The cytochrome bc_1 complex of *Rhodobacter capsulatus*: ubiquinol oxidation in a dimeric Q-cycle?

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Abstract We studied the cytochrome bc_1 complex (hereafter *bc*) by flash excitation of *Rhodobacter capsulatus* chromatophores. The reduction of the high-potential heme b_h of cytochrome *b* (at 561 nm) and of cytochromes *c* (at 552 nm) and the electrochromic absorption transients (at 524 nm) were monitored after the first and second flashes of light, respectively. We kept the ubiquinone pool oxidized in the dark and concerned for the ubiquinol formation in the photosynthetic reaction center only after the second flash. Surprisingly, the first flash caused the oxidation of about one ubiquinol per *bc* dimer. Based on these and other data we propose a dimeric Q-cycle where the energetically unfavorable oxidation of the first ubiquinol molecule by one of the *bc* monomers is driven by the energetically favorable oxidation of the second ubiquinol by the other *bc* monomer resulting in a pairwise oxidation of ubiquinol molecules by the dimeric *bc* in the dark. The residual unpaired ubiquinol supposedly remains on the enzyme and is then oxidized after the first flash.

Key words: Ubiquinone; Cytochrome bc_1 complex; Electrogenesis; Proton transfer; Electron transfer; *Rhodobacter capsulatus*

1. Introduction

Cytochrome bc_1 complexes of animals and bacteria (hereafter denoted *bc*) and cytochrome *bf* complexes of chloroplasts are membrane quinol:cytochrome *c* oxidoreductases (see [1–4] for reviews). Mitchell described their operation by a so-called Q-cycle [5]. In this concept, one out of two electrons which are released upon quinol oxidation is transferred to cyt *c* (located at the positively charged *p*-side of the membrane), whereas the second electron crosses the membrane and reduces another quinone close to the negatively charged *n*-side of the membrane. As a result, *bc* pumps protons from the *n*-side of the membrane to the *p*-side with a stoichiometry of $2H^+/e^-$ and generates the electrochemical potential difference of the proton ($\Delta\mu H^+$) with a chemical (ΔpH) and an electrical ($\Delta\psi$) component.

The *bc* of the phototrophic bacteria *Rhodobacter sphaeroides* and *Rhodobacter capsulatus* is involved in the cyclic electron transfer around the photosynthetic reaction center (RC). Upon illumination, the RC reduces ubiquinone to ubiquinol and oxidizes a water-soluble cyt c_2 that is reduced by *bc*. Studies of the operation of *bc* in response to short light flashes led to modifications of the original Q-cycle concept [6–9]. It has been suggested that two ubiquinol molecules are to be oxidized by the quinone-oxidizing center *P* to complete the turnover of *bc*: the oxidation of the first ubiquinol molecule yields a semiquinone Q_N^- in the quinone reducing center *N* and only the oxidation of the second ubiquinol molecule finally produces Q_NH_2 [6]. In the above cited works *bc* was assumed to function as a *monomer*. Two recent crystal structures of the mitochondrial *bc* [10,11] show a *dimer* which is formed from two intercalated transmembrane cyt *b* subunits each carrying one high-potential and one low-potential protoheme (b_h and b_l, respectively). The cyt c_1 subunit and the iron-sulfur Rieske protein (hereafter FeS) are attached to their respective cyt *b* subunit from the *p*-side. With specific inhibitors the quinone binding sites have been mapped: myxothiazol, stigmatellin and UHDBT bind, somewhat differently, between b_l and FeS close to the *p*-side of the membrane (center *P*), whereas antimycin A was found next to b_h, closer to the *n*-side (center *N*).

We have shown in [8] that $\Delta\psi$ generation in *bc* slowed down drastically when less than one ubiquinol per *bc* was oxidized (weak flashes were compared with saturating ones). Using a different approach in this work, we varied the amount of ubiquinol by measuring the reactions in *bc* after the first and second flashes, respectively, under conditions where the membrane ubiquinone pool was oxidized and ubiquinol was formed by the RC only after the second flash. We interpret our data in terms of a dimeric Q-cycle where the energetically unfavorable oxidation of the first ubiquinol by one of the *bc* monomers (yielding Q_N^-) is driven by the energetically favorable oxidation of the second ubiquinol molecule by another *bc* monomer (yielding Q_NH_2). As a result ubiquinol molecules are oxidized by *bc* in pairs.

2. Materials and methods

2.1. Materials

Chromatophores were isolated from the cells of *Rhodobacter capsulatus* (wild type, strain B10) grown photoheterotrophically at +30°C under high light intensity in the RCV medium [12] and disrupted by Ribi-press treatment (12000 psi) as described in [13].

2.2. Spectrophotometry

Spectrophotometric measurements were performed with the setup described in [14]. The time resolution of the setup was 1 μs. Saturating exiting flashes were provided by a xenon flash-lamp (~4 μs FWHM, Schott RG 780 nm filter). The binary oscillations of Q_B^- were monitored at 450–480 nm (to account for the contribution of electrochromic carotenoid bandshift). The voltage transients at

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Abbreviations: cyt, cytochrome; *bc*, cytochrome *bc* complex; b_l, low potential cyt b_{566} of *bc*; b_h, high potential cyt b_{561} of bc; Bchl, bacteriochlorophyll; Q, ubiquinone; QH_2, ubiquinol; Q^-, semiubiquinone anion radical; RC, photosynthetic reaction center; Q_B, secondary quinone acceptor of RC; $E_{m,8}$, electrochemical midpoint potential at pH 8.0; TMPD, *N*,*N*,*N*′,*N*′-tetramethyl-*p*-phenylenediamine; UHDBT, 5-undecyl-6-hydroxy-4,7-dioxobenzothiazole

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the chromatophore membrane were monitored by the electrochromic bandshifts of carotenoids at 524 nm. The flash-induced absorption transients of the total cyt c (c_t) at 552 nm and of b_h at 561 nm were obtained as $\Delta A_{552-570} = \Delta A_{552} - 1.42 \times \Delta A_{570}$ and $\Delta A_{561-570} = \Delta A_{561} - 1.08 \times \Delta A_{570} - 0.07 \times \Delta A_{552-570}$, respectively (the signal at 570 nm was subtracted to account for the contribution of oxidized primary donor, P^+, whereas the $\Delta A_{552-570}$ difference was subtracted to account for the small contribution of c_t at 561 nm). The concentration of Bchl in the sample was determined according to [15]. The amount of functionally active RC and *bc* was estimated as in [16]. In our preparations, the RC:*bc* monomer ratio was about 3 and the Bchl/RC ratio was 70–90. 2 mM KCN was routinely present to prevent the oxidation of the redox-buffering system through terminal oxidase (E_h of about 250 mV was usually established in the open-air cuvette). 1 μM oligomycin was added to block the proton flux through the membrane F_1F_0 ATPase and to slow down the decay of $\Delta\psi$. The dark adaptation time between two flash series was 4 min.

2.3. Kinetics

The kinetic traces were analyzed using the GIM software package kindly provided by A.L. Drachev and the Microcal Origin 4.1 software package (Microcal Software, Inc., USA).

3. Results

Fig. 1A shows that Q_B^-, the semiquinone of the secondary electron acceptor of RC, was formed after the odd-numbered flashes and disappeared after the even ones by being reduced to ubiquinol Q_BH_2. The life time of Q_B^- can be varied by addition of external electron acceptors [17]. In this work we were interested to avoid ubiquinol formation after the first flash. Hence we adjusted the concentration of methylene blue (MB, $E_{m,8} \sim -50$ mV) at 20 μM (used throughout the work) to oxidize Q_B^- with a half-life of ~5 s (see Fig. 1B). The oxidation of Q_B^- by MB in the time interval between the flashes explains the strong damping of the binary pattern in Fig. 1A. The pattern recovered already after 1 min of dark adaptation. With 4 min of dark adaptation between the flash series the concentration of Q_B^- before the first flash was expected to be negligibly small.

Fig. 2A,C shows flash-induced voltage transients in chromatophores of *Rb. capsulatus* after the first and second flash of light, respectively. The fast, here unresolved, voltage rise was due to the charge separation in the RC. After both flashes it was followed by a slower rise (traces 1) that could be suppressed by the combined action of antimycin A and myxothiazol (traces 2). Fig. 2B,D shows the flash-induced absorption changes of the 'total' cyt c component (c_t, a composite from cyt c_1, $\lambda_{max} = 552$ nm, and cyt c_2, $\lambda_{max} = 550$ nm [18]). In the absence of inhibitors the fast oxidation of c_t by P^+ was followed by a relatively slow reduction (traces 1) which was completely suppressed by antimycin A+myxothiazol (traces 2). The amount of c_t reduced after the first flash was about 1/3 of the flash-oxidized amount. Taking into account the RC:*bc* dimer ratio of 6 in our samples (see Section 2), the (molar) amount of ubiquinol oxidized after the first flash can be estimated as about one per one *bc* dimer.

The traces in the third row (Fig. 2E,F) represent the kinetics of *bc* operation. The transients after the first flash are shown by solid lines, those measured after the second flash by dashed ones. Fig. 2E shows differences between voltage transients measured in the absence of inhibitors (*bc* fully operative, traces 1 in Fig. 2A,C) and in the presence of antimycin A and myxothiazol (*bc* completely blocked, traces 2 in Fig. 2A,C). The onset rate of the electrogenic reaction in the *bc* was much slower and smaller after the first flash (solid line)

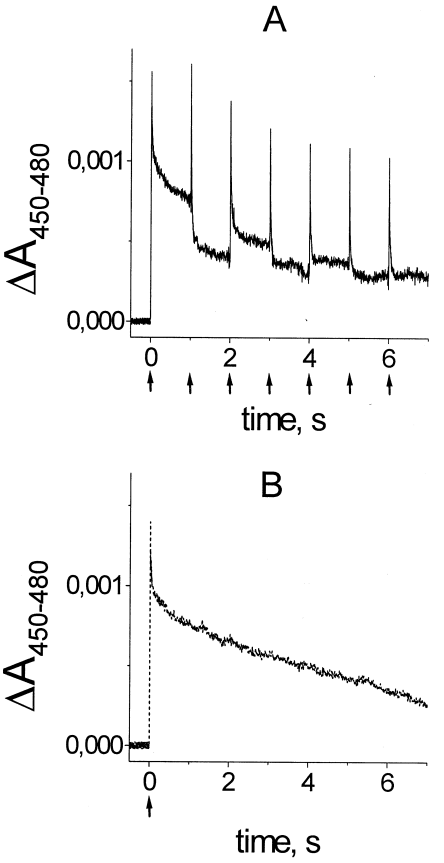

Fig. 1. Binary Q_B^- oscillations as measured at 450–480 nm. A: A series of flashes spaced 1 s were applied. B: Decay of the signal after a single flash. The incubation medium contained 2 mM $MgCl_2$, 1 mM Tris, 2 mM KCN, 2 mM potassium ferrocyanide, 2 mg/ml BSA, 1 μM oligomycin, 2 μM TMPD, 20 μM methylene blue, pH 8.1. Additions: 5 μM antimycin A, 3 μM myxothiazol.

than after the second one (dashed line). The differences between transients of c_t reduction in the absence of inhibitors (traces 1 in Fig. 2B,D) and in the presence of antimycin A and myxothiazol (traces 2 in Fig. 2B,D) are shown in Fig. 2F. They represent the rate of electron delivery from ubiquinol to c_t in the absence of inhibitors. The onset rate of c_t reduction after the first flash was slower than after the second one. Fig. 2G shows the kinetics of heme b_h reduction in the absence of inhibitors. After both flashes b_h was reduced with comparable rates ($t_{1/2} \sim 3$ ms and ~2 ms after the first and second flash, respectively). Fig. 2H shows the b_h transients in the presence of inhibitors. No b_h was reduced after the first flash in the presence of myxothiazol alone indicating that almost no ubiquinol was released from the RC after this flash (lower trace in Fig. 2H, compare with [8]). The amount of b_h that was reduced after the first flash in the presence of antimycin A was smaller than after second one and also corresponded roughly to one ubiquinol oxidized per *bc* dimer after the first flash (Fig. 2H, upper two transients).

4. Discussion

Here we showed that (1) at $E_h \sim 250$ mV, when the ubiquinone pool was expected to be fully oxidized, one ubiquinol molecule per *bc* dimer stayed reduced for several minutes and was oxidized in response to the first flash; (2) upon oxidation

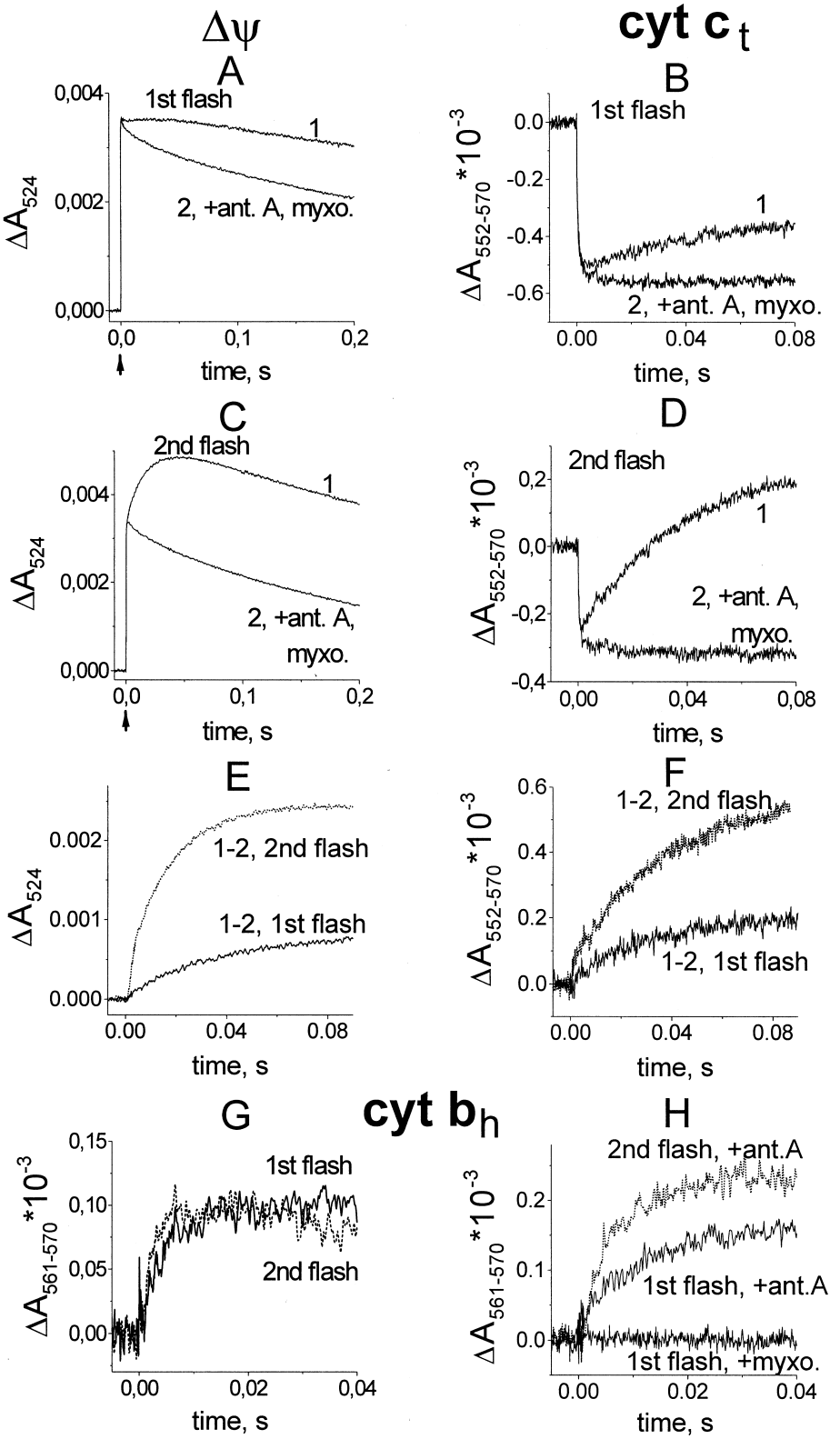

Fig. 2. Kinetics of voltage transients and redox changes of c_t and b_h after the first and second flashes (see text). 12–16 absorption transients wee averaged. Incubation medium was as in Fig. 1.

of this single ubiquinol the cyt b_h reduction was an order of magnitude faster than the cyt c_t reduction and the *bc*-attributable electrogenesis ($t_{1/2}$ of ~3 ms and ~30 ms, respectively).

That only one ubiquinol molecule (out of several formed by the previous flash series) stayed reduced during the dark adaptation time is coupled with a specific hindrance for the oxidation of the last, residual ubiquinol in the Q-cycle. The free

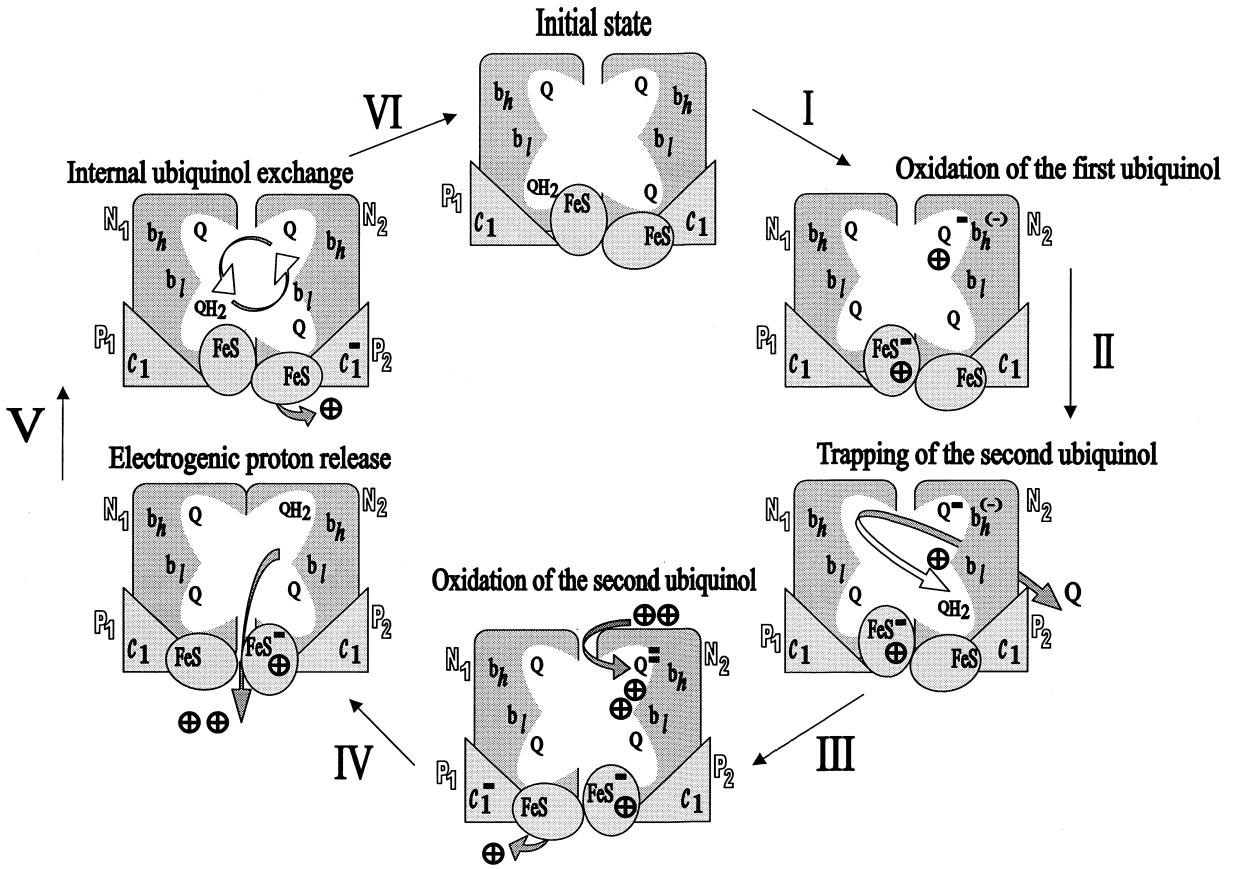

Fig. 3. Working model of the dimeric Q-cycle. The scheme shows two internal cavities as revealed in the crystal structures. They are large enough to enable an ubiquinone transfer from center N of one monomer to center P of the other and are open from the n-side of the membrane and sealed from the p-side [10]. The edge-to-edge distance of only $\sim$20 Å between two b_1 hemes in the bc dimer [10,11] is in accordance with the premise of a fast electron exchange between two centers N. The plus sign, $\oplus$, is used for a proton, the minus sign, $-$, for an electron. Only those protons and electrons that are released/accepted upon ubiquinol oxidation/reduction are depicted. The cycle starts from the oxidation of the first ubiquinol in center P^1 (reaction I) resulting in a first electron, e_1, transfer to FeS and e_2 transfer to Q_N. Two released protons stay bound by bc. To permit the transfer of the second ubiquinol to center P^2, center N^1 must be free from a bound semiquinone (the crystal structure of bc [10] and the functional data [7,34] indicate that a membrane ubiquinol enters bc from the n-side). Consequently, we hypothesize here that $Q_N{}^-$ which is formed after the oxidation of the first ubiquinol by P^1 resides not in the center N^1 of the same monomer but in the center N^2 of the other one (following the transfer of e_2 between two b_1 hemes along the membrane plane). The binding of the second ubiquinol is followed by a complex coupled reaction which we split into reactions III and IV for the purpose of illustration: (i) P^2 closes with Q_PH_2 inside; (ii) the second ubiquinol is oxidized; $e_1{}^2$ is accepted by FeS^2; (iii) two released protons stay bound by bc but without access to the ubiquinone in center N; (iv) the second electron ($e_2{}^2$) arrives on $Q_N{}^-$ and triggers binding of two protons from phase n (steps i–iv are merged in the reaction III); (v) Q_NH_2 is formed in one of the N centers; (vi) the protons bound by bc are released electrogenically into the phase p (steps v–vi are merged in reaction IV). To complete the cycle, the ubiquinol formed in N^2 is released and, perhaps, transferred to P^1 (reaction VI).

energy of the ubiquinol oxidation in center P differs depending on whether a semiquinone $Q_N{}^-$ or ubiquinol Q_NH_2 is formed in the center N. Using the estimates of midpoint potentials from [21–23] and assuming (i) that with only one molecule of ubiquinol present in the pool (>100 ubiquinone molecules per bc dimer [20]) the redox potential of the QH_2/Q pair was at least 120 mV higher than its standard $E_{m,8}$ value of 30 mV and (ii) that in a redox-equilibrated system, as in our case, the redox state of cyt c_2 is determined by E_h, we obtained following ΔG estimates for the oxidation of the first and second ubiquinol molecules by bc at $E_h = 250$ mV, respectively:

$$\mathrm{Ferri}-c_2+Q_PH_2+Q_N = \mathrm{ferro}-c_2+Q_P+Q_N^-+2H_p^+ \quad \Delta G_1 \geq +20\ \mathrm{meV} \qquad (1)$$

$$\mathrm{Ferri}-c_2+Q_PH_2+Q_N^-+2H_n^+ = \mathrm{ferro}-c_2+Q_P+2H_p^+ + Q_NH_2 \quad \Delta G_2 \leq -100\ \mathrm{meV} \qquad (2)$$

The difference between ΔG_1 and ΔG_2 is due to the higher $E_{m,8}$ value of the $Q_N{}^-/Q_NH_2$ redox pair compared with the $Q_N/Q_N{}^-$ redox pair (+150 mV and +30 mV, respectively, see [21]). Hence, under the given conditions (the ubiquinone pool oxidized and cyt c_2 mostly reduced) ubiquinol molecules are oxidized by bc in pairs: the oxidation of the first ubiquinol molecule (Eq. 1) cannot occur alone but only if it is driven by the oxidation of the second one (Eq. 2). Correspondingly, the last unpaired ubiquinol stays reduced. Conceivably, this was the one that was oxidized by the first flash in our setup. This residual ubiquinol could be pre-oxidized in the darkness by incubation of chromatophores at $E_h > 300$ mV ($\Delta G_1 < 0$).

Then the electrogenic reaction in *bc* was not observed after the first flash (data not shown) in line with previous observations with *Rb. sphaeroides* chromatophores [19,24]. The trapping of one molecule of ubiquinol per *bc* dimer at $E_h < 300$ mV corroborates the earlier observation that about half of all ubiquinol molecules leaving the RC of *Rb. sphaeroides* after the second flash (oxidizing conditions, Q_B^- binary oscillating) were not oxidized by *bc* but stayed reduced for tens of seconds [19,25]. The trapping of the residual plastoquinol on FeS in the cytochrome *bf* complex of spinach has been recently demonstrated by monitoring the EPR spectrum of FeS [26,27]. This residual plastoquinol stayed reduced in the darkness for 5–10 min, depending on the O_2 contents in the medium, but was readily oxidized by a light flash.

A functionally monomeric *bc* can hardly perform a pairwise ubiquinol oxidation. The dimeric structure of *bc* with two quinol-oxidizing centers *P*, provides a framework for a dimeric Q-cycle (shown in Fig. 3, see the caption for details). The flash-induced reactions of *bc* are triggered by the generation of P^+ in the RC which causes the complete oxidation of cyt c_2 (Fig. 2F). This brings the ΔG_1 value (see Eq. 1) below zero and drives the oxidation of the first ubiquinol (reaction I in Fig. 3). This results in the reduction of FeS by the first electron, e_1, and in the transfer of the second electron, e_2, to Q_N via b_l and b_h. That b_h stays partly reduced after the flash (see Fig. 2G) is due to the sharing of e_2 between b_h ($E_{m,8} = 20$ mV) and Q_N^- ($E_{m,8}(Q_N/Q_N^-) = 30$ mV). When only one ubiquinol is available (first flash), the turnover effectively stalls after this step. After the second flash the availability of several ubiquinol molecules formed in the RC enables the oxidation of the second and further ubiquinol molecules which causes the reduction of c_t and the voltage transient ($t_{1/2} \sim 10$ ms, Fig. 2E and 2F, dashed traces). The small extent of the very slow electrogenesis and c_t reduction still occurring after the first flash ($t_{1/2} \sim 30$ ms, Fig. 2E,F, solid traces) may be attributed to the reversibility of reaction I and to the slow redistribution of ubiquinol molecules between *bc* complexes leading to a full turnover in some of them.

The comparison of Fig. 2F,G (note the different time scales) shows that the reduction of c_t, especially after the first flash, is much slower than the reduction of b_h indicating the transient trapping of e_1 by FeS (see also [8]; it is conceivable that the stalling of e_1 prevents e_2 from slipping into FeS). From the comparative analysis of several crystal structures of *bc* it has been deduced in [11,28] that there seem to be two positions of FeS relative to cyt b_l, namely the proximal one with the distance between FeS and cyt c_1 of 31 Å not allowing the fast electron transfer to c_1 (hereafter denoted as *closed* center *P*, see [8]) and the distal one with FeS → cyt c_1 distance of about 21 Å compatible with a fast electron transfer (*open* center *P*). Based on the structural data, we attribute provisionally the stalling of the cyt c_t reduction to the trapping of FeS in the state proximal to b_l (center *P* closed, see Fig. 3)[1].

The $\Delta\psi$ onset in *bc* also contained no kinetic components corresponding to the reduction of b_h (compare traces in Fig. 2E,G). The strong kinetic discrepancy between b_h reduction and electrogenesis (of factor of 10 after the first flash) supports our earlier statement that the fast transmembrane transfer of e_2 to b_h is electrically silent (perhaps, because of a protonic re-arrangement), and that the slower electrogenic reaction is coupled with proton release from *bc* occurring concomitantly with Q_NH_2 formation (see [8,13,16] and caption to Fig. 3). Noteworthy is that under the given conditions only the latter reaction can provide the free energy for generation of $\Delta\mu H^+$ (cf. Eqs. 1 and 2).

The correlation between the rates of c_t reduction and of the voltage transient (compare Fig. 2E,F, see also [8]) indicates that the release of electrons from FeS into cyt c_t is synchronized with the oxidation of the second ubiquinol by *bc*. We hypothesize in Fig. 3 that one center *P* 'opens' leading electrons from FeS into cyt c_1 when the other one 'closes'. Such a binding change mechanism may account for the simultaneous presence of strongly and weakly bound Q_P quinones (Q_{PS} and Q_{PW}) in the *bc* of *Rb. sphaeroides* [22,29].

The dimeric Q-cycle implies that the oxidation of the first ubiquinol manifests itself only in the reduction of b_h that shares the electron with Q_N^- (reaction I in Fig. 3), whereas the electrogenesis and cyt c_t reduction lag behind being kinetically coupled with the oxidation of the second ubiquinol in center *P* and with the Q_NH_2 formation. This concept is in line not just with the data in Fig. 2, but also with various observations of the kinetic match between electrogenesis, proton release into phase *p*, cyt *c* (cyt *f*) reduction and b_h *oxidation*, in both *bc* [8,30,31] and *bf* complexes [32] and with the kinetic mismatch of the faster *reduction* of b_h with the former four reactions. Further support for the concept is provided by the observation that the site-specific mutations in cyt *f* of *Chlamydomonas reinhardtii* slowed in parallel the reduction of cyt *f* and the rise of the transmembrane voltage in the *bf* complex, whereas the faster rate of cyt *b* reduction was not retarded in these mutants [33].

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