food and energy security is focusing increasing attention on the role that agriculture can play in providing human food, animal feed, and fiber, within the constraints of renewable energy sources and environmental values. The photoautotrophic and sessile nature of plants means that, perhaps more than for many other types of organism, their redox biology is inextricably associated with their growth and defense responses, thus determining the nutritional quality of plant products. Our increasing understanding of how plants have not only mastered but also successfully exploited oxygen chemistry and light-driven redox metabolism will undoubtedly be a part of any sustainable solutions achieved to the major challenges of the 21st century.

As well as practical applications targeted to specific antioxidants, the emerging concepts of how ROS are involved in the control of plant growth and defense responses opens new avenues of potential practical development, with the targeted upregulation of suites of appropriate genes. Emerging areas of intensive interest include the PET and RET signaling pathways and epigenetic and post-transcriptional regulation of gene function, including small interfering RNAs, DNA methylation, and microRNAs. The identification of new roles for proteins such as the DELLAs, which regulate plant growth and redox-related defense processes that determine ROS accumulation, makes them attractive targets for practical applications. Similarly, this research ushers in new studies in plant biology focusing on areas that had not previously been associated with redox biology such as the role of gibberellins, which determine the lifetime of the DELLA proteins, and other signals such as those mediated by photoreceptors. Finally, the exciting discovery that plants exposed to stresses that enhance cellular oxidation inherit the memory of stress and that exposure to stress has to be persistent to maintain the same level of acclimatory responses, opens new frontiers of investigation, not only into the mechanisms that regulate the rate of homologous recombination and global genome methylation, but also of how these processes are integrated with cellular redox homeostasis.

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Abbreviations

ABA, abscisic acid; ARE, antioxidant response element; APX, ascorbate peroxidase; CBF, cytochrome b_6f complex; CSK, chloroplast sensor kinase; CAT, catalase; Cys, cysteine; CYP, cytochrome P450; DHA, dehydroascorbate; DHAR, dehydroascorbate reductase; DTT, dithiothreitol; γ-ECS, γ-glutamylcysteine synthetase; EEE, excess excitation energy; GalLDH, galactonolactone dehydrogenase; GR, glutathione reductase; GSH, glutathione; GSSG, glutathione disulfide; GT, glutathione transferases (glutathione *S*-transferases); H2DCFDA, 2′,7′-dichlorodihydrofluorecein diacetate ; JA, jasmonic acid; LHC, light harvesting complex; MDHA, monodehydroascorbate; MDHAR, monodehydroascorbate reductase; Nrf2, nuclear factor erythroid 2-related factor 2; PCD, programmed cell death; PET, photosynthetic electron transport; PGA, 3-phosphoglycerate; PQ, plastoquinone; PSI, photosystem I; PSII, photosystem II; QC, quiescent centre; RBOH, respiratory burst oxidase homolog; RET, respiratory electron transport; RuBP, ribulose-1,5-*bis*phosphate; Rubisco, RuBP carboxylase/oxygenase; ROS, reactive oxygen species; SOD, superoxide dismutase; SA, salicylic acid; SAA, systemic acquired acclimation; TRX, thioredoxin.

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