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Review

ROS-related redox regulation and signaling in plants

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ABSTRACT

As sessile oxygenic organisms with a plastic developmental programme, plants are uniquely positioned to exploit reactive oxygen species (ROS) as powerful signals. Plants harbor numerous ROS-generating pathways, and these oxidants and related redox-active compounds have become tightly embedded into plant function and development during the course of evolution. One dominant view of ROS-removing systems sees them as beneficial antioxidants battling to keep damaging ROS below dangerous levels. However, it is now established that ROS are a necessary part of subcellular and intercellular communication in plants and that some of their signaling functions require ROS-metabolizing systems. For these reasons, it is suggested that "ROS processing systems" would be a more accurate term than "antioxidative systems" to describe cellular components that are most likely to interact with ROS and, in doing so, transmit oxidative signals. Within this framework, our update provides an overview of the complexity and compartmentation of ROS production and removal. We place particular emphasis on the importance of ROS-interacting systems such as the complex cellular thiol network in the redox regulation of phytohormone signaling pathways that are crucial for plant development and defense against external threats.

Contents

1. Introduction

Like all aerobic organisms, plants benefit from oxygen while being faced with the challenge of maintaining appropriate redox conditions for their physiology and development in an oxidizing atmosphere [1]. While geological factors have affected the composition of the atmosphere, so much oxygen would not be present without the unique water-splitting activity of photosystem II (PSII). This complex first appeared in cyanobacteria and, via a process of endosymbiosis, became incorporated into the chloroplasts of eukaryotic photosynthetic organisms, including multicellular forms that are among the most important supporters of life on

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earth. The highly oxidizing species that are created when PSII absorbs light energy allow plants to access water as a source of electrons, thereby liberating oxygen, as a by-product, from which reactive oxygen species (ROS) can be formed in plant cells. ROS is a term encompassing molecules such as singlet oxygen, superoxide, hydrogen peroxide (H_2O_2), and the hydroxyl radical. These are just some of many simple reactive species produced during metabolism, all of which may impact on cell function in specific or overlapping ways [1]. Unquestionably, molecules such as H_2O_2 and singlet oxygen exert specific effects, including those we discuss in this review. Nevertheless, the term ROS continues to be useful as a generic term to describe molecules derived from ground-state oxygen whose reactivity means, first, that their accumulation must be controlled and, second, that they are excellent signaling molecules. For this latter reason, they have become woven into the fabric of the regulatory networks that underpin plant development and responses to the environment.

ROS biology and the complex associated network of redox-sensitive factors are now recognized to be important in cellular regulation, with molecules such as H_2O_2 playing crucial roles in oxidative signaling [2–4]. As we emphasize in this review, ROS biology in plants encompasses numerous processes found in animals while including many specific features that may explain the relative complexity of redox regulation in plants. Plant growth is driven by the highly energetic reactions of photosynthesis, which involves continuous generation of both oxygen and ROS in a light-dependent manner. Plants have limited control over light intensity and temperature, both of which fluctuate widely in nature. The capacity of metabolism, which is influenced by temperature, to consume the products of photosynthetic electron transport, which is light-dependent, can vary widely, even in the short-term. Powerful and flexible regulatory mechanisms are therefore required to match photosynthetic light use to the prevailing irradiance and temperature regimes. The need for flexibility has been a more significant evolutionary driving factor than metabolic efficiency in plants, which may contrast with the situation in animals. This concept influences much of current research thinking in plants, and has opened up exciting possibilities for improving crop photosynthesis [4,5].

Another important feature that distinguishes plants from animals is their sessile life habit. Thus, in plants confronted by stressful conditions, short-term escape strategies are limited to organellar, cellular or organ levels. However, much of the developmental programme of plants is indeterminate, meaning that the development of new organs can be more flexibly tailored to the external conditions than in many animals. These factors probably explain the particularly influential role of ROS and related redox changes in plants as an interface between internal physiology and the environment [3]. Such an interface is only possible in the presence of an intricate and complex antioxidative network that dictates the extent to which ROS are allowed to accumulate and that may be important in relaying ROS signals.

In this review, our aim is to outline the fundamentals of current knowledge of ROS biology in plants and to re-evaluate current concepts of ROS-antioxidant relationships. We provide an update on the reactions that produce and regulate ROS to allow appropriate signaling in response to environmental and developmental cues.

2. The basics of ROS formation in plants

While the chloroplast is a key site of ROS production in photosynthetic cells, plants can produce these compounds through respiratory processes that have also been well studied in animals [1]. (Fig. 1). Superoxide is generated by the plant mitochondrial respiratory electron transport chain [6] and by NADPH oxidases, which have a catalytic subunit that is homologous to mammalian Respiratory Burst Oxidases, and which are probably mainly located at the plasma membrane [7]. However, as discussed below, various plant-specific pathways add extra layers of biochemical complexity.

2.1. Singlet oxygen

This reactive non-radical molecule is thought to be largely formed in the PSII reaction centre by photodynamic activation of ground-state oxygen that reacts with triplet chlorophyll [8]. Singlet oxygen production is minimized by several carotenoid-dependent quenching mechanisms that dissipate excess light energy as heat [9], but these regulatory mechanisms have limits [4]. If these limits are exceeded, singlet oxygen kick-starts lipid peroxidation reactions, which may be the main cause of stress-induced photooxidation [10]. Singlet oxygen signaling, through lipid-derived metabolites, is an important control of gene expression and cell fate [11–13]. Chemical scavenging of singlet oxygen by carotenoids generates breakdown products that act as signals [14,15]. This is one example of how signals may be generated during ROS processing by antioxidants.

2.2. Reduction of oxygen to hydrogen peroxide (H_2O_2)

In addition to singlet oxygen generation, ROS can be formed in plants through many reactions in which O_2 undergoes reduction to superoxide or H_2O_2. Superoxide can be chemically reduced or dismutated to H_2O_2, a reaction that is accelerated by superoxide dismutases (SODs). Plants contain both "prokaryotic" and "eukaryotic" type SODs. The former are based on Fe or Mn co-factors and are found in the chloroplasts, peroxisomes, and mitochondria (Fig. 2). Eukaryotic Cu-ZnSODs are found in various compartments, including the chloroplast and the apoplast. As well as generation via superoxide, H_2O_2 can be produced by two-electron reduction of O_2 through various oxidases such as glycolate oxidase (GOX) located in the peroxisomes [3]. Class III heme peroxidases are yet another potentially important source of superoxide and H_2O_2 in plants [16]. These proteins are encoded by >70 genes in the model plant Arabidopsis: while their biochemical properties remain in many cases to be elucidated, at least some of them can catalyze superoxide formation from O_2 as well as, or instead of, H_2O_2 reduction to water [17].

From a purely quantitative point of view, the major players in superoxide/H_2O_2 generation in plants are probably the photosynthetic electron transport chain, photorespiratory GOX, the respiratory electron transport chain, and NADPH oxidases located at the plasmalemma (Fig. 1). However, the contributions of these different sources are highly conditional and, also, dependent on plant species. High rates of H_2O_2 generation in plants [3,18], as well as the associated signaling and biochemical functions, probably account for the plethora and variety of enzyme systems able to use this molecule as a substrate (see Section 3). Acting alongside proteins such as ferritins that chaperone transition metals, maintenance of H_2O_2 at low levels prevents generation of the hydroxyl radical at excessive rates. It should be noted, however, that even the highly reactive hydroxyl radical plays important roles in plants, notably in cell wall metabolism and structure [19].

3. Plant antioxidative/ROS-processing systems

Plants are exceptionally rich in many compounds of low molecular weight that have antioxidative activity [20,21]. Antioxidants include basic housekeeping compounds, such as amino acids and sugars, pigments that play important roles in the regulation of photosynthesis, such as carotenoids, as well as secondary compounds

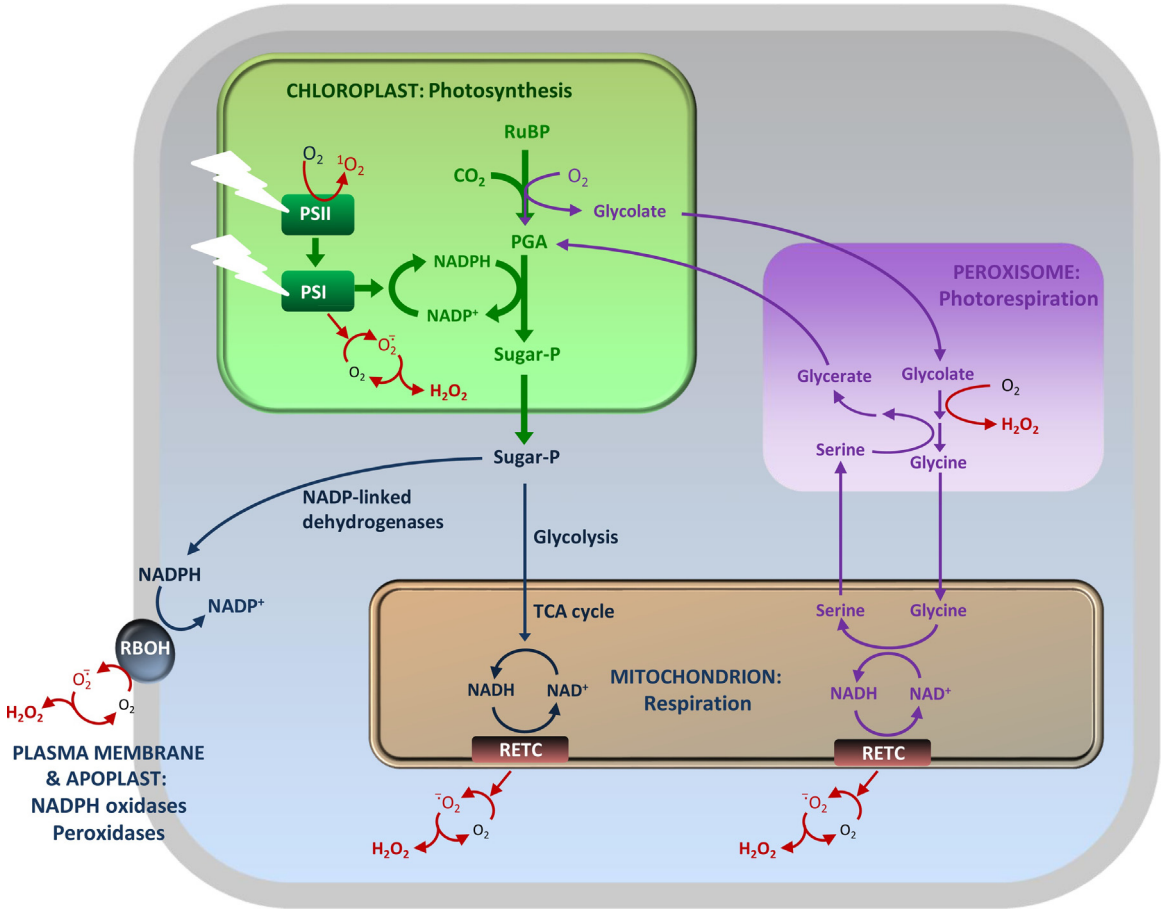

Fig. 1. The basics of ROS formation in plants. The chloroplast is the main site of singlet oxygen formation whereas ROS generation by reduction of molecular oxygen occurs at several subcellular and extracellular sites. Although mitochondrial ROS are shown being released into the cytosol, they may also be released into the matrix. PGA, 3-phosphoglycerate. PSI/II, photosystem I/II. RBOH, respiratory burst oxidase homologue. RETC, respiratory electron transport chain. RuBP, ribulose 1,5-bisphosphate. Sugar-P, sugar phosphate.

like flavonoids. Among the rich tapestry of antioxidant molecules found in plants, ascorbate and glutathione are key players because (1) specific enzyme systems (peroxidases) enable them to react rapidly with H_2O_2 and (2) their oxidized forms are regenerated by high capacity reductases and associated systems that draw on reductants generated in photosynthesis or by respiratory dehydrogenases. These features distinguish ascorbate and glutathione from sacrificial antioxidants because they allow repeated redox-cycling to effectively regulate overall cell redox state [22]. The pools of these antioxidants are likely to be highly reduced (over 95%) within the cytosol, chloroplasts and mitochondria [23], with the oxidized forms accumulating only in compartments that lack efficient redox-recycling mechanisms, such as the vacuole and apoplast [24,25].

As in animals [1], glutathione plays important antioxidant roles, as a reductant of superoxide or as a substrate for certain peroxidases or other enzymes like methionine sulfoxide reductases (MSR) [26–28]. In plants, ascorbate is probably a more important co-factor for peroxidases and is also important in direct chemical removal of ROS, as well as regeneration of tocopherols and production of xanthophylls involved in excitation energy quenching. Up to 10 genes encode ascorbate peroxidases (APX) in Arabidopsis. The main role of glutathione in H_2O_2 metabolism may be in regenerating ascorbate from one of its oxidized forms, dehydroascorbate (DHA), either chemically or via dehydroascorbate reductases (DHAR [22]). Indeed, a recent report provides in planta evidence that the main route of glutathione oxidation when H_2O_2 metabolism is increased is via the ascorbate-glutathione pathway rather than more directly via GSH-dependent peroxidases [29].

Despite the importance of the ascorbate-glutathione pathway, recent evidence also points to an overlapping role for peroxiredoxins (PRX) in maintaining appropriate levels of H_2O_2, at least in the chloroplast [30]. By contrast, catalases are the major players in H_2O_2 metabolism in the peroxisomes [31]. Functional studies are gradually adding to information from genomics to provide insight into the workings of the plant antioxidative system. Together with information from localization studies, a picture is emerging in which a complex, compartmentalized network of several types of proteins (Fig. 2) ensures redox homeostasis and allows ROS-triggered signaling (Fig. 3).

Some of the enzymes listed in Fig. 2 can be encoded by more than gene per compartment (e.g., CAT) while for others a single gene directs the encoded protein to more than one location (e.g., GR). Intricate redox control in organelles such as chloroplasts and mitochondria might reflect their roles as redox sensing hubs, i.e., sites where rates of local ROS production reflect the balance between the plant's environment and the internal metabolic and devlopmental status. The importance of these two organelles as sites of redox signal integration and transmission may also be related to integration of the expression of their genomes with nuclear gene expression [32]. Nevertheless, organelles do not function in isolation, and several transporter systems connect redox reactions occurring in the compartments shown in Figs. 2 and 3 [33,34].

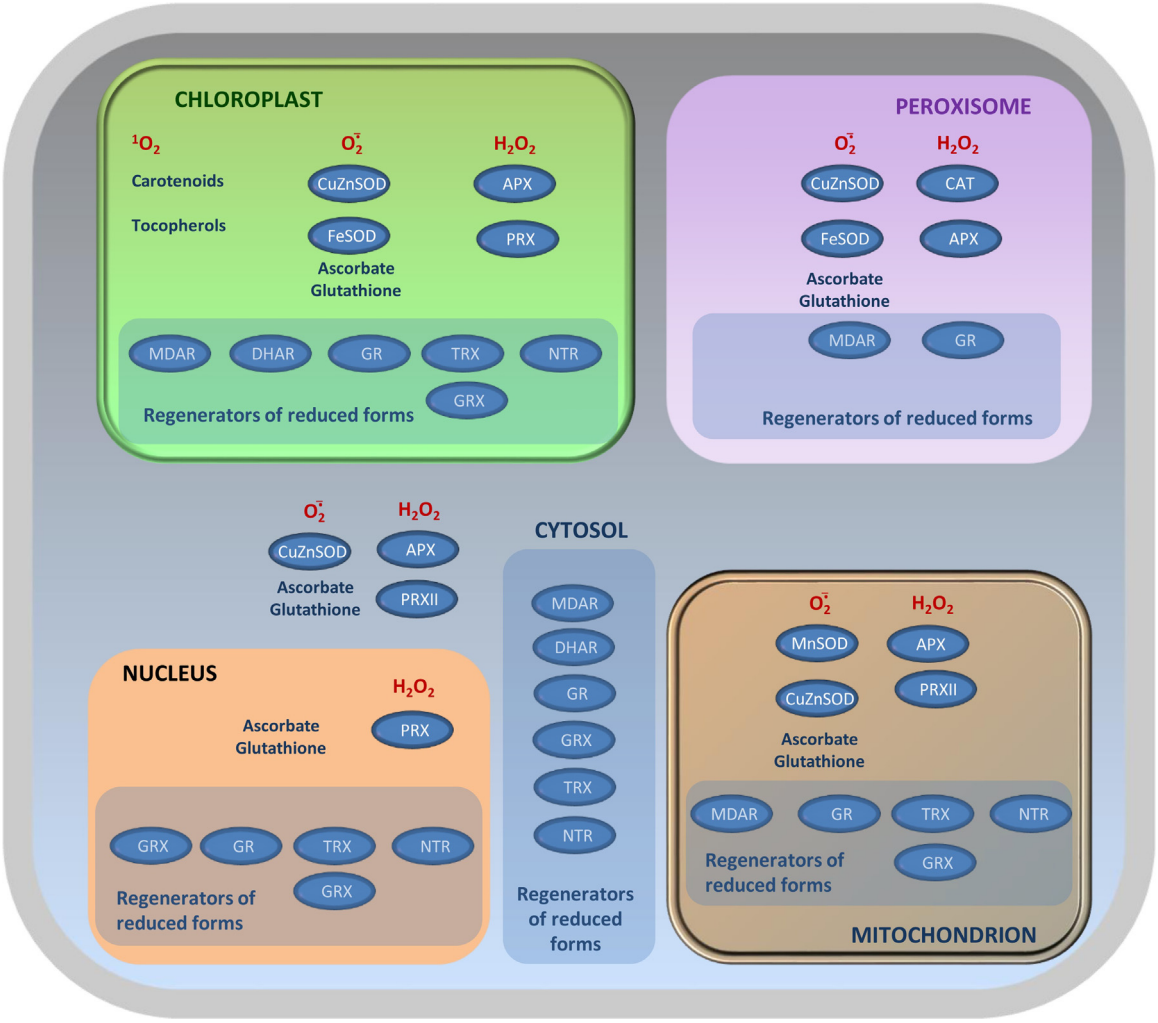

Fig. 2. Plant antioxidative systems and where they are found within the cell. The figure summarizes available information for Arabidopsis on the subcellular localization of antioxidative enzymes and related proteins. The information is not exhaustive, and other proteins may be involved. APX, ascorbate peroxidase. CAT, catalase. DHAR, dehydroascorbate reductase. GR, glutathione reductase. GRX, glutaredoxin. MDAR, monodehydroascorbate reductase. NTR, NADPH-thioredoxin reductase. PRX, peroxiredoxin. SOD, superoxide dismutase. TRX, thioredoxin.

Antioxidants not only function to keep ROS low but may also be involved in regulating ROS-dependent signaling. Examples are the roles of carotenoid degradation products in chloroplast singlet oxygen-triggered signaling to the nucleus [14,15] and the importance of cytosolic DHARs and glutathione reductase (GR) in determining glutathione status and thereby coupling H_2O_2-induced oxidative stress to phytohormone signaling [29,35–37]. The repertoire of glutathione-related signaling is extended by interactions with nitric oxide (NO) through the *S*-nitrosoglutathione (GSNO) and GSNO reductase system [38].

While the roles of thiol-disulfide systems are discussed further in Section 4.2, recent work has identified nucleus-located antioxidant and thiol reductase systems that could be particularly important in different aspects of redox regulation. Among them, thioredoxins (TRX) and glutaredoxins (GRX) are involved in the regulation plant of development through interaction with distinct transcription regulators [39–42]. Moreover nuclear TRX-dependent 1-Cys-PRX functions in wheat embryo cells undergoing oxidative stress [43]. Although synthesized in the chloroplast and cytosol [44], glutathione has been proposed to be recruited into the nucleus and to undergo a redox cycle during the cell cycle [45,46].

4. ROS and redox signaling during stress and development: a coming of age

A dominant concept in redox signaling is that there is a balance between ROS on one side and antioxidants on the other (Fig. 4A). According to this concept, oxidative signaling shifts this balance so that ROS accumulate, either through an increase in their production or a decrease in antioxidant capacity. The resulting enhanced oxidation entrains programmed cell death and/or acclimation and improved stress tolerance, according to its intensity (Fig. 4A). While this concept continues to be useful, recent work suggests that it needs to be updated, notably to take account of the complexity and specificity of plant antioxidative systems as well as their roles in signaling.

4.1. ROS-antioxidative interplay in stress signaling and acclimation

Studies involving targeted down-regulation of antioxidant systems support the broad concept that enhanced oxidation is a key part of stress signaling. For instance, both ozone exposure and down-regulation of catalase are sufficient to mimic several effects of pathogen challenge and to induce cell death and biotic defense

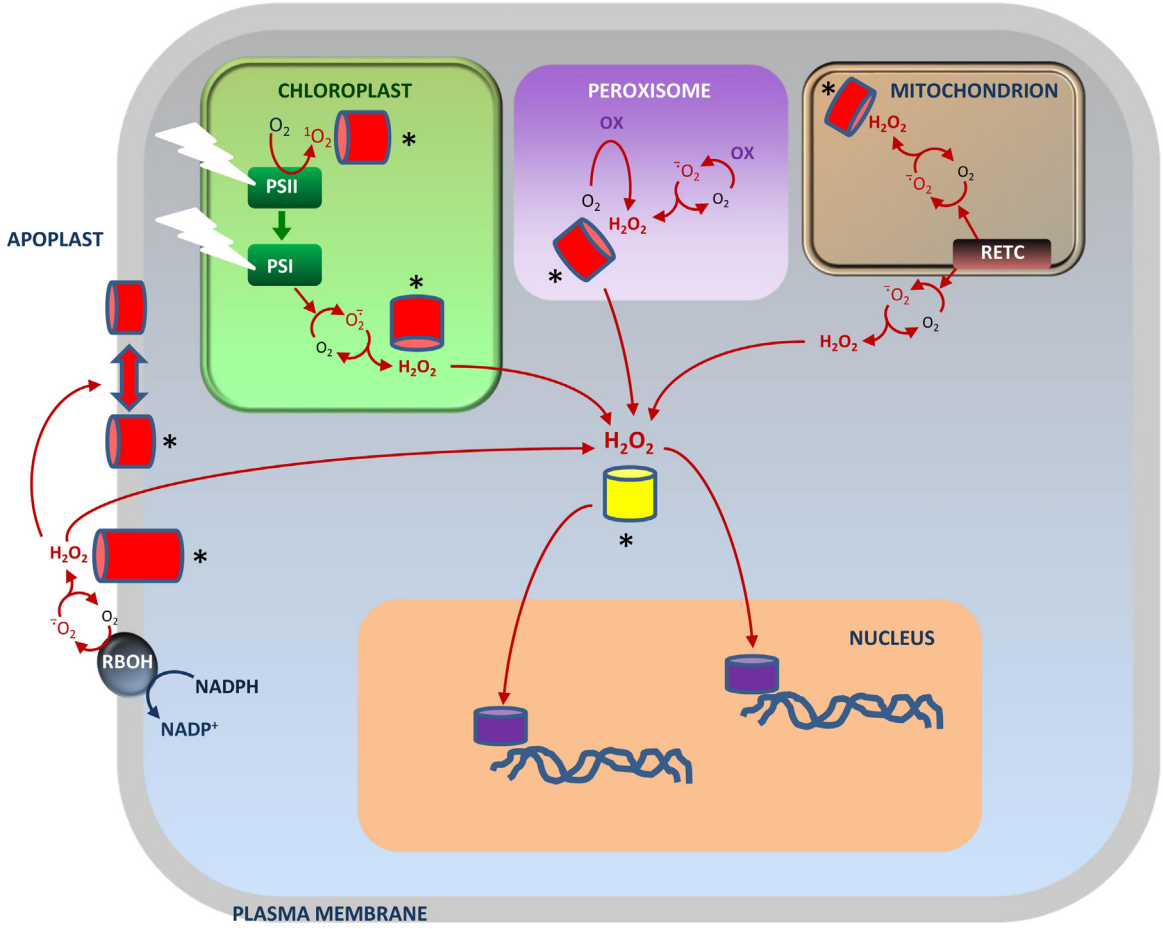

Fig. 3. Integration of multiple pathways of ROS signaling in plant cells. The most stable ROS, H_2O_2, can move from the compartments in which it is mainly produced to alter cytosolic and nuclear redox states, which can be perceived by receptor proteins (yellow barrel). In addition, site-specific receptor systems (red barrels) may perceive singlet oxygen- or H_2O_2-driven redox changes more locally, leading to (in)activation of signaling networks (*). This might involve redox modifications of protein–protein interactions or second messengers. Ultimately, gene expression will be modified by altered activity of transcription factors (purple) that may nor may not themselves be redox-modified. This extensive vocabulary of generic and site-specific signaling confers both specificity and flexibility in the redox regulation of gene expression.

responses [47,48]. Simultaneous knockdown of catalase and APX leads to induction of novel mechanisms that can strengthen resistance to DNA damage [49]. However, it is clear from other studies that the concept of a simple balance in which signal output is modulated only by the intensity of oxidative stress needs to be updated (Fig. 4B). We now know that multiple mechanisms can produce ROS at various cellular sites while the antioxidant system is not only intricate (Fig. 2) but also multifunctional. Recent work shows that defense hormone signaling elicited by catalase deficiency is partly dependent on H_2O_2-induced changes in glutathione status [35–37]. Modifications in glutathione status triggered when H_2O_2 metabolism shifts from catalase to reductive pathways require DHARs located in the cytosol. Knocking out these enzymes weakens, rather than reinforces, cell death and defense responses induced by catalase deficiency [29]. A related enzyme, monodehydroascorbate reductase (MDHAR), can act as a pro-oxidant rather than an antioxidant in certain conditions [50,51] while catalase, despite its well-attested role in H_2O_2 processing, has been reported to promote autophagy-dependent cell death [52].

The potency and redundancy of antioxidative enzymes is required to build robustness into the system, but also to allow acclimation through multiple redox signaling pathways. As shown in Fig. 4B, some signaling pathways may require the metabolism of ROS, with modified flux and/or changes in antioxidant status being the signal that is perceived. Hence, the function of some antioxidative enzymes may not only be to keep ROS low but also to allow the cell to sense and signal altered ROS availability and redox perturbations. In such cases, «ROS-processing enzyme» might be a more accurate descriptor than «antioxidative enzyme».

The ability of the cell to switch between ROS-processing pathways may restrict ROS accumulation, and so localized stress-induced changes in these molecules may not be easily detectable on a tissue level. In animal cells, physiologically beneficial levels of H_2O_2, a relatively stable oxidant, are considered to be in the low nanomolar range, with concentrations above 100 nM being deleterious [2]. Despite this, many measurements of H_2O_2 in plants imply mM concentrations in some compartments [53]. While plant cells may tolerate somewhat higher values than animal cells, inferred values for peroxisomal H_2O_2 concentrations suggest that they are unlikely to exceed 10 μM even at high rates of production [3]. Further work is required to resolve this issue [25], which is important to understanding oxidative stress signaling within the metabolic context of the plant cell.

The same signaling pathways are involved in ROS-mediated acclimation in response to stress and in the control of growth and development. For instance, catalase-deficient plants show morphological changes that are dependent on key growth regulators and phytohormones such as auxins [54–57], as well as other hormones that are most often associated with biotic stress defense but that are also involved in the control of development [37,48]. A differential distribution of superoxide and H_2O_2 is required for root growth and development. This distribution is regulated by the transcrip-

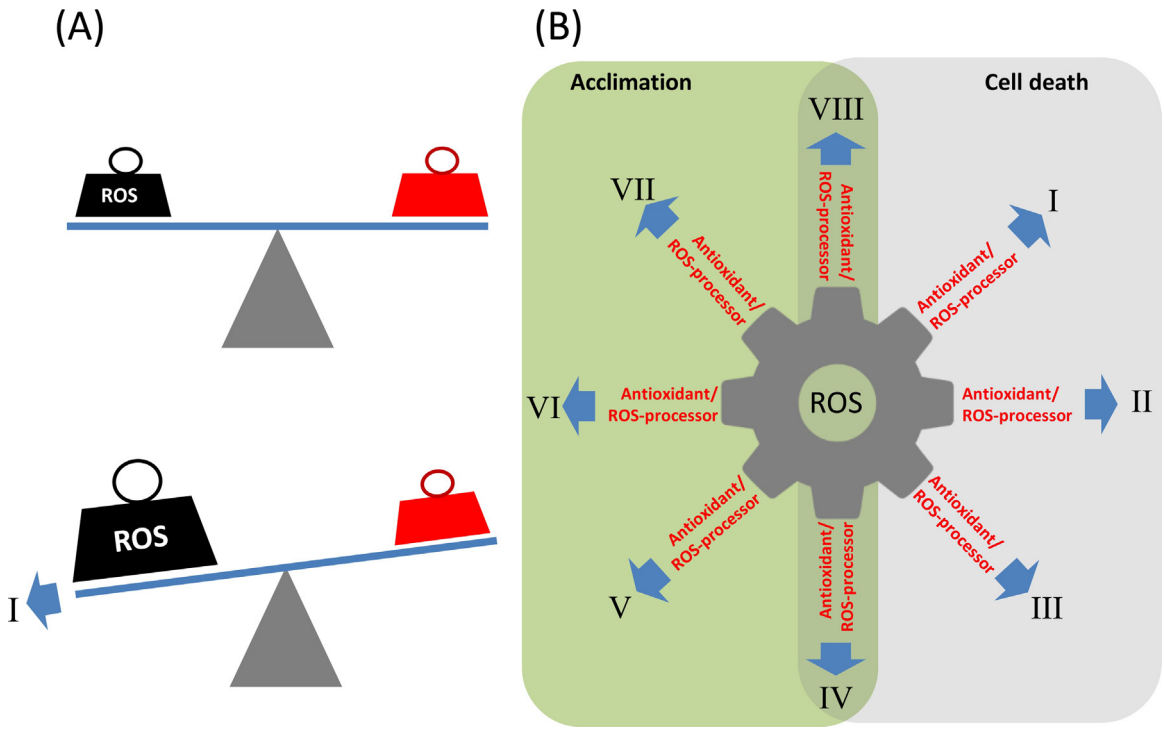

Fig. 4. Classical and updated models for ROS-antioxidant interplay in stress signaling. A, Simple balance model showing that when ROS are increased, there is a signaling change through a single pathway independent of antioxidants. B, More realistic model showing that antioxidants act as ROS processing and signaling mediators, allowing different options for signal transduction. Roman numerals indicate different possible pathways but are not mutually exclusive. The model indicates that loss of any one of these antioxidant components would tend to drive processing and signaling through the other pathways. As discussed in the text, ROS-induced changes in the status of thiols such as glutathione is particularly important.

tional regulation of distinct peroxidases [58,59]. This underlines the plasticity of plant morphology as a key response to environmental challenge, and the central role of ROS-related redox signaling in directing this plasticity.

4.2. The importance of cellular thiols

As noted in the previous section, glutathione may be important in the transmission of ROS signaling as an interfacing molecule between ROS, NO, and protein Cys groups. Particularly in plants, cellular thiols are a key redox buffer and their homeostasis is highly affected by excess ROS generated during stress. Glutathione is the most abundant and widely distributed low-molecular thiol compound of the cell. In most subcellular compartments, it is found in a large proportion in its reduced form (GSH). However, despite the presence of a large panel of reduction or conjugation systems, excess ROS can cause substantial accumulation of glutathione disulfide (GSSG) in plants [24]. Perturbing the redox state of glutathione during stress impacts different aspects of cell metabolism, as shown in studies of mutants with lower levels of glutathione or with perturbed GR capacities, which are more sensitive to oxidative stress and are affected in different aspects of development [60–65]. Glutathione may play a signaling role during stress by conjugating to various thiol residues, particularly cysteine residues of proteins, a reaction called *S*-glutathionylation [66]. Some studies have shown that such Cys modification can affect the protein function. One known example is the redox regulation of the chloroplastic glyceraldehyde-3-phosphate dehydrogenase involved in the Calvin-Benson cycle, in which *S*-glutathionylation of the catalytic Cys residue leads to inhibition of the enzyme activity [67]. While hundreds of proteins are suspected to be prone to *S*-glutathionylation, the impact of this modification on function remains to be established for most of them [68].

The role of glutathione in plant defense responses to stress and plant development also relates to its important role as a modulator of plant stress response hormones salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) signaling pathways [35–37,69] and plant development hormones auxin and abscisic acid (ABA) [70–72]. Although the underlying regulatory mechanisms are not fully resolved yet, it is likely that glutathione redox imbalance triggers oxidation of key components of hormonal signaling pathways. Indeed, GSNO-mediated oxidation through *S*-nitrosylation has been described for the auxin receptors TRANSPORT INHIBITOR RESPONSE 1 (TIR1) and the SA signaling regulator NONEXPRESSOR OF PATHOGENESIS-RELATED GENE1 (NPR1) [73–75].

As well as its close interactions with ascorbate, glutathione is a reducer of GRX, for which more than 40 members are found in Arabidopsis [76]. In addition to their role as antioxidants, GRX notably act as regenerators of PRXs and MSRs in the chloroplast and cytosol [77–79]. They also play important roles in redox signaling. A plant-specific class of GRX (called ROXYs) has a nucleocytosolic localization and the capacity to interact with TGA members of transcription factors and to act on gene transcription. In relation to this activity, ROXYs members have a strong influence on specific developmental process like flower development, gamete maturation, meristem activity, and phyllotaxy [80–84], and also function in hormonal and stress responses [85,86]. In addition to their thiol reduction activities, discrete GRX isoforms have the capacity to coordinate iron-sulfur clusters and to transfer them to acceptor proteins [87–89]. Genetic evidence shows that these isoforms are required for oxidative stress responses and in different checkpoints of plant development [40,90–92].

As well as glutathione/GRX systems, TRX are important regulators of thiol-disulfide status and also play roles in ROS processing. Similarly to GRX, plant TRX also form a complex multigenic family for which distinct isoforms are located in almost all cell compart-

ments (Fig. 2). Remarkably, at least 20 different TRX isoforms are found in the chloroplast, some of them (CDSP32, TRX x, y, z or NTRC) acting as major reducers of different types of PRX, DHAR or MSR, while other isoforms regulate key enzymes of the Calvin-Benson cycle [93,94]. Knock-out mutants in specific chloroplastic TRX show severe chloroplast development phenotypes and are sensitive to ROS-generating stress conditions (reviewed in [95]). A different situation is found in mitochondria, where a single dedicated TRX isoform (TRXo) acts as a regenerator of PRX as well as a regulator of different enzymes of the TCA cycle and the electron transport chain [96–98]. Thioredoxins also play a major role in plant defense responses. The best described example is the involvement of the cytosolic TRXh5 in regulating the SA-induced systemic acquired resistance pathway through modulation of the oligomerization of NPR1, a key actor in the plant immunity response [73,75,99].

Although each has specific functions, there is significant crosstalk between the GSH/GRX and TRX systems, which increases the robustness of thiol redox control in plants. Thus, GSH together with TRX reductases maintain thiol redox states in the cytosol and control different plant development programs [100–102].

4.3. The key role of the plasma membrane-apoplast interface in systemic signaling

While the plant immune system is not directly comparable to that in animals, it has been clearly demonstrated that responses to biotic and abiotic threats trigger local and systemic signaling and prime generic defense responses that increase resistance to subsequent threats. Both signaling processes and defense priming require ROS and associated redox processes. Sequential oxidative bursts in the plant apoplast are a key feature of systemic stress responses (Fig. 5). Challenge with either biotic or abiotic stress can lead to acquired resistance, even at sites that are distal to the challenge. In the case of biotic stress this is called systemic acquired resistance, whereas abiotic stresses such as high light induce systemic acquired acclimation. These pathways share common features, particularly related to ROS production by RBOH-type NADPH oxidases. While Arabidopsis contains ten genes encoding RBOH, most attention has focused on two (*AtRBOHD* and *AtRBOHF*) that have been implicated in a variety of stress-related and developmental responses in plants, including stomatal closure induced by abscisic acid, pathogenesis-related responses, and systemic signaling to stresses such as wounding, drought, and salt [48,103–105]. A key current concept is the reciprocal interactions between calcium and ROS in transmission of signals from cell to cell [106]. As illustrated in Fig. 5, oxidation of the apoplast is involved in the transmission of light signals from the plant apex to the systemic shaded leaves, leading to a faster induction of photosynthesis and hence a better adaptation to the fluctuating light environment [107]. Moreover, simply changing the redox balance of the cell wall and apoplast determines the extent of acclimation to high light and the susceptibility of photosynthesis to high light-induced inhibition [108]. The exact nature of the redox-sensitive components involved in such signaling remains to be fully established.

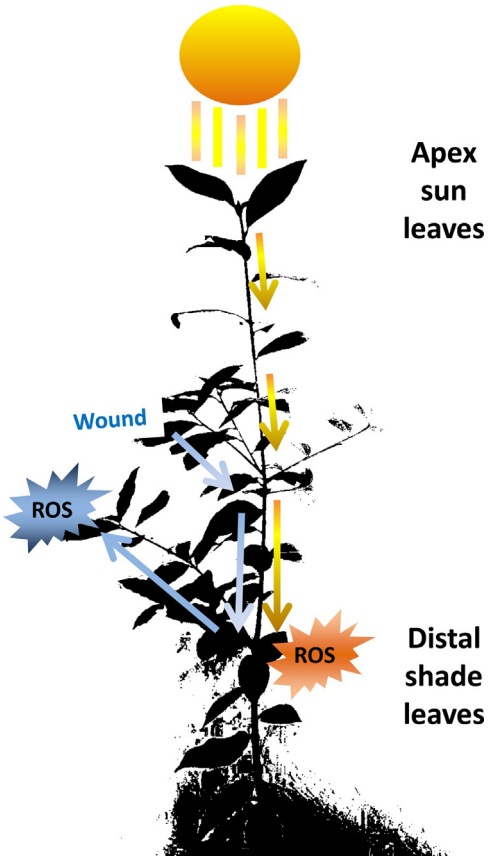

Fig. 5. Examples of systemic signaling pathways in plants. Yellow arrows: light perceived by the apex is transmitted via phytochrome and auxin to distal leaves leading to localized ROS bursts and more rapid induction of photosynthesis on subsequent exposure to high light. Blue arrows: wounding by herbivores or pathogens leads to signal transduction to distal leaves, where localized ROS bursts induce defenses to prepare for possible attack.

5. Concluding remarks

It is now clear that redox regulation is tightly embedded into almost every aspect of plant development and environmental responses. Changes in influential factors such as ROS concentrations or glutathione redox states can profoundly influence plant functions and cell fate. Outstanding questions include the mechanistic details by which redox regulation occurs. No specific ROS receptor or specific signaling transduction pathway has yet been elucidated. As we have emphasized here, it is no longer useful to view ROS as stress-inducing compounds and antioxidants as beneficial guardians: these compounds form a nexus of interacting processes in which ROS metabolism may be required to entrain oxidative signaling. While numerous peroxidases, reductases, and dehydrogenases potentially involved in processing ROS and related molecules are now annotated in several plant genomes, the biochemical reactions they catalyze in vivo are not established in many cases. Another unresolved issue, related to the existence of multiple genes encoding ROS-processing enzymes in plants, is the physiological importance of specific members in terms of their potential influence on stress resistance, phytohormone signaling, and developmental programs.

Although mechanisms similar to those recently described for oxygen sensing [109] may await discovery, ROS and related redox processes can exercise signaling functions by modulating phytohormone synthesis and signaling pathways. Multiple reports show that ROS and redox components interact closely with master coordinators of plant development such as auxins, abscisic acid, and cytokinins [54–56,71,72,74,102,103,110], as well as plant defense regulators like salicylic acid, jasmonic acid, and ethylene [29,35–37,47,69,73,75,85,99]. Several of these studies have assigned important functions to factors such as TRX, GRX, glutathione, and GSNO, and the coming years will no doubt witness further exciting developments in this area. For example, a very recent report implicates a member of the plant-specific class III GRX in long-distance signaling of nitrogen availability [111], a key factor in determining plant growth and yield.

Progress in understanding the complexity of redox signaling in plants has been greatly accelerated by work on the model plant, Arabidopsis. The unique tools and rich information available should keep this plant at the forefront of fundamental research for some years to come, but questions remain concerning the transposition of this information to other plants, particularly those that are taxonomically distant or that show specific traits. Nevertheless, available knowledge suggests that many redox mechanisms show a substantial degree of conservation between different plant groups, and that the goal of improving crop performance will ultimately benefit from the fundamental knowledge generated for model plants. Within the context of a changing climate, intriguing questions are how ROS and redox-triggered processes may affect plant responses to abiotic and biotic stress in a higher-CO_2 world [112–114]. Improved knowledge of this and other features of redox signaling will help to inform attempts to exploit the flexibility of plant growth and development to optimize crop performance in present and future field conditions [4,5].

Acknowledgments

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