The \textit{pgr1} Mutation in the Rieske Subunit of the Cytochrome $b_{6f}$ Complex Does Not Affect PGR5-dependent Cyclic Electron Transport around Photosystem I*  

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Although photosystem I (PSI) cyclic electron transport is essential for plants, our knowledge of the route taken by electrons is very limited. To assess whether ferredoxin (Fd) donates electrons directly to plastoquinone (PQ) or via a Q-cycle in the cytochrome (cyt) $b_{6f}$ complex in PSI cyclic electron transport, we characterized the activity of PSI cyclic electron transport in an \textit{Arabidopsis} mutant, pgr1 (proton gradient regulation). In pgr1, Q-cycle activity was impaired. Furthermore, the simultaneous determination of the quantum yields of both photosystems indicated that the ratio of linear and PSI cyclic electron transport in \textit{vivo}. In contrast to the pgr5 defect, the pgr1 defect did not show any synergistic effect on the quantum yield of photosystem II in \textit{vivo}, a mutant in which NDH (NAD(P)H dehydrogenase) activity was impaired. Furthermore, the simultaneous determination of the quantum yields of both photosystems indicated that the ratio of linear and PSI cyclic electron transport was not significantly affected in pgr1. All the results indicated that the pgr1 mutation did not affect PGR5-dependent PQ reduction by Fd, which functions in PSI cyclic electron transport \textit{in vivo}. The phenotypic differences between pgr1 and pgr5 indicate that maintenance of the proper balance of linear and PSI cyclic electron transport is essential for preventing over-reduction of the stroma.

Photosynthetic electron transport consists of two main routes, linear electron transport and photosystem I (PSI) \textsuperscript{1} cyclic electron transport. In linear flow, electrons excited from water at PSII are ultimately transferred to NADP$^+$. Electron transport between PSII and PSI is mediated by the cyt $b_{6f}$ complex and coupled to translocation of protons across the thylakoid membranes from the stroma to the lumen. The resulting $\Delta \text{pH}$ is utilized in ATP synthesis. In PSI cyclic electron transport, however, electrons are recycled from either NAD(P)H or Fd to PQ, generating $\Delta \text{pH}$ without accumulation of NADPH. Both linear electron transport and PSI cyclic electron transport have been shown to be essential for photoprotection and photosynthesis in higher plants (1, 2).

In higher plants, PSI cyclic electron transport consists of two pathways, both of which partly share the route with linear electron transport. The NDH-dependent pathway was originally discovered in cyanobacteria (3, 4) and was also shown to operate in PSI cyclic electron transport in the chloroplast (5, 6). Although the NDH complex is a major player in PSI cyclic electron transport in cyanobacteria, its contribution is minor in higher plants. Instead, the Fd-dependent route of PSI cyclic electron transport, which was discovered in the 1950s (7), contributes significantly to $\Delta \text{pH}$ generation in higher plants. Despite this long history of interest (8) and documented physiological significance (1, 2), our knowledge of the machinery of Fd-dependent electron transport is limited. Recently, we showed that a small thylakoid protein, PGR5, is essential for Fd-dependent PSI cyclic electron transport (1), although the function of the PGR5 protein is still unclear.

The cyt $b_{6f}$ complex plays a key role in $\Delta \text{pH}$ generation in both linear and PSI cyclic electron transport (9, 10). The complex comprises four major subunits, the Rieske subunit, cyt $f$, cyt $b_{6}$, and subunit IV, as well as four small subunits, PetL, PetG, PetM, and PetN. Fig. 1 outlines a scheme for electron transport through the cyt $b_{6f}$ complex, called the Q-cycle. Although the Rieske subunit binds an Fe$2$S$_2$ cluster, cyt $b_{6}$ is essential for its high and low potential hemes, $b_{6i}$ and $b_{6j}$. The Rieske subunit catalyzes the oxidation of plastoquinol (PQH$_2$) at the Q$_i$ site, which is associated with the reduction of both cyt $f$ via the Fe$2$S$_2$ cluster and $b_{6j}$, resulting in the release of two protons to the thylakoid lumen. Bifurcated electron transport consists of a high potential chain to plastocyanin via the Fe$2$S$_2$ cluster and cyt $f$ and a low potential chain via cyt $b_{6i}$, allowing electron transfer across thylakoid membranes to PQ at the Q$_i$ site near the stroma side.

Following discovery of Fd-dependent PSI cyclic electron transport (7), there has been continuing debate as to whether reduced Fd donates electrons to the cyt $b_{6f}$ complex and then to PQ via the Q-cycle (Fig. 1, \textit{route 1}) or directly to PQ (Fig. 1, \textit{route 2}). In the latter case a putative Fd-PQ reductase (FQR) is required (8), whereas for the former the cyt $b_{6f}$ complex would be involved. Recently, serious consideration of the idea that Fd donates electrons to the cyt $b_{6f}$ complex (route 1) was revisited based on the findings that the Fd-NADP$^+$ reductase associates with the cyt $b_{6f}$ complex (11) and also that the cyt $b_{6f}$ complex contains an unexpected heme, heme $x$, which is not conserved in the cyt $b_{6c}$ complex in mitochondria (12, 13). To assess the possibility that Fd donates electrons to the cyt $b_{6f}$ complex, we characterized electron transport in an \textit{Arabidopsis thaliana} mutant, pgr1, in which Q-cycle activity is conditionally impaired because of an amino acid alteration in the Rieske subunit of the cyt $b_{6f}$ complex (14, 15).

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1 The abbreviations used are: PSI, photosystem I; cyt, cytochrome; ETR, electron transport rate; NDH, NAD(P)H dehydrogenase; Fd, ferredoxin; FQR, ferredoxin-plastoquinone reductase; PAM, pulse amplitude modulation; PSI, photosystem II; PQ, plastoquinone; PQH$_2$, plastoquinol; Q$_i$, quinone-reducing site of the cytochrome $b_{6f}$ complex; Q$_{o}$, quinol-oxidizing site of the cytochrome $b_{6f}$ complex; MOPS, 4-morpholinepropanesulfonic acid; MES, 4-morpholineethanesulfonic acid.
In Vitro Assay of PSI Cyclic Electron Transport Activity—Fd-dependent PQ reduction activity was measured in ruptured chloroplasts as described previously (1, 16). Media were buffered with 50 mM HEPES/NaOH for pH 7.6, 5 mM MOPS/NaOH for pH 7.0 and 6.5, and 5 mM MES/NaOH for pH 6.0 and 5.5.

In Vivo Analysis of Chlorophyll Fluorescence—Chlorophyll fluorescence parameters were measured using a MINI-PAM system (WALZ, Effeltrich, Germany).

In Vitro Assay of Linear Electron Transport—Intact chloroplasts were osmotically ruptured in the assay media used for PSI cyclic electron transport activity (see below). Linear electron transport activity was measured using 25 μM methyl viologen as an electron acceptor in the presence of 5 mM NH4Cl. Activity was determined by the quantum yields of PSII (ΦPSII) at 144 μmol of photons m⁻² s⁻¹ using a MINI-PAM (pulse amplitude modulation) portable chlorophyll fluorometer (WALZ, Effeltrich, Germany).

In Vitro Assay of PSI Cyclic Electron Transport Activity—Fd-dependent PQ reduction activity was measured in ruptured chloroplasts as described previously (1, 16). Media were buffered with 50 mM HEPES/NaOH for pH 7.6, 5 mM MOPS/NaOH for pH 7.0 and 6.5, and 5 mM MES/NaOH for pH 6.0 and 5.5.

In Vivo Analysis of Chlorophyll Fluorescence—Chlorophyll fluorescence parameters were measured using a MINI-PAM system. The minimum chlorophyll fluorescence at the open PSII center (F₀) was determined by measuring light at a light intensity of 0.05–0.15 μmol of photons m⁻² s⁻¹. A saturating pulse of white light (800 ms) was applied to determine the maximum chlorophyll fluorescence at the closed PSI center in the dark (Fₐ) and during actinic light illumination (Fₐ'). The steady state of chlorophyll fluorescence level was recorded during actinic light illumination (20–100 μmol of photons m⁻² s⁻¹). Efficiency of the PSI photochemistry (ΦPSII) was calculated by an equation, (Fₐ'/Fₐ) × F₀/Fₐ'. Relative electron transport rate (ETR) was calculated as ΦPSII × light intensity (μmol of photons m⁻² s⁻¹).

Simultaneous Determination of Quantum Yields of Both Photosystems—Changes in chlorophyll fluorescence and P700 absorbance at 830 nm were simultaneously monitored during a PAM chlorophyll fluorometer (WALZ) equipped with emitter detector units ED101 for chlorophyll fluorescence emitted from PSII. In this assay system, NADPH is essential for electron donation to Fd through the reverse reaction of Fd-NADPH oxidoreductase (17). Although addition of NADPH did not reduce Fd, it was rapidly reduced by subsequent addition of Fd. Both PGR5- and NDH-dependent pathways contribute in this electron donation to PQ (2). Because the NDH-dependent pathway also requires Fd in our assay system, we refer to the Fd-dependent, anticyanin A-sensitive pathway as the PGR5-dependent pathway hereafter to avoid confusion.

In Vivo Assay of PSI Cyclic Electron Transport Activity in pgr1—To study a possible defect in PSI cyclic electron transport activity in pgr1, electron donation to PQ from Fd was compared using ruptured chloroplasts isolated from the wild type and pgr1. Whether Fd donates electrons directly to PQ or indirectly via the cyt b₆f complex (Fig. 1, routes 2 and 1, respectively), PQ reduction can be monitored as an increase in chlorophyll fluorescence emitted from PSI. In this assay system, NADPH is essential for electron donation to Fd through the reverse reaction of Fd-NADPH oxidoreductase (17). Although addition of NADPH did not reduce Fd, it was rapidly reduced by subsequent addition of Fd. Both PGR5- and NDH-dependent pathways contribute in this electron donation to PQ (2). Because the NDH-dependent pathway also requires Fd in our assay system, we refer to the Fd-dependent, anticyanin A-sensitive pathway as the PGR5-dependent pathway hereafter to avoid confusion.

To monitor the effect of impaired Q-cycle activity in pgr1 on the PGR5-dependent PQ reduction, the reaction medium was acidified from pH 7.6 to 5.5 in a stepwise manner. Unexpectedly, the activity of PQ reduction was drastically affected even at pH 7.0 and completely inhibited at pH 5.5 in the wild type (Fig. 3), implying that electron transfer from NADPH to PQ requires slightly alkaline pH (pH 7.6). This pH sensitivity of PQ reduction was observed both in pgr5 (data not shown) and crr2-2 (chlororespiratory reduction) (Fig. 4). NDH activity is specifically affected in crr2-2, in which the expression of the...
We conclude that the of PGR5-dependent PQ reduction in the stroma is slightly alkaline. Although the thylakoid lumen is acidified in the light, the pendent activity and completely impaired by addition of 10 μM antimycin A. In the double mutant, the PQ reduction activity was completely impaired in the double mutant lacking NDH activity. In the double mutant, the PQ reduction activity was drastically impaired even at pH 7.0.

To study the possible effect of the pgr1 mutation on PSI cyclic electron transport, the pH dependence of PQ reduction activity was compared between pgr1 and wild type. In pgr1, the activity of PQ reduction was severely affected at pH 7.0 and reduced to almost undetectable levels at pH 5.5 (Fig. 3). However, this pH dependence was similar to that in the wild type, suggesting that the hypersensitivity of the Q-cycle did not affect the activity of PQ reduction in pgr1.

Because PQ reduction activity consists of two redundant pathways, the PGR5- and NDH-dependent pathways may be influenced by NDH activity. Based on analogy with the mitochondrial and bacterial NADH dehydrogenase, it is highly possible that the chloroplast NDH will donate electrons directly to PQ, and thus NDH activity is unlikely to be affected by the pgr1 mutation. To simplify the assay system, we used crr2-2, in which NDH activity was specifically impaired (18). To evaluate the effect of the pgr1 mutation within the crr2 mutant background, a double mutant, crr2-2 pgr1, was compared with a single mutant, crr2-2. The PQ reduction activity remaining under the crr2-2 mutant background was because of PGR5-dependent activity and completely impaired by addition of 10 μM antimycin A (Fig. 4). Even in the absence of antimycin A, the activity of PQ reduction was completely impaired in the double mutant crr2-2 pgr5 (2). As in the wild type, PQ reduction activity was drastically affected even at pH 7.0 in crr2-2 (Fig. 4), indicating that PGR5-dependent activity requires a slightly alkaline pH. In the double mutant crr2-2 pgr1, the activity of PQ reduction was also reduced upon a decrease in pH of the reaction medium. However, we did not find any difference in sensitivity to the medium pH between crr2-2 and crr2-2 pgr1.

We conclude that the pgr1 mutation does not affect the activity of PGR5-dependent PQ reduction in the in vitro assay system using ruptured chloroplasts.

In Vivo Assay of PSI Cyclic Electron Transport Activity in pgr1—Although the thylakoid lumen is acidified in the light, the stroma is slightly alkaline in vivo. In our in vitro assay system, however, the stromal side was also acidified, which may have introduced an artificial effect on electron transport. In fact, the activity of PQ reduction by Fd in ruptured chloroplasts was sensitive to a pH of less than 7.0 in both wild type (Fig. 3) and crr2-2 (Fig. 4). To eliminate this as a possible problem, the ETR was calculated from a chlorophyll fluorescence parameter of Fd-NADP⁺ reductase (19), is sensitive to pH lower than 7.0.

As reported previously, ETR was specifically impaired at high light intensity in pgr1 (14), the phenotype being explained by the hypersensitivity of the Q-cycle to lumen acidification (15). Although pgr5 exhibited a similar phenotype in ETR to pgr1, the defect was because of different physiological events (1, 2). The pgr5 defect in PSI cyclic electron transport leads to a reduction in the ATP/NADPH production ratio during photosynthesis, which causes the over-reduction of the stroma. The resulting depletion of NADP⁺ limits linear electron transport specifically at high light intensity. The contribution of the NDH-dependent pathway for ATP synthesis is much lower compared with the PGR5-dependent pathway. Therefore, crr2-2 did not show a defect in ETR at any light intensity, consistent with results for other mutants lacking NDH activity (5, 18, 20). In the double mutant, crr2-2 pgr5, however, ETR was drastically impaired even at a low light intensity of less than 100 μmol of photons m⁻² s⁻¹. This result indicates that the NDH-dependent pathway also contributes to ATP synthesis, a function that is essential under the pgr5 mutant background (2). Thus, crr2-2 is an enhancer of a defect in the PGR5-dependent pathway. If PGR5-dependent PQ reduction is through the cyt b₅f complex (Fig. 1, route 1) and is affected by the pgr1 mutation in the Rieske subunit, the pgr1 phenotype is a mixture of defects in Q-cycle activity and the PGR5-dependent PSI cyclic pathway. Within the crr2-2 mutant background, a possible defect in the PGR5-dependent PSI cyclic pathway in pgr1 may be enhanced, as occurred for the double mutant crr2-2 pgr5.
In crr2-2 pgr1, however, light intensity dependence of ETR was identical to that in pgr1 (Fig. 5). In contrast to the synergistic effect of the crr2-2 defect on pgr5, the crr2-2 defect did not enhance the pgr1 phenotype. This result indicates that the activity of PGR5-dependent PSI cyclic electron transport was not impaired at any light intensity in vivo in pgr1. Considering this together with the results of in vitro analysis (Figs. 3 and 4), we concluded that the PGR5-dependent PSI cyclic electron transport was not affected by the pgr1 mutation in the Rieske subunit.

**In Vivo Assay of the Ratio between Linear and PSI Cyclic Electron Transport in pgr1**—The crr2-2 mutation did not enhance the pgr1 phenotype, indicating that PGR5-dependent PSI cyclic electron transport was not affected in pgr1. Even assuming that the cyt bcf complex is not involved in the activity of PGR5-dependent PQ reduction by Fd, PQH2 is reoxidized at the Qb site of the cyt bcf complex, indicating that the entire pathway of PSI cyclic electron transport is inevitably through the cyt bcf complex. Therefore, it is likely that both the PSI cyclic and linear electron pathways are limited at the cyt bcf complex at high light intensity in pgr1. To evaluate the effect of the pgr1 defect on both electron transport pathways quantitatively, the ratio between linear and PSI cyclic electron transport was compared between pgr1 and wild type (Fig. 6). Although PSI photochemistry is preferentially involved in linear electron transport, PSI photochemistry mediates both linear and PSI cyclic electron transport. Recently, we established a method for the simultaneous determination of PSI and PSII photochemical efficiencies, $\Phi_{\text{PSI}}$ and $\Phi_{\text{PSII}}$. 2 Fig. 6, A and B, shows the quantum yields of both photosystems at each light intensity. In pgr1 both quantum yields were decreased, especially at the higher light intensities of 270 and 560 μmol of photons m$^{-2}$ s$^{-1}$. Unexpectedly, the efficiency of PSI photochemistry was higher in pgr1 at low light intensity (Fig. 6B). Fig. 6C shows two-dimensional plots of the quantum yields of both photosystems. When the ratio is 1.0, all of the photochemical reactions of PSI are linked to the PSII photochemical reactions, indicating that there is no contribution of PSI cyclic electron transport. At all light intensities used, $\Phi_{\text{PSI}}/\Phi_{\text{PSII}}$ was $\sim 1.3$, which was not influenced in pgr1. This result indicates that the ratio of PSI cyclic electron transport was $\sim 30\%$ of linear electron transport in the wild type. Although the pgr1 defect affected the quantum yields of both photosystems, the ratio of linear and PSI cyclic electron transport was not significantly affected. This result is consistent with our conclusion that the pgr1 defect does not affect PGR5-dependent PQ reduction by Fd.

**DISCUSSION**

From the first discovery of PSI cyclic electron transport, the idea that Fd directly donates electrons to the cyt bcf complex has been widely discussed. In early publications this idea was partly based on the finding that PGR5-dependent PSI cyclic electron transport was sensitive to antimycin A (8, 21). Antimycin A binds to the Q site of the cyt bc1 complex and inhibits the donation of electrons to quinone (22). Because antimycin A inhibits PGR5-dependent PSI cyclic electron transport, it was plausible that Fd directly donates electrons to the cyt bcf complex and subsequently to PQ via the Q-cycle at the Qb site in PSI cyclic electron transport. However, it was subsequently revealed that antimycin A did not impair the Q-cycle in the cyt bcf complex (23). Thus antimycin A somehow specifically inhibits PSI cyclic electron transport rather than pleiotropically via inhibition of the Q-cycle (1, 24). We are still not sure how antimycin A inhibits PGR5-dependent PSI cyclic electron transport, and the route of electron flow from Fd to PQ in the PSI cyclic electron transport must be seriously reconsidered.

To assess the possibility that Fd directly donates electrons to the cyt bcf complex, we characterized the activity of PGR5-dependent PQ reduction in pgr1, in which the Q-cycle is conditionally impaired. Neither in vitro (Figs. 3 and 4) nor in vivo assays (Figs. 5 and 6) showed any evidence that the cyt bcf complex is involved in PGR5-dependent PQ reduction. The most straightforward explanation for these results is that Fd directly donates electrons to PQ (Fig. 1, route 2) and not to the cyt bcf complex. For this model, a putative enzyme, FQR, must be hypothesized (8), but it has not been characterized thus far. PGR5 is a thylakoid membrane protein essential for PQ reduction and may provide a clue as to the identity of FQR. However, PGR5 is unlikely to bind any prosthetic group, suggesting that other proteins are directly involved in electron transport and that PGR5 is an accessory protein of the FQR complex.

It is also still possible that the pgr1 mutation did not affect electron transport from Fd to PQ through the cyt bcf complex, even though the Q-cycle was severely impaired. Both hemes, bH and x, may be required for reduction of PQ without electrons passing through the cyt bcf complex in the PGR5-dependent PSI cyclic electron transport. This idea would explain why photosynthetic systems contain heme x, as it would be required to gate the two electron transfer reactions. Fd may reduce two hemes at the Qb center, and these may then reduce PQ. Our results on pgr5 (1) are not necessarily inconsistent with this
idea. PGR5 may be a factor facilitating the access of Fd to the cyt b6f complex. However, PGR5 is stable even in the mutant background completely lacking the cyt b6f complex, suggesting that PGR5 is not a subunit of the cyt b6f complex (1).

The classical view that Fd donates electrons to the cyt b6f complex was at least partly based on the idea that antimycin A inhibited the reduction of PQ at the Q site in the cyt b6f complex, which was found to be incorrect. However, the recent discovery of heme x (heme c1) made us reconsider this possibility. Heme x may provide clarification of the route of PGR5-dependent PQ reduction, especially in light of suggestions that heme x is involved in electron transfer from Fd to PQ (12, 13, 25). Unfortunately, in Chlamydomonas reinhardtii, site-directed mutagenesis of cytochrome b6, which altered cysteine635 to valine in the covalent link to heme x, generated a mutant in which the cyt b6f complex could not be assembled (26). At present, there seems to be no evidence suggesting that heme x is involved in direct electron donation from Fd to the cyt b6f complex. Taken together with the results from pgr1, we prefer the idea that Fd directly donates electrons to PQ as a working model (Fig. 1, route 2).

Regardless of whether Fd donates electrons to the cyt b6f complex or PQ in PGR5-dependent PSI cyclic electron transport, PQH2 is reoxidized at the Qb site of the cyt b6f complex, the reaction that is shared with direct electron transport. However, we could not find any alteration in \( \Phi_{\text{PSII}}/\Phi_{\text{PSI}} \) in pgr1 except for the unexpected high ratio at low light intensity (Fig. 6), indicating that the ratio of linear and PSI cyclic electron transport was not significantly disturbed. Although we could not eliminate the possibility that the high-level reduction of the PQ pool during photosynthesis would have modified the ratio slightly in pgr1, the effect was rather minor. In contrast, \( \Phi_{\text{PSII}}/\Phi_{\text{PSI}} \) was significantly reduced in pgr5. These results indicate that pgr1 is defective in the route of electron transfer shared by linear and PSI cyclic electron transport, that is, in the Q-cycle. In contrast, the PSI cyclic pathway is specifically impaired in pgr5, resulting in indirect effects on linear electron transport because of over-reduction of the stroma. This conclusion is consistent with the observation that there was no synergistic effect of the crr2 defect on pgr1 (Fig. 5) and also in vitro assays (Figs. 3 and 4).

In contrast to pgr5, the balance of PSI cyclic and linear electron transport was not significantly affected in pgr1 (Fig. 6C). We consider that the ATP/NADPH production ratio is not drastically disturbed in pgr1, although the production rate of ATP and NADPH was decreased at high light intensity by the impaired Q-cycle activity. This idea is consistent with the fact that P700 was slightly more oxidized in pgr1 during photosynthesis (14), the change in oxidation level being explained by the limitation of electron transport at the cyt b6f complex. A similar phenotype was observed in pgr3 (27), in which the level of the cyt b6f complex was reduced, and also in paa1 (28), which has dramatically lowered levels of plastocyanin. In contrast, when the ATP production ratio is decreased, depletion of NADPH+ causes reduction of P700+ due to charge recombination, as was observed in pgr5 (1). The sensitivity of PSI to high light intensity in pgr5 can be ascribed to this over-reduction of the stroma. pgr1 and pgr5 have been shown to exhibit similar phenotypes in terms of chlorophyll fluorescence, lack of NPF induction, and specific reduction in ETR level at high light intensity. However, pgr5 is much more sensitive to excessive light stresses because of the defect in preventing stromal over-reduction, and its phenotype is drastically enhanced under the crr2 mutant background (2). We consider that there is a strong requirement for plants to maintain the balance of linear and PSI cyclic electron transport.

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