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Forum Review

Chloroplast Redox Control of Nuclear Gene Expression—A New Class of Plastid Signals in Interorganellar Communication

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ABSTRACT

Chloroplasts are genetically semiautonomous organelles that contain their own subset of 100–120 genes coding for chloroplast proteins, tRNAs, and rRNAs. However, the great majority of the chloroplast proteins are encoded in the nucleus and must be imported into the organelle after their translation in the cytosol. This arrangement requires a high degree of coordination between the gene expression machineries in chloroplasts and nucleus, which is achieved by a permanent exchange of information between both compartments. The existence of such coordinating signals has long been known; however, the underlying molecular mechanisms and signaling routes are not understood. The present data indicate that the expression of nuclear-encoded chloroplast proteins is coupled to the functional state of the chloroplasts. Photosynthesis, which is the major function of chloroplasts, plays a crucial role in this context. Changes in the reduction/oxidation (redox) state of components of the photosynthetic machinery act as signals, which regulate the expression of chloroplast proteins in both chloroplasts and nucleus and help to coordinate the expression both in compartments. Recent advances in understanding chloroplast redox regulation of nuclear gene expression are summarized, and the importance for intracellular signaling is discussed. *Antioxid. Redox Signal.* 5, 95–101.

INTRODUCTION

CHLOROPLASTS, THE TYPICAL ORGANELLES of higher plants and green algae, provide all structural and functional properties necessary for photosynthesis, a process that converts light energy into chemical energy. Changes in environmental parameters such as light intensity, light quality, or temperature affect the photosynthetic electron transport and subsequently change the efficiency of light energy fixation. Photosynthetic organisms therefore developed several mechanisms to acclimate to a wide range of environmental conditions and to maintain the photosynthetic efficiency as high as possible (2, 4, 20).

The light-driven chemistry of photosynthesis involves a series of redox steps in structural components or functionally coupled pools of redox-active compounds, such as thioredoxin or glutathione. An increasing number of reports show that environmentally induced changes in the redox state of these electron transport components act as signals that regulate the expression of proteins of the photosynthetic machinery (for reviews, see 3, 9, 26, 42). This feedback mechanism couples the present function of photosynthesis to the expression of its structural constituents and thus acclimates photosynthesis to changing environmental cues. As the photosynthetic machinery consists of both plastid and nuclear encoded products, this requires a high coordination in the expression of these genes. Chloroplast redox signals play an important role in this intracellular coordination; their impact on different levels of plastid gene expression is also reviewed in this issue. This review focuses on the role of chloroplast redox signals in nuclear gene expression. It summarizes the present knowledge about such pathways and describes the interaction with other signaling cascades, as well as specific problems in the transduction of these signals.

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NUCLEAR GENE EXPRESSION—A TARGET FOR CHLOROPLAST REDOX CONTROL

Light regulation of nuclear gene expression is an extensively investigated field of research in plant biology, and the impact of photoreceptor-controlled signaling cascades is well documented (for reviews, see in 1, 5, 14, 16, 25, 33, 47). Chloroplast redox control, however, is a new concept of how light can influence the expression of nuclear genes. By this way, photosynthesis contributes important information to the regulation of nuclear gene expression that is not sensed by cytosolic photoreceptors. Through this, the chloroplast serves as a sensor for environmental changes and can induce physiological acclimation reactions (Fig. 1). The activation of transcription of nuclear encoded photosynthesis genes by illumination is a long-known phenomenon, but the first clear evidence that such regulation can be coupled to photosynthetic electron transport came from studies with the unicellular algae *Dunaliella tertiolecta* and *Dunaliella salina* (17, 28).

Escoubas *et al.* (17) demonstrated that transcription of the *Lhcb* genes [encoding chlorophyll-binding proteins of light-harvesting complex II (LHCII)] is stimulated when high light-acclimated cells are exposed to low-light intensities. By performing the same experiments in the presence of the site-specific electron transport inhibitors 3-(3′,4′-dichlorophenyl)-1,1′-dimethylurea (DCMU) and 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone (DBMIB) (51), they could show that this increase is coupled to photosynthetic electron transport. In addition, they identified the redox state of the plastoquinone pool (PQ) as the controlling parameter: an oxidized PQ pool (generated by low light or DCMU treatment) activates *Lhcb* transcription, whereas a reduced one (generated by high light or DBMIB treatment) represses it.

In an independent study, Maxwell *et al.* (28) came to similar conclusions. They analyzed *Lhcb* transcription and LHCII apoprotein content in response to varying light intensities under controlled temperature environments. This approach is based on the observation that light-intensity effects also depend on ambient temperature. For instance, the same light intensity that represents low-light condition at a high temperature can also represent high-light condition under low temperature. The enzymatic steps in the photosynthetic dark reaction become rate-limiting under low temperature, thus generating a higher excitation pressure. In their experiments, Maxwell *et al.* (28) found that relaxation of high excitation pressure both thermodynamically (shifting cells from 13°C to 30°C under constant light) and photodynamically (shifting cells from light to dark growth regimes under constant temperature) results in an increase of *Lhcb* transcription and LHCII protein accumulation. They concluded that the redox poise of intersystem electron transport represents a common sensing/signaling pathway for *Lhcb* transcription under various stress conditions.

A recent study using *Lemna perpusilla* as model organism demonstrated that *Lhcb* transcript abundance and LHCII protein content are increased under low-light intensities also in a higher plant (52). Using a cytochrome $b_6 f$ complex ($cyt\ b_6 f$) deficient mutant, Yang *et al.* (52) showed that this regulation is coupled to photosynthetic electron transport. The mutant failed to respond to changing light intensities and exhibited always a high light-acclimated phenotype. Further investigations with DCMU in combination with results from the photosynthetic mutant led to the conclusion that the redox state of the PQ pool represents the controlling redox parameter in this regulation, which is fully consistent with the observations described above.

High-light treatment was also used to demonstrate the existence of another type of chloroplast redox signals in the higher plant *Arabidopsis thaliana* (22). An increase of incident light intensity from 200 to 2,000 μE increased the transcript level of two genes for cytosolic ascorbate peroxidases (*Apx1*, *Apx2*) within 15 min. DCMU and DBMIB treatments

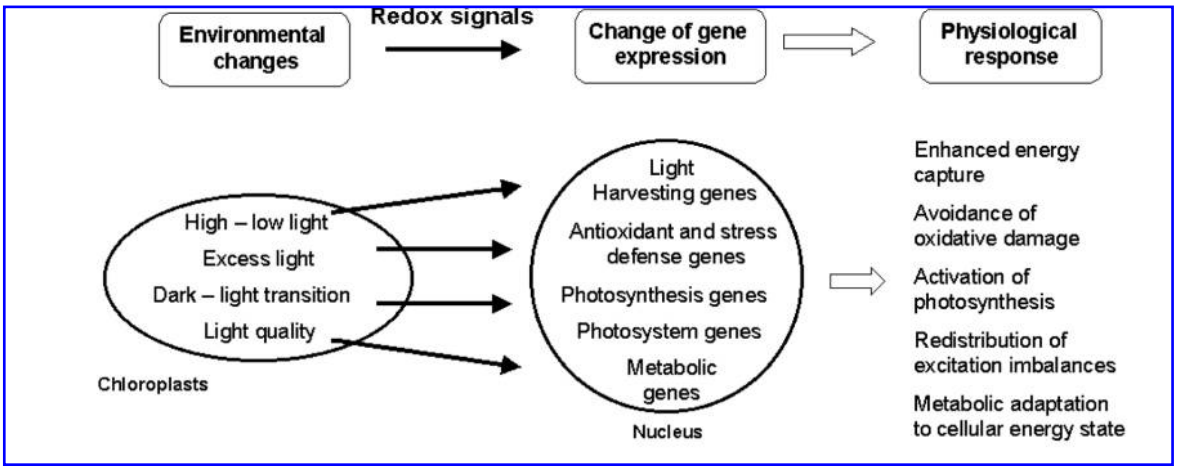

FIG. 1. Physiological role of chloroplast redox signals. Fluctuating environmental conditions, especially changes in illumination, result in changes of photosynthetic efficiency. Concomitantly, the redox state of chloroplast components is affected. Such redox changes represent a sensor for the environmental fluctuation and serve as signals that influence the expression of nuclear genes whose products, in turn, are involved in an appropriate physiological response to counteract the limitation of photosynthesis by the environment. A general scheme for this is given at the top of the figure; several concrete examples are summarized below. For more details, see text.

of leaf discs indicated that this signaling pathway originates from the PQ pool; however addition of reduced glutathione abolished this signal, suggesting that the redox state of the glutathione pool may play an interacting regulatory role in this context. Both ascorbate peroxidases and glutathione are scavengers of reactive oxygen species (ROS) that are generated preferentially under stress conditions, such as high light. Further experiments with transgenic *Arabidopsis* plants containing *Apx2* promoter:luciferase fusions demonstrated that the high light-induced signal can be transported from a high light-treated tissue to an untreated tissue plant probably via high light-generated hydrogen peroxide (H_2O_2) (23). This phenomenon, called "systemic acquired acclimation," suggests that redox signals in higher plants not only are transduced from chloroplasts to the nucleus of the same cell, but also function in a tissue overriding manner. Further investigations demonstrated that also transcripts for a peroxisomal catalase (*Cat2*), the chloroplast glutathione peroxidase (*Gpx2*), and glutathione *S*-transferase (*Gst*), as well as the pathogenesis-related type 2 protein (*Pr2*), were induced by light stress, but interestingly only in systemic, nonilluminated leaves (31).

In transgenic tobacco plants harboring a pea ferredoxin transcribed region (*Fed1*) under the control of the cauliflower 35S promoter, the abundance of this *Fed1* message was four- to fivefold increased in reilluminated plants compared with dark-adapted plants (38). The light-induced increase was shown to be coupled to photosynthetic electron transport because DCMU treatment could abolish the light effect. This response is gene-specific because *Lhcb* transcript accumulation was not affected by the drug. In addition, it could be demonstrated that light-dependent polyribosome loading of *Fed1* mRNA, an important prerequisite for efficient translation, was also diminished by the DCMU treatment. The same was observed for the *Lhcb* message. Further experiments indicated that interruption of photosynthetic electron transport by either dark or DCMU treatment led to a rapid destabilization of the *Fed1* mRNA (39). Taken together, these observations demonstrate that chloroplast redox signals affect also post-transcriptional events besides transcription.

In mustard photosystem stoichiometry adjustment in response to varying light-quality conditions was shown to be regulated by PQ redox control of transcription of the chloroplast genes *psbA* and *psaAB* [encoding reaction center proteins of photosystem II (PSII) and photosystem I (PSI)] (41). In a recent study using transgenic tobacco plants harboring promoters of nuclear-encoded PSI genes *petE*, *petH*, *psaD*, and *psaF* [encoding plastocyanin (PC), the ferredoxin:NADP:oxidoreductase (FNR), and the PSI subunits *PsaD* and *PsaF*] fused to the *uidA* gene, it could be demonstrated that the same light-quality variations also affect nuclear gene expression (43). In combination with DCMU and DBMIB treatments, these experiments showed that the PC promoter activity is controlled by the redox state of the PQ pool, whereas the *psaD* and *psaF* promoters are under the control of a yet unidentified downstream component of the photosynthetic electron transport chain. All three promoters have in common that they are activated by the reduction of photosynthetic electron transport components. The FNR promoter revealed no regulation under any condition. A further study using the same experimental approach showed that the *Arabidopsis* nitrate reductase (*Nia2*) promoter also responds to the light-induced redox signals. However, it is activated by a predominant oxidation of the electron transport components either by PSI light or by DCMU or DBMIB treatment (45). Beside transcription, the nitrate reductase enzyme activity was regulated in exactly the same way. The use of a *cyt* b_6 *f*-deficient *Lemna aequinoctialis* mutant (8) confirmed that the photosynthetic electron transport regulates nitrate reductase activity. The mutant showed constitutive high activity under all conditions similar to PSI light-acclimated wild type. These results show that redox signals that are shown to act within the organelle (41) can also extend to the nucleus and that the same signal can have different gene-specific effects.

These examples show that chloroplast redox signals affect nuclear gene expression under various physiological conditions and at different levels of expression. The increasing number of reports describing such regulation pathways suggests that a complex network of redox signals rather than a single signaling pathway couples chloroplast function to the nuclear gene expression machinery.

INTERACTION OF REDOX SIGNALS WITH OTHER SIGNALING PATHWAYS

All chloroplast redox signals described in the previous section are connected to illumination and photosynthesis. As photosynthesis is the central point of plant energy metabolism that is connected with almost all processes in the cell, it should be expected that there exist interactions with other regulating signals (Fig. 2). An example for this can be found in a recent study with an *Arabidopsis* cell culture (36). It is well known that high external sugar concentrations repress nuclear photosystem gene expression (46). Oswald *et al.* (36) found that the increase in transcription of *Lhcb* and *RbcS* genes, which is normally observed when the external sugar is removed, can be blocked by addition of DCMU to the culture medium. The same experiments performed with transgenic *Arabidopsis* lines carrying *Lhcb2* and PC promoters fused to

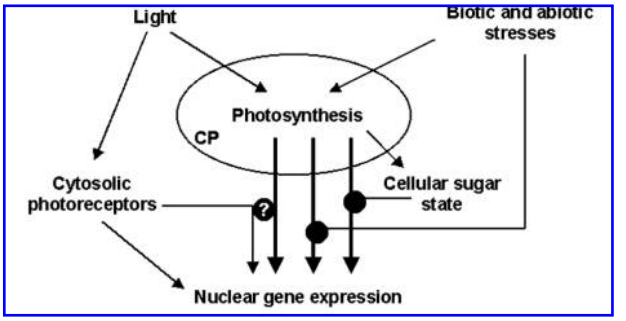

FIG. 2. Known and putative interactions of plastid redox signals with other signaling pathways during control of nuclear gene expression. Thick arrows indicate redox signals; thin arrows are influences of various parameters on cellular components or processes. Black circles indicate interactions of signaling pathways. CP, chloroplast. For more details, see text.

the luciferase reporter gene confirmed these results *in planta*. Further investigations using the sugar-insensitive *Arabidopsis* mutant *sun6* also carrying the PC-LUC construct revealed that the PC promoter activity is increased by sugars and that DCMU treatment has no effect on this response. Oswald *et al.* (36) postulated that under weak light a redox signal from photosynthetic electron transport activates the derepression of nuclear photosynthetic genes when the strong antagonistic suppression by external sugars is removed. This signal is not active once derepression has occurred. This suggests that photosynthetic gene expression is balanced by cellular sugar status and photosynthetic activity to fulfil the demands of energy metabolism. If this model also accounts for high-light conditions has to be tested.

As described above, excess-light-induced ROS formation activates nuclear antioxidant genes (22); however, the generation of ROS can be also observed under other abiotic or biotic stresses, such as chilling, wounding, or pathogen attack. It has been shown that transcripts for several components of the cytosolic ROS-scavenging system are induced by such stresses (for review, see 31). ROS formation also affects the degree of reduction of major antioxidant pools, such as glutathione, which is regarded as a key component of antioxidant defense in plants (18). In general, there is increasing evidence that ROS and the redox states of antioxidant pools regulate the expression of nuclear antioxidant genes. In this instance, redox signals via ROS serve as integrating components of several signaling pathways that help the plant to adapt to various types of stresses. As the cell is not able to identify the respective type of stress only by recognizing the ROS levels in the stroma of the plastid or in the cytoplasm, further signals are required. For instance, in the case of systemic acquired resistance, ROS provide the common signal for stress, but the response appears to be elicited only in co-action with nitric oxide (13). Therefore, ROS-mediated redox signals may interact with many other signals to define changes in environmental conditions. The importance for a sensitive sensing of cellular ROS levels has been demonstrated also in transgenic tobacco plants overexpressing a plastid γ-glutamylcysteine synthetase. Despite the potentially higher ROS scavenging capacity, these plants suffered from strong oxidative stress that is caused most probably by their inability to sense changes in ROS formation (12).

Redox signals induced by the photosynthetic process are strictly light-dependent. From the present data, it appears that different types of signals can be generated depending on the incident-light quantity, and a model has been proposed in which such different signals operate in a hierarchical order (42). It appears likely that under certain conditions, different redox-controlled signaling pathways may interact with each other.

Light-regulated nuclear gene expression is largely affected by the red and blue light-absorbing cytosolic photoreceptors (see Introduction). Furthermore, there exists increasing evidence that plastid signals interact with phytochrome signaling pathways that also influence expression of nuclear genes for plastid proteins (27). At present, it is unclear if chloroplast redox signals interfere or interact with cytosolic photoreceptor cascades. The availability of photoreceptor mutants provides a useful tool to solve this problem. First results show that phytochrome A- and phytochrome B-deficient *Arabidopsis* mutants respond in the same way to light quality-induced redox signals as the wild type, which indicates that the redox signaling cascade is functional in phytochrome A- and phytochrome B-deficient *Arabidopsis* mutants (Fey and Pfannschmidt, unpublished observations).

CHLOROPLAST REDOX SIGNALS AND THEIR ROLE AS "PLASTID FACTOR"

Early investigations in the 1980s especially with norflurazon, an inhibitor of phytoene desaturase, gave the first hints that chloroplast function or development influences nuclear gene expression. This led to the postulation of a so-called "plastid factor" that couples the functional state of the plastids to the expression of nuclear encoded plastid proteins (for reviews, see 34, 50). Norflurazon-treated plants exhibit a white phenotype that is caused by an arrest in chloroplast biogenesis through photobleaching because the inhibitor blocks chlorophyll biosynthesis. As a concomitant effect, an inhibition of the expression of some nuclear genes (*i.e., RbcS*, *Lhcb*) encoding chloroplast proteins was observed. The same results were obtained when chloroplast gene expression was blocked by inhibitors such as tagetitoxin, rifampicin, or cycloheximide (for review, see 19), suggesting the involvement of (a) chloroplast gene product(s) in this retrograde signaling. Further studies point to intermediates or enzymatic components of the chlorophyll biosynthesis pathway as putative plastid regulators of nuclear gene expression (for reviews, see 6, 21, 37, 44).

The molecular nature of the "plastid factor(s)," as well as the way this signal is transduced across the chloroplast envelope into the cytosol, is still unknown. Recent studies with *Arabidopsis cue1*, *gun5*, and *laf6* mutants, however, have shed more light on this complex signaling network. Their defects are located in the phosphoenolpyruvate/phosphate translocator (*cue1*), the *H* subunit of the magnesium chelatase (*gun5*), and a new ABC transporter protein (*laf6*). All three components are located in the chloroplast envelope, suggesting that the defect interrupts the transport of one or several plastid factor(s), and models of respective signaling pathways have been proposed (29, 30, 48). Earlier studies with mustard (35, 40) and a recent study on the pea *lip1* mutant (49), which shows photomorphogenic development in the dark, demonstrated that nuclear photosynthesis gene expression depends on plastid translation or transcription in a light-independent manner. This suggests that the plastid factor(s) is not necessarily coupled to light, thylakoid formation, or photosynthesis. At present, it is not clear how this light-independent signal relates to the signaling pathways identified in the *Arabidopsis* mutants; however, as redox signals from photosynthesis are strictly light-dependent, they represent a different class of plastid signals.

Mutants with defects in chloroplast-to-nucleus signaling often show strong developmental defects (19, 44). Especially in the beginning of cell or tissue differentiation, the developmental state of plastids may be a very important parameter for further development of its host cell. Early plastid signals

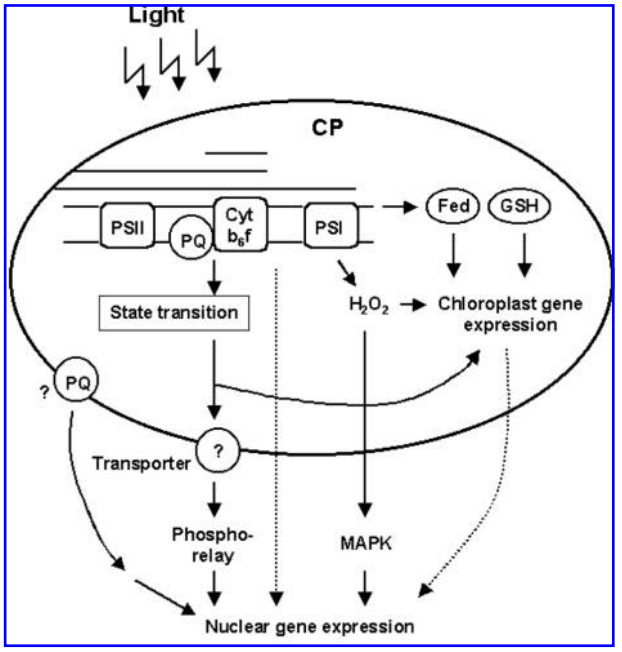

FIG. 3. Putative transduction mechanisms of chloroplast redox signals to the nucleus. Photosynthetic electron transport components are sketched within a chloroplast (CP). Horizontal lines above them represent the thylakoid membrane system and indicate its connection with the envelope of the chloroplast. Arrows represent redox signals; dotted arrows represent putative redox signaling pathways. MAPK, mitogen-activated protein kinase. For details, see text.

therefore may represent light-independent developmental signals, whereas plastid signals in fully developed tissue report the actual physiological state of the organelle. From the present data, it is apparent that photosynthetic redox signals play an important role in this second class of signals (compare Fig. 1). In addition, it can be assumed that cytosolic photoreceptors may play an important role in gene expression control of plastid proteins during greening of seedlings and that its role is overtaken by other factors such as chloroplast redox signals in fully green plants.

Although the function of chloroplast redox signals appears to be relatively clear, the molecular mechanisms of its transduction toward the nucleus are only poorly understood (Fig. 3). Best models for signal transduction exist for H_2O_2–mediated redox signals. H_2O_2 is known to be membrane-permeable and may pass the chloroplast envelope without directed transport. In the cytosol it induces the respective change in gene expression by activating a mitogen-activated protein kinase cascade (24). For redox signals starting from the PQ pool, the transduction is less clear. It is likely that the same signaling pathway that regulates the state transition also regulates chloroplast gene expression (3), and it is conceivable that an additional branch of this pathway extends to the nucleus. How the signal passes the envelope, for instance via a transporter, is unknown to date. However, there exists experimental evidence that the PQ redox signal is transformed into a phosphorylation signal in the cytosol (10, 17). As a second possibility, PQ redox signals may be sent directly to the nucleus via PQ molecules located in the envelope membrane. As the inner-chloroplast membrane system is in close contact to the envelope and envelope-located electron transport has been found (32), such PQ molecules could report the redox state of the PQ pool to a putative cytosolic receptor. Chloroplast redox signals may be also reported to the nucleus indirectly by their effects on chloroplast gene expression or by affecting the biochemistry of the organelle. As outlined above, there exist plastid signals that tightly couple nuclear gene expression to the function of chloroplast gene expression and therefore to inner-chloroplast redox signals. Besides these possible transduction pathways, several redox-regulated gene expression events are described from which the transduction of the redox signals is completely unknown.

TABLE 1. SUMMARY OF REDOX-CONTROLLED NUCLEAR GENES SORTED BY TYPE OF REGULATING REDOX SIGNAL

*Gene and gene class**	*Activating redox control parameter* *Oxidation signals*	*Organism and reference*
Lhcb (LH)	Oxidized PQ pool	*Dunaliella tertiolecta* (17)
Lhcb (LH)	Oxidized intersystem electron carrier	*Dunaliella salina* (28)
Lhcb (LH)	Oxidized PQ pool	*Lemna perpusilla* (52)
Apx1, Apx2 (AOS)	Reduced PQ pool, high H_2O_2 concentration	*Arabidopsis, Arabidopsis* (transgenic) (22, 23)
Gpx2, Cat2, Gst, Pr2 (AOS)	High H_2O_2 concentration	*Arabidopsis* (31)
Lhcb2, RbcS (PSY)	Photosynthetic electron transport (oxidized)	*Arabidopsis* (cell culture) (36)
Nia2 (M)	Photosynthetic electron transport (oxidized)	Tobacco (transgenic) (45)
	Reduction signals	
Fed1 (PSY)	Photosynthetic electron transport (reduced)	Tobacco (transgenic) (38)
PetE (PSY)	Photosynthetic electron transport (reduced)	*Arabidopsis* (transgenic) (36)
PetE (PSY)	Reduced PQ pool	Tobacco (transgenic) (43)
PsaD, PsaF (PS)	Photosynthetic electron transport (reduced)	Tobacco (transgenic) (43)

*For physiological details and encoded proteins, see text. LH, light-harvesting genes; AOS, antioxidant and stress genes; M, metabolic genes; PSY, photosynthesis genes; PS, photosystem genes.

PERSPECTIVES

The increasing number of reports describing chloroplast redox control of nuclear gene expression suggests that there exist more signals than previously anticipated and underlines the importance of such signals for the development and metabolism of the cell. Currently, we have more questions than answers; however, the present data already show that redox signals are involved in many signaling pathways, such as light, stress, energy, and metabolic signaling, and more are expected. In addition, redox signals from other cell components, *i.e.,* mitochondria and peroxisomes (for review, see 11), may interact with those from the chloroplast. Furthermore, the identity and number of all redox-controlled genes are unknown to date. Genomic approaches using microarray techniques will give us a more complete picture within the next few years. A first study using a H_2O_2-treated *Arabidopsis* cell culture showed that at least 175 different open reading frames responded to oxidative stress (15). As the number and identities of redox-responsive genes will vary depending on the physiological test systems, the presently known genes (Table 1) may represent only the tip of the iceberg. It will be fascinating work to unravel all the roles that redox signals play in the intracellular signaling network.

ACKNOWLEDGMENTS

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ABBREVIATIONS

cyt b_6 *f,* cytochrome b_6 *f* complex; DBMIB, 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone; DCMU, 3-(3′,4′-dichlorophenyl)-1,1′-dimethylurea; FNR, ferredoxin:NADP: oxidoreductase; H_2O_2, hydrogen peroxide; LHCII, light-harvesting complex of photosystem II; PC, plastocyanin; PQ, plastoquinone; PSI, photosystem I; PSII, photosystem II; ROS, reactive oxygen species.

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