ANTIOXIDANTS & REDOX SIGNALING
Volume 8, Numbers 9 & 10, 2006

Forum Review

Reactive Oxygen Species Signaling in Plants

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ABSTRACT

The evolution of aerobic metabolism such as respiration and photosynthesis resulted in the generation of reactive oxygen species (ROS). A common property of all ROS types is that they can cause oxidative damage to proteins, DNA, and lipids. This toxicity of ROS explains the evolution of complex arrays of nonenzymatic and enzymatic detoxification mechanisms in plants. However, increasing evidence indicates that plants also make use of ROS as signaling molecules for regulating development and various physiological responses. In this review, novel insights into the mechanisms of how plants sense and respond to ROS are discussed in the context of the biological effects and functions of ROS in plants. *Antioxid. Redox Signal.* 8, 1757–1764.

INTRODUCTION

In plants, reactive oxygen species (ROS) are continuously produced as byproducts of various metabolic pathways that are localized in different cellular compartments (19). Under physiological steady-state conditions, these molecules are scavenged by different antioxidative defense components that are often confined to particular compartments (2). The balance between production and scavenging of ROS may be perturbed by a number of adverse environmental factors. As a result of these disturbances, intracellular levels of ROS may rapidly rise (18, 36, 52, 64). External conditions that adversely affect the plants can be biotic, imposed by other organisms, or abiotic, arising from an excess or deficit in the physical or chemical environment.

In the presence of transition metal ions, hydrogen peroxide may be reduced by superoxide to hydroxyl radicals. Since there are no known scavengers of hydroxyl radicals, the only way to avoid oxidative damage through this radical would be to control the reactions that lead to its generation. Thus, cells had to evolve sophisticated strategies to keep the concentrations of superoxide, hydrogen peroxide, and transition metal ions such as Fe^{2+}, Fe^{3+}, and Cu^{2+} under tight control.

(a) Nonenzymatic antioxidants include ascorbate and glutathione (GSH), but also tocopherol, flavonoids, alkaloids, and carotenoids. Ascorbate and GSH are major cellular redox buffers. Mutants with decreased ascorbic acid levels (8) or altered GSH content (24) are hypersensitive to stress.

(b) Enzymatic ROS scavenging mechanisms in plants include superoxide dismutase (SOD), ascorbate peroxidase (APX), glutathione peroxidase (GPX), and catalase (CAT). SODs act as the first line of defense against ROS, dismutating superoxide to H_2O_2. APX, GPX, and CAT subsequently detoxify H_2O_2. Unlike most other organisms, plants have multiple genes encoding SOD and APX isoforms that are specifically targeted to chloroplasts, mitochondria, peroxisomes, as well as to the cytosol and apoplast (5).

ROS SIGNALING IN PLANTS

Rapid progress has been made in recent years in defining ROS as a major signal in diverse biological processes in plants. ROS can react with a large variety of biomolecules that can cause irreversible damage and lead to necrosis and death (22, 55). On the other hand, ROS can influence the expression of a number of genes and signal transduction pathways, suggesting that cells have evolved strategies to utilize ROS as signals that control various biological programs (10). ROS are ideally suited to act as such signaling molecules

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Table 1. Half-Life and Diffusion Distances for Prominent ROS Signals that are of Biological Significance

ROS	*Half-life*	*Diffusion distance*
$^1O_2$	1.4 μsec	0.8 μm
O_2^-	1 sec	8 mm
H_2O_2	∞	—
•OH	1 – 0.01 μsec	0.5 μm

since they are small and can diffuse over short distances (Table 1). Among the different ROS, only H_2O_2 can cross plant membranes and can therefore directly function in cell-to-cell signaling. Moreover, cells have evolved several mechanisms for rapid and controllable ROS production and removal. This concept requires cells to possess specific ROS sensors that process and translate this information into the respective biological output programs. In several systems, it has been shown that ROS can modulate the activity of specific transcription factors, thereby directly affecting gene expression. In addition, nonenzymatically generated ROS oxidation products may act as second messengers that trigger biological responses. For example, polyunsaturated fatty acids are a preferred target of ROS attack, and several lipid oxidation products are biologically active in that they can change gene expression (25). ROS can also modulate gene expression by changing the cellular redox state. Changes in the redox state of chloroplasts strongly affect organellar enzyme activities and gene expression programs. Although many aspects of ROS signaling are still unclear, distinct modes of ROS action have been identified in different processes and will be discussed here.

ROS AND STOMATA

Recent work has shown ROS to be essential signals mediating stomatal closure induced by ABA (Fig. 1 and Table 2). The phytohormone abscisic acid (ABA) accumulates in response to dehydration and induces a range of stress adaptation responses including stomatal closure. Earlier work had shown that H_2O_2 induces stomatal closure (39) and that guard cells synthesize ROS in response to elicitor challenge (1, 34). H_2O_2 is an endogenous component of ABA signaling in *Arabidopsis* guard cells. ABA-stimulated ROS accumulation induces stomatal closure via activation of plasma membrane calcium channels (50). ABA-induced ROS synthesis also occurs in *Vicia faba* (70), where ROS production takes place at the plasma membrane and in the chloroplast. This study indicates the complexity of ROS signaling in this system. Various *Arabidopsis* mutants have been used to dissect ABA and ROS signaling in guard cells. The *gca2* (guard cell associated protein 2) mutant displays normal ABA-induced ROS production, but no H_2O_2-induced calcium channel activation, nor stomatal closure (50).

Protein phosphorylation is also involved in guard cell signaling, as shown by analysis of the ABA-insensitive *abi1* and *abi2* mutants (Fig. 1). *ABI1* and *ABI2* encode protein phosphatase 2C enzymes, whose phosphatase activities are strongly reduced in the *abi1* and *abi2* point mutants. Whereas ABA-induced ROS generation was absent in *abi1*, *abi2* mutants synthesized ROS but could not respond to H_2O_2 (45). Therefore, ABI1 may act upstream and ABI2 downstream of ROS signaling.

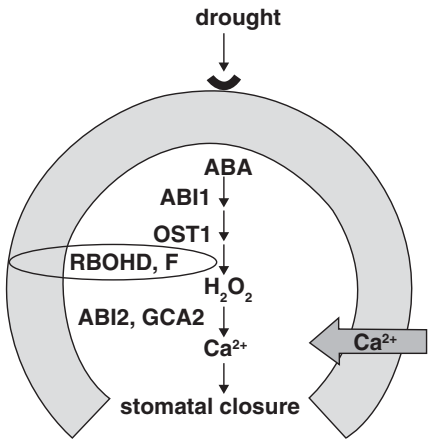

FIG. 1. ROS signaling in stomatal closure. Dehydration is perceived via an unknown receptor mediating ABA synthesis in guard cells. Increased ABA levels trigger the production of H_2O_2, which in turn mediates the activation of Ca^{2+} channels at the plasma membrane, resulting in stomatal closure. Mutants blocked at various steps of the signaling pathway are shown.

In an elegant screen for mutants affected in drought-induced stomatal closure, *OST1* (open stomata 1) was identified. Positional cloning and mutant analysis characterized OST1 as a protein kinase that functions between ABA perception and ROS signaling of stomata regulation (46). OST1 is activated by ABA in guard cell protoplasts of wild type but not of *ost1* plants. Moreover, ABA-induced ROS synthesis was absent from *ost1* plants, although *ost1* stomata still closed in response to H_2O_2. The plasma membrane-located NADPH oxidases AtrbohD and AtrbohF were found to be required for ABA-induced stomatal ROS production (33); and mutant plants in these NADPH oxidases were similarly defective in stomatal closure as *ost1* plants. These findings suggest that OST1 regulates ROS production via these NADPH oxidases.

It was recently found that ethylene receptor 1 (ETR1), a member of the two-component histidine kinase family that functions in ethylene signaling in *Arabidopsis*, is also involved in ROS signaling of stomata (16). In prokaryotes and fungi, two-component systems are well-known redox sensors (54, 68). In prokaryotes, two-component systems usually consist of a histidine kinase that senses the signal and a response regulator that functions as transcription factor. The transmembrane sensory kinase functions through its capacity to autophosphorylate a histidine residue in response to the presence or absence of an external stimulus. The phosphoryl group is subsequently transferred from the histidine to an aspartate residue in the response regulator. The induced conformational change in the response regulator alters its DNA

TABLE 2. GENES INVOLVED IN *ARABIDOPSIS* ROS SIGNALING

Mutation	*Process affected*	*Phenotype*	*Protein function*	*Ref.*
abi1 point mutant	Stomatal closure	No ROS generation upon ABA	PP2C	45
abi2 point mutant		Normal ROS generation upon ABA but no response to H2O2	PP2C	
rbohD and *AtrbohF* T-DNA k.o.		No ROS generation upon ABA	Plasma membrane-located NADPH oxidases	33
ost1 no kinase activity		No ABA-induced ROS synthesis but normal stomatal closure upon H2O2	Kinase	
etr1 C65S point		Completely abrogates ROS signaling in stomata	Two-component histidine kinase	
etr1 lacking C-term. kinase domain		Normal ROS signaling, no ethylene signaling		
gca2		Normal ROS generation upon ABA but no H_2O_2-induced calcium channel activation and stomatal closure	Homology to calmodulin-binding protein	50
rhd2	Root hair development	No ROS in the growing tips	NADPH oxidase	20
oxi1 T-DNA k.o.		Fewer and shorter root hairs No MPK3,6 activation upon ox stress	Protein kinase	56
CITRX silencing (tomato)	Pathogen defense	Enhanced pathogen-induced ROS accumulation, calcium-dependent protein kinase activation and defense gene expression	Thioredoxin	57
npr1	SAR control	No SA- or pathogen-induced PR gene expression	Redox-sensitive transcription factor	43
rbohD and *rbohF* T-DNA k.o.	Pathogen defense	Reduced ROS generation and PCD following bacterial challenge	NADPH oxidase	63

binding affinity and thereby promotes gene expression of certain promoters. In budding and fission yeast, two component histidine kinases can function as sensors of oxidative stress, but modify gene expression through activation of mitogen-activated protein kinase signal transduction cascades (Fig. 2 and Ref. 60). Similar to fungi, plants also contain a number of two-component histidine kinases that are not found in the animal kingdom (29).

Interestingly, ethylene and ROS seem to activate ETR1 by distinct mechanisms. ETR1 carries an N-terminal input domain that is separated from a kinase domain by a hydrophobic transmembrane region. The response regulatory domain is also located at the C-terminal cytoplasmic face of ETR1. Point mutation and deletion analyses revealed that a change of cysteine-65 to tyrosine in ETR1 completely abrogates ROS signaling in stomata, strongly suggesting that this particular cysteine residue might be involved in ROS sensing by ETR1. Interestingly, the C-terminal kinase domain of ETR1 is dispensable for ROS but not for ethylene signaling. These results suggest that ROS and ethylene signaling might involve interaction with different downstream partners. Considering that the kinase domain of ETR1 is not required for ROS signaling in plants, it is surprising that ETR1 can functionally replace yeast double mutants lacking the ROS sensing histidine kinase Sln1 and the downstream response regulator Ssk1 (Fig. 2 and Ref. 16). For budding yeast, it has been shown that signaling requires the kinase activity of Sln1 for transferring a

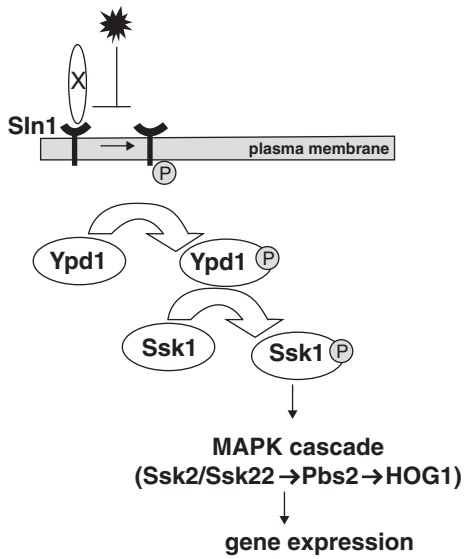

FIG. 2. Two-component system in budding yeast. Upon binding of an unknown extracellular signal (X), Sln1 changes to an autophosphorylated active form, which mediates the transfer of a phosphate group (P) to the response regulator Ssk1 via the intermediate Ypd1. Activated Ssk1 triggers a MAPK cascade leading to changes in expression of target genes. Stress signals (*e.g.*, ROS) prevent Sln1 autophosphorylation, thus retaining the cascade in an inactive state.

phosphoryl group via the intermediary component Ypd1 to the response regulator Ssk1. Stress inhibits autophosphorylation of Sln1, resulting in accumulation of nonphosphorylated Ssk1, which can activate the mitogen-activated protein kinase HOG1 cascade (27). Further investigations are needed to clarify the differences in the ROS signaling mechanisms in plants and yeast.

ROS AND ROOT HAIR DEVELOPMENT

Root hairs assist water and nutrient uptake from the soil and help to anchor the plant in the soil. Because root hairs are not essential for plant growth under laboratory conditions, they have become an attractive model to study cell polarity and development in plants. Recently, *root hair defective 2* (*rhd2*), an *Arabidopsis* mutant forming root hair bulges but no elongated root hairs, was found to be defective in NADPH oxidase (20). In this study, ROS were localized in the growing tips of root hairs of wild-type plants but not in the root hair bulges of the *rhd2* mutant. Possibly, NADPH oxidase is controlled via small GTPases of the Rop family in analogy to mammalian cells, where the small GTPase Rac regulates NADPH oxidase and ROS production (69). Two recent reports (4, 56) have revealed that OXI1 (oxidative stress inducible 1) belonging to the AGC family of protein kinases is involved in root hair growth. Mutant plants defective in OXI1 show strong reduction in number and length of root hairs. OXI1 kinase activity is induced by phosphatidic acid (4) and by hydrogen peroxide (56), and both factors have been shown to be regulating root hair growth. A T-DNA *oxi1* null mutant is impaired in the activation of the two *Arabidopsis* MAPKs, MPK3 and MPK6, upon oxidative stress (56), suggesting that OXI1 functions downstream of ROS but upstream of the MAPK module. Interestingly, a previous study had already linked the stress-induced *Medicago* MAPK SIMK, a close homolog of *Arabidopsis* MPK6, to root hair growth. In this work, inhibition of MAPK activity resulted in abrogation of root hair growth, and expression of constitutively active SIMK was correlated with increased root hair length (59). These findings point to the existence of a ROS signaling pathway as a regulator of root hair development that depends on a number of distinct protein kinases.

ROS AND BIOTIC STRESS

The importance of ROS in root hair growth and stomatal regulation has become clear by now. However, originally ROS were attributed a role in plant pathogen defense. Although cell wall-bound peroxidases and apoplastic amine oxidases also contribute to ROS production in response to pathogen attack, ROS are clearly produced during pathogen defense by the very same NADPH oxidases that regulate root hair growth (24, 28). In contrast to superoxide, H_2O_2 can diffuse into cells and activate many of the plant defenses, including PCD (programmed cell death) (11). During plant–pathogen interactions, the activities and levels of the ROS detoxifying enzymes APX and CAT are downregulated by SA and NO (31). Because the plant simultaneously produces more ROS while decreasing its ROS scavenging capacities, accumulation of ROS and activation of PCD occurs. The suppression of ROS detoxifying mechanisms is crucial for the onset of PCD. ROS production at the apoplast alone without suppression of ROS detoxification does not result in the induction of PCD (14, 41), indicating an absolute requirement for the coordinated production of ROS and downregulation of ROS scavenging mechanisms.

Induction of PCD is thought to potentially limit the spread of disease from the infection point. During incompatible reactions, when host defense responses including PCD are induced, H_2O_2 production occurs in a biphasic manner. The initial and very rapid accumulation of H_2O_2 is followed by a second and prolonged burst of H_2O_2 production. During compatible interactions, only the first peak of H_2O_2 accumulation occurs and the pathogen can systemically infect the host plant (6). The oxidative burst in pathogen-challenged *Arabidopsis* leaves was found to induce a subsequent burst in distal parts of the plant, leading to systemic immunity via the expression of defense-related genes (3). Although the oxidative burst is a primary response following pathogen challenge leading to PCD (7), and H_2O_2 was shown to induce PCD in various systems (15, 35, 61), in some cases H_2O_2 is apparently not sufficient (23) or not required (30) for PCD induction.

A certain exposure time of cells to H_2O_2 involving *de novo* transcription and translation is required for PCD to occur (15, 61). Pharmacological data indicate that removal of ROS during pathogen or elicitor challenge reduces PCD (15, 35), and tobacco plants with reduced CAT or APX expression levels show enhanced PCD upon exposure to low doses of bacteria (41). On the other hand, elicitor-challenged tobacco suspension cells undergo PCD independent of H_2O_2 production (30). Tobacco plants silenced for a catalase gene accumulate H_2O_2 during photosynthesis (65). Using high light (HL) conditions, endogenous H_2O_2 production can be achieved, leading to cell death. Short HL preexposure (1–2 h) triggered an acclimatory effect that protected the plant against subsequent long HL stress treatment. These findings suggest that whether H2O2 has a beneficial or harmful effect is dose-dependent and depends on when and for how long it occurs.

It is not clear yet to what extent PCD in plants and animals share similar mechanisms. Expression of the animal cell death suppressor genes *Bcl-xl* and *Ced-9* in tobacco plants suppressed oxidative stress-induced cell death (40). In animals, mitochondria play a primary role in ROS production and in triggering apoptosis. Mitochondrial ROS production has also been implicated in eliciting ROS-induced cell death in plants (37, 61). Although the majority of ROS in plants appears to be produced in chloroplasts and peroxisomes, *Arabidopsis* knock-out mutants lacking functional *rboh* genes display reduced ROS generation and PCD following bacterial challenge (62). These data clearly point to an essential role of membrane-located NADPH oxidases in pathogen defense in plants that is reminiscent of the function of these enzymes during apoptosis in animals.

Signaling through two-component histidine kinases is not the only way by which oxidative stress can be translated into MAPK cascade activation. An interesting mechanism has been uncovered in animals for the ROS-activated MAPK kinase kinase ASK1. In unstimulated cells, ASK1 is kept inac-

tive through interaction with thioredoxin (58). Upon oxidative stress, thioredoxin becomes oxidized, resulting in dissociation from ASK1. Homodimerization of the free ASK1 subsequently results in activation of the MAPK pathway. Although it is presently unknown whether thioredoxin-mediated MAPK activation also exists in plants, a recent study revealed an essential role of the CITRX thioredoxin in regulating pathogen defense (57). The particular thioredoxin was originally identified in a yeast two-hybrid screen for proteins interacting with the tomato Cf9 resistance protein that is required for defense against the fungal pathogen *Cladosporium fulvum* carrying the corresponding *avr9* gene. Although the exact mechanism is unclear, silencing of CITRX enhanced pathogen-induced ROS accumulation, calcium-dependent protein kinase activation and defense gene expression. This study shows that CITRX acts as a negative regulator of defense responses mediated by Cf9. However, it remains to be shown whether this function of CITRX actually requires its redox-regulatory activity.

ROS-induced gene expression in prokaryotes, fungi, and plants also show common mechanisms (21). The transcription factors OxyR in *E. coli* and Yap1 in budding yeast have been found to be of paramount importance in oxidative stress signaling (71). OxyR and Yap1 are redox-sensitive transcription factors and modulate gene expression in response to oxidative stress. The activity of the transcription factors is regulated through ROS-mediated oxidation of their cysteine thiol (12, 13). Recently, NPR1 was identified as a redox-sensitive transcription factor in plants (43). NPR1 is an essential regulator of plant systemic acquired resistance (SAR). In the oxidized state, which corresponds to an unchallenged situation, NPR1 occurs as oligomers in the cytosol. After pathogen challenge, a biphasic change in cellular reduction potential occurs, resulting in the reduction of NPR1 to a monomeric form that accumulates in the nucleus and that activates gene expression. An interacting partner of NPR1 is the transcription factor TGA1, which is involved in SAR-mediated regulation of gene expression. Before the induction of SAR, TGA1 carries an intramolecular disulfide bridge between two conserved cysteine residues (17). During SAR, SA accumulation causes the reduction of this disulfide bridge, resulting in enhanced interaction of TGA1 with NPR1. This interaction enhances the DNA-binding capacity of TGA1, resulting in the activation of numerous pathogenesis-related (PR) genes.

ROS AND ABIOTIC STRESS

In plants, reactive oxygen species are continuously produced predominantly in chloroplasts, peroxisomes, and mitochondria. Production and removal of ROS must be strictly controlled. The equilibrium between production and scavenging of ROS, however, may be perturbed by a number of adverse abiotic stress factors such as high light, drought, and low or high temperatures (18, 36, 52, 64).

Whereas in mammalian cells the mitochondria are the major source of ROS, the relative contribution of mitochondria to ROS production in green tissues seems to be very low (53). One reason that plant mitochondria do not produce more ROS could be the presence of the alternative oxidase (AOX) that catalyzes the reduction of O_2 by ubiquinone. The AOX competes for electrons, and in this way may help to reduce ROS production in mitochondria. This suggestion has been supported by the findings that H_2O_2 induces the synthesis of AOX (67) and overproduction of AOX in transgenic cell lines reduces ROS production, whereas antisense cells with reduced levels of the alternative oxidase (AOX) accumulate five times more ROS than control cells (38).

Oxygen is continuously produced during light-driven photosynthetic electron transport and at the same time removed from chloroplasts by reduction and assimilation. The formation of ROS during photosynthesis occurs by direct photoreduction of O_2 by reduced electron transport components associated with PSI and reactions linked to the photorespiratory cycle. When plants are exposed to light intensities that exceed the capacity of CO_2 assimilation, over-reduction of the electron transport chain leads to the inhibition of photosynthesis. To protect the photosynthetic apparatus against photoinhibition, thermal dissipation of excess excitation energy and transfer of electrons to various acceptors within the chloroplast occurs (49).

In general, the function of ROS during abiotic stresses appears to be opposite to that during pathogen defense. Upon abiotic stresses, ROS scavenging enzymes are induced to decrease the concentration of toxic intracellular ROS levels. The differences in the function of ROS between biotic and abiotic stresses might result from the action of hormones and crosstalk between different signaling pathways or from differences in the locations where ROS are produced and/or accumulate during different stresses. These considerations raise the question of how plants can regulate ROS production and scavenging mechanisms when they are exposed simultaneously to pathogen attack and abiotic stress. Evidence for the significance of such conflicting situations comes from experiments with tobacco plants that showed reduced PCD after exposure to oxidative stress (41). The oxidative stress pretreatment resulted in increased levels of ROS scavenging enzymes, thereby abrogating the plants' ability to build up sufficient ROS for inducing PCD. In accordance with this model, CAT overproducing plants show decreased resistance to pathogen infection (51), wounding (48), and high light treatment (44).

MAPK signaling modules are involved in eliciting responses to various signals including oxidative stresses (47). In *Arabidopsis,* H_2O_2 activates the MAPKs (MPK3 and MPK6) via the MAPK kinase kinase (MAPKKK) ANP1 (32). Overexpression of ANP1 in transgenic plants resulted in increased tolerance to heat shock, freezing, and salt stress (32). H_2O_2 also increases expression of the *Arabidopsis* nucleotide diphosphate (NDP) kinase 2 (42). Overexpression of At-NDPK2 reduced accumulation of H_2O_2 and enhanced tolerance to multiple stresses including cold, salt, and oxidative stress. The effect of NDPK2 might be mediated by the MAPKs, MPK3 and MPK6, because NDPK2 can interact and activate these MAPKs.

Because H_2O_2 is a mild oxidant that can oxidize thiol residues, it has been speculated that H_2O_2 is sensed via modification of thiol groups in certain proteins. Recent work has identified human protein tyrosine phosphatase PTP1B to be modified by H_2O_2 at the active site cysteine (66). Interest-

ingly, inactivation of PTP1B by H_2O_2 is reversible and can be brought about by incubation with glutathione. A similar regulation is likely to occur in plants because PTP1, an *Arabidopsis* PTP that can inactivate the *Arabidopsis* MPK6 can be inactivated by H_2O_2 (26).

OPEN QUESTIONS

An unavoidable consequence of adapting life to an oxygen-containing environment was the continuous production of ROS as toxic byproducts of metabolism. It is likely that the evolution of mechanisms for ROS detoxification evolved step by step with those for sensing and signaling of ROS at various sites where ROS were produced. Later cells had sophisticated mechanisms to produce and use ROS as secondary messengers for various tasks ranging from coping with environmental challenges to making developmental decisions. By uncovering novel biological roles for ROS, it becomes clear that our understanding of ROS signaling is more than superficial and that a number of questions await clarification. Among these questions is: How can a given ROS specify different biological responses? Do different signaling cascades exist for ROS originating from different cellular sites? Can different types of intracellular ROS induce distinct biological outputs in the same cellular system?

ACKNOWLEDGMENTS

The work was supported by projects of the Austrian Science Fund, the Vienna Science and Technology Fund and the European Union Marie Curie Training and Quality of Life programs.

ABBREVIATIONS

ABA, abscisic acid; ABI1,2, ABA-insensitive; AGC, cAMP/cGMP-dependent protein kinase; ANP1, NPK1-related protein kinase 1; AOX, alternative oxidase; APX, ascorbate peroxidase; ASK1, *Arabidopsis* serine/threonine kinase 1; AtrbohD,F, respiratory burst oxidase protein F; AVR9, avirulence-gene 9; BCL-Xl; B-cell lymphoma; CAT, catalase; CED-9, cell death protein 9; CITRX, Cf-9-interacting thioredoxin; ETR1, ethylene receptor 1; GCA2, guard cell associated protein 2; GPX, glutathione peroxidase; GSH, glutathione; H_2O_2, hydrogen peroxide; HL, high light; HOG1, high osmolarity glycerol 1; M(A)PK, mitogen-activated protein kinase; MAPKK(K), MAPK kinase kinase (kinase); NDPK2, nucleotide diphosphate kinase; NO, nitric oxide; NPK1, *Nicotiana* protein kinase 1; NPR1, nonexpressor of pathogenesis-related proteins 1; OST1, open stomata 1; OXI1, oxidative stress inducible 1; OxyR, oxidative stress transcriptional regulator; PCD, programmed cell death; PP2C, protein phosphatase 2c; PR, pathogenesis-related; PTP1, protein tyrosine phosphatase 1; RHD2, root hair defective 2; ROP, Ras-homology-like small G protein; ROS, reactive oxygen species; SA, salicylic acid; SAR, systemic acquired resistance; SLN1, synthetic lethal of N-end rule 1; SOD, superoxide dismutase; SSK1, suppressor of sensor kinase 1; T-DNA, transfer DNA; TGA1, TGACG motif-binding transcription factor 1; YAP1, AP-1-like transcription factor 1; YPD1, tyrosine phosphatase dependent 1.

REFERENCES

1. Allan AC and Fluhr R. Two distinct sources of elicited reactive oxygen species in tobacco epidermal cells. *Plant Cell* 9: 1559–1572, 1997.
2. Alscher RG, Donahue JH, and Cramer CL. Reactive oxygen species and antioxidants: relationships in green cells. *Physiol Plantarum* 100: 224–233, 1997.
3. Alvarez ME, Pennell RI, Meijer P-J, Ishikawa A, Dixon RA, and Lamb C. Reactive oxygen intermediates mediate a systemic signal network in the establishment of plant immunity. *Cell* 92: 773–784, 1998.
4. Anthony RG, Henriques R, Helfer A, Meszaros T, Rios G, Testerink C, Munnik T, Deak M, Koncz C, and Bogre L. A protein kinase target of a PDK1 signalling pathway is involved in root hair growth in Arabidopsis. *EMBO J* 23: 572–581, 2004.
5. Asada K and Takahashi M. Production and scavenging of active oxygen in photosynthesis. In: *Photoinhibition,* edited by Kyle DJ, et al. Elsevier, 1987, pp.227–287.
6. Baker MA and Orlandi EW. Active oxygen in plant pathogenesis. *Annu Rev Phytopathol* 33: 299–321, 1995.
7. Bolwell GP. Role of active oxygen species and NO in plant defense responses. *Curr Opin Plant Biol* 2: 287–294, 1999.
8. Conklin PL, Williams EH, and Last RL. Environmental stress sensitivity of an ascorbic acid-deficient *Arabidopsis* mutant. *Proc Natl Acad Sci USA* 93: 9970–9974, 1996.
9. Creissen G. Firmin J, Fryer M, Kular B, Leyland N, Reynolds H, Pastori G, Wellburn F, Baker N, Wellburn A, and Mullineaux P. Elevated glutathione biosynthetic capacity in the chloroplasts of transgenic tobacco plants paradoxically causes increased oxidative stress. *Plant Cell* 11: 1277–1292, 1999.
10. Dalton TP, Shertzer HG, and Puga A. Regulation of gene expression by reactive oxygen. *Annu Rev Pharmacol Toxicol* 39: 67–101, 1999.
11. Dangl JL and Jones JDG. Plant pathogens and integrated defense responses to infection. *Nature* 411: 826–833, 2001.
12. Delaunay A, Isnard A-D, and Toledano MB. H_2O_2 sensing through oxidation of the Yap1 transcription factor. *EMBO J* 19: 5157–5166, 2000.
13. Delaunay A, Pflieger D, Barrault MB, Vinh J, and Toledano MB. A thiol peroxidase is an H_2O_2 receptor and redox-transducer in gene activation. *Cell* 111: 471–481, 2002.
14. Delledonne M, Zeier J, Marocco A, and Lamb C. Signal interactions between nitric oxide and reactive oxygen intermediates in the plant hypersensitive disease resistance response. *Proc Natl Acad Sci USA* 98: 13454–13459, 2001.
15. Desikan R, Reynolds A, Hancock JT, and Neill SJ. Harpin and hydrogen peroxide both initiate programmed cell death but have differential effects on gene expression in *Arabidopsis* suspension cultures. *Biochem J* 330: 115–120, 1998.
16. Desikan R, Hancock JT, Bright J, Harrison J, Weir I, Hooley R, and Neill SJ. A role for ETR1 in hydrogen peroxide

signaling in stomatal guard cells. *Plant Physiol* 137: 831–834, 2005.
17. Despres C, Chubak C, Rochon A, Clark R, Bethune T, Desveaux D, and Fobert PR. The Arabidopsis NPR1 disease resistance protein is a novel cofactor that confers redox regulation of DNA binding activity to the basic domain/leucine zipper transcription factor TGA1. *Plant Cell* 15: 2181–2191, 2003.
18. Elstner EF. Mechanisms of oxygen activation in different compartments of plant cells. In: *Active Oxygen/Oxidative Stress in Plant Metabolism*, edited by Pelland EJ and Steffen KL. American Society of Plant Physiologists, Rockville, MD. 1991, pp. 13–25.
19. Foyer CH and Harbinson JC. Oxygen metabolism and the regulation of photosynthetic electron transport. In: *Causes of Photooxidative Stress and Amelioration of Defense Systems in Plants,* edited by Foyer CH and Mullineaux PM. Boca Raton Fl: CRC Press. 1994, pp. 1–42.
20. Foreman J, Demidchik V, Bothwell JH, Mylona P, Miedema H, Torres MA, Linstead P, Costa S, Brownlee C, Jones JD, Davies JM, and Dolan L. Reactive oxygen species produced by NADPH oxidase regulate plant cell growth. *Nature* 422: 442–446, 2003.
21. Georgiou G. How to flip the (redox) switch. *Cell* 111: 607–610, 2002.
22. Girotti AW. Photosensitized oxidation of membrane lipids: reaction pathways, cytotoxic effects and cytoprotective mechanisms. *J Photochem Photobiol* 63: 103–113, 2001.
23. Glazener JA, Orlandi EW, and Baker CJ. The active oxygen response of cell suspensions to incompatible bacteria is not sufficient to cause hypersensitive cell death. *Plant Physiol* 110: 759–763, 1996.
24. Grant JJ and Loake GJ. Role of reactive oxygen intermediates and cognate redox signaling in disease resistance. *Plant Physiol* 124: 21–29, 2000.
25. Grether–Beck S, Bonizzi G, Schmitt–Brenden H, Felsner I, Timmer A, et al. Non-enzymatic triggering of the ceramide signaling cascade by solar UVA radiation. *EMBO J* 19: 5793–5800, 2000.
26. Gupta R and Luan S. Redox control of protein tyrosine phosphatases and mitogen-activated protein kinases in plants. *Plant Physiol* 132: 1149–1152, 2003.
27. Gustin MC, Albertyn J, Alexander M, and Davenport K. MAP kinase pathways in the yeast *Saccharomyces cerevisiae*. *Microbiol Mol Biol Rev* 62: 1264–1300, 1998.
28. Hammond–Kosack K and Jones JDG. Responses to plant pathogens. In: *Biochemistry and Molecular Biology of Plants*, edited by Buchanan BB, Gruissem W, and Jones RL. American Society of Plant Physiologists, Rockville, MD. 2000, pp. 1102–1156.
29. Hwang I, Chen H-C, and Sheen J. Two-component signal transduction pathways in *Arabidopsis*. *Plant Physiol* 129: 500–515, 2002.
30. Yuki Ichinose Y, Andi S, Doi R, Tanaka R, Taguchi F, Sasabe M, Toyoda K, Shiraishi T, and Yamada T. Generation of hydrogen peroxide is not required for harpin-induced apoptotic cell death in tobacco BY-2 cell suspension cultures. *Plant Physiol Biochem* 39: 771–776, 2001.
31. Klessig DF, Durner J, Noad R, Navarre DA, Wendehenne D, Kumar D, Zhou JM, Shah J, Zhang S, Kachroo P, Trifa Y, Pontier D, Lam E, and Silva H. Nitric oxide and salicylic acid signaling in plant defense. *Proc Natl Acad Sci USA* 97: 8849–8855, 2000.
32. Kovtun Y, Chiu W-L, Tena G, and Sheen J. Functional analysis of oxidative stress-activated mitogen-activated protein kinase cascade in plants. *Proc Natl Acad Sci USA* 97: 2940–2945, 2000.
33. Kwak JM, Mori IC, Pei ZM, Leonhardt N, Torres MA, Dangl JL, Bloom RE, Bodde S, Jones JD, and Schroeder JI. NADPH oxidase AtrbohD and AtrbohF genes function in ROS-dependent ABA signaling in *Arabidopsis*. *EMBO J* 22: 2623–2633, 2003.
34. Lee S, Choi H, Suh S, Doo IS, Oh KY, Choi EJ, Schroeder Taylor AT, Low PS, and Lee Y. Oligogalacturonic acid and chitosan reduce stomatal aperture by inducing the evolution of reactive oxygen species from guard cells of tomato and *Commelina communis*. *Plant Physiol* 121: 147–152, 1999.
35. Levine A, Tenhaken R, Dixon R, and Lamb C. H_2O_2 from the oxidative burst orchestrates the plant hypersensitive disease resistance response. *Cell* 79: 583–593, 1994.
36. Malan C, Gregling MM, and Gressel J. Correlation between CuZn superoxide dismutase and glutathione reductase and environmental and xenobiotic stress tolerance in maize inbreds. *Plant Sci* 69: 157–166, 1990.
37. Maxwell DP, Nickels R, and McIntosh L. Evidence of mitochondrial involvement in the transduction of signals required for the induction of genes associated with pathogen attack and senescence. *Plant J* 29: 269–279, 2002.
38. Maxwell DP, Wang Y, and McIntosh L. The alternative oxidase lowers mitochondrial reactive oxygen production in plant cells. *Proc Natl Acad Sci USA* 96: 8271–8276, 1999.
39. McAinsh MR, Clayton H, Mansfield TA, and Hetherington AM. Changes in stomatal behavior and guard cell cytosolic free calcium in response to oxidative stress. *Plant Physiol* 111: 1031–1042, 1996.
40. Mitsuhara I, Malik KA, Miura M, and Ohashi Y. Animal cell-death suppressors Bcl-$x_L$and Ced-9 inhibit cell death in tobacco plants. *Curr Biol* 9: 775–778, 1999.
41. Mittler R, Herr EH, Orvar BL, van Camp W, Willekens H, Inze D, and Ellis BE. Transgenic tobacco plants with reduced capability to detoxify reactive oxygen intermediates are hyperresponsive to pathogen infection. *Proc Natl Acad Sci USA* 96: 14165–14170, 1999.
42. Moon H, Lee B, Choi G, Shin D, Prasad DT, Lee O, Kwak SS, Kim DH, Nam J, Bahk J, Hong JC, Lee SY, Cho MJ, Lim CO, and Yun DJ. NDP kinase 2 interacts with two oxidative stress-activated MAPKs to regulate cellular redox state and enhances multiple stress tolerance in transgenic plants. *Proc Natl Acad Sci USA* 100: 358–363, 2003.
43. Mou Z, Fan W, and Dong X. Inducers of plant systemic acquired resistance regulate NPR1 function through redox changes. *Cell* 113: 935–944, 2003.
44. Mullineaux P and Karpinski S. Signal transduction in response to excess light: getting out of the chloroplast. *Curr Opin Plant Biol* 5: 43–48, 2002.
45. Murata Y, Pei Z–M, Mori IC, and Schroeder J. Abscisic acid activation of plasma membrane Ca^{2+} channels in guard cells requires cytosolic NAD(P)H and is differentially disrupted upstream and downstream of reactive oxygen species production in *abi1-1* and *abi2-1* protein phosphatase 2C mutants. *Plant Cell* 13: 2513–2523, 2001.

46. Mustilli A-C, Merlot S, Vavasseur A, Fenzi F, and Giraudat J. *Arabidopsis* OST1 protein kinase mediates the regulation of stomatal aperture by abscisic acid and acts upstream of reactive oxygen species production. *Plant Cell* 14: 3089–3099, 2002.
47. Nakagami H, Pitzschke A, and Hirt H. Emerging MAP kinase pathways in plant stress signalling. *Trends Plant Sci* 10: 339–346, 2005.
48. Orozco–Cardenas ML, Ryan C. Hydrogen peroxide is generated systemically in plant leaves by wounding and systemin via the octadecanoid pathway. *Proc Natl Acad Sci USA* 96: 6553–6557, 1999.
49. Ort RD and Baker NR. A photoprotective role for O_2 as an alternative electronic sink in photosynthesis? *Curr Opin Plant Biol* 5: 193–198, 2002.
50. Pei ZM, Murata Y, Benning G, Thomine S, Klusener B, Allen GJ, Grill E, aand Schroeder JI. Calcium channels activated by hydrogen peroxide mediate abscisic signaling in guard cells. *Nature* 406: 731–734, 2000.
51. Polidoros AN, Mylona PV, and Scandalios JG. Transgenic tobacco plants expressing the maize *Cat2* gene have altered catalase levels that affect plant–pathogen interactions and resistance to oxidative stress. *Transgenic Res* 10: 555–569, 2001.
52. Prasad TK, Anderson MD, Martin BA, and Stewart CR. Evidence for chilling-induced oxidative stress in maize seedlings and a regulatory role for hydrogen peroxide. *Plant Cell* 6: 65–74, 1994.
53. Purvis AC. Role of the alternative oxidase in limiting superoxide production by plant mitochondria. *Physiol Plant* 100:165–170, 1997.
54. Quinn J, Findlay VJ, Dawson K, Millar JB, Jones N, Morgan BA, and Toone WM. Distinct regulatory proteins control the graded transcriptional response to increasing H(2)O(2) levels in fission yeast *Schizosaccharomyces pombe*. *Mol Biol Cell* 13: 805–816, 2002.
55. Rebeiz CA, Montazer–Zouhoor A, Mayasich JM, Tripathy BC, Wu S–M, and Rebeiz C. Photodynamic herbicides. Recent developments and molecular basis of selectivity. *CRC Crit Rev Plant Sci* 6: 385–436, 1988.
56. Rentel MC, Lecourieux D, Ouaked F, Usher SL, Petersen L, Okamoto H, Knight H, Peck SC, Grierson CS, Hirt H, and Knight MR. OXI1 kinase is necessary for oxidative burst-mediated signalling in *Arabidopsis*. *Nature* 427: 858–861, 2004.
57. Rivas S, Rougon–Cardoso A, Smoker M, Schauser L, Yoshioka H, and Jones JD. CITRX thioredoxin interacts with the tomato Cf-9 resistance protein and negatively regulates defence. *EMBO J* 23: 2156–2165, 2004.
58. Saitoh M, Nishitoh H, Fujii M, Takeda K, Tobiume K, Sawada Y, Kawabata M, Miyazono K, and Ichijo H. Mammalian thioredoxin is a direct inhibitor of apoptosis signal-regulating kinase (ASK) 1. *EMBO J* 17: 2596–2606, 1998.
59. Samaj J, Ovecka M, Hlavacka A, Lecourieux F, Meskiene I, Lichtscheidl I, Lenart P, Salaj J, Volkmann D, Bogre L, Baluska F, and Hirt H. Involvement of the mitogen-activated protein kinase SIMK in regulation of root hair tip growth. *EMBO J* 21: 3296–3306, 2002.
60. Singh KK. The *Saccharomyces cerevisiae* SLN1P-SSK1P two-component system mediates response to oxidative stress and in an oxidant-specific fashion. *Free Rad Biol Med* 29: 1043–1050, 2000.
61. Solomon M, Belenghi B, Delledonne M, Menachem E, and Levine A. The involvement of cysteine proteases and protease inhibitor genes in the regulation of programmed cell death in plants. *Plant Cell* 11: 431–443, 1999.
62. Tiwari BS, Belenghi B, and Levine A. Oxidative stress increased respiration and generation of reactive oxygen species, resulting in ATP depletion, opening of mitochondrial permeability transition, and programmed cell death. *Plant Physiol* 128: 1271–1281, 2002.
63. Torres MA, Dangl JL, and Jones JDG. *Arabidopsis* gp91^{phox} homologues *AtrbohD* and *AtrbohF* are required for accumulation of reactive oxygen intermediates in the plant defense response. *Proc Natl Acad Sci USA* 99: 517–522, 2002.
64. Tsugane K, Kobayashi K, Niwa Y, Ohba Y, Wada K, and Kobayashi H. A recessive *Arabidopsis* mutant that grows enhanced active oxygen detoxification. *Plant Cell* 11: 1195–1206, 1999.
65. Vandenabeele S, Vanderauwera S, Vuylsteke M, Rombauts S, Langebartels C, Seidlitz HK, Zabeau M, Van Montagu M, Inze D, and Van Breusegem F. Catalase deficiency drastically affects gene expression induced by high light in *Arabidopsis* thaliana. *Plant J* 39: 45–58, 2004.
66. van Montfort RL, Congreve M, Tisi D, Carr R, and Jhoti H. Oxidation state of the active-site cysteine in protein tyrosine phosphatase 1B. *Nature* 423: 773–777, 2003.
67. Wagner AM. A role for active oxygen species as second messengers in the induction of alternative oxidase gene expression in *Petunia hybrida* cells. *FEBS Lett* 368: 339–342, 1995.
68. Whistler CA, Corbell NA, Sarniguet A, Ream W, and Loper JE. The two-component regulators GacS and GacA influence accumulation of the stationary-phase sigma factor sigmaS and the stress response in *Pseudomonas fluorescens* Pf-5. *J Bacteriol* 180: 6635–6641, 1998.
69. Gu Y, Vernoud V, Fu Y, and Yang Z. ROP GTPase regulation of pollen tube growth through the dynamics of tip-localized F-actin. *J Exp Bot* 54 :93–101, 2003.
70. Zhang X, Zhang L, Dong F, Gao J, Galbraith DW, and Song C-P. Hydrogen peroxide is involved in abscisic acid-induced stomatal closure in *Vicia faba*. *Plant Physiol* 126: 1438–1448, 2001.
71. Zheng M, Aslund F, and Storz G. Activation of the OxyR transcription factor by reversible disulfide bond formation. *Science* 279: 1718–1721, 1998.

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Date of first submission to ARS Central, March 24, 2006; date of acceptance, April 6, 2006.

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1. Hua-Sen Wang, Zhu-Jun Zhu, Zhen Feng, Shi-Gang Zhang, Chao Yu. 2012. Antisense-mediated depletion of GMPase gene expression in tobacco decreases plant tolerance to temperature stresses and alters plant development. *Molecular Biology Reports* . [CrossRef]
2. Francisco Javier Cejudo, Julia Ferrández, Beatriz Cano, Leonor Puerto-Galán, Manuel Guinea. 2012. The function of the NADPH thioredoxin reductase C-2-Cys peroxiredoxin system in plastid redox regulation and signalling. *FEBS Letters* **586**:18, 2974-2980. [CrossRef]
3. Dagan Sade, Assaf Eybishtz, Rena Gorovits, Iris Sobol, Henryk Czosnek. 2012. A developmentally regulated lipocalin-like gene is overexpressed in Tomato yellow leaf curl virus-resistant tomato plants upon virus inoculation, and its silencing abolishes resistance. *Plant Molecular Biology* . [CrossRef]
4. Manoj Kumar Sekhwal, Ajit Kumar Swami, Renu Sarin, Vinay Sharma. 2012. Identification of salt treated proteins in sorghum using gene ontology linkage. *Physiology and Molecular Biology of Plants* **18**:3, 209-216. [CrossRef]
5. Deepak Kumar, Preeti Singh, Mohd Aslam Yusuf, Chandrama Prakash Upadhyaya, Suchandra Deb Roy, Thomas Hohn, Neera Bhalla Sarin. 2012. The Xerophyta viscosa Aldose Reductase (ALDRXV4) Confers Enhanced Drought and Salinity Tolerance to Transgenic Tobacco Plants by Scavenging Methylglyoxal and Reducing the Membrane Damage. *Molecular Biotechnology* . [CrossRef]
6. N. Garg, H. Kaur. 2012. Response of Antioxidant Enzymes, Phytochelatins and Glutathione Production Towards Cd and Zn Stresses in Cajanus cajan (L.) Millsp. Genotypes Colonized by Arbuscular Mycorrhizal Fungi. *Journal of Agronomy and Crop Science* n/a-n/a. [CrossRef]
7. Mengmeng Zhu, Shaojun Dai, Sixue Chen. 2012. The stomata frontline of plant interaction with the environment-perspectives from hormone regulation. *Frontiers in Biology* **7**:2, 96-112. [CrossRef]
8. Andreas M. Fischer. 2012. The Complex Regulation of Senescence. *Critical Reviews in Plant Sciences* **31**:2, 124-147. [CrossRef]
9. Yuxiang Qin, Mengcheng Wang, Yanchen Tian, Wenxing He, Lu Han, Guangmin Xia. 2012. Over-expression of TaMYB33 encoding a novel wheat MYB transcription factor increases salt and drought tolerance in Arabidopsis. *Molecular Biology Reports* . [CrossRef]
10. S. Besseau, J. Li, E. T. Palva. 2012. WRKY54 and WRKY70 co-operate as negative regulators of leaf senescence in Arabidopsis thaliana. *Journal of Experimental Botany* . [CrossRef]
11. D. I. Bashmakov, N. A. Pynenkova, K. A. Sazanova, A. S. Lukatkin. 2012. Effect of the synthetic growth regulator Cytodef and heavy metals on oxidative status in cucumber plants. *Russian Journal of Plant Physiology* **59**:1, 59-64. [CrossRef]
12. Mirko Zaffagnini, Mariette Bedhomme, Stéphane D. Lemaire, Paolo Trost. 2012. The emerging roles of protein glutathionylation in chloroplasts. *Plant Science* . [CrossRef]
13. Ruth C Martin, Kira Glover-Cutter, James C Baldwin, James E Dombrowski. 2012. Identification and characterization of a salt stress-inducible zinc finger protein from Festuca arundinacea. *BMC Research Notes* **5**:1, 66. [CrossRef]
14. François Anquez, Ikram El Yazidi-Belkoura, Stéphane Randoux, Pierre Suret, Emmanuel Courtade. 2011. Cancerous Cell Death from Sensitizer Free Photoactivation of Singlet Oxygen. *Photochemistry and Photobiology* no-no. [CrossRef]
15. Alberto Milli, Daniela Cecconi, Luisa Bortesi, Anna Persi, Sara Rinalducci, Anita Zamboni, Gianni Zoccatelli, Arianna Lovato, Lello Zolla, Annalisa Polverari. 2011. Proteomic analysis of the compatible interaction between Vitis vinifera and Plasmopara viticola. *Journal of Proteomics* . [CrossRef]
16. Prakasini Satapathy, V. Mohan Murali Achary, Brahma B. Panda. 2011. Aluminum-induced abiotic stress counteracts Fusarium infection in Cajanus cajan (L.) Millsp. *Journal of Plant Interactions* 1-8. [CrossRef]
17. Karim Benabdellah, Younes Abbas, Mohamed Abourouh, Ricardo Aroca, Rosario Azcón. 2011. Influence of two bacterial isolates from degraded and non-degraded soils and arbuscular mycorrhizae fungi isolated from semi-arid zone on the growth of Trifolium repens under drought conditions: Mechanisms related to bacterial effectiveness. *European Journal of Soil Biology* . [CrossRef]
18. Patricia Berjak, Sershen, Boby Varghese, N.W. Pammenter. 2011. Cathodic amelioration of the adverse effects of oxidative stress accompanying procedures necessary for cryopreservation of embryonic axes of recalcitrant-seeded species. *Seed Science Research* 1-17. [CrossRef]
19. Q. Zheng, D. J. Oldenburg, A. J. Bendich. 2011. Independent effects of leaf growth and light on the development of the plastid and its DNA content in Zea species. *Journal of Experimental Botany* **62**:8, 2715-2730. [CrossRef]

20. Neera Garg, Priyanka Singla. 2011. Arsenic toxicity in crop plants: physiological effects and tolerance mechanisms. *Environmental Chemistry Letters* . [CrossRef]
21. L. Contreras-Porcia, D. Thomas, V. Flores, J. A. Correa. 2011. Tolerance to oxidative stress induced by desiccation in Porphyra columbina (Bangiales, Rhodophyta). *Journal of Experimental Botany* **62**:6, 1815-1829. [CrossRef]
22. Neera Garg, Nalini Aggarwal. 2011. Effects of Interactions Between Cadmium and Lead on Growth, Nitrogen Fixation, Phytochelatin, and Glutathione Production in Mycorrhizal Cajanus cajan (L.) Millsp. *Journal of Plant Growth Regulation* . [CrossRef]
23. M. Yusuf, Q. Fariduddin, S. Hayat, A. Ahmad. 2011. Nickel: An Overview of Uptake, Essentiality and Toxicity in Plants. *Bulletin of Environmental Contamination and Toxicology* **86**:1, 1-17. [CrossRef]
24. Yushuang Guo, Changjun Huang, Yan Xie, Fengming Song, Xueping Zhou. 2010. A tomato glutaredoxin gene SlGRX1 regulates plant responses to oxidative, drought and salt stresses. *Planta* **232**:6, 1499-1509. [CrossRef]
25. Hironaka Tsukagoshi, Wolfgang Busch, Philip N. Benfey. 2010. Transcriptional Regulation of ROS Controls Transition from Proliferation to Differentiation in the Root. *Cell* **143**:4, 606-616. [CrossRef]
26. Damla BilginROS, Oxidative Stress and Engineering Resistance in Higher Plants 205-227. [CrossRef]
27. P. Pulido, M. C. Spinola, K. Kirchsteiger, M. Guinea, M. B. Pascual, M. Sahrawy, L. M. Sandalio, K.-J. Dietz, M. Gonzalez, F. J. Cejudo. 2010. Functional analysis of the pathways for 2-Cys peroxiredoxin reduction in Arabidopsis thaliana chloroplasts. *Journal of Experimental Botany* **61**:14, 4043-4054. [CrossRef]
28. Geert Potters, Nele Horemans, Marcel A.K. Jansen. 2010. The cellular redox state in plant stress biology – A charging concept. *Plant Physiology and Biochemistry* **48**:5, 292-300. [CrossRef]
29. Helen H. Tai, Kevin E. Percy, David F. Karnosky. 2010. DNA damage in Populus tremuloides clones exposed to elevated O3. *Environmental Pollution* **158**:4, 969-976. [CrossRef]
30. Y. Wang, W. H. Wu. 2010. Plant Sensing and Signaling in Response to K+-Deficiency. *Molecular Plant* **3**:2, 280-287. [CrossRef]
31. C. Bianco, R. Defez. 2009. Medicago truncatula improves salt tolerance when nodulated by an indole-3-acetic acid-overproducing Sinorhizobium meliloti strain. *Journal of Experimental Botany* **60**:11, 3097-3107. [CrossRef]
32. Georg Zeller, Stefan R. Henz, Christian K. Widmer, Timo Sachsenberg, Gunnar Rätsch, Detlef Weigel, Sascha Laubinger. 2009. Stress-induced changes in the Arabidopsis thaliana transcriptome analyzed using whole-genome tiling arrays. *The Plant Journal* **58**:6, 1068-1082. [CrossRef]
33. J. M. Perez-Ruiz, M. Gonzalez, M. C. Spinola, L. M. Sandalio, F. J. Cejudo. 2009. The Quaternary Structure of NADPH Thioredoxin Reductase C Is Redox-Sensitive. *Molecular Plant* **2**:3, 457-467. [CrossRef]
34. C. Chang, I. Damiani, A. Puppo, P. Frendo. 2009. Redox Changes during the Legume-Rhizobium Symbiosis. *Molecular Plant* **2**:3, 370-377. [CrossRef]
35. Juan Manuel Pérez-Ruiz, Francisco Javier Cejudo. 2009. A proposed reaction mechanism for rice NADPH thioredoxin reductase C, an enzyme with protein disulfide reductase activity. *FEBS Letters* **583**:9, 1399-1402. [CrossRef]
36. A. Gutsche, T. Heng-Moss, G. Sarath, P. Twigg, Y. Xia, G. Lu, D. Mornhinweg. 2009. Gene expression profiling of tolerant barley in response to Diuraphis noxia (Hemiptera: Aphididae) feeding. *Bulletin of Entomological Research* **99**:02, 163. [CrossRef]
37. Gabriella Szalai, Tibor Kell#s, Gábor Galiba, Gábor Kocsy. 2009. Glutathione as an Antioxidant and Regulatory Molecule in Plants Under Abiotic Stress Conditions. *Journal of Plant Growth Regulation* **28**:1, 66-80. [CrossRef]
38. P.L. Gratão, C.C. Monteiro, A.M. Antunes, L.E.P. Peres, R.A. Azevedo. 2008. Acquired tolerance of tomato (Lycopersicon esculentum cv. Micro-Tom) plants to cadmium-induced stress. *Annals of Applied Biology* **153**:3, 321-333. [CrossRef]
39. Sujon Sarowar, Jae -Yong Lee, Eu -Ree Ahn, Hyun -Sook Pai. 2008. A role of hexokinases in plant resistance to oxidative stress and pathogen infection. *Journal of Plant Biology* **51**:5, 341-346. [CrossRef]
40. H.-C. Jing, R. Hebeler, S. Oeljeklaus, B. Sitek, K. Stühler, H. E. Meyer, M. J. G. Sturre, J. Hille, B. Warscheid, P. P. Dijkwel. 2008. Early leaf senescence is associated with an altered cellular redox balance in Arabidopsis cpr5 / old1 mutants. *Plant Biology* **10**, 85-98. [CrossRef]
41. K. Kirchsteiger, P. Pulido, M. Gonzalez, F. J. Cejudo. 2008. NADPH Thioredoxin Reductase C Controls the Redox Status of Chloroplast 2-Cys Peroxiredoxins in Arabidopsis thaliana. *Molecular Plant* **2**:2, 298-307. [CrossRef]

42. Priscila L. Gratão, Georgia B. Pompeu, Flávia R. Capaldi, Victor A. Vitorello, Peter J. Lea, Ricardo A. Azevedo. 2008. Antioxidant response of Nicotiana tabacum cv. Bright Yellow 2 cells to cadmium and nickel stress. *Plant Cell, Tissue and Organ Culture* **94**:1, 73-83. [CrossRef]

43. C. A. Cullis, B. J. Vorster, C. Van Der Vyver, K. J. Kunert. 2008. Transfer of genetic material between the chloroplast and nucleus: how is it related to stress in plants?. *Annals of Botany* **103**:4, 625-633. [CrossRef]

44. Vadim Demidchik, Frans J. M. Maathuis. 2007. Physiological roles of nonselective cation channels in plants: from salt stress to signalling and development. *New Phytologist* **175**:3, 387-404. [CrossRef]

45. TRACEY ANN CUIN, SERGEY SHABALA. 2007. Compatible solutes reduce ROS-induced potassium efflux in Arabidopsis roots. *Plant, Cell & Environment* **30**:7, 875-885. [CrossRef]

46. James A. McCubrey , Richard A. Franklin . 2006. Reactive Oxygen Intermediates and Signaling Through Kinase Pathways. *Antioxidants & Redox Signaling* **8**:9-10, 1745-1748. [Citation] [Full Text PDF] [Full Text PDF with Links]