Reduction of cytochrome *b*-561 through the antimycin-sensitive site of the ubiquinol-cytochrome c_2 oxidoreductase complex of *Rhodopseudomonas sphaeroides*

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Cytochrome *b*-561 of the ubiquinol-cytochrome c_2 oxidoreductase complex of *Rhodopseudomonas sphaeroides* is reduced after flash illumination in the presence of myxothiazol in an antimycin-sensitive reaction. Flash-induced reduction was observed over the redox range in which cytochrome *b*-561 and the Q-pool are both oxidized before the flash. The extent of reduction increased with increasing pH, and was maximal at pH >10.0 where the extent approached that observed in the presence of antimycin following a group of flashes. Reduction of cytochrome *b*-561 in the presence of myxothiazol showed a lag of ~1 ms after the flash, followed by reduction with $t_{1/2}$ ~6 ms; by analogy with the similar kinetics of the quinol oxidase site, we suggest that the rate is determined by collision with the QH_2 produced in the pool on flash excitation.

Ubiquinol-cytochrome c_2 *oxidoreductase* *Antimycin-sensitivity* *Myxothiazol* *Electrogenic process* *Cytochrome* b-*561* (Rps. sphaeroides)

1. INTRODUCTION

Several Q-cycle type mechanisms have been proposed to account for the function of the ubiquinol-cytochrome *c* oxidoreductase complex [1–8]. The models emphasize the existence of separate ubiquinol oxidase and the ubiquinone reductase sites within the oxidoreductase complex. There is considerable agreement on the reaction catalysed by the ubiquinol oxidase site, in which ubiquinol delivers one electron to a high-potential chain consisting of Rieske iron-sulfur center and *c*-type cytochromes, and the second electron to the *b*-type cytochromes. However, the reaction at the quinone reductase site is not well understood.

Specific inhibitors, which react at separate sites of the complex, have been used in studies of the mechanism of action of the complex [7,8]. Here, we have used myxothiazol, which inhibits the ubiquinol oxidase site, to study electron transfer through the antimycin-sensitive site of the complex. We show that cyt *b*-561 can be reduced after flash illumination in the presence of myxothiazol in both chromatophores and spheroplasts in a reaction which is antimycin sensitive. The redox characteristics, pH dependence, kinetics, and possible mechanisms of cyt *b*-561 reduction in the presence of myxothiazol in both chromatophores and spheroplasts, and the correlation of these properties with the redox properties of the semiquinone signal ($Q_c^{\cdot -}$) will be discussed.

2. MATERIALS AND METHODS

Chromatophores of *Rhodopseudomonas sphaeroides* strain Ga were prepared as in [9] and

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Abbreviations: Bchl, bacteriochlorophyll; Mops, 3-(*N*-morpholino)propanesulfonic acid; Hepps, *N*-2-hydroxyethylpiperazine-*N'*-2-propanesulfonic acid; Cyt, cytochrome; FeS, Rieske-type iron-sulfur center; Q, QH_2, ubiquinone, ubiquinol

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were further purified by centrifugation on a continuous sucrose gradient in the range 0.6–1.4 M sucrose in 50 mM Mops buffer and 100 mM KCl, pH 7.0. Spheroplast derived vesicles (referred to as spheroplasts in the text) were prepared according to [10].

The reduction of cyt *b*-561, the concentrations of reaction center, cyt *b*-561, and cytochromes c_1 and c_2, flash-induced spectra, redox poising, and bacteriochlorophyll content were measured as in [8,11–14].

The anaerobic reaction mixture consisted of chromatophores or spheroplasts suspended in a medium containing 50 mM buffer [Mops (pH 7.0), Hepps (pH 7.7), Bis-Tris-propane (pH 8.7), glycine (pH 9.9), or threonine (pH 10.5)], 100 mM KCl, redox mediators (at the concentrations indicated in the figure legends), valinomycin and nigericin at 2 μM, and inhibitors when indicated. The concentration of chromatophores or spheroplasts was adjusted so that a single flash induced turnover in more than 90% of the reaction centers. In all experiments, a dark period of 1 min was allowed between flashes or groups of flashes.

Myxothiazol was a kind gift from Drs Reichenbach, Thierbach, and Trowitzsch, and nigericin was a gift from E. Lilly Pharmaceuticals. Valinomycin and antimycin A were obtained from Sigma.

3. RESULTS

Fig.1 shows typical traces of reduction of cyt *b*-561 in the presence of myxothiazol and/or antimycin. At pH 7.0 and 7.7 a maximal extent of the reduction was obtained after the second flash, at pH 8.7, after the third flash. The extent of the change increased with increasing pH. At pH 8.7 the change after the first flash approaches about 30%, and the maximal change approaches about 60%, of the change obtained on flash illumination by a group of flashes in the presence of antimycin alone. The change obtained in the presence of myxothiazol was completely abolished when both myxothiazol and antimycin were present. The maximal extent of reduction of cyt *b*-561 in the presence of antimycin alone (lower trace) was the same at all values of pH in the range tested.

Fig.2 shows the spectra of flash-induced changes measured in chromatophores (fig.2A,C) and spheroplasts (fig.2B,D) in the presence of myxothiazol, at E_h = 140 mV ± 3 mV, at pH 8.7, over

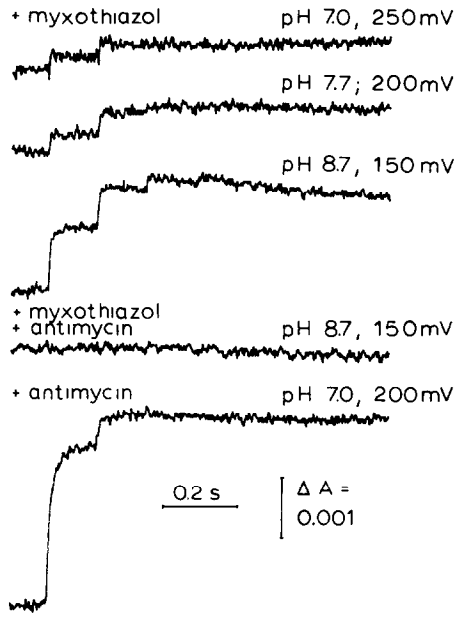

Fig.1. The reduction of cyt *b*-561 in the presence of inhibitors. The reduction in the presence of myxothiazol was measured at 3 pH values: 7.0, 7.7, 7.8. The reduction in the presence of both myxothiazol and antimycin was measured at pH 8.7 and the reduction in the presence of antimycin alone was measured at pH 7.0. Chromatophores were suspended as described in section 2. Concentrations of redox mediators were: 1 μM each of phenazine methosulfate, phenazine ethosulfate, pyocyanine at pH 7.0, 0.5 μM at pH 7.7, and 0.2 μM at pH 8.7; concentration of 2,3,5,6-tetramethyl-*p*-phenylenediamine (DAD) was 2 μM at pH 7.0, 0.5 μM at pH 7.7, and 0.2 μM at pH 8.7; 1,2-naphthoquinone, 1,4-naphthoquinone, *p*-benzoquinone and duroquinone were at 10 μM at all values of pH. Concentration of BChl in the cuvette was 25 μM; reaction center, 0.26 μM; flash-reducible cyt *b*-561, 0.16 μM; cyt ($c_1 + c_2$), 0.23 μM; 3 μM myxothiazol and 10 μM antimycin A were present when indicated. Traces were average of 2, sweep 1 s full scale, time constant 1 ms.

the range 540–575 nm. The spectra shown in fig.2A,B by inverted triangles represent the spectra corrected for the change due to reaction center. The spectra in fig.2C,D are corrected for reaction center, and additionally for the change due to cyt $c_1 + c_2$ in chromatophores and cyt c_1 in spheroplasts, and show the characteristic appearance of cyt *b*-561. The peak due to cyt *b*-561 was not seen if the ambient potential was below the value at which reduction of cytochrome *b*-561 in the presence of myxothiazol titrated out, or when

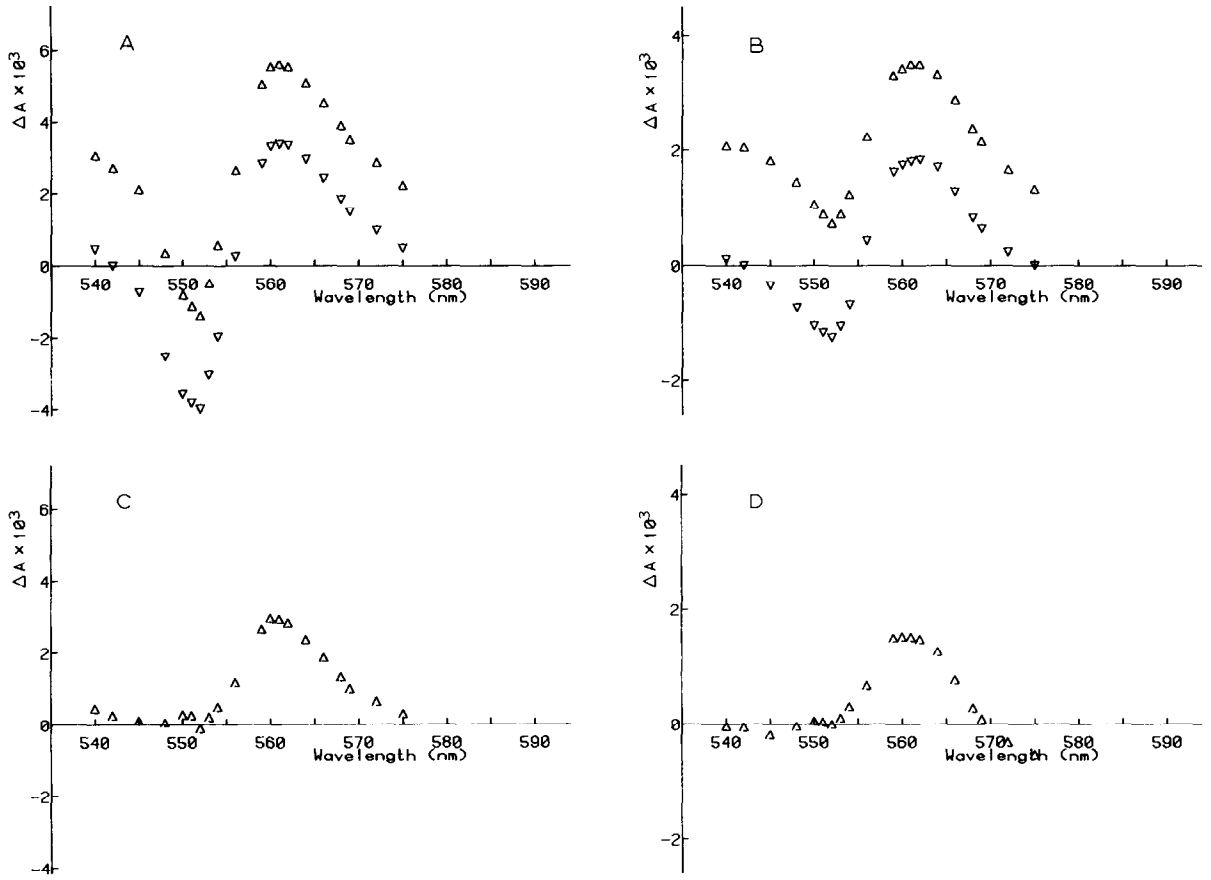

Fig.2. Spectra of the flash-induced changes for chromatophores (A,C) and spheroplasts (B,D) in the presence of myxothiazol. Spectra were obtained at pH 8.7, at 140 mV. Experimental conditions were as described in fig.1. In chromatophores, the concentration of reaction center was 0.36 μM; in spheroplasts, 0.22 μM. Note the different scales for the chromatophore and spheroplast spectra. Inverted triangles in panels A and B show the flash-induced spectra corrected for the change due to reaction center; panels C and D show the spectra corrected for reaction center and additionally for cyt ($c_1 + c_2$) in chromatophores (C), and for cyt c_1 in spheroplasts (D). Correction factors were as in [6,11,14,19,21]. The change was measured after group of 4 flashes spaced 0.13 s apart, immediately after the fourth flash.

antimycin was present in addition to myxothiazol (not shown).

The redox titration curves for the extent of reduction of cyt *b*-561 in the presence of myxothiazol at different pH values are shown in fig.3A. On lowering the E_h below 300 mV at pH 7.0, the extent of the flash-induced reduction titrated in to reach a maximal extent at E_h value of about 220 mV. On lowering the E_h below 220 mV, the extent of the change decreased and titrated out at an E_h value of about 150 mV, corresponding to the ambient potential at which the first QH_2 became chemically reduced in the pool before the flash. The extent of the change was strongly pH dependent, increasing by about 8-fold as the pH was increased from 7.0 to 10.4. The maximal extent appeared at lower E_h values on increasing the pH, and the change also titrated out at lower values of E_h. The titration curves appear to show a two-electron process ($n = 2$). A maximal extent after one flash, corresponding to about 60% of the amount of cyt *b*-561 which could be reduced by a group of flashes in the presence of antimycin, was obtained at an ambient potential of $E_h \sim 100$ mV, at pH 9.9 and at $E_h \sim 70$ mV at pH 10.4, 10.6. Fig.3B shows the midpoint potentials taken from the titration curves shown in fig.3A. The dashed curve shows as a function of pH theoretical values

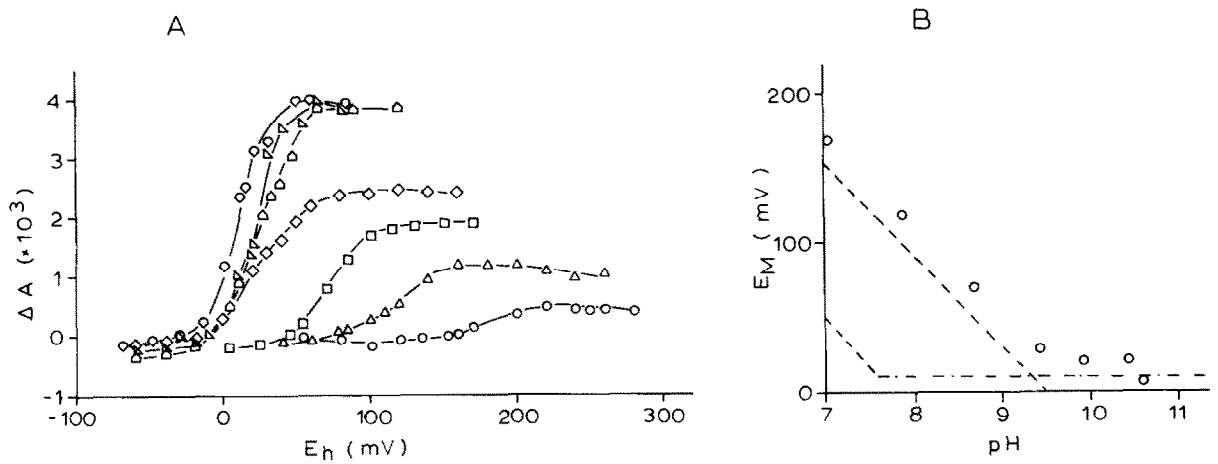

Fig.3. Redox titration of the extent of reduction of cyt *b*-561 in the presence of myxothiazol. Panel A shows the redox titration curves at different values of pH: 7.0 (○), 7.85 (△), 8.7 (□), 9.45 (◇), 9.9 (▵), 10.4 (▹), 10.6 (○). Experimental conditions were as described in section 2 and in fig.1, except that at pH 9.9, 10.4 and 10.6, 1 μM *N,N,N',N'*-tetramethyl-*p*-phenylene diamine (TMPD) was included in the reaction mixture. Each point was an average of 2, at sweep of 1 s full scale and time constant 1 ms. The changes were measured 10 ms after the first flash at pH 7, 7.85 and 8.7, 20 ms at pH 9.45, and 50 ms at pH 9.9, 10.4 and 10.6, at which times reduction was maximal. In panel B, midpoint potential values measured from the curves shown in A are shown as a function of pH. The theoretical curve (– – –) shows the E_h value at which 0.5 QH_2/complex is present in the pool due to chemical reduction before the flash, and the theoretical curve (-----) shows midpoint potential values for cyt *b*-561 at different pH values.

of the potential at which 0.5 QH_2/complex would be reduced chemically in the pool before the flash, assuming $E_{m,7}$ for the Q/QH_2 couple to be 90 mV, and 60 $(Q + QH_2)$/complex in the pool. The other curve shows the midpoint potential of cyt *b*-561 at varying values of pH [15]. Although the E_m values calculated from the redox titration curves in fig.3 are slightly higher than the more positive of the values from the theoretical curves at any pH, a reasonable correlation is shown between the ability to reduce cyt *b*-561 on flash excitation, and the availability of both a fully oxidized quinone pool, and oxidized cyt *b*-561 before the flash.

Fig.4 shows the kinetics of reduction of cyt *b*-561 in the presence of myxothiazol or antimycin, and in the absence of the inhibitors, in chromatophores (fig.4A,C) and in spheroplasts (fig.4B,D). The change was measured at pH 8.7, E_h = 140 mV on excitation by a group of 4 flashes (A,B), or on a faster time scale, after a single flash (C,D). In chromatophores, the initial kinetics obtained in the presence of myxothiazol are very similar to the kinetics in the presence of antimycin, and slightly slower than the change in the absence of inhibitors. The traces show a rise phase with $t_{1/2}$ of about 6 ms, which occurs after a brief lag. In spheroplasts, however, the initial rate in the presence of antimycin was inhibited as compared to the initial rates in the presence of myxothiazol or in the absence of inhibitors. This is probably due to the fact that the high potential components of the complex cannot be rapidly oxidized in spheroplasts because of the lack of cyt c_2. As shown on a faster time scale, in both chromatophores and spheroplasts, the reduction of cyt *b*-561 in the presence of myxothiazol follows a lag of about 1 ms, indicating that the rate of reduction is determined by the diffusion of QH_2 from the reaction center through the pool [6,7]. The similarity of the kinetics of reduction of cyt *b*-561 through both sites suggests that both are governed by a similar set of constraints.

4. DISCUSSION

The reaction at the site of the ubiquinol-cyt c_2 oxidoreductase complex at which cyt *b* is oxidized is not well understood. In chromatophores and mitochondria, most authors suggest that antimycin is a specific inhibitor of all reactions at the site [2,3,7,8]. Antimycin inhibits reoxidation of cyt *b*-561 after a flash, and of cyt *b*-566 if cyt *b*-561 is

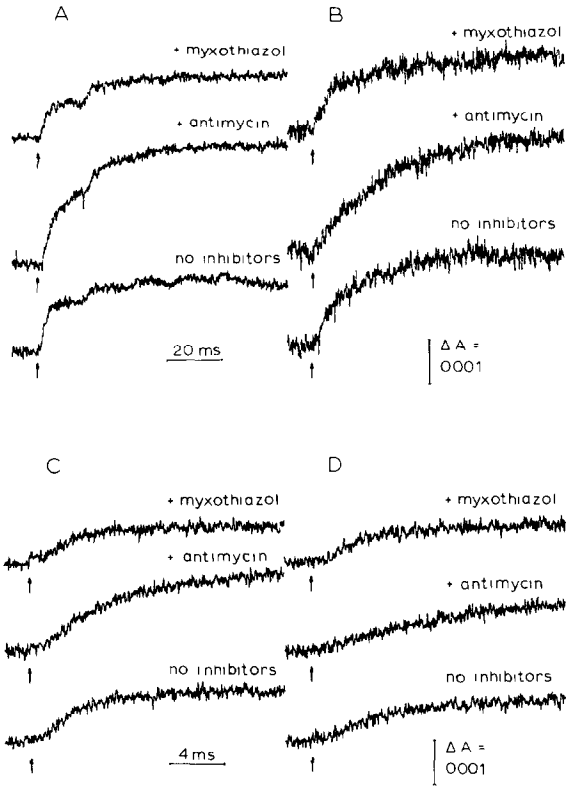

Fig.4. Kinetics of the reduction of cyt *b*-561 in the presence and absence of inhibitors in chromatophores (A,C) and spheroplasts (B,D). The measurements were performed at pH 8.7, at E_h = 140 mV. Experimental conditions were as described in fig.1. Myxothiazol (3 μM) or antimycin (10 μM) were added where indicated. A and B, concentration of BChl in the cuvette in the chromatophore suspension was 22.4 μM, and in the spheroplast suspension, 20.2 μM; A and D, concentration of reaction center in the chromatophore suspension was 0.19 μM, and in the spheroplast suspension was 0.24 μM. Experiments shown in panel A and B were done as an average of 8 and 4, respectively, sweep 100 ms full scale, time constant 100 μs, and show excitation by a group of four flashes. The traces shown in panels C and D were initiated by a single flash and were an average of 16, sweep 20 ms, time constant 20 μs.

previously reduced [16]. It has been shown earlier in mitochondria [17], that in the presence of myxothiazol, which is a specific inhibitor of the ubiquinol oxidase site of the complex [17,18], reduction of cyt *b*-561 by QH_2 may occur without involving a concerted reduction of the high-potential chain of the complex. Flash-induced reduction of the cyt *b*-562 (the mitochondrial analogue of cyt *b*-561) through an antimycin-sensitive process which did not involve a concerted reaction at the quinol oxidase site, has been previously observed in a hybrid system [19,20]. Here, we show that cyt *b*-561 can be reduced after flash illumination in the presence of myxothiazol in both chromatophores and spheroplasts of *Rps. sphaeroides*, and that the change is sensitive to antimycin. We have characterized the reduction of cyt *b*-561 through this site with respect to its redox properties, pH dependence and kinetics. Recently, an antimycin-sensitive ubisemiquinone radical ($Q_c^{\cdot -}$) of the ubiquinol-cyt c_2 oxidoreductase complex of *Rps. sphaeroides* has been characterized [21]. The redox properties have been interpreted as showing formation from a ubiquinone/quinol couple with an E_m of 150 mV at pH 7. Observation of a similar signal in the R 126 mutant of *Rps. capsulata* [22], which does not contain a functional ubiquinol oxidase site [22,23], indicated that the $Q_c^{\cdot -}$ signal originates from another site. The maximal extent of the antimycin-sensitive $Q_c^{\cdot -}$ signal was reached in the pH range 9.5–10.7 at a value of about 0.4 ($Q_c^{\cdot -}$) per complex [21]. The maximal extent of the reduction of cyt *b*-561 in the presence of myxothiazol upon illumination with a single flash under conditions when 1 QH_2 is produced in the oxidized pool per oxidoreductase complex is reached in the same pH range as the $Q_c^{\cdot -}$ signal, with a stoichiometry of about 0.6 cyt *b*-561 reduced per complex.

In summary, the redox properties, pH dependence and kinetics of the antimycin-sensitive cyt *b*-561 reduction in the presence of myxothiazol, together with the experiments discussed above from other laboratories [19–22] point to the conclusion that there is a site, distinct from the quinol oxidase site, at which electrons can be transferred to cyt *b*-561 from quinol in the pool, in a reversal of the reaction by which ferrocytochrome *b* is normally oxidized and quinol produced. However, the mechanism of the reaction at the site is still unclear. Several variants of the Q-cycle [1,2] have been proposed with different mechanisms for oxidation of the *b*-cytochrome chain [1–6,24–27]. Each of the above mechanisms may be represented as involving the production of quinol in the pool by oxidation of cyt *b*, and our present results could therefore be interpreted as a reversal of such a reaction; they do not therefore exclude any of the

proposed mechanisms. However, they do allow us to reach some definite conclusions about the reaction catalysed by the site.

(a) The kinetics of oxidation of quinol are similar to those of the myxothiazol-sensitive quinol oxidase site when the latter are measured at the same ambient potential. This suggests that at both sites a second-order reaction occurs (first-order in quinol from the pool), with a value for k_2 of about $2 \times 10^{-5}\ M^{-1} \cdot s^{-1}$, the value obtained for the quinol oxidase site [6].

(b) The flash-induced reduction of cyt *b*-561 is most simply interpreted as reflecting a response to the change in concentration of QH_2 in the pool resulting from flash illumination. Reduction of cyt *b*-561 occurs on production of ~1 QH_2/complex in the fully oxidized pool (~60 Q/complex) [6], suggesting a relatively high value for the equilibrium constant favoring cyt *b*-561 reduction. When the pool is partly reduced, the photochemical production of 1 QH_2/complex would give rise to a much smaller change in poise of the Q/QH_2 couple, and this could in part explain why the flash-induced change becomes smaller as the pool becomes reduced.

Values for K can be calculated from the results for various putative reaction mechanisms.

(i) The following reaction represents the simplest mechanism for reduction of cyt *b*-561 through the antimycin-sensitive site:

$$QH_2 + 2 \text{ ferricyt } b\text{-}561 \rightleftharpoons Q + 2 \text{ ferrocyt } b\text{-}561 \text{ (H)} \quad (1)$$

Assuming that after a single flash, 1 QH_2/complex is present in the pool, the value for K_1 obtained from our results is considerably larger over the pH range from 7 to 8.0 than that calculated using $E_{m,7}$ values for the couples Q/QH_2 and ferri/ferrocyt *b*-561 of 90 and 50 mV, respectively, appropriately adjusted for pH; this suggests either that a different reaction occurs, or that the values assumed are wrong. At higher values of pH, where the E_m value of the Q/QH_2 couple continues to decline with pH, but that for the cyt *b*-561 becomes independent of pH [15], the measured and calculated equilibrium constants approach each other, reaching the same value at about pH 9.5.

In view of the above difficulty, several alternative explanations of the results observed may be suggested.

(ii) In many laboratories (review [27]), redox titrations of chromatophores from *Rps. sphaeroides* have shown the presence of a high potential form of cyt *b*-561 ($E_{m,7}$ = 150 mV) (cyt *b*-561 (HP)) of unknown function. If cyt *b*-561 (HP) were the acceptor of electrons from QH_2 in the above reaction, calculated values for the equilibrium constant would be much closer to the measured values at the lower end of the pH range. However, functional preparations of the complex contain only two *b*-type hemes per complex, corresponding to cyt *b*-566 and cyt *b*-561 [27,28] and the extent of reduction of cyt *b*-561 through the antimycin-sensitive site observed at higher values of pH exceeds the stoichiometry of cyt *b*-561 (HP).

(iii) An alternative mechanism may be framed in terms of the equation:

$$QH_2 + Q_c \cdot \text{ferricyt } b\text{-}561 \rightleftharpoons Q_c^{\cdot -} \cdot \text{ferrocyt } b\text{-}561(H) + H^+ + Q \quad (2)$$

This reaction may be considered as the sum of two partial reactions, involving displacement of Q from the complex by QH_2 followed by electron transfer:

$$QH_2 + Q_c \cdot \text{ferricyt } b\text{-}561 \rightleftharpoons Q_cH_2 \cdot \text{ferricyt } b\text{-}561 + Q \quad (2a)$$

$$Q_cH_2 \cdot \text{ferricyt } b\text{-}561 \rightleftharpoons Q_c^{\cdot -} \cdot \text{ferrocyt } b\text{-}561(H) + H^+ \quad (2b)$$

The equilibrium constant (K_2) for the overall reaction includes the binding constants for Q and QH_2, and the equilibrium constant for electron transfer, none of which are known independently. However, a tighter binding of QH_2 than Q (cf. [21]) and a relatively stable semiquinone would both help to displace the overall reaction to the right. The mechanism of eq.2 suggests an explanation for the simultaneous formation of ferrocyt *b*-561 and $Q_c^{\cdot -}$, found in kinetic experiments previously reported using the mitochondrial complex [24], and inferred from the experiments reported here and from the data in [21,22]. The mechanism could also account for the cyt *b*-561 (HP) found in redox titrations, in terms of a reduction of cyt *b*-561 by the first QH_2 produced in the pool on chemical reduction. If such a reduction occurred, it would account for the fact that the flash-induced change also titrated out over the range in which the first quinol appeared in the pool.

(iv) Alternatively, the reaction of eq.2 could involve a mechanism in which quinone was present at the antimycin site as a relatively tightly bound species. The bound quinone and ferricytochrome *b*-561 could then act as a two-electron accepting system for oxidation of quinol from the pool. To account for the observed equilibrium constant, this mechanism would require that the couple $Q_c/Q_c^{\cdot -}$ has a relatively high redox potential ($E_{m,7} > 175$ mV from the data of fig.3), indicating a relatively stable semiquinone species. This mechanism has the additional advantage over the mechanism in (iii) above of explaining the observation that isolated ubiquinol:cyt c_2 (or cyt *c*) oxidoreductase complexes always have associated with them about 0.6 equivalents/complex of bound quinone [29].

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REFERENCES

[1] Mitchell, P. (1975) FEBS Lett. 56, 1–6.
[2] Mitchell, P. (1976) J. Theor. Biol. 62, 327–367.
[3] Wikström, M. and Krab, K. (1980) Curr. Top. Bioeng. 10, 51–101.
[4] Van Ark, G., Raap, A.., Berden, J.A. and Slater, E.C. (1981) Biochim. Biophys. Acta 637, 34–42.
[5] De Vries, S., Albracht, S.P.J., Berden, J.A. and Slater, E.C. (1982) Biochim. Biophys. Acta 681, 41–53.
[6] Crofts, A.R., Meinhardt, S.W., Jones, K.R. and Snozzi, M. (1983) Biochim. Biophys. Acta 723, 202–218.
[7] Crofts, A.R. and Wraight, C.A. (1983) Biochim. Biophys. Acta 726, 149–185.
[8] Crofts, A.R. (1983) in: The Enzymes of Biological Membranes, 2nd edn (Martonosi, A.N. ed.) pp.347–382, Plenum, New York.
[9] Bowyer, J.R., Tierney, G.V. and Crofts, A.R. (1979) FEBS Lett. 101, 201–206.
[10] Takemoto, J. and Bachmann, R.C. (1979) Arch. Biochem. Biophys. 195, 526–534.
[11] Bowyer, J.R., Meinhardt, S.W., Tierney, G.V. and Crofts, A.R. (1981) Biochim. Biophys. Acta 635, 167–186.
[12] Dutton, P.L., Petty, K.M., Bonner, H.S. and Morse, S.D. (1975) Biochim. Biophys. Acta 387, 536–556.
[13] Clayton, R.K. (1963) in: Bacterial Photosynthesis (Gest, H. et al. eds) p.498, Antioch Press, Yellow Springs.
[14] Snozzi, M. and Crofts, A.R. (1984) Biochim. Biophys. Acta, in press.
[15] Petty, K.M. and Dutton, P.L. (1976) Arch. Biochem. Biophys. 172, 346–353.
[16] Meinhardt, S.W. and Crofts, A.R. (1983) Biochim. Biophys. Acta 723, 219–230.
[17] Von Jagow, G. and Engel, W.D. (1981) FEBS Lett. 136, 19–24.
[18] Meinhardt, S.W. and Crofts, A.R. (1982) FEBS Lett. 149, 217–222.
[19] Matsuura, K. and Dutton, P.L. (1981) in: Chemiosmotic Proton Circuits in Biological Membranes (Skulachev, V.P. and Hinkle, P.C. eds) pp.259–270, Addison-Wesley, Reading, MA.
[20] Zhu, Q.S. (1984) PhD Thesis, University of Amsterdam, Amsterdam.
[21] Robertson, D.E., Prince, R.C., Bowyer, J.R., Matsuura, K., Dutton, P.L. and Ohnishi, I. (1984) J. Biol. Chem. 259, 1758–1763.
[22] Robertson, D.E., Davidson, E., Prince, R., Van der Berg, W.H., Marrs, B.L. and Dutton, P.L. (1984) Fed. Proc. 43, 2000 (Abstr.3385).
[23] Bowyer, J.R. and Crofts, A.R. (1981) Biochim. Biophys. Acta 636, 218–233.
[24] De Vries, S., Albracht, S.P.J., Berden, J.A., Marres, C.A.M. and Slater, E.C. (1983) Biochim. Biophys. Acta 723, 91–103.
[25] Garland, P.B., Clegg, R.A., Boxer, D., Downie, J.A. and Haddock, B.A. (1975) in: Electron Transfer Chains and Oxidative Phosphorylation (Quagliariello, E. et al. eds) pp.351–358, North-Holland, Amsterdam.
[26] Cramer, W.A. and Crofts, A.R. (1982) in: Photosynthesis: Energy Conversion by Plants and Bacteria (Govindjee, ed.) vol.1, pp.387–467, Academic Press, New York.
[27] Dutton, P.L. and Prince, R.C. (1978) in: The Photosynthetic Bacteria (Clayton, R.K. and Siström, W.R. eds) pp.525–570, Plenum, New York.
[28] Gabellini, N. and Hauska, G. (1983) FEBS Lett. 153, 146–150.
[29] Hauska, G., Hunt, E., Gabbellini, N. and Lockau, W. (1983) Biochim. Biophys. Acta 726, 97–133.