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Forum Editorial

Lessons from Redox Signaling in Plants

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ALL LIVING ORGANISMS are oxidation–reduction (redox) systems. They use anabolic, reductive processes to store energy and catabolic, oxidative processes to release it. It is plants that set this global wheel in motion. By harnessing light energy to drive biochemistry, photosynthetic organisms have perfected the art of redox control. Indeed, it is now widely accepted that redox signals are key regulators of plant metabolism, morphology, and development. These signals exert control on nearly every aspect of plant biology from chemistry to development, growth, and eventual death. It is therefore timely and appropriate that the current state of scientific advancement is assessed and reviewed in this volume. We suggest that "redox signaling" was the first type of sensory regulation that evolved in nature, because it prevented uncontrolled "boom and bust" scenarios in energy availability, utilization, and exchange. There followed further pathways of redox control through regulation of gene expression at many levels. It is likely that intermediates and other systems of signal transduction arose from this central core. In eukaryotic plants and algae, the green, cytoplasmic organelle of photosynthesis is the chloroplast. This forum issue offers an opportunity to dissect the complex systems of plant redox control, particularly the systems in the chloroplast that sense redox changes and control redox homeostasis.

Plants are autotrophic organisms powered by photosynthesis. Photosynthesis is light-driven redox chemistry, and it should be no surprise that redox signals from the light reactions of photosynthesis initiate profound changes in gene function (7). These changes encompass posttranslational modification of proteins by phosphorylation (8), redox modulation of assimilatory reactions (24, 25), and control of gene transcription and translation (10, 12, 17, 23). Interestingly, photosynthetic control of gene expression can now be described for the genes located in chloroplasts themselves, at both transcriptional (17) and posttranscriptional (10) levels. Redox signals also leave the chloroplast to provide a decisive input into transcriptional control in the cell nucleus (23).

The persistence of redox control of gene expression within chloroplasts (17, 22) is evidence that redox signaling is the primary function of the chloroplast's small, specialized, but vital cytoplasmic genome (2, 6). Mitochondria, like chloroplasts, originated as bacterial endosymbionts, and retain their own genomes. Redox signaling as the function of the mitochondrial genome (2) has wide implications that extend beyond plant biology into subjects such as aging and evolution of sex (3). The "free radical" and "mitochondrial" theories of aging (3) have consequences for the technology of somatic cloning, where human, reproductive clones should be expected to age prematurely for the same reason as "Dolly," the cloned sheep (5).

Around two billion years ago, molecular oxygen became intimately involved with the essential energy exchange reactions on which life is based, allowing use of the very high electrochemical potential (E_{m7} = +815 mV) of the O_2/H_2O redox couple. Cyanobacteria, the prokaryotic cousins of plant chloroplasts, created the Earth's oxygen-rich atmosphere through oxygenic photosynthesis (11). Oxygenic photosynthesis and aerobic respiration now deal with concerted, four-electron exchange between water and oxygen, without release of reactive, partially reduced intermediates. However, many processes in plants catalyze only partial reduction of oxygen, and so generate superoxide, hydrogen peroxide (H_2O_2), and hydroxyl radicals. Photosynthesis has a high capacity for the production of these active oxygen species (AOS) and also singlet oxygen, but the intracellular levels of these oxidants are tightly controlled by an antioxidant system, comprising a network of enzymatic and nonenzymatic components (13, 18). In most cases, such defense reactions are linked to the two major redox buffers of plant cells, ascorbate and glutathione pools (18). The essential photoelectrochemistry of photosynthesis first evolved in an anaerobic world (11). Photosynthesis produces superoxide as result of direct electron transfer to oxygen (4, 13, 14). However, quantitatively it is the source of even greater amounts of H_2O_2 by virtue of the glycolate oxidase reaction of photorespiration (20). It is important to note that photosynthesis generates reductants with potentials far lower than even the most reducing part of the mitochondrial respiratory chain, the donor couple NADH/NAD^+ (E_{m7} = −324 mV). Electron flow from chloroplast photosystem I (primary acceptor, $E_{m7} < -900$ mV) to oxygen is thermodynamically favorable for reduction of oxygen to superoxide

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(E_{m7} = −330 mV). Bipyridyl mediators such as paraquat (methyl viologen) have long been known to remove almost all kinetic barriers to this lethal electron flux (1). On a scale of oxidative stress, such herbicidal action on plants in normal sunlight surely ranks second only to incineration.

Redox signals are central to defense responses and cross-tolerance phenomena, enabling a general acclimation of plants to stressful conditions (21). H_2O_2 has long been recognized as a signal-transducing molecule in the activation of defense responses in plants. It mediates intra- and extracellular communication during plant reactions to pathogens, and several studies have suggested a role in systemic acquired resistance. It is involved in the adaptation of leaves to high light. H_2O_2 has a strong regulatory influence on fluxes through Ca^{2+} channels and on Ca^{2+} concentrations indifferent cellular compartments.The role of mitogen-activated protein kinases in oxidative stress signaling has recently been demonstrated in the model plant *Arabidopsis thaliana* (15).

H_2O_2 is a secondary messenger in many hormone-mediated events, such as stomatal movement, cell growth, and tropic responses (21). Hence, redox signals interact closely with other signaling systems. They also influence and modify the action of secondary messengers, such as nitric oxide. It also now appears that the simultaneous generation of nitric oxide and AOS is required to trigger cell death cascades in response to pathogen attack (12).

Plants are much more tolerant of H_2O_2 than animals, and their antioxidant systems appear to have been designed to ensure control of the cellular redox state rather than to facilitate the complete elimination of H_2O_2 (18–20, 26). Ascorbate, glutathione, and associated antioxidant enzymes determine the lifetime of H_2O_2 *in planta* (15). Plant cells are strongly redox-buffered and contain very large quantities of ascorbate (10–100 m*M*) and glutathione (1–10 m*M*) (18). Most of their intracellular compartments hence have the capacity to deal with even very high fluxes of H_2O_2 production (18). Rapid compartment-specific differences in redox state (and hence signaling) that influence the operation of many fundamental processes in plants can be achieved by modifying AOS (particularly H_2O_2) production or by repressing or activating antioxidant defenses. Recent evidence suggests that glutathione and ascorbate are key components of redox signaling in plants (9, 15, 19, 21). Specific compartment-based signaling and regulation of gene expression can be achieved via differential compartment-based changes in either the absolute concentrations of ascorbate and glutathione or the ascorbate/dehydroascorbate and GSH/GSSG ratios, which are very high and stable in the absence of stress (9, 13, 19).

The original articles and authoritative reviews that constitute this forum issue provide an account of the ultimate origin of oxidative stress and an analysis of the steps taken to deal with it at source. Plants have created the aerobic world in which we live. It is therefore no surprise to find that plants have already tackled the key problems of living with oxygen, and found solutions in antioxidants and in redox signaling.

ABBREVIATIONS

AOS, active oxygen species; H_2O_2, hydrogen peroxide; redox, oxidation–reduction.

REFERENCES

1. Allen JF. Superoxide and photosynthetic reduction of oxygen. In: *Superoxide and Superoxide Dismutases*, edited by Michelson AM, McCord JM, and Fridovich I. London and New York: Academic Press, 1977, pp. 417–436.
2. Allen JF. Control of gene expression by redox potential and the requirement for chloroplast and mitochondrial genomes. *J Theor Biol* 165: 609–631, 1993.
3. Allen JF. Separate sexes and the mitochondrial theory of ageing. *J Theor Biol* 180: 135–140, 1996.
4. Allen JF. Superoxide as an obligatory, catalytic intermediate in photosynthetic reduction of oxygen by adrenaline and dopamine. *Antioxid Redox Signal* 5: 7–14, 2003.
5. Allen JF and Allen CA. A mitochondrial model for premature ageing of somatically cloned mammals. *IUBMB Life* 48: 369–372, 1999.
6. Allen JF and Raven JA. Free-radical-induced mutation versus redox regulation: costs and benefits of genes in organelles. *J Mol Evol* 42: 482–492, 1996.
7. Allen JF, Alexciev K, and Håkansson G. Photosynthesis. Regulation by redox signalling. *Curr Biol* 5: 869–872, 1995.
8. Aro E-M and Ohad I. Redox regulation of thylakoid protein phosphorylation. *Antioxid Redox Signal* 5: 55–67, 2003.
9. Baier M, Noctor G, Foyer CH, and Dietz, KJ. Antisense suppression of 2-cys peroxiredoxin in *Arabidopsis thaliana* specifically enhances the activities and expression of enzymes associated with ascorbate metabolism, but not glutathione metabolism. *Plant Physiol* 124: 823–832, 2000.
10. Barnes D and Mayfield SP. Redox control of posttranscriptional processes in the chloroplast. *Antioxid Redox Signal* 5: 89–93, 2003.
11. Blankenship RE. *Molecular Mechanisms of Photosynthesis.* London: Blackwell Science, 321 pp. 2002.
12. Delledonne M, Polverari A, and Murgia I. The functions of nitric oxide-mediated signaling and changes in gene expression during the hypersensitive response. *Antioxid Redox Signal* 5: 33–41, 2003.
13. Foyer CH and Noctor G. Oxygen processing in photosynthesis: regulation and signalling. *New Phytol* 146: 359–388, 2000.
14. Ivanov B and Khorobrykh S. Participation of photosynthetic electron transport in production and scavenging of reactive oxygen species. *Antioxid Redox Signal* 5: 43–53, 2003.
15. Kiddle G, Pastori GM, Bernard S, Pignocchi C, Antoniw J, Verrier PJ, and Foyer CH. Effects of leaf ascorbate signaling on defense and photosynthesis gene expression in *Arabidopsis thaliana. Antioxid Redox Signal* 5: 23–32, 2003.
16. 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 U S A* 97: 2940–2945, 2000.
17. Link G. Redox regulation of chloroplast transcription. *Antioxid Redox Signal* 5: 79–87, 2003.
18. Noctor G and Foyer CH. Ascorbate and glutathione: keeping active oxygen under control. *Annu Rev Plant Physiol Plant Mol Biol* 49: 249–279, 1998.

19. Noctor G, Veljovic-Jovanovic S, and Foyer CH. Peroxide processing in photosynthesis: antioxidant coupling and redox signalling. *Philos Trans Soc Lond Biol* 355: 1465–1475, 2000.
20. Noctor G, Veljovic-Jovanovic S, Driscoll S, Novitskaya L, and Foyer CH. Drought and oxidative load in wheat leaves: a predominant role for photorespiration? *Ann Bot (Lond)* 89: 841–850, 2002.
21. Pastori GM and Foyer CH. Common components, networks and pathways of cross tolerance to stress: the central role of "Redox" and ABA-mediated controls. *Plant Physiol* 129: 460–468, 2002.
22. Pfannschmidt T, Nilsson A, and Allen JF. Photosynthetic control of chloroplast gene expression. *Nature* 397: 625–628, 1999.
23. Pfannschmidt T, Schütze K, Fey V, Sherameti I, and Oelmüller R. Chloroplast redox control of nuclear gene expression—a new class of plastid signals in interorganellar communication. *Antioxid Redox Signal* 5: 95–101, 2003.
24. Rouhier N, Vlamis-Gardikas A, Lillig CH, Berndt C, Schwenn J-D, Holmgren A, and Jacquot J-P. Characterization of the redox properties of poplar glutaredoxin. *Antioxid Redox Signal* 5: 15–22, 2003.
25. Schürmann P. Redox signaling in the chloroplast: the ferredoxin/thioredoxin system. *Antioxid Redox Signal* 5: 69–78, 2003.
26. Veljovic-Jovanovic S, Noctor G, and Foyer CH. Are leaf hydrogen peroxide concentrations commonly overestimated? Interference by tissue ascorbate. *Plant Physiol Biochem* 40: 501–507.

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