A Novel Mechanism of Nuclear Photosynthesis Gene Regulation by Redox Signals from the Chloroplast during Photosystem Stoichiometry Adjustment*

Thomas Pfannschmidt‡, Katia Schütze, Meta Brost, and Ralf Oelmüller

From the Institute of General Botany, Department of Plant Physiology, University of Jena, Dornburger Straße 159, 07743 Jena, Germany

Photonsynthetic organisms acclimate to long term changes in the environmental light quality by an adjustment of their photosystem stoichiometry to maintain photosynthetic efficiency. By using light sources that predominantly excite either photosystem I (PSI) or photosystem II (PSII), we studied the effects of excitation imbalances between both photosystems on nuclear PSI gene transcription in transgenic tobacco seedlings with promoters:β-glucuronidase gene fusions. Shifts from PSI to PSII light sources (and vice versa) induced changes in the reduction/oxidation state of intersystem redox components, and acclimation of tobacco seedlings to such changes were monitored by changes in chlorophyll a/b ratios and in vivo chlorophyll a fluorescence. The ferredoxin-NADPH⁺-oxidoreductase gene promoter did not respond to these treatments, those from the genes for subunits PsAD and PsAF of PSI are activated by a reduction signal, and the plastocyanin promoter responded to both reduction and oxidation signals. Additional experiments with photosynthetic electron transport inhibitors 3-(3',4'-dichlorophenyl)-1,1'-dimethyl urea and 2,5-dibromo-3-methyl-6-isopropyl-p-benzoquinone demonstrated that the redox state of the plastocyanine pool controls the activity of the plastocyanin promoter, whereas subunit PsAD and PsAF gene transcription is regulated by other photosynthesis-derived signals. Thus, the expression of nuclear-encoded PSI genes is controlled by diverse light quality-dependent redox signals from the plastids during photosystem stoichiometry adjustment.

The photosynthetic apparatus in the chloroplasts of higher plants and algae is comprised of a patchwork of nuclear- and plastochloroplast-encoded components. Nuclear-encoded genes for structural components of the photosynthetic machinery are either new or a result of a gene transfer from the endosymbiotic ancestor of chloroplasts to the nucleus of the host cell (1–4). The enormous differences in gene copy number between both compartments require a highly coordinated regulation in their expression during development and acclimation of the organism to environmental cues. This coordination is controlled by the nucleus at many levels (5) but also involves signals from the plastids, which influence the expression of nuclear genes for plastid proteins (6–9). The exact nature of the plastid-derived signal(s) is still elusive. Inhibition of either plastid transcription or translation or photo-oxidative destruction of chloroplasts prevents the transcription of several nuclear-encoded photosynthesis genes (10–13). Other crucial components involved in this interorganellar cross-talk are intermediates and/or components of the tetrapyrrol biosynthesis pathway (14–18) or the availability of phosphoenolpyruvate (19). One of the central players in this scenario is light, and it seems to regulate the expression of several nuclear genes for plastid proteins via the same cis-active elements as the plastid-derived signal(s) (20). Besides cytosolic photoreceptors plants sense changes in light quantity or quality via the modulation of the reduction/oxidation (redox) state of various chloroplast molecules involved in photosynthesis. This redox signaling provides a feedback link between the degree of photosynthetic efficiency and the expression of nuclear photosynthesis genes (21–23), which helps to acclimate the photosynthetic process to varying environmental conditions. A few examples for this are known. Acclimation to different light intensities and temperatures of the unicellular algae Dunaliella tertiolecta and Dunaliella salina involves changes in the transcription of the nuclear-encoded chlorophyll-binding proteins of the light-harvesting complex (LHC) genes, which are regulated by the redox state of the PQ¹ pool (24, 25). Dark/light shift experiments with transgenic tobacco revealed that photosynthetic electron transport controls also transcription of the pea ferredoxin (FED1) gene as well as FED1 mRNA ribosome loading (26), and the inhibition of photosynthetic electron transport by DCMU also destabilizes the FED1 mRNA (27). Under high light stress, Arabidopsis showed an increase in the transcription of the nuclear APX genes (encoding cytosolic ascorbate peroxidases) probably controlled by the PQ redox state and H₂O₂ (28, 29). Furthermore, an interaction between photosynthetic electron transport and sugar regulation on nuclear photosynthesis gene expression has been postulated. In an Arabidopsis cell culture the observed increase in LHCb and PETE (encoding plastocyanin) transcription after sugar repletion was diminished in the presence of DCMU (30).

Illumination conditions that predominantly excite photosystems I (PSI) or II (PSII) generate an imbalance in excitation energy distribution between PSI and PSII, which results in a decrease of the photosynthetic efficiency. Such light quality gradients that typically appear in canopies of trees or forests,
in dense plant populations, and aquatic environments are counterbalanced by plants with an initial short term response called state transitions, and a following long term response that causes a readjustment of PS stoichiometry. This readjustment requires changes in photosynthesis gene expression (31, 32). With light sources that predominantly excite PSI or PSII we could demonstrate that the redox state of the PQ pool controls transcription of the chloroplast-encoded PS genes psbA and psaAB in mustard (33, 34). However, at least half of the structural compounds of the photosynthetic apparatus are encoded in the nucleus (35, 36), and nothing is known about how their expression is coupled to the changed expression in chloroplasts under varying light quality. In this study, we focus on the nuclear-encoded PSI genes PSAD (encoding subunit PsaD), PSAF (encoding subunit PsaF), PETF, and PETH (encoding ferredoxin-NADP⁺-oxidoreductase) to address the question of whether their expression also responds to changes of plastid redox parameters. These genes encode crucial components of the PSI complex. The subunits PsaD and FNR are located at the stromal side of the complex and provide an essential site for ferredoxin docking (37–39) and the electron transfer from ferredoxin to the electron acceptor NADP⁺. The subunits PsaF and plastocyanin are located at the luminal side. The mobile electron carrier plastocyanin transfers electrons from the cytochrome b6f complex to PSI, and PsaF provides a docking site for this protein (40–42). Recent studies using promoter:β-glucuronidase (GUS) fusions in transgenic tobacco revealed that these four promoter constructs respond to light and the plastid signal(s) (19, 43, 44). To test whether promoter activation of these genes is coupled to the photosynthetic electron transport, we grew transgenic tobacco seedlings under the above mentioned PS-specific light sources. We found that the promoters of the genes PSAD, PSAF, and PETF but not PETH respond to light-induced redox signals from the plastid. Furthermore, inhibitor assays revealed that transcription of the genes PSAD, PSAF, and PETF is coupled to photosynthetic electron transport via different redox systems. This demonstrates that redox signals do not only operate within the chloroplast (33) but also affect nuclear-encoded photosystem genes with the aim to coordinate nuclear and chloroplast PSI gene expression during PS stoichiometry adjustment.

EXPERIMENTAL PROCEDURES

Plant Material and Growth Conditions—Plants were grown in a temperature-controlled growth chamber at 22 °C. Mustard seedlings (Sinapis alba L.) were grown on vermiculite in plastic boxes under continuous light. They were grown under PSI or PSII light for 7 days or 5 days in PSI light followed by 2 days of PSII light and vice versa as described (33, 34). Tobacco wild-type (SamsunNN) and transgenic lines were germinated and grown on 1/2 Murashige and Skoog medium containing 1.35% sucrose and, in case of transgenic lines, 80 mg/ml kanamycin in Petri dishes. Generation of the transgenic lines used here have been described earlier (45–48). Promoter:GUS fusion constructs introduced into tobacco contained the following cis-active regions relative to the transcription start site (+1): −1802 to +55 (PSAD), −1074 to +163 (PSAF), −1126 to +60 (PETF), and −731 to +251 (PETH). Tobacco seedlings were grown for 18 days under continuous PSI or PSII light or 14 days in PSI light followed by 4 days in PSII light or vice versa. Spectral quality and light quantity of the PSI and PSII light sources have been described (33). White-light control plants were illuminated continuously for 7 days (mustard) or 14 days (tobacco) with 30 W white-stripe lamps (OSRAM) with a photon flux density of 100 μmol/m²s.

Inhibitor Treatments—The electron transport inhibitors DCMU and DBMIB (Sigma) have been applied to the plants grown in Petri dishes by spraying 0.5 ml of indicated concentrations on the leaves using a 10-ml fine sprayer. Control seedlings were treated with the solvent without inhibitors. Wild-type seedlings were illuminated with white light until Chl fluorescence measurements were performed. Transgenic tobacco lines grown 14 days under PSI or PSII light sources were treated with inhibitors in the same way directly before the respective light switch to PSI or PSII light. The DBMIB treatment was repeated every 3–4 h during the last 96 h of experimentation. Stock solutions of DCMU were 10 mm in 50% ethanol and of DBMIB were 100 mm in 10% Me₂SO in ethanol. The different inhibitor concentrations were prepared by dilution in sterile water/1×

RESULTS

Acclimation to PSI and PSII Light Sources—The acclimation of higher plants to light sources favoring either PSI or PSII can be monitored easily by determination of the Chl a/b ratio, which represents a rough estimate of changes in PS stoichiometry (cf. Ref. 34). Tobacco seedlings were grown under PSI and PSII light (PSI and PSII plants) for 14 days, which resulted in an acclimation (Fig. 1A, 1st acclimation) to the respective illumination conditions as indicated by a low Chl a/b ratio for PSI plants and a high ratio for PSII plants (Fig. 1A). A shift of the seedlings to the other light source forces the plants to acclimate to the new illumination conditions (Fig. 1A, 2nd acclimation). Such shifts were shown to induce state transitions (34) and to influence the redox state of the PQ pool (50). A shift from PSI to PSII light results in a more reduced PQ pool (Fig. 1A, reduction signal), whereas a shift from PSII to PSI light influences a more oxidized state of PSI (Fig. 1A, oxidation signal). 96 h after the light shift, tobacco seedlings are completely acclimated to the new light conditions. This process occurs fast at the beginning (first 24 h) and significantly slower towards the end of the treatment (Fig. 1B). PSI fluorescence measurements with fully acclimated tobacco plants confirm differences in the photosynthetic electron transport efficiency, which result from the different acclimations. Representative Chl fluorescence induction curves of PSI and PSII light-acclimated tobacco obtained by standard pulse amplitude-modulated fluorescence measurements (Fig. 1B) demonstrate a higher level in steady-state fluorescence F₇ in PSI plants when compared with PSII plants. A more detailed analysis of photosynthetic parameters is given in Table I. Mustard, for which changes in PSI stoichiometry in response to the same PSI or PSII light sources have been described (33, 34), were used as a...
plants. In contrast, the effective quantum yield, $\Phi_H$, is higher in PSII plants than in PSI plants and changes accord-
ingly after a shift of the seedlings to the respective other light source. Taken together, these data indicate a limited electron transport capacity in PSI and PSII→PSI plants in comparison with the capacity measured in PSII and PSI→PSII plants. This is consistent with results from mustard; the PSI/PSII ratio is high after acclimation to PSI light and low after acclimation to PSII light (33, 34), causing respective changes in electron transport capacity.

**Differential Activation of Nuclear PSI Gene Promoters**—To analyze the putative impact of chloroplast redox signals on the expression of the nuclear PSI genes PETH, PSAD, and PETH, transgenic tobacco lines harboring the PSI promoter-GUS gene fusions were grown under PSI or PSII light (Fig. 2). The PETH promoter showed the same activity under all light sources tested. In contrast, the PSAD promoter exhibited higher activity in PSII light than in PSI light. 4 days under PSII light stimulates the promoter activity to the level detectable in continuous PSI light; however, no reduction in the activity was observed after a PSI→I light shift. The PSAD promoter behaves similarly except that it exhibits lower activity under control white light. Similarly, the PETH promoter shows low activity under PSI light, high activity under PSII light, and activation after a PSI→II light shift. Furthermore, in contrast to the other lines, the GUS activity declined after a PSII→I light shift. Thus, at least three different response patterns to chloroplast redox signals can be distinguished.

Sucrose has been shown to repress photosynthesis gene expression (54) and to interact with chloroplast redox signals (30). Because we grew seedlings on a medium containing 1.35% sucrose, we tested a possible role of sucrose by transferring seedlings to a sucrose-free medium before shifting them to the other light regime. The GUS activity was determined after 96 h. We found no qualitative changes; however, in general the GUS activities were 20–30% lower on the sugar-free medium (data not shown). Thus we conclude that sucrose has no effect on the light quality-dependent redox signals under our conditions.

**Coupling of Nuclear Gene Transcription to Photosynthetic Electron Transport**—The electron transport inhibitors DCMU and DBMIB inhibit electron flow before and behind the PQ pool, respectively, thus resulting in the oxidation or reduction of it (55). We sprayed these inhibitors exogenously onto the seedlings to show a coupling of the expression of PSI genes in the nucleus to the photosynthetic electron transport. Initially, we determined the optimal inhibitor concentrations by adjusting them such that their effects match the observed effects of the light sources on the Chl fluorescence parameters (Fig. 3). Treatment with DCMU (10 and 40 $\mu M$) resulted in a decrease in the $\Phi$PSII value within 15 min. After 4 and 96 h these values decreased slightly, presumably because of a delayed inhibitory effect caused by the penetration of the substance. Detailed dose-response analyses uncovered that a concentration of 10 $\mu M$ DCMU results in a reduction of electron transport, which is identical to the effect of a PSI→I light shift. In addition, a DBMIB concentration of 25 $\mu M$ caused a similar effect as a PSI→PSII redox signal; however, the component is labile, and the photosynthetic parameters could only be maintained over the 96-h period when DBMIB was reapplied every 3–4 h. Spraying of control plants with the solvent alone had no effect on the photosynthetic electron flow or the expression of the reporter genes when compared with untreated plants (data not shown). With regard to the redox state of the PQ pool, we used the inhibitors as a reduction (DBMIB) or oxidation (DCMU) signal itself or as antagonists to the respective light-induced redox signals (Fig. 4). The tobacco lines harboring the PETH promoter construct did not respond to any of the inhibitor control. Both PSI and PSII plants show no significant changes in the maximal quantum yield after acclimation to the respective other light sources. In addition, the Fv/Fm value of both species is between 0.80 and 0.84 under all conditions tested, which is typical for dark-acclimated, healthy, and unstressed plants. However, the Fs/Fm ratio is high in PSI plants and maximal fluorescence, Fm, are indicated.

![FIG. 1. Acclimation of tobacco seedlings to PSI and PSII light sources](image)

A tobacco wild-type seedlings were grown sterile 14 days under PSI or PSII light. Plants were then switched to the respective other light source, and plant material was taken at indicated points of time for Chl a/b determination. A PSI→PSII light shift represents a reduction signal, and a PSII→PSI light shift represents an oxidation signal for the redox state of the PQ pool. This definition is used throughout the article if not indicated otherwise. Plants for zero values were harvested directly before the light switch. Values represent the mean of two independent experiments performed in triplicate ($n = 6$). B, representative in vivo Chl fluorescence of 18-day-old tobacco plants acclimated to PSI and PSII light. Details of the measurements are given under “Experimental Procedures.” The steady-state fluorescence, Fs, and maximal fluorescence, Fm, are indicated.
treatments (data not shown), which is consistent with the results obtained with the light regimes. The other three promoter constructs responded to the inhibitor treatments, however, in a differential way. In all instances, DCMU inhibited the promoter activation by a reduction signal of PSII light on PSI plants. In addition, the DCMU alone down-regulated the promoter activity of PSAD, PSAF, and PETE in PSII plants. Thus expression of these genes is coupled to photosynthetic electron transport in chloroplasts. The promoter activity of PSAD and PSAF is not down-regulated by PSI light in PSII plants, which is in contrast to the results obtained for PETE (compare with Fig. 2). The application of DBMIB in co-action with PSI light resulted in a down-regulation of PSAD and PSAF promoter activity. In contrast, the down-regulation of PETE promoter activity by the PSI light-induced oxidation signal was inhibited to over 50% by the DBMIB treatment. Application of the drug alone on PSI plants, however, resulted in no significant reaction in all three cases. These data indicate that the promoter activity of PETE is controlled by the redox state of the PQ pool, whereas PSAD and PSAF gene expression is redox-controlled; however, a clear initiator component within the chloroplast could not be identified so far.

**DISCUSSION**

We demonstrate that chloroplast redox signals participate in light regulation of nuclear PSI genes by using PS-specific light sources originally established for mustard (33). Our studies showed that tobacco seedlings represent a useful model organism for such studies. In addition, this is the first report describing the expression of nuclear PSI genes during photosystem stoichiometry adjustment. We show that redox signals previously found to control gene expression within chloroplasts also control gene expression in the nucleus. The four promoters analyzed in this study showed three different responses to the light sources: no response (PETH), activation (PSAF and

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**TABLE I**

Photosynthetic efficiency of mustard and tobacco seedlings after acclimation to different light qualities

| Light | Fv/Fm Mustard | Fv/Fm Tobacco | 1-qP Mustard | 1-qP Tobacco | dPSII Mustard | dPSII Tobacco |
|-------|---------------|---------------|--------------|--------------|---------------|---------------|
| PSI   | 0.836 ± 0.025 | 0.815 ± 0.006 | 0.284 ± 0.044 | 0.247 ± 0.03 | 0.313 ± 0.06 | 0.416 ± 0.068 |
| PSI → II | 0.826 ± 0.003 | 0.809 ± 0.042 | 0.103 ± 0.021 | 0.139 ± 0.034 | 0.106 ± 0.025 | 0.207 ± 0.036 |
| PSII  | 0.807 ± 0.01  | 0.803 ± 0.06  | 0.059 ± 0.004 | 0.134 ± 0.027 | 0.085 ± 0.026 | 0.274 ± 0.026 |
| PSII → I | 0.81 ± 0.01   | 0.816 ± 0.019 | 0.179 ± 0.03  | 0.251 ± 0.036 | 0.206 ± 0.077 | 0.484 ± 0.054 |

**FIG. 2.** PSI promoter utilization during acclimation to PSI and PSII light. The illumination conditions of plant growth are indicated across the top. The identity of the gene promoters is given inside the panels. White, white-light control.

**FIG. 3.** Effects of exogenously applied electron transport inhibitors on photosynthetic efficiency of tobacco. 14-day-old white-light-grown wild-type tobacco seedlings were treated with increasing concentrations (given on top) of DCMU or DBMIB, and the inhibitory effects on electron transport were quantified by measuring the Chl fluorescence parameter, dPSII (effective quantum yield), at the indicated times after application. As control (C), the plants were treated in the same way with the solvent but without any inhibitor. The values represent means of three independent measurements. Variations were in the same range as shown in Table I.
photosynthetic electron transport. The optimal concentrations
amplitude-modulated fluorometer measurements demonstrate
over the whole period of experimentation,
hibitor effects were optimized such that they are compa-
hibitor assay with DCMU and DBMIB confirmed the coupling
PSAD
 parallels.

FIG. 4. Effects of electron transport inhibitors in interaction
with PS-specific light sources on PSI promoter activities. Growth conditions and inhibitor treatments were as indicated across
the top. The identity of gene promoters is given inside the panels. The respective promoter activation of PSII light on PSI plants (compare with Fig. 1, reduction signal) was arbitrarily set to 100%, and changes in promoter utilization induced by switches between light sources and inhibitor treatments were expressed in percent. The values represent
the mean of three independent experiments performed with 3–4
parallels.

PSAD), and reversible activation/inactivation (PETE). An in-
hibitor assay with DCMU and DBMIB confirmed the coupling
of the promoter response to photosynthetic electron transport. The inhibitor effects were optimized such that they are compara-
ble with the effects of the light sources and that they persist
over the whole period of experimentation, i.e. 96 h. The pulse
amplitude-modulated fluorometer measurements demonstrate
that the inhibitor concentrations mimic the light effects on
photosynthetic electron transport. The optimal concentrations
(10 μM DCMU and 25 μM DBMIB) differed from those used by
Karpinski et al. (28); they used 4 and 14 μM, which were
effective after infiltration into Arabidopsis leaf discs. Because we sprayed the inhibitors on the cotyledons, the effective con-
centrations within the tissue are not known, but they must be
considerably lower when compared with the concentration ap-
plied by the infiltration technique. 10 μM DCMU, for instance,
completely inhibits electron transport when applied to isolated
chloroplasts (data not shown). DBMIB is a light-labile com-

PSAD, and PETE promoter constructs are activated by PSII light-generated reduction signals that can be blocked by a DCMU-generated oxidation signal. This oxidation signal alone is also able to
reduce the activity of all three promoters, demonstrating that they
can be regulated by both reduction and oxidation signals.
A PSI light-induced oxidation signal reduced the PETE pro-
moter-driven GUS activity but had no influence on the GUS
activities driven by the PSAD and PSAF promoters. DBMIB
treatment had only an effect in co-action with the PSI light-
generated oxidation signal but not alone. In the cases of PSAD
and PSAF this results in a decrease of the GUS activities as
observed after DCMU treatment. This suggests that these two
promoters respond to redox signals originating between the PQ
pool and PSI or to the electron transport capacity in general. In
contrast, because DBMIB prevents the PETE promoter deacti-
vation induced by PSI light, the redox state of the PQ pool
seems to regulate this promoter. The different response pattern
of the promoters can be caused by different sensitivities to
chloroplast redox signals. Activation by a dark/light transition
of seedlings is a strong environmental signal and represents an
off/on mechanism. PSI and PSII light-induced redox signals are
a different type of environmental signal, and responses to them
represent a modulation mechanism under persistent illumina-
tion. The additive effect of PSI light and DBMIB on PSAD and
PSAF promoter activity suggests the existence of a threshold
value in these cases that may be not reached when PSI light or
DBMIB suppress electron transport alone. The signaling redox
components remain unclear; however, the inhibitor experi-
ments demonstrate that PSAF and PSAD promoter activity is
clearly connected to the photosynthetic electron transport.

The chloroplast redox signals represent a new facet in the
light regulation of nuclear photosynthesis genes. Results with
dark-grown pea LIP1 and Arabidopsis COP1-4 mutants imply
that the plastid-derived signal(s) is light-independent and not
coupled to photosynthesis (13). The chloroplast redox signals
investigated in this study are both light-dependent and coupled
to photosynthesis, thus representing a new type of plastid-
derived signal. Physiologically, this light quality-dependent
regulation of PSAD, PSAF, and PETE makes sense. The gene
products of PSAD and PSAF are structural components of PSI.
Thus, chloroplast redox signals adjust the expression of nuclear
PSI genes to the overall need of the photosynthetic apparatus
and might be a course-regulated process. The fine-tuning of PS
stoichiometry adjustment is then achieved by the rapid and
direct redox-regulated synthesis of the PsA and PsAB reaction
center subunits (33), which occurs in the same time scale as
state transitions (59). In contrast, plastocyanin is not an inte-
grated component of PSI, and the fine tuning of its expression might occur differently, in particular because this polypeptide operates as an electron transfer component between the two photosystems. The present study provides new insights into chloroplast-nuclear signaling. Our current investigations focus on the impact of redox signals on other nuclear genes, the velocity of signal transduction in comparison to inner chloroplast redox signals, and the way how these signals are transduced.

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Redox Control of Photosynthesis Gene Expression

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Redox Control of Photosynthesis Gene Expression