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SHORT REVIEW

Cytochrome b_{50} as a Proton Carrier in the Photosynthetic Redox Chain of Purple Bacteria

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Abstract

Recent data on the proton-translocating activity of *b* cytochromes in chromatophores of purple bacteria and their arrangement in the photosynthetic redox chain are discussed. These data appear to support the concept of the b_{50} and b_{-90} cytochrome doublet spanning the membrane. Current schemes of H^+ transport by *b* cytochromes are considered, and the scheme of H^+ translocation by cytochrome b_{50} taking up H^+ at the outer side of the membrane and a quinone delivering them from this cytochrome to the inner space of the chromatophore is favored as the most probable in the light of recent findings. This scheme is applicable both to Crofts' linear model of the redox chain and to Mitchell's Q cycle. Kinetic discrepancies between H^+ uptake and cytochrome b_{50} reduction at high ambient redox potentials are interpreted in terms of a special, cytochrome b_{50}-independent, yet Rieske FeS-protein-dependent mode of H^+ transport.

Key Words: Cyclic photosynthetic redox chain; reaction center; ubiquinone; cytochrome c_2 reductase; cytochrome *b*; purple bacteria.

Introduction

Cytochromes of the *b* type form part of the electron transfer chains of mitochondria, chloroplasts, and bacterial membranes. Oxidation or reduction of these cytochromes entails shifts in their p*K* values, enabling the *b* type cytochromes to function as proton carriers in redox chains (Papa *et al.*, 1973; Papa, 1976, 1982; Petty and Dutton, 1976). H^+ transfer by *b* cytochromes has been proposed for the mitochondrial ubiquinone:cytochrome *c* reductase complex (Skulachev, 1971; Wikström and Berden, 1972; Papa *et al.*, 1973; Von Jagow and Engel, 1980; 1981a; Konstantinov *et al.*, 1982) and its

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analogue, the ubiquinone:cytochrome c_2 reductase complex of purple bacteria (Petty and Dutton, 1976; Dutton and Prince, 1978; Wraight, 1979). Generally speaking, three possible mechanisms for the protonmotive activity of the b–c_1 complexes of redox chains have been taken into account in the literature so far: (i) protonation/deprotonation reactions accompanying cytochrome *b* oxidoreduction are of no functional relevance, and all the H^+ transfer across the membrane is carried out by some other component(s); this possibility is exemplified by Mitchell's Q cycle (in its original form), which attributes the H^+ transfer in the mitochondrial ubiquinone:cytochrome *c* reductase solely to the ubiquinone (Mitchell, 1976, 1980). The two other possibilities implicate the participation of *b* type cytochromes in *vectorial* H^+ transfer: (ii) cytochrome(s) *b* and the other H^+ carriers are arranged in parallel. Thus each of them carries out transmembrane H^+ transfer *per se;* this is postulated in the hypothesis of Von Jagow and Engel (1980, 1981a), and earlier such an arrangement of H^+ carriers was suggested by Papa (1976); (iii) cytochromes *b* and the other carriers are arranged in series, and transmembrane H^+ flow includes several stages. This was suggested by Skulachev (1971) and by Wikström and Berden (1972) for mitochondria and by Prince and Dutton (1976) for chromatophores; in both cases, a *b* type cytochrome accepts H^+ ions from ubiquinol and releases them into an aqueous phase. Konstantinov *et al.* (1982) proposed that H^+ uptake from the inner space of mitochondria is accomplished by a *b* cytochrome which thereupon donates H^+ to ubiquinone. Despite these suggestions, the possibility of a sequential H^+ transport by cytochromes *b* and other H^+ carriers has been somewhat overshadowed in the literature; however, this is the scheme which appears to be applicable to the chromatophore redox chain, and this review discusses relevant recent data and concepts concerning the possibility of a relay transfer of H^+ by cytochrome *b* (b_{50}) and a quinone component in chromatophores.

Cytochrome b_{50} is a component of the ubiquinone:cytochrome c_2 reductase of *Rhodopseudomonas sphaeroides* (Petty and Dutton, 1976), *Rhodopseudomonas capsulata* (Evans and Crofts, 1974), and *Rhodospirillum rubrum* (Niedermann *et al.*, 1980). Cytochrome b_{50} has much in common with cytochrome *b*-560 of the sulfur purple bacterium *Chromatium vinosum* (Takamiya and Hanada, 1980). Cytochrome b_{50} is analogous to cytochrome *b*-562 of the mitochondria (Packham *et al.*, 1980) as well as to cytochrome b_6 of chloroplasts (Trebst, 1980). Of the two types of the latter revealed recently (Hurt and Hauska, 1981; Hauska *et al.*, 1982), the relatively high potential type apparently bears more similarity to the chromatophore cytochrome dealt with here. Apart from the similarity of cytochromes *b* in various biological energy-transducing systems, we should point out what whole quinol:cytochrome $c(f)$ reductase complexes from mitochondria, purple bacteria, spinach chloroplasts, and the cyanobacterium *Anabaena variabilis* in general

follow the same model in their subunit structure (Hauska *et al.*, 1982), and such complexes can perform their function in chimerical systems (Packham *et al.*, 1980; Matsuura *et al.*, 1981; Prince *et al.*, 1982). These structural similarities were confirmed by the analysis of the polypeptide composition of the isolated and purified ubiquinone:cytochrome c_2 reductase from purple bacteria (Hauska *et al.*, 1982; Takamiya *et al.*, 1982; Yu and Yu, 1982).

Functional Characterization of the Components of the Chromatophore Redox Chain

This section describes the general scheme of the light-induced electron transfer in the membranes of purple bacteria. The light-excited bacteriochlorophyll dimer of the photochemical reaction center P870 reduces the primary, tightly bound quinone Q_I to semiquinone via the short-lived intermediary acceptor I (Olson and Thornber, 1979; Wraight, 1979; Dutton *et al.*, 1982). Semiquinone Q_I passes its electron to the secondary quinone Q_{II}. The bacteriochlorophyll dimer is reduced by cytochrome c_2 (E_m 360 mV), the oxidant of cytochrome c_1 with an E_m value of 290 mV (Wood, 1980a, b). Cytochrome c_1 oxidizes the special quinol Q_zH_2 to semiquinone $Q_zH^{\cdot}$ (Prince and Dutton, 1977; Prince *et al.*, 1978; Van den Berg *et al.*, 1979; Takamiya *et al.*, 1979) via the Rieske FeS-protein (Prince and Dutton, 1976; Bowyer *et al.*, 1980). The E_m values of the couples Q_z/Q_zH_2, $Q_zH^{\cdot}/Q_zH_2$, and $Q_z/Q_zH^{\cdot}$ are equal to 155 (Prince and Dutton, 1977; Prince *et al.*, 1978), $\geq$300, and $\leq$0 mV (Van den Berg *et al.*, 1979), respectively, based on experimental data or calculations.

The arrangement of *b* cytochromes in the redox chain remains a controversial point. Cytochrome b_{50} is characterized by a midpoint potential of 50 mV at pH 7 and an α-band in the absorption spectrum with a maximum at 560 nm (Petty and Dutton, 1976). Reduced cytochrome b_{50} is in the protonated state, its pK value being 7.4. The oxidation of the heme leads to deprotonation of the cytochrome at pH $\leq$ 7.4. At higher pH values, oxidoreduction of the cytochrome does not involve protonation or deprotonation (Petty and Dutton, 1976).

With single-turnover flashes, cytochrome b_{50} has been shown to accept and lose an electron with half times ($t_{1/2}$) of about 1.5 and 2–3 ms, respectively (Dutton and Jackson, 1972; Crofts *et al.*, 1974; Petty and Dutton, 1976; Dutton and Prince, 1978; O'Keefe and Dutton, 1981), after the exciting flash. A light flash also causes redox changes in another cytochrome *b* species, cytochrome b_{-90} (Bowyer and Crofts, 1981). The E_m value of cytochrome b_{-90} is equal to -90 mV at pH 7; its α-band has an absorption maximum at 566 nm with a shoulder at 558.5 nm (Bowyer *et al.*, 1981). The spectral characteris-

tics of cytochrome b_{-90} resemble those of the mitochondrial cytochrome *b*-566 (Packham *et al.*, 1980). Based on the data on cytochrome b_{-90} reduction upon membrane potential generation by inorganic pyrophosphate hydrolysis (Dutton and Baltscheffsky, 1972), the cytochrome is arranged close to the inner side of the chromatophore membrane.

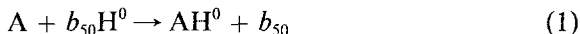
Reduction of cytochrome b_{50} by a light flash is accompanied by a red electrochromic shift of the carotenoid band in the absorption spectrum (phase III), indicative of a transmembrane outward electron translocation (Dutton and Prince, 1978). Interestingly, when the bulk of the cytochrome b_{50} molecules is already reduced in the dark and only oxidation of the cytochrome is observed upon a flash, the phase of the carotenoid band shift is markedly decreased in extent (O'Keefe and Dutton, 1981). This suggests that cytochrome b_{50} is located close to the outer phase. Thus, a number of recent data obtained in experiments with chromatophores lend support to the concept of the cytochrome *b* doublet spanning the membrane, which was originally suggested for mitochondria by Mitchell (1976), Papa (1976, 1982), and Wikström *et al.* (1981) (these references discuss the concept in detail). The electron transfer from cytochrome c_2 to quinone Q_I (implicating the participation of the bacteriochlorophyll dimer and I) and from cytochrome b_{-90} to cytochrome b_{50}, due to its electrogeneity, may be responsible for energy conservation in the $\Delta\bar{\mu}e^-$ form, which is thereupon transduced into a transmembrane H^+ gradient.

In a recent work (Crofts *et al.*, 1982), cytochrome b_{50} is suggested to react with cytochrome b_{-90} in an electron transfer process oriented perpendicularly to the membrane plane and sensitive to myxothiazol, which is thought to inhibit also electron flow between the two mitochondrial *b* type hemes (Von Jagow and Engel, 1981b). The half-time value for the cytochrome b_{-90} and b_{50} interaction, its equilibrium constant, and the forward and backward reaction rate constants are estimated to be 0.3 ms, 2.15, 2×10^5 M^{-1} s^{-1}, and 9.3×10^{-4} M^{-1} s^{-1}, respectively (Crofts *et al.*, 1982).

Involvement of Cytochrome b_{50} in H^+ Binding by the Chromatophores of Purple Bacteria

The operation of the chromatophore redox chain involves H^+ binding from the external, aqueous phase. The process includes two stages with $t_{1/2}$ values of 0.1 and 1.5–2 ms, respectively (termed H_I^+ and H_{II}^+), in *Rps. sphaeroides* at E_h below 150 mV and pH 7 (Petty *et al.*, 1977, 1979). The first stage occurs following the reduction of the secondary quinone and is probably due to this component. The nature of the agent binding H_{II}^+ with a $t_{1/2}$ value of 1.5–2 ms has not yet been elucidated (Petty *et al.*, 1979; Wraight, 1979).

Nevertheless, there is a considerable body of evidence suggesting the involvement of cytochrome b_{50} in this process: (i) the pK value of the $H_{II}{}^+$ binding agent is 7.5, practically coinciding with that of reduced cytochrome b_{50} (Petty *et al.*, 1979); (ii) the kinetics of $H_{II}{}^+$ binding follows that of cytochrome b_{50} reduction (Petty *et al.*, 1979; Wraight, 1979); (iii) inhibition of Q_{II} reduction by *o*-phenanthroline suppresses the binding of only one of the two H^+ (Cogdell *et al.*, 1972; Halsey and Parson, 1974; Carithers and Parson, 1975); this perturbation of the operation of the redox chain, as experiments with ametryne (acting similarly to *o*-phenanthroline) have revealed, does not change the extent of the reduction of cytochrome b_{50} (Bowyer and Crofts, 1981); (iv) dibromothymoquinone and 7-*n*-heptylquinoline quinone, which totally inhibit the reduction of cytochrome b_{50} at high concentrations, abolish the binding of only one of the two H^+ (Dutton and Prince, 1978); (v) after the extraction of loosely bound quinones, H^+ binding by chromatophores is accompanied by cytochrome b_{50} reduction (Halsey and Parson, 1974; Wraight, 1979). Cytochrome b_{50} reoxidation is, however, not accompanied by H^+ release into the incubation medium (Petty and Dutton, 1976; Petty *et al.*, 1979). Therefore, it seems likely that the H^+ liberated by the cytochrome considered is transferred in addition to an electron to acceptor (A) oxidizing cytochrome b_{50}:



$$A + b_{50}H^0 \rightarrow AH^0 + b_{50} \tag{1}$$

H^+ Transport by Cytochrome b_{50} in Different Schemes of the Photosynthetic Redox Chain

Two alternative schemes of electron transfer in the bacterial photosynthetic redox chain have been proposed in the literature: the linear (Crofts *et al.*, 1974, 1975; Dutton and Prince, 1978; Bowyer and Crofts, 1981) and the parallel, which is also termed the protonmotive quinone cycle (Mitchell, 1976; Dutton and Prince, 1978; O'Keefe and Dutton, 1981; Crofts *et al.*, 1982). In these schemes, different components are supposed to donate reducing equivalents to and accept them from *b* cytochromes (Fig. 1).

According to the linear model, quinone component Q_{II} is in the semiquinone form in the dark. The semiquinone is thermodynamically stable, due to its complexation in the reaction center (its stability constant K_{st} exceeds unity, and thus the E_m ($Q_{II}H^{\cdot}/Q_{II}H_2$) value is higher than the E_m ($Q_{II}/Q_{II}H^{\cdot}$) value). The photoreduced Q_I converts $Q_{II}H^{\cdot}$ to quinol, which is protonated by an H^+ from the outer aqueous phase and thereupon oxidized by a *b* type cytochrome arranged near the inner side of the chromatophore membrane, which obviously requires a transmembrane inward flow of reducing equivalents. For the reasons mentioned above, we suggest that this *b* cytochrome is cytochrome

b_{-90}. Cytochrome b_{50} located close to the outer surface accepts an electron from cytochrome b_{-90} and passes it to semiquinone $Q_zH^{\cdot}$, as proposed by Crofts *et al.* (1975) and more recently by Bowyer and Crofts (1981). The E_m ($Q_{II}H^{\cdot}/Q_{II}H_2$) value at pH 7 is equal to 20 mV (Rutherford and Evans, 1980), exceeding the midpoint potential of cytochrome b_{-90} by 110 mV. The thermodynamically unfavorable reduction of cytochrome b_{-90} will be facilitated by the following processes resulting in free energy release: cytochrome $b_{-90} \rightarrow$ cytochrome b_{50} ($\Delta E = -140$ mV) and cytochrome $b_{50} \rightarrow Q_zH^{\cdot}$ ($\Delta E = -250$ mV).

The H^+ transport by cytochrome b_{50} can briefly be described as follows. This cytochrome donates a neutral H^0 equivalent ($e^- + H^+$) to semiquinone $Q_zH^{\cdot}$ upon H^+ uptake from the outer aqueous phase. At $E_h > E_m$ ($Q_{II}/Q_{II}H^{\cdot}$), which according to Rutherford and Evans (1980) is equal to 100 mV at pH 7, the secondary quinone is fully oxidized in the dark and, upon a light flash, only semiquinone is formed, which is unable to reduce the *b* cytochromes considered. In line with these data, the light-induced reduction of cytochrome b_{50} is drastically slowed down in the E_h range of 100–150 mV (Bowyer and Crofts, 1981). At $E_h > 150$ mV, H^+ binding with a $t_{1/2}$ value of 1.5–2 ms is not observed (Petty *et al.*, 1977, 1979).

The parallel scheme assumes that the secondary quinone remains fully oxidized in the dark and is sequentially reduced by $Q_I^{\cdot}$ and cytochrome *b* (b_{50}) to quinol upon a light flash (Dutton and Prince, 1978; Crofts *et al.*, 1982). Cytochrome b_{50} is reduced by cytochrome b_{-90} which accepts electrons from Q_z in cooperation with cytochrome c_2. Quinone Q_z is rereduced to Q_zH_2 by $Q_{II}H_2$. It is also possible that whole $Q_{II}H_2$ molecules move toward the site of Q_z in the membrane to replenish the Q_z pool, as suggested by Remennikov and Samuilov (1979).

In terms of the hypothesis of H^+ transfer by cytochrome b_{50}, the cytochrome involved is protonated and transfers its H^+ together with an electron to semiquinone $Q_{II}H^{\cdot}$. At $E_h > 155$ mV, the bulk of the Q_z pool is fully oxidized and cannot reduce cytochrome species, which accounts for the observed retardation of cytochrome b_{50} reduction and the lack of H^+ binding

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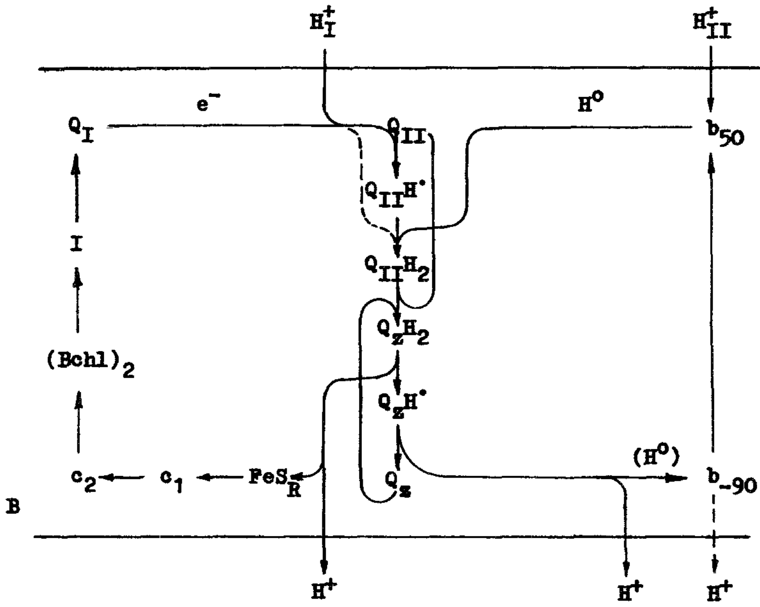
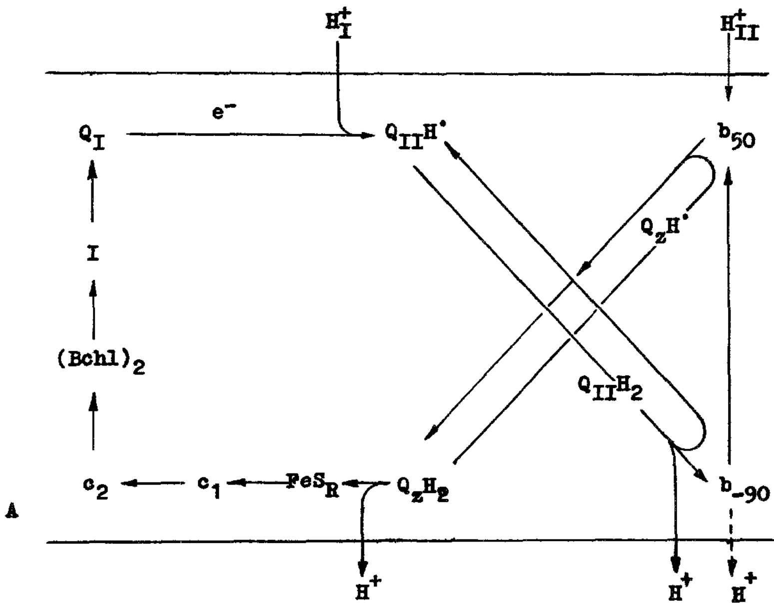
Fig. 1. Schemes of the linear (A) and the parallel (B) electrogenic cyclic redox chain in chromatophores of the nonsulfur purple bacteria. Unlike analogous schemes considered previously (Petty *et al.*, 1977; Dutton and Prince, 1978; Remennikov and Samuilov, 1979; Bowyer and Crofts, 1981), the schemes proposed contain the following modifications: (i) the primary intermediate step in charge separation between $(BChl)_2$ and I is electrogenic (Borisov *et al.*, 1980; Kotova *et al.*, 1981; Samuilov, 1982); (ii) cytochromes b_{-90} and b_{50} are arranged close to the inner and the outer sides of the membrane, respectively; (iii) cytochrome b_{50} functions as a redox-linked proton translocator, and not merely as an electron carrier. Designations: $(BChl)_2$, P870 reaction center bacteriochlorophyll dimer; I, short-lived intermediary electron acceptor; Q_I, Q_{II}, and Q_z, quinones; c_1 and c_2, *c*-type cytochromes; b_{50} and b_{-90}, *b*-type cytochromes; FeS_R, Rieske FeS-protein.


H^+_I
H^+_{II}
e⁻
H^o
Q_I
Q_{II}
b_{50}
$Q_{II}H^\cdot$
I
$Q_{II}H_2$
Q_zH_2
$(Bchl)_2$
$Q_zH^\cdot$
(H^o)
c_2
c_1
FeS_R
Q_z
b_{-90}
B
H^+
H^+
H^+


H^+_I
H^+_{II}
e⁻
Q_I
$Q_{II}H^\cdot$
b_{50}
$Q_zH^\cdot$
I
$(Bchl)_2$
$Q_{II}H_2$
c_2
c_1
FeS_R
Q_zH_2
b_{-90}
A
H^+
H^+
H^+

with a $t_{1/2}$ value of 1.5–2 ms. The E_h dependence of cytochrome b_{50} reduction and of millisecond H^+ binding interpreted above in terms of the linear scheme is explainable on the basis of the parallel scheme as well, if only H^+ transfer by cytochrome b_{50} is assumed in both schemes discussed. Currently, there is good reason for suggesting that the redox chain easily switches over from one schemes to the other (Dutton *et al.*, 1982). The choice of either the parallel or the linear scheme of electron transfer is presumably subject to regulation by the redox state of cytochrome b_{50} (O'Keefe and Dutton, 1981) and/or that of the secondary quinone (Remennikov and Samuilov, 1979; Rutherford and Evans, 1980).

It is possible that cytochrome b_{-90} accepts H^0 from $Q_{II}H_2$ in the linear scheme or $Q_zH^{\cdot}$ in the parallel one, subsequently releasing H^+ into the interior of the chromatophore. Similarly to cytochrome b_{50}, cytochrome b_{-90} might also transfer H^+, substituting the suggested "output proton well" for the quinone redox center(s) (Konstantinov *et al.*, 1982).

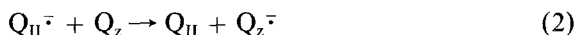
Special Modes of H^+ Transfer

A. In the presence of antimycin A, $H_{II}{}^+$ binding is inhibited (Petty *et al.*, 1977, 1979), the level of cytochrome b_{50} reduction is increased, and its subsequent reoxidation is retarded (Dutton and Jackson, 1972; Crofts *et al.*, 1974; Prince and Dutton, 1975; Petty and Dutton, 1976; Van den Berg *et al.*, 1979; Takamiya *et al.*, 1979; Bowyer and Crofts, 1981). Supposedly, it is the secondary quinone (in the $Q_{II}H^{\cdot}$ or $Q_{II}H_2^{\cdot}$ form) that directly reduces cytochome b_{50} (Van den Berg *et al.*, 1979; Bowyer and Crofts, 1981). A lack of $H_{II}{}^+$ binding can be accounted for by H^0 transfer from component Q_{II} to cytochrome b_{50}. At pH > pK ($b_{50}{}^{red}$), cytochrome b_{50} only accepts an electron; thus, in the presence of an uncoupler, H^+ bound to the secondary quinone is free to reappear in the incubation medium after Q_{II} oxidation by cytochrome b_{50} (Petty and Dutton, 1976; Dutton and Prince, 1978; Petty *et al.*, 1979).

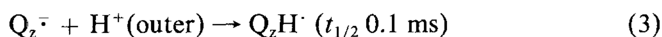
B. With the loosely bound quinones extracted, cytochrome b_{50} probably accepts an electron from semiquinone $Q_I{}^{\bar{\cdot}}$ which is formed upon a light flash. Subsequent protonation of cytochrome b_{50} may be the reason for the H^+ binding observed in this case (Halsey and Parson, 1974; Wraight, 1979). However, there is evidence (Baccarini-Melandri *et al.*, 1982) suggesting the dependence of cytochrome b_{50} reduction in extracted chromatophores on the remaining Q_{II}, and thus the proposal of a "short-circuit" between Q_I and this cytochrome is unnecessary. Nonetheless, by supposing electron transfer between $Q_I{}^{\bar{\cdot}}$ and cytochrome b_{50}, one can also explain the H^+ binding seen after the inhibition of the reaction between Q_I and Q_{II} (Cogdell *et al.*, 1972; Halsey and Parson, 1974; Carithers and Parson, 1975). However, cytochrome

b_{50} can be readily reduced by $Q_zH^·$ generated according to the parallel scheme of the redox chain.

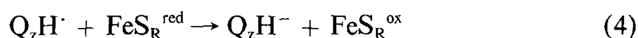
C. At high E_h values of the incubation medium (150–300 mV), H_{II}^+ rapidly binds with a $t_{1/2}$ value of 0.2 ms (Petty *et al.*, 1977, 1979), even though the overall turnover rate of the cyclic photosynthetic redox chain is drastically retarded. This puzzling fact can be explained by a hypothetical redox mechanism implicating the following reaction sequence:



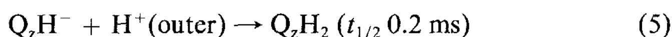
$$Q_{II}^{\overline{\cdot}} + Q_z \rightarrow Q_{II} + Q_z^{\overline{\cdot}} \quad (2)$$



$$Q_z^{\overline{\cdot}} + H^+(\text{outer}) \rightarrow Q_zH^{\cdot}\ (t_{1/2}\ 0.1\ \text{ms}) \quad (3)$$



$$Q_zH^{\cdot} + FeS_R^{red} \rightarrow Q_zH^- + FeS_R^{ox} \quad (4)$$



$$Q_zH^- + H^+(\text{outer}) \rightarrow Q_zH_2\ (t_{1/2}\ 0.2\ \text{ms}) \quad (5)$$

The key element of this hypothetical scheme is the suggested reversal of the "usual" reduction of the Rieske FeS-protein by the Q_z quinone [scheme (4)]. Thermodynamically, this reversal is permissible, since the midpoint potentials of the reactants are quite close to each other. Evidence for the suggested involvement of the Rieske protein in semiquinone reduction and (indirectly) in H_{II}^+ binding comes from numerous observations on the dependence of both processes on the redox state of the iron-sulfur protein (Bowyer *et al.*, 1979; Verkhovsky *et al.*, 1980; O'Keefe *et al.*, 1981). At redox potentials above 280–300 mV, with the Rieske protein essentially oxidized, semiquinone accumulates in chromatophores upon each odd-numbered flash, suggesting that some reaction normally leading to reduction of the semiquinone to quinol is precluded. Upon an even-numbered flash, semiquinone produces quinol, presumably by dismutation (Bowyer *et al.*, 1979; Verkhovsky *et al.*, 1980; O'Keefe *et al.*, 1981). The reason why component Q_z takes up H^+ from the outer aqueous phase may be that an "input proton well" carries out H^+ transfer between this phase and the Q_z site in the membrane (Konstantinov *et al.*, 1982). Some recent data raise the possibility that quinones Q_{II} and Q_z share a single redox center in the membrane, which could minimize the need of spatial translocation of the semiquinones involved (Rutherford and Evans, 1980; Wikström and Krab, 1980; Konstantinov *et al.*, 1982).

Besides the protonmotive activity of cytochrome b_{50}, it is conceivable that the FeS-protein, which releases an H^+ upon oxidation at pH > 8 (Prince and Dutton, 1976), is also involved in H^+ translocation at these pH values and at high redox potentials. This protein, which is suggested by us to reduce $Q_zH^·$, could then donate the H^+ ejected to the resulting quinol anion, thus producing Q_zH_2 without H^+ uptake from the external aqueous phase. This may account for the observed lack of the binding of the second H^+ at the pH and E_h values specified (Petty *et al.*, 1979).

In summary, the present paper is concerned with the mechanism of H^+ transfer by cytochrome b_{50}. This cytochrome binds a proton ($H_{II}{}^+$) following the reduction of its heme and donates an H^0 equivalent to a quinone component, thereby playing the role of a peculiar proton well operative between the outer aqueous phase and the quinone redox center of the chromatophore membrane. The most striking feature of the photosynthetic electron transport chain is the dynamism of the redox machinery, which readily switches over from one mode of action to another in response to redox potential changes or disruption of the electron transfer processes in some of the segments of the chain.

References

Baccarini-Melandri, A., Gabellini, N., Melandri, B. A., Jones, K. R., Rutherford, A. W., Crofts, A. R., and Hurt, E. (1982). *Arch. Biochem. Biophys.* **216,** 566–580.

Borisov, A. Yu., Godik, V. I., Kotova, E. A., and Samuilov, V. D. (1980). *FEBS Lett.* **119,** 121–124.

Bowyer, J. R., and Crofts, A. R. (1981). *Biochim. Biophys. Acta* **636,** 218–233.

Bowyer, J. R., Dutton, P. L., Prince, R. C., and Crofts, A. R. (1980). *Biochim. Biophys. Acta* **592,** 445–460.

Bowyer, J. R., Meinhardt, S. W., Tierney, G. V., and Crofts, A. R. (1981). *Biochim. Biophys. Acta* **635,** 167–186.

Bowyer, J. R., Tierney, G. V., and Crofts, A. R. (1979). *FEBS Lett.* **101,** 201–206.

Carithers, P. P., and Parson, W. W. (1975). *Biochim. Biophys. Acta* **347,** 194–211.

Cogdell, R. J., Jackson, J. B. and Crofts, A. R. (1972). *J. Bioenerg. Biomembr.* **4,** 413–429.

Crofts, A. R., Crowther, D., and Tierney, G. V. (1975). In *Electron Transfer Chains and Oxidative Phosphorylation* (Quagliariello, E., Papa, S., Palmieri, F., Slater, E. C., and Siliprandi, N., eds.), Elsevier-North Holland, Amsterdam, pp. 233–241.

Crofts, A. R., Evans, E. H., and Cogdell, R. J. (1974). *Ann N.Y. Acad. Sci.* **227,** 227–243.

Crofts, A. R., Meinhardt, S. W., Snozzi, M., and Jones, K. R. (1982). In *2nd European Bioenergetics Conference* Bernard-Lyon, France, pp. 327–328.

Dutton, P. L. and Baltscheffsky, M. (1972). *Biochim. Biophys. Acta* **267,** 172–178.

Dutton, P. L., and Jackson, J. B. (1972). *Eur. J. Biochem.* **30,** 495–510.

Dutton, P. L., and Prince, R. C. (1978). In *The Photosynthetic Bacteria* (Clayton, R. K., and Sistrom, W. R., eds.). Plenum Press, New York, pp. 525–570.

Dutton, P. L., Mueller, P., O'Keefe, D. P., Packham, N. K., Prince, R. C., and Dutton, P. L. (1982). In *Current Topics in Membranes and Transport* (Slayman, C., ed.), Vol. 16, Academic Press, pp. 323–343.

Evans, E. H., and Crofts, A. R. (1974). *Biochim. Biophys. Acta* **357,** 78–88.

Halsey, Y. D., and Parson, W. W. (1974). *Biochim. Biophys. Acta* **347,** 404–416.

Hauska, G., Gabellini, N., Hurt, E., Krinner, M., and Lockau, W. (1982). *Biochem. Soc. Trans.* **10,** 340–341.

Hurt, E., and Hauska, G. (1981). *Eur. J. Biochem.* **117,** 591–599.

Konstantinov, A., Kunz, W. S., and Kamensky, Yu. A. (1982). In *Chemiosmotic Proton Circuits in Biological Membranes* (Skulachev, V. P., and Hinkle, P. C., eds.). Addison-Wesley, London, pp. 123–146.

Kotova, E. A., Samuilov, V. D., Godik, V. I., and Borisov, A. Yu. (1981). *FEBS Lett.* **131,** 51–54.

Matsuura, K., Packham, N. K., Mueller, P., and Dutton, P. L. (1981). *FEBS Lett.* **131,** 54–58.

Mitchell, P. (1976). *J. Theor. Biol.* **62,** 327–367.

Mitchell, P. (1980). *Ann. N.Y. Acad. Sci.* **341,** 564–584.

Niedermann, R. A., Hunter, C. N., Mallon, D. E., and Jones, O. T. G. (1980). *Biochem, J.* **186,** 453–459.
O'Keefe, D. P., and Dutton, P. L. (1981). *Biochim. Biophys. Acta* **635,** 149–166.
O'Keefe, D. P., Prince, R. C., and Dutton, P. L. (1981). *Biochim. Biophys. Acta* **637,** 512–522.
Olson, J. M., and Thornber, J. P. (1979). In *Membrane Proteins in Energy Transduction* (Capaldi, R. A., ed.), Marcel Dekker, New York, pp. 279–340.
Packham, N. K., Tiede, M. D., Mueller, P., and Dutton, P. L. (1980). *Proc. Natl. Acad. Sci. U.S.A.* **77,** 6339–6343.
Papa, S. (1976). *Biochim. Biophys. Acta* **456,** 39–84.
Papa, S. (1982). *J. Bioenerg. Biomembr.* **14,** 69–86.
Papa, S., Guerrieri, F., Simone, S., and Lorusso, M. (1973). In *Mechanisms in Bioenergetics* (Azzone, G. F., Ernster, L., Papa, S., Quagliariello, E., and Siliprandi, N., eds.), Academic Press, New York, London, pp. 451–472.
Petty, K. M., and Dutton, P. L. (1976). *Arch. Biochem. Biophys.* **172,** 346–353.
Petty, K. M., Jackson, J. B., and Dutton, P. L. (1977). *FEBS Lett.* **84,** 299–303.
Petty, K. M., Jackson, J. B., and Dutton, P. L. (1979). *Biochim. Biophys. Acta* **546,** 17–42.
Prince, R. C., and Dutton, P. L. (1975). *Biochim. Biophys. Acta* **387,** 609–613.
Prince, R. C., and Dutton, P. L. (1976). *FEBS Lett.* **36,** 169–179.
Prince, R. C., and Dutton, P. L. (1977). *Biochim. Biophys. Acta* **462,** 731–747.
Prince, R. C., Bashford, C. L., Takamiya, K. I., van den Berg, W. H., and Dutton, P. L. (1978). *J. Biol. Chem.* **253,** 4137–4142.
Prince, R. C., Matsuura, K., Hurt, E., Hauska, G., and Dutton, P. L. (1982). *J. Biol. Chem.* **257,** 3379–3381.
Remennikov, V. G., and Samuilov, V. D. (1979). *Biochim. Biophys. Acta* **548,** 216–223.
Rutherford, A. W., and Evans, M. C. W. (1980). *FEBS Lett.* **110,** 257–261.
Samuilov, V. D. (1982). *Usp. Sovrem. Biol.* **92,** 46–63.
Skulachev, V. P. (1971). *Curr. Top. Bioenerg.* **4,** 127–190.
Takamiya, K. -I., Doi, M., and Okimatsu, H. (1982). *Plant Cell Physiol.* **23,** 987–997.
Takamiya, K. I., and Hanada, H. (1980). *Plant Cell Physiol.* **21,** 979–988.
Takamiya, K. I., Prince, R. C., and Dutton, P. L. (1979). *J. Biol. Chem.* **254,** 11307–11311.
Trebst, A. (1980). In *Cell Compartmentation and Metabolic Chanelling* (Nover, L., Lynen, F., and Mothers, K., eds.), VEB Gustav Fischer Verlag, Jena (GDR) and Elsevier-North-Holland, Amsterdam, pp. 201–213.
Van den Berg, W. H., Prince, R. C., Bashford, C. L., Takamiya, K. I., Bonner, W. D., Dutton, Jr., and Dutton, P. L. (1979). *J. Biol. Chem.* **254,** 8594–8604.
Verkhovsky, M. I., Grishanova, N. P., Kaurov, B. S., and Shinkarev, V. N. (1980). *Biol. Nauki* **8,** 35–37.
Von Jagow, G., and Engel, W. D. (1980). *FEBS Lett.* **111,** 1–5.
Von Jagow, G., and Engel, W. D. (1981a). In *Vectorial Reactions in Electron and Ion Transport in Mitochondria and Bacteria* (Palmieri, F., eds.), Elsevier–North-Holland, Amsterdam, pp. 411–422.
Von Jagow, G., and Engel, W. D. (1981b). *FEBS Lett.* **136,** 19–24.
Wikström, M. K. F., and Berden, J. A. (1972). *Biochim. Biophys. Acta* **283,** 403–420.
Wikström, M. K. F., and Krab, K. (1980). *Curr. Top. Bioenerg.* **10,** 51–101.
Wikström, M. K. F., Krab, K., and Saraste, M. (1981). *Annu. Rev. Biochem.* **50,** 623–635.
Wood, P. M. (1980a). *Biochem. J.* **189,** 385–391.
Wood, P. M. (1980b). *Biochem, J.* **192,** 761–764.
Wraight, C. A. (1979). *Photochem. Photobiol.* **30,** 767–776.
Yu, L., and Yu, C. -A. (1982). *Biochem. Biophys. Res. Commun.* **108,** 1285–1292.