Biochimica et Biophysica Acta, 725 (1983) 138–145
Elsevier

BBA 41386

KINETIC EVIDENCE FOR INVOLVEMENT OF TWO CYTOCHROME *b*-563 HEMES IN PHOTOSYNTHETIC ELECTRON TRANSPORT *

JEFFREY P. HOUCHINS and GEOFFREY HIND

Biology Department, Brookhaven National Laboratory, Upton, NY 11973 (U.S.A.)

(Received May 20th, 1983)

Key words: Cytochrome b-563; Q-cycle; Electron transport; Photosynthesis; (Anabaena)

The effect of 2-(*n*-heptyl)-4-hydroxyquinoline *N*-oxide (HQNO) on the kinetics of cytochrome *b*-563 and cytochrome *c* † turnovers following single-turnover flashes was measured in isolated heterocysts. Low concentrations of HQNO (below 3 μM) blocked reoxidation of cytochrome *b*-563, whereas higher concentrations (above 5 μM) resulted in additional inhibition of cytochrome *b*-563 oxidation and also inhibited reduction of cytochrome *b*-563 and cytochrome *c*. Similar effects on cytochrome *b*-563 reduction and reoxidation were obtained with a combination of 5 μM HQNO and 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone (1–7 μM). In HQNO-inhibited heterocysts, cytochrome *c* reduction following a flash occurred in three phases with half-times of 0.5, 2.8 and 45 ms. The second phase nearly equalled the cytochrome *b*-563 reduction in half-time and magnitude. In the presence of HQNO, the reoxidation of cytochrome *b*-563 following two closely spaced actinic flashes displayed biphasic kinetics. The two phases correspond to reoxidation of cytochrome *b*-563 in which one or both of the cytochrome *b*-563 hemes in the cytochrome *b-f* complex are reduced. These results are interpreted in terms of a Q-loop in which HQNO, at low concentrations, blocks the site of rapid cytochrome *b*-563 reoxidation and at higher concentrations, also inhibits the site of electron donation by plastoquinol to the cytochrome *b-f* complex.

† **Cyanobacteria contain a membrane-bound cytochrome *f*-556.5 and a soluble cytochrome *c*-553. Because it is frequently impossible to distinguish between these components in kinetic experiments, the term cytochrome *c* will be used to refer to these components collectively.**

Introduction

Cytochrome complexes that catalyze the oxidation of quinol have been identified in a number of energy-transducing membranes including mitochondria [1], bacteria [2,3] and chloroplasts [4,5]. In all cases these complexes contain one *c*-type cytochrome, one high-potential Fe-S center and two cytochrome *b* hemes which differ in midpoint potential by about 100 mV [2]. A proposed mechanism for the oxidation of quinol and subsequent release of two protons by these complexes involves a concerted electron transfer in which donation of one electron from QH_2 to the Fe-S center and ultimately to cytochrome *c* generates a semiquinone which then reduces cytochrome *b* [6]. The reoxidation of cytochrome *b* by Q occurs at a

Abbreviations: DBMIB, 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone; HQNO, 2-(*n*-heptyl)-4-hydroxyquinoline *N*-oxide; Chl, chlorophyll; Hepes, *N*-2-hydroxyethylpiperazine-*N'*-2-ethanesulfonic acid; PQ, plastoquinone.

0005-2728/83/$03.00

second quinone-binding site after transfer of the electron across the membrane through the second cytochrome *b* heme. This mechanism is supported in part by studies with antimycin A which blocks the reoxidation of cytochrome *b* in mitochondria and bacteria [7,8].

Antimycin A does not block the reoxidation of cytochrome *b* in cyanobacteria, but we have recently found that HQNO inhibits at this site [9], and a similar site of action was recently reported in chloroplast membranes [10]. This inhibitor allows measurement of the cytochrome *b*-563 reduction rate, following a single-turnover flash, by suppressing interference from the reoxidation kinetics; it therefore provides a valuable tool for the study of electron transfer in these systems.

In a number of filamentous, N_2-fixing cyanobacteria, vegetative cells can differentiate into heterocysts. These cells are the site of N_2 fixation and as such possess a number of modifications that protect nitrogenase against inactivation by O_2 [11]. Photosystem II is eliminated during differentiation, whereas Photosystem I and the associated electron-transfer components are retained [12,13]. Because these cells have much diminished levels of accessory pigments [14] and a simplified photosynthetic system, while still possessing a higher-plant type of Photosystem I [15], they represent excellent subjects for the study of electron flow around Photosystem I. We have previously characterized the flash-induced absorbance changes in isolated heterocysts [9]. Here we report the effects of HQNO on the turnover kinetics of cytochrome *b*-563 and cytochrome *c* following single-turnover flashes, and provide evidence that two separate sites of inhibition exist in the cytochrome *b*-*f* complex. Evidence is also presented, based on the relaxation kinetics of cytochrome *b*-563, that both cytochrome *b*-563 hemes are reduced by successive flashes in the presence of HQNO.

Methods

Anabaena sp. strain 7120 (ATCC 27893) was grown in batch cultures as described previously [16] at a Cu^{2+} concentration of 80 nM unless stated otherwise. If Cu^{2+}-free cultures were desired, all glassware was rinsed with 12 M HCl prior to preparation of medium, and quarter-strength medium of Allen and Arnon [17] minus Cu^{2+} was used. Heterocysts were isolated under H_2 [16] and stored under Ar at 0°C until use.

Flash-induced absorbance changes were measured as described previously [9]. Samples contained 17 μg Chl/ml, as isolated heterocysts, in 1.5 ml of reaction medium consisting of 50 mM KCl, 1 mM $MgCl_2$, 7% Ficoll, 25 μM KCN, 5 μM valinomycin, 5 μM nigericin and 40 mM Hepes (pH 7.5), under a gas phase of 90% H_2 plus 10% O_2. Actinic flashes were provided by two EG and G FX-201 xenon flashlamps which were fired simultaneously or, in double-flash experiments (Figs. 6 and 7) 20 ms apart. Cytochrome *c* was monitored at 554 nm and cytochrome *b*-563 at 563–572 nm.

Rise and decay times, and corresponding amplitudes of flash-induced cytochrome turnovers were evaluated by a least-squares fit of the data to an equation of the type shown below for a coupled exponential rise and decay:

$$A = \frac{k_1 A_2}{k_1 - k_2}(e^{-k_2 t} - e^{-k_1 t}) + \frac{k_1 A_3}{k_1 - k_3}(e^{-k_3 t} - e^{-k_1 t}) \qquad (1)$$

Eqn. 1 defines a monophasic rise and biphasic decay where A is the absorbance change at time t, k_1 the rate constant for the rise, k_2 and k_3 the rate constants for the two phases of decay, and A_2 and A_3 the amplitudes corresponding to k_2 and k_3. If necessary an additional term was added to analyze for triphasic decay.

Results

Following flash excitation of heterocysts under 90% H_2 plus 10% O_2, very little cytochrome *b*-563 turnover is visible [9]. HQNO inhibits the reoxidation of cytochrome *b*-563 [9], permitting estimation of the total active cytochrome *b*-563 pool and its rate of reduction. The observed light-induced responses at different concentrations of HQNO can then be analyzed by use of Eqn. 1. The data require the assumption of a fast and a slow phase of cytochrome *b*-563 relaxation; the magnitudes of these phases, calculated from Eqn. 1, are plotted separately in Fig. 1. In the absence of HQNO, rapid reoxidation of cytochrome *b*-563 occurs almost simultaneously with its reduction ($t_{0.5} = 1$–2

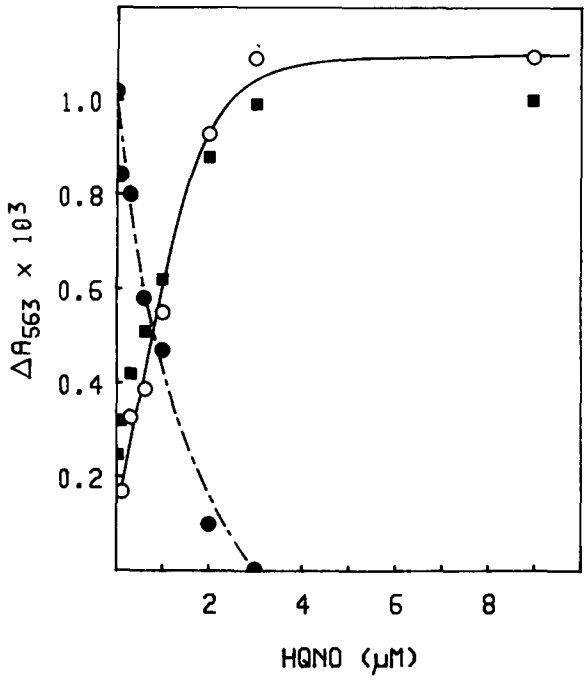

Fig. 1. Effect of HQNO on the observed amplitude of the cytochrome *b*-563 response following a flash, and on the calculated magnitudes of the fast and slow phases of cytochrome *b*-563 reoxidation. (■) Maximum observed absorbance change at 563 nm, (●) amplitude of fast phase, (○) amplitude of slow phase. Calculations were based upon Eqn. 1 as described in Methods.

ms for both), thus accounting for the small apparent turnover. At 3 μM HQNO the fast phase is completely eliminated and only a slow relaxation remains, which increases in parallel with the observed extent of the light-induced response.

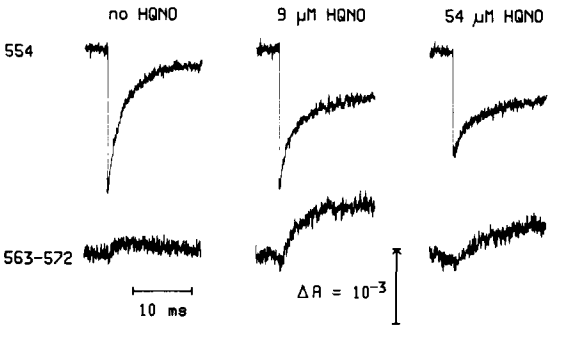

Fig. 2. Effect of HQNO on the reduction of cytochrome *c* and cytochrome *b*-563. Traces are averages of 64 events. Flash frequency = 0.3 Hz.

We have previously demonstrated that low concentrations of HQNO have little effect on the rates of reduction of cytochrome *c* and cytochrome *b*-563 following a flash [9]. At higher concentrations of HQNO, however, both of these reactions are slowed as shown in Fig. 2. In *Anabaena* strain 7120 either plastocyanin or cytochrome *c*-553 will function as the mediator between cytochrome *bf* and P-700 depending on Cu^{2+} levels in the growth medium [18]. When cells are grown on Cu^{2+}-free medium, cytochrome *c*-553 functions exclusively in this role, and the transfer of electrons through the Rieske Fe-S center and cytochrome *f* can be entirely accounted for by following the absorbance change at 554 nm. Plastocyanin, in contrast, has a weak and broad difference absorption band which is less suitable for spectroscopic studies. Because of the relative rates of cytochrome *c*-553 oxidation and reduction, the extent of cytochrome *c*-553 turnover in Cu^{2+}-free heterocysts is nearly equal to the amount of P-700 oxidized by a flash.

The traces shown in Fig. 2 were collected from heterocysts grown on Cu^{2+}-free medium. In the

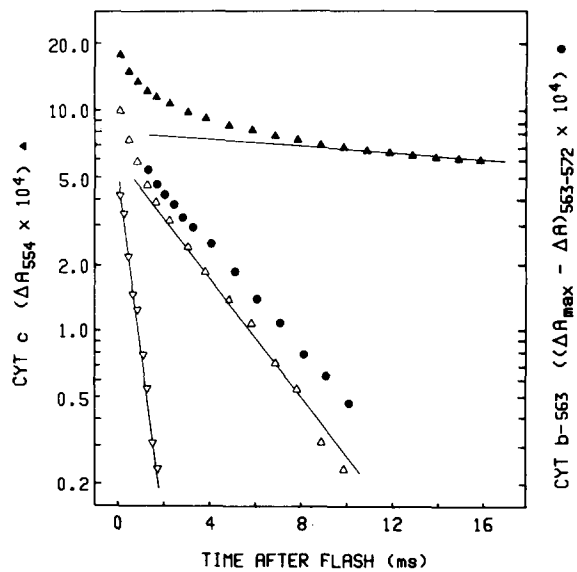

Fig. 3. Kinetic resolution of the reduction time courses of cytochrome *c* and cytochrome *b*-563. Data were obtained in the presence of 9 μm HQNO under the conditions used in Fig. 2. Cytochrome *c* reduction is resolved into three components by sequential semilogarithmic plots. (▲) Cytochrome *c* reduction, (△ and ▽) replots of the faster phases of cytochrome *c* reduction, (●) cytochrome *b*-563 reduction.

absence of HQNO little cytochrome *b*-563 reduction is visible and cytochrome *c* relaxation is rapid and complete within 20 ms. The site of rapid cytochrome *b*-563 reoxidation is completely blocked by 9 μM HQNO, and the full extent of cytochrome *b*-563 reduction is visible at this inhibitor concentration. At 54 μM, HQNO also inhibits the reductions of cytochrome *c* and cytochrome *b*-563.

The extent of cytochrome *c* oxidation in the presence of 9 μM HQNO is approx. 2.5-times that of cytochrome *b*-563 reduction, indicating that the ratio of P-700:cytochrome *bf* turning over is approx. 2.5 and that multiple turnovers of the cytochrome *b-f* complex are required for complete relaxation of the cytochrome *c* pool. Exponential analysis (Fig. 3) of the cytochrome *c* reduction reveals three phases of relaxation at 9 μM HQNO. The fastest phase, with a half-time of about 0.5 ms, probably corresponds to electron transfer from the Rieske Fe-S protein into the cytochrome *c* pool. This electron transfer has been reported to occur in less than 1 ms in spinach chloroplasts [19] and photosynthetic bacteria [20]. The second phase of cytochrome *c* reduction ($t_{0.5} = 2.8$ ms) probably corresponds to transfer of an electron from PQH_2 into the cytochrome *c* pool. This phase nearly equals the cytochrome *b*-563 reduction in both magnitude and half-time (Fig. 3) in support of the Q-loop model [6] which states that plastosemiquinone generated in this step serves as electron donor for cytochrome *b*-563 reduction. If multiple turnovers of the cytochrome *b-f* complex are prevented by the addition of HQNO, a large slow phase of cytochrome *c* relaxation ($t_{0.5} = 45$ ms) appears.

The need for multiple turnovers of the cyto-

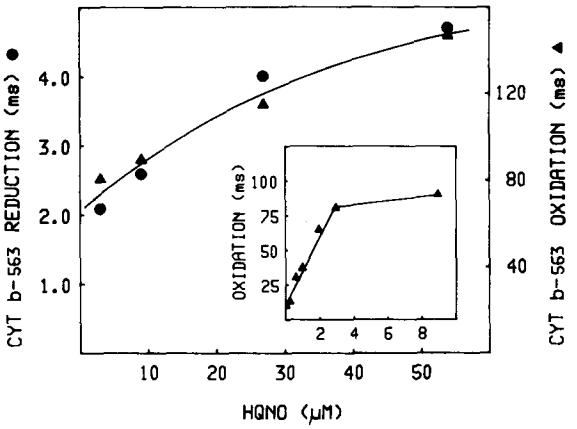

Fig. 5. Effect of HQNO on the half-times of reduction and reoxidation of cytochrome *b*-563. (●) Cytochrome *b*-563 reduction, (▲) cytochrome *b*-563 reoxidation. The inset shows the effect of low concentrations of HQNO on the half-time of the slow phase of cytochrome *b*-563 oxidation.

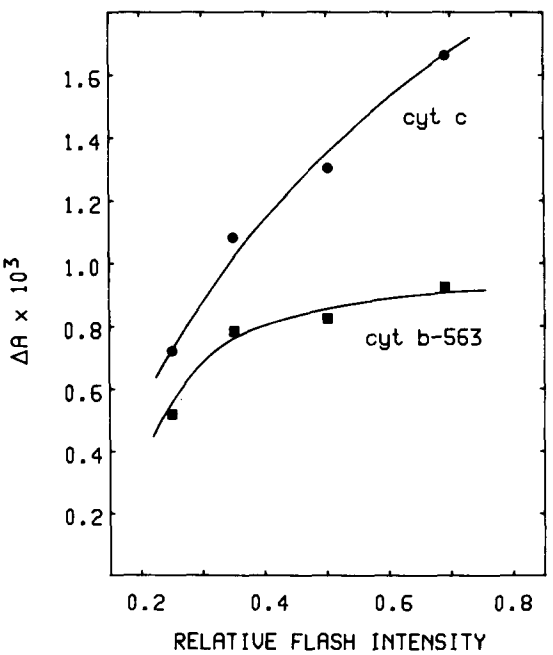

Fig. 4. Extents of cytochrome turnover as a function of relative intensity of the actinic flash. (■) Increase in absorbance at 563–572 nm (cytochrome *b*-563), (●) bleaching at 554 nm (cytochrome *c*). Saturating flashes, used elsewhere in this work, correspond to a relative intensity of 1.0.

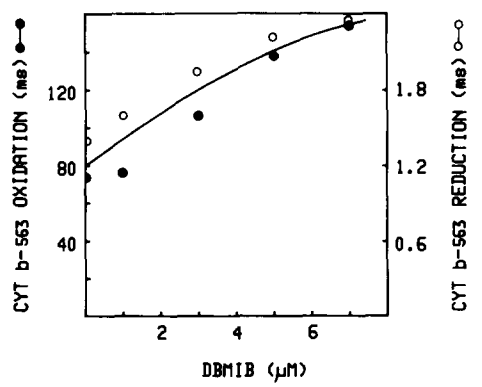

Fig. 6. Effect of DBMIB on the half-times of reduction and reoxidation of cytochrome *b*-563 in heterocysts inhibited with 5 μM HQNO.

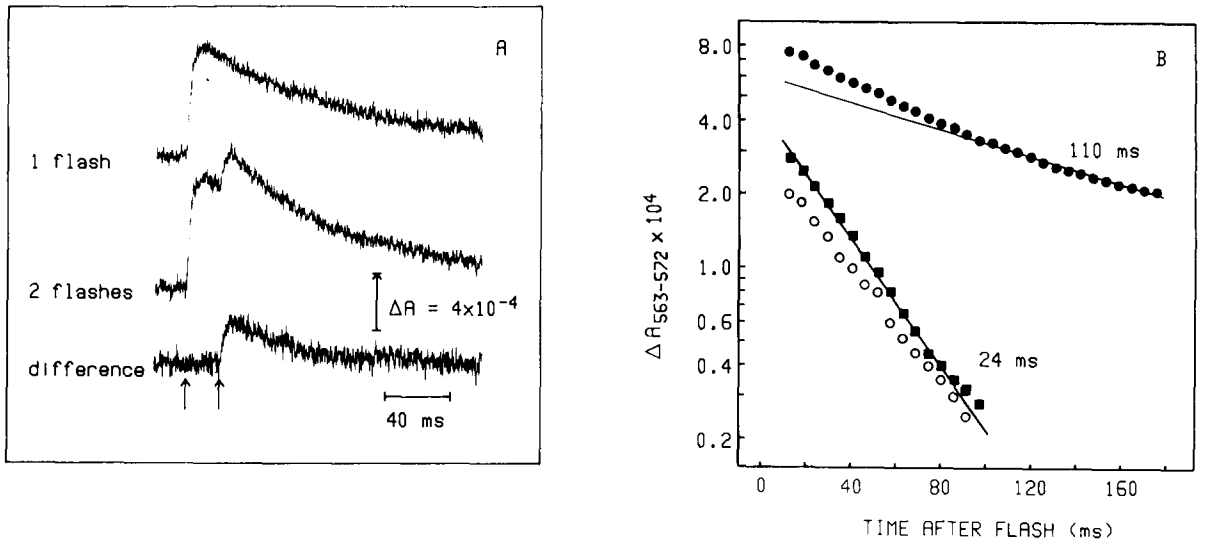

Fig. 7. Kinetics of cytochrome *b*-563 oxidation after one or two actinic flashes in the presence of 5 μM HQNO. (A) Cytochrome *b*-563 turnovers measured at 563–572 nm. Traces are averages of 128 events. Flash frequency = 1.0 Hz. Actinic flashes separated by 20 ms were delivered at the arrows. (B) Semilogarithmic plots of cytochrome *b*-563 reoxidation. (●) One flash, (○) replot of the fast phase of reoxidation after a single flash, (■) decay of two-flash minus one-flash difference shown in A.

chrome *b*-*f* complex can be eliminated if the amount of P-700 turnover is decreased by lowering the intensity of the actinic flash. Fig. 4 shows the magnitude of the cytochrome *b*-563 and cytochrome *c* turnovers, in the presence of 5 μM HQNO, at subsaturating flash intensities. The magnitude of the cytochrome *b*-563 turnover remains constant as long as there is excess turnover of cytochrome *c* (and thus, P-700); without such excess, the cytochrome *b*-563 turnover declines along with that of cytochrome *c*. At the lowest flash intensity shown in Fig. 4, the slowest phase of cytochrome *c* relaxation (Fig. 3) was completely eliminated. Two faster phases remained; the half-time for the slower of these was nearly equal to the half-time for cytochrome *b*-563 reduction (data not shown).

Although the rapid phase of cytochrome *b*-563 reoxidation is prevented by 3 μM HQNO, slow reoxidation ($t_{0.5} \approx 80$ ms) still occurs at this inhibitor concentration. The inset in Fig. 5 shows the effect of low concentrations of HQNO on the rate of cytochrome *b*-563 reoxidation following a flash. A sharp break in the inhibition profile occurs at a concentration above 3 μM. Fig. 5 shows that additional, albeit less dramatic, inhibition of reoxidation occurs at higher HQNO concentrations, sug-

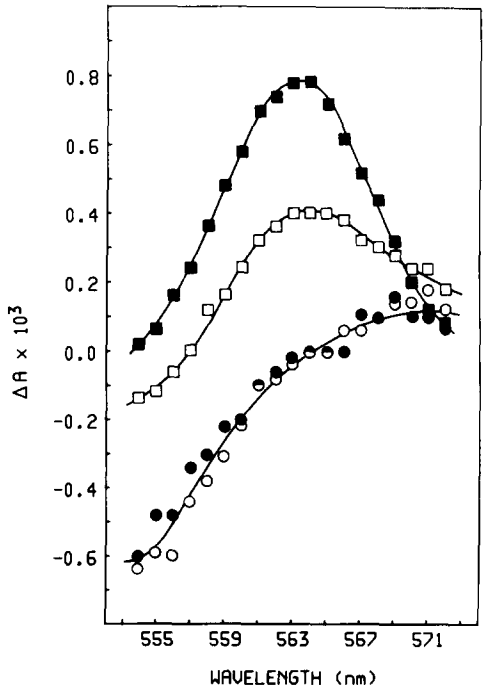

Fig. 8. Time-resolved, flash-induced difference spectra following the first or second flashes. Absorbance changes for the first and second flashes were measured relative to the absorbance values immediately before each flash. (●) 0.7 ms after first flash, (■) 5.3 ms after first flash, (○) 0.7 ms after second flash, (□) 5.3 ms after second flash.

gesting a second site of inhibition that is blocked only at high HQNO levels. Parallel inhibition of cytochrome *b*-563 reduction and reoxidation by these high HQNO concentrations (Fig. 5) suggests that reoxidation of cytochrome *b*-563 may involve a back-reaction through the PQH_2 donation site.

Further evidence for this reoxidation pathway can be obtained with a combination of HQNO and DBMIB, as shown in Fig. 6. HQNO at 5 μM blocks the site of rapid cytochrome *b*-563 reoxidation and DBMIB acts at the site of electron donation by PQH_2 to the cytochrome *b-f* complex [21]. Increasing concentrations of DBMIB inhibit both the reduction of cytochrome *b*-563 and also the HQNO-insensitive reoxidation of cytochrome *b*-563.

When two closely spaced actinic flashes are applied to HQNO-inhibited heterocysts, the additional cytochrome *b*-563 reduction elicited by the second flash has reoxidation kinetics that are distinct from those obtained with a single flash. Cytochrome *b*-563 turnovers obtained with one or two flashes are shown in Fig. 7A, and Fig. 7B shows semilogarithmic plots of the reoxidation kinetics from the first or second flashes. After a single flash, two phases of decay are apparent. The slower phase ($t_{0.5} \approx 110$ ms) accounts for about 70% of the total decay and the remainder has a $t_{0.5}$ of 24 ms. The additional cytochrome *b*-563 reduction on the second flash decays monophasically with the faster half-time. Flash-induced difference spectra are shown in Fig. 8. On both flashes there is a rapid oxidation of cytochrome *c*, followed by a slower reduction of cytochrome *b*-563 observed at 5.3 ms.

Discussion

The dramatic increase in the magnitude of apparent cytochrome *b*-563 turnover, which results from addition of HQNO, suggests that only a fraction of the true cytochrome *b*-563 turnover is observed in its absence. Attempts to measure cytochrome *b*-563 reduction rates under such conditions lead to a substantial underestimation of the cytochrome *b*-563 reduction half-time; quite different values are found if an inhibitor of cytochrome *b*-563 reoxidation is provided. This is illustrated in Fig. 9, which shows how the reoxidation kinetics can interfere with an accurate measurement of both the magnitude and the half-times of cytochrome responses. It can be seen in the figure that considerably less time is required to achieve 50% of the maximum apparent reduction if a substantial fraction of the turnover is obscured by the competing reoxidation reaction.

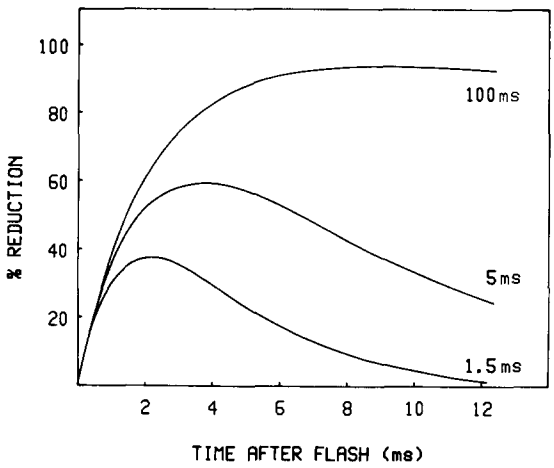

Fig. 9. Theoretical profiles of cytochrome turnovers that would be observed with a fixed rise time and varying decay times. These data were generated from a one-term equation of the type shown in Methods with a monophasic reduction half-time of 1.5 ms and monophasic reoxidation half-times as shown in the figure.

Cytochrome turnover kinetics measured in the presence of HQNO should be interpreted with caution, since this inhibitor, at higher concentrations, can also block electron flow from PQH_2 to the cytochrome *b-f* complex. Selak and Whitmarsh [10] observed a similar effect in spinach chloroplasts where 50 μM HQNO was found to inhibit cytochrome *f* reduction as well as cytochrome *b*-563 reoxidation.

In all cytochrome *b-f* complexes studied so far, the two cytochrome *b* hemes differ in midpoint potential by about 100 mV, and this was recently confirmed in the cytochrome *b-f* complex from *Anabaena variabilis* [22]. Therefore, we will refer to the more positive component as cytochrome b_H and the more negative component as cytochrome b_L. We believe that the slow phase of cytochrome *b*-563 reoxidation seen in Fig. 7B is attributable to oxidation of cytochrome b_H, and the fast phase, which occurs when both cytochrome *b*-563 hemes

in the cytochrome *b-f* complex are reduced, corresponds to the oxidation of cytochrome b_L. The occurrence of both phases of decay following the first flash is a result of multiple turnovers of the cytochrome *b-f* complex with each flash. Although the stoichiometry of P-700:cytochrome *bf* turning over with each flash would require more than two turnovers of the cytochrome *b-f* complex, only partial reduction of the cytochrome b_L appears to occur. This probably results from an equilibrium between plastosemiquinone generated by the second turnover and reduced cytochrome b_L. The additional cytochrome *b*-563 reduction on the second flash consists only of reduction of cytochrome b_L in those complexes where cytochrome b_H is already reduced.

The slowest phase of cytochrome *c* relaxation that is observed in the presence of HQNO also results from the unequal stoichiometries of P-700:cytochrome *bf*. If cytochrome *b*-563 is reduced and its reoxidation is prevented by HQNO, electron donation to the cytochrome *c* pool is impeded. This slow phase of cytochrome *c* reduction ($t_{0.5}$ = 40–50 ms) may require electron donation from plastosemiquinone to the Rieske iron-sulfur center and ultimately to the cytochrome *c* pool, or alternatively may first require release of the semiquinone from the cytochrome *b-f* complex and binding of a second molecule of PQH_2 to serve as electron donor.

Using chemical titrations we have measured P-700:cytochrome *bf* ratios in heterocysts of 2.0–2.5 (unpublished data); a similar value was reported by Almon and Böhme [13]. A comparable P-700:cytochrome *bf* ratio is observed in flash-induced turnovers, although only about 50% of the chemically titratable P-700 or cytochrome *bf* is found to participate.

In chloroplasts, the P-700:cytochrome *bf* ratio is close to 1.0 [23]; however, the heterogeneous lateral distribution of membrane complexes between grana and stroma thylakoids gives rise to localized regions of unequal stoichiometry. Anderson [23] reported a P-700:cytochrome *f* ratio greater than 2.0 in stroma thylakoids, and this might result in unequal stoichiometries of cytochrome *b-f* complex and P-700 participating in single-turnover flashes. In such circumstances the rate of cytochrome *f* reduction following a flash would not accurately

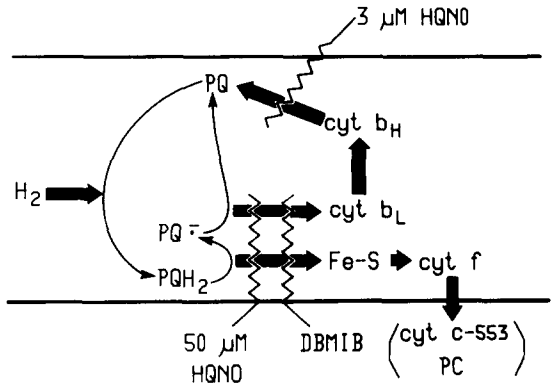

Fig. 10. A proposed sequence of electron transfer depicting the sites of HQNO inhibition in the cyanobacterial cytochrome *b-f* complex. Electron transfers are indicated by heavy arrows.

reflect the rate of electron transfer from PQH_2 to the cytochrome *b-f* complex, since rapid equilibration of cytochrome *f* with plastocyanin would necessitate more than one turnover of the cytochrome *b-f* complex for full relaxation of cytochrome *f* and would lead to spuriously low rates of apparent cytochrome *f* reduction. This may explain why the reduction of cytochrome *b*-563 in chloroplasts appears to precede cytochrome *f* reduction [10] in conflict with the expectation based on a Q-loop mechanism. The ability to manipulate the heterocyst system by growth on Cu^{2+}-free medium, so that plastocyanin is replaced by cytochrome *c*-553, allows the accurate measurement of electron flow from PQH_2 into the cytochrome *c* pool and demonstration (Fig. 3) that the kinetics of cytochrome *b*-563 reduction coincide with the kinetics of (one phase of) cytochrome *c* reduction. Hence, it is unnecessary to postulate a Q-loop mechanism [10] in which reduction of cytochrome *b*-563 precedes reduction of cytochrome *f*.

A proposed sequence of electron transfers through the cytochrome *b-f* complex and the sites of HQNO inhibition are shown in Fig. 10. The basic sequence of electron transfer is very similar to that proposed initially for mitochondria [6] with PQH_2 first reducing the Rieske Fe-S center and plastosemiquinone reducing cytochrome b_L. The relative positions of cytochrome b_L and cytochrome b_H in this scheme are based on proposals for the analogous components in mitochondria

where semiquinone is presumed to react with cytochrome *b*-566 ($E_{m,7} = -40$ mV [2]) which subsequently reduces cytochrome *b*-562 ($E_{m,7} = +40$ mV). The reduction of PQ by cytochrome b_H and release of PQH_2 could be achieved by successive electron transfers from cytochrome b_H requiring two turnovers of the cytochrome *b-f* complex. Hydrogenase probably reduces PQ by an independent pathway. The sites of HQNO inhibition in Fig. 10 are consistent with the observation that low HQNO concentrations block cytochrome *b*-563 reoxidation without affecting reduction of cytochrome *b*-563 and cytochrome *c*, whereas higher HQNO concentrations inhibit the reduction of cytochrome *c* and cytochrome *b*-563. This scheme is fully consistent with the observed kinetics of cytochrome turnovers following single-turnover flashes, and the results presented herein provide evidence for the existence of a Q-loop mechanism in the photosynthetic electron-transfer chain of cyanobacteria.

Acknowledgements

We thank Robert D. Clark and Keith Thompson for assistance with data analysis. This work was supported by the Competitive Research Grant Office of the U.S. Department of Agriculture/Science and Education Administration. It was conducted at Brookhaven National Laboratory under the auspices of, and with additional support from, the United States Department of Energy.

References

1 Trumpower, B.L. and Katki, A.G. (1979) in Membrane Proteins in Energy Transduction (Capaldi, R.A., ed.), pp. 89–200, Marcel Dekker, New York
2 Crofts, A.R., Meinhardt, S.W. and Bowyer, J.R. (1982) in Function of Quinones in Energy Conserving Systems (Trumpower, B.L., ed.), pp. 477–498, Academic Press, New York
3 Krinner, M., Hauska, G., Hurt, E. and Lockau, W. (1982) Biochim. Biophys. Acta 681, 110–117
4 Nelson, N. and Neumann, J. (1972) J. Biol. Chem. 247, 1817–1824
5 Hurt, E. and Hauska, G. (1981) Eur. J. Biochem. 117, 591–599
6 Mitchell, P. (1976) J. Theor. Biol. 62, 327–367
7 Bowyer, J.R. and Trumpower, B.L. (1981) J. Biol. Chem. 256, 2245–2251
8 Bowyer, J.R. and Crofts, A.R. (1981) Biochim. Biophys. Acta 636, 218–233
9 Houchins, J.P. and Hind G (1983) Arch. Biochem. Biophys. 224, 272–282
10 Selak, M.A. and Whitmarsh, J. (1982) FEBS Lett. 150, 286–292
11 Stewart, W.D.P. (1980) Annu. Rev. Microbiol. 34, 497–536
12 Tel-Or, E. and Stewart, W.D.P. (1977) Proc. R. Soc. B 198, 61–86
13 Almon, H. and Böhme, H. (1980) Biochim. Biophys. Acta 592, 113–120
14 Peterson, R.B., Dolan, E., Calvert, H.E. and Ke, B. (1981) Biochim. Biophys. Acta 634, 237–248
15 Cammack, R., Luijk, L.J., MaGuire, J.J., Fry, I.V. and Packer, L. (1979) Biochim. Biophys. Acta 548, 267–275
16 Houchins, J.P. and Hind, G. (1982) Biochim. Biophys. Acta 682, 86–96
17 Allen, M.B. and Arnon, D.I. (1955) Plant Physiol. 30, 366–372
18 Sandman, G. and Böger, P. (1980) Plant Sci. Lett. 17, 417–424
19 Whitmarsh, J., Bowyer, J.R. and Crofts, A.R. (1982) Biochim. Biophys. Acta 682, 404–412
20 Meinhardt, S.W. and Crofts, A.R. (1982) FEBS Lett. 149, 217–222
21 Trebts, A. (1980) Methods Enzymol. 69, 675–715
22 Hurt, E.C. and Hauska, E. (1983) FEBS Lett. 153, 413–419
23 Anderson, J.M. (1982) FEBS Lett. 138, 62–66