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Mechanism of electron transfer in the cytochrome *b/f* complex of algae: Evidence for a semiquinone cycle

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ABSTRACT **The most widely accepted mechanism of electron and proton transfer within the cytochrome (Cyt) *b/f* complex derives from the Q-cycle hypothesis originally proposed for the mitochondrial Cyt b/c_1 complex by Mitchell [Mitchell, P. (1975) *FEBS Lett.* 57, 135–137]. In chloroplasts, the Cyt *b/f* complex catalyzes the oxidation of a plastoquinol at a site, Q_o (the plastoquinol binding site), close to the inner aqueous phase and the reduction of a quinone at a site, Q_i (the plastoquinone binding site), close to the stromal side of the membrane. In an alternative model, the semiquinone cycle [Wikström, M. & Krab, K. (1986) *J. Bioenerg. Biomembr.* 18, 181–193], a charged semiquinone formed at site Q_o is transferred to site Q_i where it is reduced into quinol. Flash-induced kinetics of the redox changes of Cyt *b* and of the formation of a transmembrane potential have been measured in *Chlorella sorokiniana* cells incubated in reducing conditions that induce a full reduction of the plastoquinone pool. The experiments were performed in the presence of an uncoupler that collapses the permanent electrochemical proton gradient and thus accelerates the rate of the electrogenic processes. The results show that the electrogenic reaction driven by the Cyt *b/f* complex precedes the processes of reduction or oxidation of the *b*-hemes. This electrogenic process is probably due to a transmembrane movement of a charged semiquinone, in agreement with the semiquinone-cycle hypothesis. This mechanism may represent an adaptation to reducing conditions when no oxidized quinone is available at the Q_i site.**

The cytochrome (Cyt) *b/f* complex mediates the electron transfer reactions between photosystem (PS) II and PS I reaction centers in the thylakoid membrane of chloroplasts. It catalyzes the oxidation of plastoquinol and the reduction of plastocyanin. The electron transfer within the Cyt *b/f* complex induces proton pumping and the formation of a membrane potential occurring in the millisecond range (for a review, see ref. 1). Two electron-transfer chains operate within the complex: the high-potential chain is located close to the lumenal aqueous phase and includes an iron–sulfur protein (FeS) and Cyt *f* ($E_{m,7} \approx 340$ mV); the low-potential chain, in transmembrane position, consists of two hemes: Cyt b_h and Cyt b_l of midpoint potential −50 and −150 mV, respectively (2). Two sites, Q_o (the plastoquinol binding site) and Q_i (the plastoquinone binding site), located close to the lumenal and stromal faces of the membrane are able to bind quinol and quinone molecules, respectively. For a unicellular algae, such as *Chlorella* and *Chlamydomonas*, a fifth electron carrier, G (3), in equilibrium with the Cyt *b* chain, is located on the stromal side of the complex and connected to the low-potential chain. G is a hemoprotein with a reduced minus oxidized difference spectrum that displays a negative peak at 424 nm and a broad weak band in the green region. Its midpoint potential was estimated to be ≈20 mV higher than that of Cyt b_h (4). The equilibrium constant between G and Cyt b_h depends upon the value of the membrane potential that induces the oxidation of G and the reduction of Cyt b_h (3). In the most oxidized state of the low-potential chain that we were able to achieve in living cells, G and Cyt b_h in the same Cyt *b/f* complex share one positive charge (Scheme I, equilibrium 1). This suggests that electrostatic interactions rend difficult the oxidation of both G and Cyt b_h belonging to the same Cyt *b/f* complex. G is probably involved in a cyclic electron transfer around PS I.

The mechanism of the Q cycle (5), as modified by Crofts *et al.* (6), is widely accepted as a description of the process of electron and proton transfer in the Cyt *b/f* complex. This process is initiated by a charge separation in PS I that leads to the transfer of a positive charge to the high-potential chain via plastocyanin. The oxidation of a quinol occurs at site Q_o associated with the concerted transfer of electrons toward the high- and low-potential chains, release of quinone to the pool, and release of two protons into the lumen (reaction 2). The transfer of a second positive charge to the high-potential chain (reaction 3) induces the oxidation of a second quinol through a second concerted process (reaction 4), which leads to the reduction of Cyt b_l. Quinone from the pool (or formed at site Q_o) is transferred to site Q_i (reaction 5) where it induces the oxidation of both *b*-hemes (reaction 6). An alternative model, the semiquinone cycle, has been proposed by Wikström and Krab (7). The Q cycle and semiquinone cycle have in common the concerted reaction that leads to the reduction of Cyt b_h (reaction 2). In the semiquinone cycle, however, the transfer of a second positive charge to the high-potential chain results in the formation of a semiquinone anion (PQ^-) at site Q_o (reaction 4′), which is then transferred to site Q_i (reaction 5′) and reduced by Cyt b_h (reaction 6′).

In the Q cycle, the electrogenic steps associated with the turnover of Cyt *b/f* complex are exclusively linked to the electron-transfer reactions through the low-potential chain. In contrast, in the semiquinone cycle, a different electrogenic process is also involved that corresponds to the transfer of PQ^- from site Q_o to site Q_i. We have reported (4, 8) experiments performed under reducing conditions that led us to favor the semiquinone cycle. The present experiments have been done in similar conditions, focusing on the different kinetics observed for the electrogenic process and electron transfer in the low-potential chain.

MATERIALS AND METHODS

The experiments were performed with PS II-lacking mutant strains of *Chlorella sorokiniana* (S8 and S52), isolated by Bennoun and coworkers (9). S52 is a double mutant lacking a large part of the chlorophyll antenna pigments. The algae were suspended in 50 mM phosphate buffer, pH 7.2/10% (wt/wt) Ficoll. Carbonylcyanide 4-trifluoromethoxyphenylhydrazone (FCCP, 5 μM) was added in all experiments

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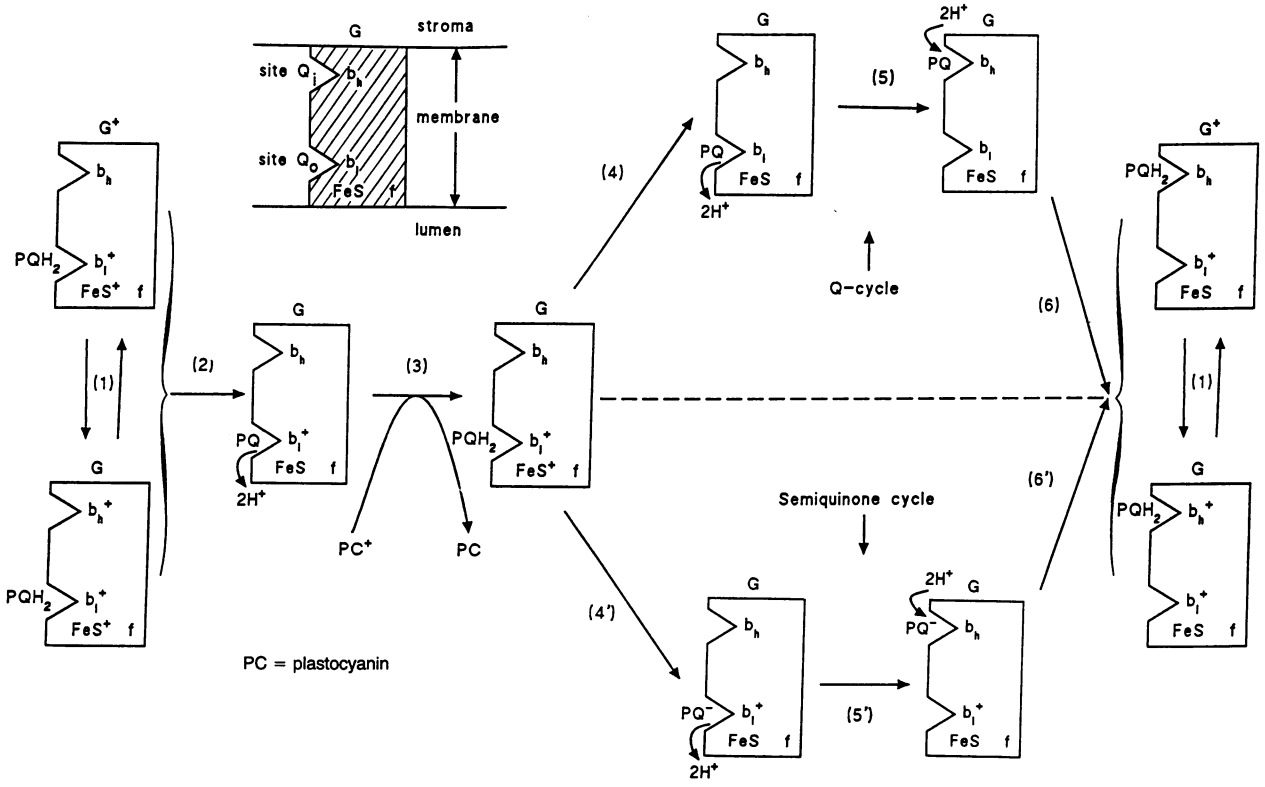
Abbreviations: Cyt, cytochrome; PS, photosystem; PQ, plastosemiquinone; FCCP, carbonylcyanide 4-trifluoromethoxyphenylhydrazone; NQNO, 2-*n*-nonyl-4-hydroxyquinoline 1-oxide.



Scheme I

except those shown in Fig. 1. Spectrophotometric measurements were performed with an apparatus similar to that described in ref. 10. The algae in the measuring cuvette (0.4 ml) can be rapidly exchanged with those of a larger reservoir (≈40 ml) maintained under anaerobic conditions by an argon flux. This device allows repetitive experiments (9-s interval) with fully dark-adapted cells. FCCP, which is slowly consumed in the reservoir, was repeatedly added to maintain a constant decay of the membrane potential. Actinic excitation was provided by a xenon flash (3-μs duration at half-height) filtered through red filters (Schott RG665, Kodak Wratten 97). The membrane potential was measured by the electrochromic shift as the difference $\Delta I/I$ (515 nm − 494 nm), which yields a linear response with respect to the membrane potential (11). In the presence of saturating concentrations of inhibitors such as the dinitrophenyl ether of iodonitrothymol or stigmatellin, which block the electron transfer at site Q_o, the fast absorption change (phase a, <100 μs) associated with the PS I charge separation is followed by a rise phase ($t_{1/2} \approx$ 170 μs), the amplitude of which is 4.7% of phase a. This signal is not an electrochromic shift and was subtracted from all membrane potential measurements. The slow electrogenic phase (phase b) has been corrected for the decay of the membrane potential. In the presence of 5 μM FCCP, the membrane potential decays according to a first-order kinetics ($t_{1/2} \approx$ 50 ms), and we determined its rate constant for each experiment. Corrected kinetics of phase b was then computed assuming that the rate of the decay of the membrane

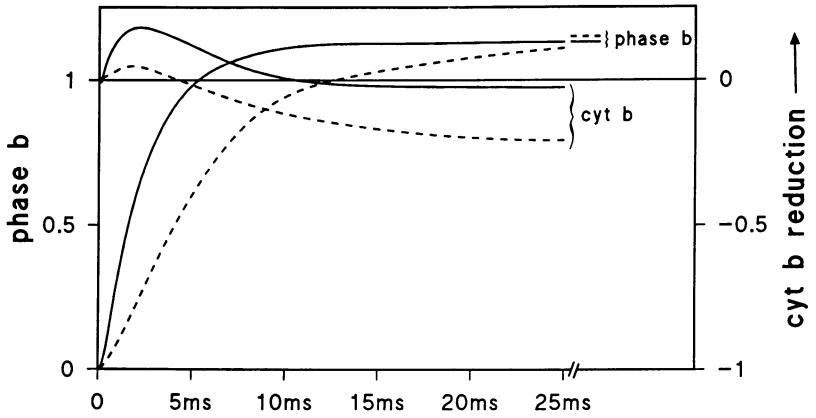


FIG. 1. Effect of an uncoupler on the kinetics of phase b and of Cyt *b* redox changes induced by nonsaturating repetitive flashes (1.8 s apart). The flashes hit 33% of PS I reaction centers. Dashed lines, 20 mM glucose; solid lines, 5 μM FCCP. The left ordinate shows the amplitude of the membrane potential associated with the electron transfer reactions within the Cyt *b*/*f* complex divided by the amplitude of phase a. Phase b was corrected for the decay of the membrane potential ($t_{1/2} \approx$ 300 ms or ≈ 50 ms in the absence or the presence of uncoupler, respectively). The right ordinate shows the amplitude of the redox changes of Cyt *b* expressed as the amount of reduced or oxidized Cyt *b* divided by the amount of positive charges formed by PS I reaction.

potential was linearly related to the actual value of the membrane potential. Cyt *b* redox changes were measured as the difference $\Delta I/I$ (564 nm − 573 nm) with ε = 18.7 $mM^{-1}\cdot cm^{-1}$; at these wavelengths, the contribution of Cyt *f* is negligible; the signal was corrected for a small contribution of the electrochromic shift [2.5% of $\Delta I/I$ (515 nm − 494 nm)]. Redox changes of Cyt *f* were measured as the difference between the absorption at 554 nm and a baseline was drawn between 545 nm and 572 nm with ε = 19.2 $mM^{-1}\cdot cm^{-1}$. The total concentration of Cyt *f* was measured in the presence of the dinitrophenyl ether of iodonitrothymol at 30 μM in algae submitted to a strong continuous illumination that induces the full oxidation of primary and secondary PS I donors. On this basis, the concentration of the Cyt *b/f* complexes was estimated to 70% of that of PS I reaction centers, measured as the difference $\Delta I/I$ (702 nm − 729 nm) with ε = 68 $mM^{-1}\cdot cm^{-1}$. The difference between the absorption peaks of Cyt b_h and Cyt b_l (0.5 nm) is too small to spectrally separate these two components. The low equilibrium constant between G and Cyt b_h provides a way to discriminate between redox changes involving specifically Cyt b_h rather than Cyt b_l and is discussed in *Results and Discussion*.

RESULTS AND DISCUSSION

The flash-induced membrane potential rise observed in algae displays two well-separated phases: a fast phase (phase a) is associated with the photochemical charge separation; a subsequent slow phase (phase b) corresponds to a transmembrane charge transfer related to the turnover of the Cyt *b/f* complex (12). In algal mutant strains lacking PS II, the amplitude of phase a, measured 100 μs after the flash, is proportional to the number of positive charges appearing on the donor side of PS I. In Figs. 1–4, the amplitude of phase b has been normalized to phase a. In the same way, the amount of oxidized or reduced Cyt *b* has been divided by the number of positive charges formed by PS I charge separation. In all the experiments described in this paper, the algae were incubated for >30 min under anaerobic conditions, which causes a full reduction of the plastoquinone pool. The experiments shown in Figs. 1–3 were performed using nonsaturating flashes hitting 33% of the reaction centers to minimize the occurrence of double turnovers that would have complicated the interpretation of the kinetic data.

In Fig. 1, the kinetics of phase b and of the redox changes of Cyt *b*, induced by repetitive nonsaturating flashes, were measured in the presence or absence of an uncoupler. We showed previously that, after dark adaptation of *Chlorella* cells to anaerobic conditions, the hydrolysis of ATP generates a large electrochemical proton gradient across the membrane (11). ATP is formed by a fermentation process that is stimulated by the addition of glucose. In the absence of an uncoupler, phase b occurs with $t_{1/2} \approx 5$ ms and corresponds to the transfer of 1.15 charges across the membrane per PS I reaction. This large electrogenic phase is accompanied by minor redox changes of Cyt *b*. The addition of an uncoupler that collapses the permanent proton gradient dramatically accelerates phase b ($t_{1/2} \approx 1.75$ ms) with no significant change in its amplitude, as reported by Bouges-Bocquet (13). This acceleration is due to an increase of the rate of PQ oxidation at site Q_o. Both the initial rate and the amplitude of the transient reduction of Cyt *b* are increased. Increasing the concentration of FCCP beyond 5 μM induced no further acceleration of phase b, which suggests that this concentration is sufficient to fully collapse the permanent proton gradient. The addition of dicyclohexyl-18-crown-6, instead of FCCP, led to similar effects on both phase b and Cyt *b* kinetics (data not shown). All subsequent experiments were performed in the presence of 5 μM FCCP—i.e., under conditions where the electrogenic phase largely precedes the oxidation of Cyt *b*.

In Figs. 2 and 3 *A–C*, the kinetics of phase b and of Cyt *b* changes were measured for different initial redox states of Cyt *b*.

For conditions A (Figs. 2 and 3*A*), algae were dark-adapted in the presence of a low concentration of a redox mediator (1 μM safranine T, $E_{m,7}$ = −289 mV). The addition of safranine induced in ≈1 h a full reduction of both *b*-hemes. No change in the final redox level of the *b*-hemes was observed upon further addition of 15 μM sodium dithionite (data not shown). We checked that after addition of sodium dithionite, the redox potential of the medium was lower than −350 mV, indicating that reduced sodium dithionite was in excess. Under these conditions, a Q cycle, which requires the presence of an oxidized Cyt b_l, cannot operate. In contrast, reactions 4′, 5′, and 6′ in the semiquinone cycle can operate regardless of whether one or both Cyts *b* are reduced.

For conditions B (Figs. 2 and 3*B*), algae were dark-adapted in the absence of mediator. The fraction of oxidized Cyt *b* per complex was determined from the difference between the absorption spectra recorded in the dark under conditions B and A in the 545-nm to 573-nm range. This difference displays an oxidized minus reduced Cyt *b* spectrum, the amplitude of which was normalized to the maximum Cyt *f* signal. This spectrum shows no contribution of oxidized G; this suggests that only Cyt b_l (not in equilibrium with G) is oxidized. Besides, we measured under these conditions ≈1.45 reduced Cyt *b* per complex. One thus expects that 45% of the

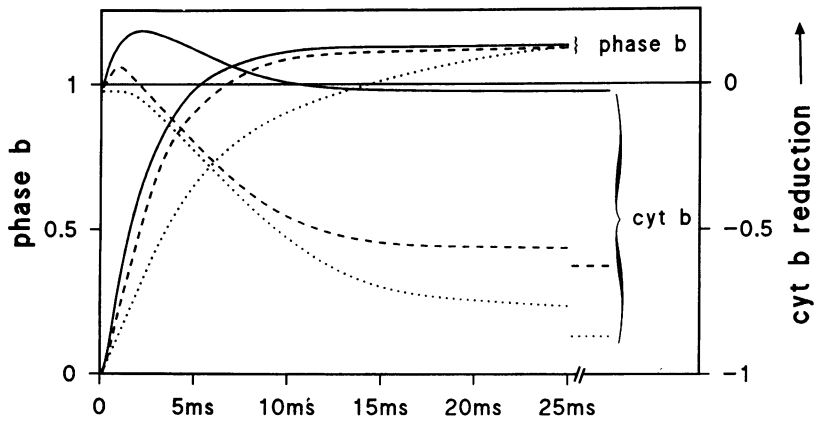


FIG. 2. Effect of the initial redox state of the low-potential chain on the kinetics of phase b and of redox changes of Cyt *b* at 5 μM FCCP. Same ordinates and same flash energy as in Fig. 1. Dotted lines, 1 μM safranine T and algae dark-adapted for >2 h; dashed lines, no mediator and algae dark-adapted for >30 min; solid lines, same experiment as in Fig. 1 (solid lines). The data points illustrate conditions A–C and are shown in Fig. 3 *A–C*.

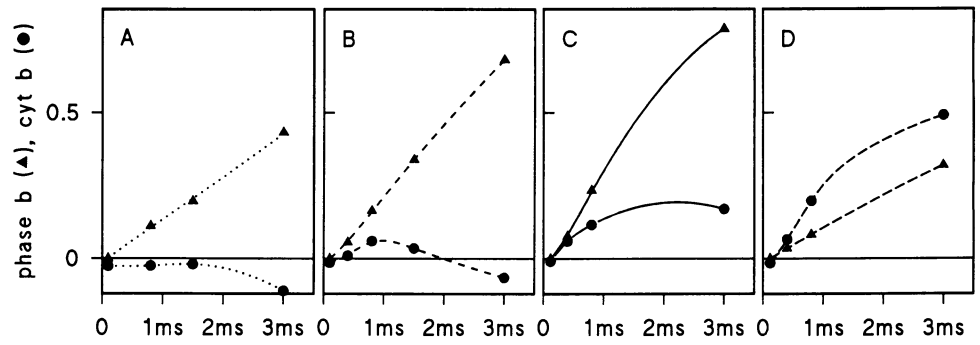


FIG. 3. (*A–C*) Same experiments as in Fig. 2, conditions A–C, respectively, on an expanded time scale. (*D*) 2-*n*-Nonyl-4-hydroxyquinoline 1-oxide (NQNO; 2 μM). Repetitive flashes were 1.8 s apart. The maximum level of Cyt *b* reduction (0.65) is reached in 5 ms. The small negative signal observed 100 μs after the flash in the Cyt *b* kinetics is due to oxidized plastocyanin.

complexes have both *b*-hemes reduced and 55% have Cyt b_l oxidized and Cyt b_h reduced. For this last fraction of the complexes, a Q cycle could in principle operate; the electron transfer within the Cyt *b/f* complex would then start by a concerted process leading to the reduction of Cyt b_l (reaction 4). The half-time of this reaction is ≈1.2 ms (8). On the basis of experiments performed with bacterial Cyt b/c_1 complex similar in structure to Cyt *b/f*, Glaser and Crofts (14) and Robertson and Dutton (15) concluded that the reduction of Cyt b_l is not electrogenic. The concerted reaction would be followed by the transfer of PQ from site Q_o to site Q_i and then by the oxidation of both *b*-hemes (reaction 6). Even if PQ were instantaneously transferred from site Q_o to site Q_i, reaction 6—which is the only electrogenic process—would present a lag determined by the time course of reaction (4). In contradiction with the Q-cycle hypothesis, we actually observed a lag phase in the Cyt *b* oxidation but not in phase b (see Figs. 2 and 3*B*).

For conditions C (Figs. 2 and 3*C*), algae were illuminated by repetitive nonsaturating flashes given 1.8 s apart. We estimated (from spectral measurements similar to those outlined above) that, beyond the third exciting flash, 75% of the complexes contain Cyt b_l oxidized and Cyt b_h reduced and 25% contain both *b*-hemes oxidized. According to Robertson and Dutton (15), the reduction of Cyt b_h generates a membrane potential that is 60% of the potential corresponding to the transfer of one electron across the membrane. If a Q cycle were operating, the ratio between the initial rate of phase b and of Cyt *b* reduction should be $R = 0.25 \times 0.6 = 0.15$, much lower than the measured value, which is ≈1.2.

The experiments performed in conditions A–C show that, regardless of the initial redox state of the low-potential chain, an electrogenic process occurs in addition to that associated with the reduction of the *b*-hemes. The occurrence of this additional electrogenic process is predicted by a semiquinone cycle (reaction 5′). The half-time of phase b depends upon the initial redox state of Cyt *b* (4.1, 2.3, and 1.8 ms for conditions A–C, respectively).

In Fig. 3*D*, algae were submitted to repetitive nonsaturating flashes 1.8 s apart, in the presence of a saturating concentration of NQNO, an inhibitor of site Q_i (16). The addition of NQNO slightly stimulates the initial rate of Cyt *b* reduction while it inhibits phase b by ≈60%. In a semiquinone cycle, we expect that the addition of NQNO, which binds to the Q_i site, will prevent the transmembrane movement of PQ^-. Therefore, the electrogenic phase should be exclusively associated with the reduction of Cyt *b* by the concerted reaction. The low value of *R* (0.36) is compatible with this hypothesis.

In Fig. 4 absorption changes in the blue region of the spectrum are shown. To improve the signal-to-noise ratio, these experiments were performed with mutant S52, which lacks a major fraction of the light-harvesting complexes. Spectrum 1 was obtained upon illumination by a single nonsaturating flash given in conditions where both *b*-hemes are in their reduced form. This spectrum is mainly an oxidized minus reduced spectrum of Cyt *b*. Only a minor absorption change is observed at 424 nm, which implies that, under these conditions, the oxidation of Cyt *b* is not associated with a significant oxidation of carrier G. Spectrum 2 was obtained using dark-adapted material in the absence of mediator [i.e., under conditions (B) where a fraction of Cyt b_l is oxidized prior to the flash]. This spectrum displays a large negative signal at 424 nm and corresponds to the superimposition of the oxidized minus reduced spectra of Cyt *b* and G (4).

In Table 1, the relative amounts of oxidized G and Cyt *b* have been estimated as a function of the flash energy under conditions A, where both *b*-hemes are initially reduced. These amounts are proportional to the absorption changes at 425 nm and 435 nm, isobestic points for Cyt *b* and G, respectively. At these wavelengths, the extinction coefficients of G and Cyt *b* are close to each other (4). The fraction of oxidized G increases with the energy of the flash and is negligible for the weakest intensity. By taking into account that the midpoint potentials of Cyt b_h and G are close (Scheme I, equilibrium 1) and that there is a large gap (≈90

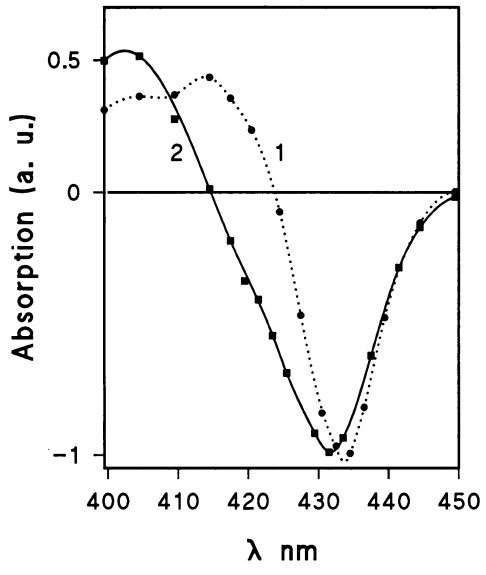


FIG. 4. Flash-induced difference spectra using S52 mutant strain. The flash hit ≈40% of PS I reaction centers. The absorption was measured 200 ms after a single exciting flash and 5 μM FCCP. Spectra: 1, 1 μM safranine T and algae dark-adapted for >2 h; 2, no mediator. Note that the amount of oxidized Cyt *b* is not the same in spectra 1 and 2. a.u., Arbitrary unit(s).

Table 1. Relative amounts of G and Cyt *b* oxidized by flashes of various energies

		$\Delta I/I$ ($\times 10^6$)		
	A	G^+	Cyt b^+	G^+/Cyt b^+
Safranine T (0.8 μM)	1	780	4433	0.18
	0.42	179	2024	0.09
	0.18	20	893	0.02
No safranine T	0.42	526	1563	0.34

Dark-adapted S52 mutant and 5 μM FCCP were used. The amounts of G^+ and Cyt b^+ are expressed by the $\Delta I/I$ ($\times 10^6$), measured 900 ms after the exciting flash at 425 nm and 435 nm, respectively. *A* is the fraction of PS I centers that undergoes a charge separation. For a saturating flash, $A = 1$. In the presence of safranine T, the amounts of G^+ and Cyt b^+ are constant from 500 ms to 2 s.

mV) between midpoint potentials of Cyt b_h and Cyt b_l (2), one can conclude that the weakest flash predominantly induced the oxidation of Cyt b_l. Flashes of higher energy induce a partial oxidation of G and Cyt b_h in addition to the oxidation of Cyt b_l, due to the occurrence of double turnovers at sites Q_o and Q_i. As expected, in conditions where a fraction of Cyt b_l is oxidized prior to the flash, illumination by a weak flash induces a partial oxidation of both Cyt b_h and G (Fig. 4, spectrum 2, and Table 1).

It is worth pointing out that in the Q-cycle framework, one can envisage several mechanisms that would lead to the formation of a quinone at site Q_o under conditions where both *b*-hemes are reduced prior to illumination. Among the possible hypotheses is the sequential transfer of two positive charges to the same high-potential chain, which could then lead to the formation of a quinone at site Q_o. A disproportionation involving two semiquinones formed at the Q_o site of two complexes belonging to a functional dimer would also lead to the formation of a quinone. The transfer of this quinone at site Q_i would then induce the oxidation of both Cyt b_h and Cyt b_l. These hypotheses can be excluded since we have established that weak flash excitation induces the oxidation of Cyt b_l exclusively (one-electron process). In the same line of evidence, we observed that the normalized amplitude of phase b increases while its half-time decreases when the energy of the flash is decreased. A maximum amplitude (1.5) is reached for flashes hitting ≈10% of PS I centers. This result excludes any hypothesis in which cooperation is required between several positive charges.

CONCLUSION

The experiments presented above have led us to conclude that electron transfer within the Cyt *b/f* complex operates according to a semiquinone cycle, at least in conditions where the plastoquinone pool is fully reduced. We have proposed (17) that PQ^- has a larger affinity for the Q_i than for the Q_o site. This would provide a driving force for the rapid transfer of PQ^- from Q_o to Q_i. We have also suggested (17) that plastoquinone is not an efficient electron acceptor at site Q_i, which implies that the semiquinone cycle may also operate under oxidizing conditions.

According to a semiquinone cycle, the normalized amplitude of phase b in highly reducing conditions should be close to 2 (transfer of PQ^- from the Q_o to the Q_i site plus transfer of one electron from Cyt b_l to site Q_i plus transfer of two protons from the outer face of the membrane to site Q_i), while the maximum value under weak-flash excitation is 1.55. We also observed that the initial slope of phase b is 1.7 times lower under conditions A than under conditions B. These anomalies could be explained if Cyt b_l in its reduced form makes the electrostatic environment of site Q_o more negative, which would stabilize the protonated form of semiquinone ($PQH^\cdot$). If a part of the semiquinones moves across the membrane as a neutral species, the rate and the yield of the electrogenic reaction and of proton pumping would be decreased.

A critical point in the semiquinone cycle is the mechanism by which a charged species such as PQ^- can move rapidly in an environment of low dielectric constant. For thermodynamic reasons, it is unlikely that PQ^- is released in the lipid phase as a free-diffusing species and we favor a model in which the semiquinone remains trapped in the Cyt *b/f* complex. Polarizable or positively charged amino acid residues on the edge or within the Cyt *b/f* complex ["Q-pocket" (7)] would stabilize PQ^- and facilitate its transfer from the Q_o to the Q_i site by increasing the local dielectric constant. Moreover, one cannot exclude that these two sites are close to each other, which would also facilitate the transfer of PQ^-.

The semiquinone cycle appears especially well adapted to operate under anaerobic conditions in a cyclic pathway around PS I, since it does not require the presence of an oxidized quinone at the Q_i site. It is worth pointing out that a Cyt *b/f* complex was recently identified in a strictly anoxygenic green photosynthetic bacterium, *Heliobacterium* (18). On the other hand, Cyt b/c_1 complex in mitochondria or purple bacteria could operate according to a Q cycle, better suited to conditions where a fraction of the quinone pool is oxidized.

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