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CYTOCHROME FUNCTION IN THE CYCLIC ELECTRON TRANSPORT PATHWAY OF CHLOROPLASTS

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Summary

Flash excitation of isolated intact chloroplasts promoted absorbance transients corresponding to the electrochromic effect (*P*-518) and the α-bands of cytochrome b_6 and cytochrome *f*. Under conditions supporting coupled cyclic electron flow, the oxidation of cytochrome b_6 and the reduction of cytochrome *f* had relaxation half-times of 15 and 17 ms, respectively. Optimal poising of cyclic electron flow, achieved by addition of 0.1 μM 3-(3,4-dichlorophenyl)-1,1-dimethylurea, increased phosphorylation of endogenous ADP and prolonged these relaxation times. The presence of NH_4Cl, or monensin plus NaCl, decreased the half-times for cytochrome relaxation to approximately 2 ms. Uncouplers also revealed the presence of a slow rise component in the electrochromic absorption shift, with formation half-time of about 2 ms. The inhibitors of cyclic phosphorylation antimycin and 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone abolished the slow rise in the electrochromic shift and prolonged the uncoupled relaxation times of cytochromes b_6 and *f* by factors of ten or more.

These observations indicate that cytochrome b_6, plastoquinone and cytochrome *f* participate in a coupled electron transport process responsible for cyclic phosphorylation in intact chloroplasts. Estimations of cyclic phosphorylation rates from 40 to 120 μmol ATP/mg chlorophyll per h suggest that this process can provide a substantial fraction of the ATP needed for CO_2 fixation.

Abbreviations: DBMIB, 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone; DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea; CCCP, carbonylcyanide-*m*-chlorophenylhydrazone; *P*-700, reaction center pigment for Photosystem I.

Introduction

Studies of intact chloroplasts and thylakoid membranes reconstituted with ferredoxin have shown that cyclic photophosphorylation driven by Photosystem I is inhibited by the plastoquinone antagonist 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone (DBMIB) [1,2] and by antimycin [2—4]. The observation that linear and cyclic electron flows are similarly sensitive to DBMIB and uncouplers prompted the suggestion [1,5] that these two processes share a common coupling site between plastoquinone and cytochrome *f*. Antimycin, by contrast, is a specific inhibitor of the cyclic system; at antimycin concentrations which inhibit cyclic phosphorylation [2,3] or other energy-dependent parameters [6], linear electron flow is neither uncoupled nor inhibited [7]. The inhibition site for antimycin has thus been ascribed to a part of the cyclic pathway not shared with the linear pathway [3,4,7]. By analogy with mitochondria [8] and photosynthetic bacteria [9,10], this site is presumed to lie between the *b*- and *c*-type chloroplast cytochromes.

Currently, only scant kinetic evidence has been presented for the participation of cytochromes b_6 and *f* in the chloroplast cyclic system [11], and there has been no evidence for an electrogenic loop between the chloroplast cytochromes, such as that observed in bacterial chromatophores [9,10] or with algae [31—33]. However, similar complex kinetics have been noted for the flash-induced 515 nm absorbance change in intact chloroplasts [34]. The results here form a preliminary kinetic study of cytochrome b_6 and *f* turnovers and of the electrochromic effect in intact chloroplast preparations known to have cyclic activity.

Materials and Methods

Intact CO_2-fixing chloroplasts were isolated from spinach as described previously [12]. Measurements at 18°C were performed with samples composed of chloroplasts in 'reaction buffer' consisting of 0.36 M sorbitol, 50 mM Tricine and 0.3 mM K_2HPO_4 adjusted to pH 8.1. Samples also contained 1200 units of catalase/ml to prevent H_2O_2 buildup during illumination [13]. The chlorophyll concentrations were 70 μg/ml for steady state and 50 μg/ml for flash-induced absorption change measurements. Steady-state spectra were obtained by the technique of Rapp and Hind [14] with the spectrophotometer in single beam mode. Flash-induced absorption changes with peaks at 518, 554 and 564 nm were kinetically resolved by the procedure of Dolan and Hind [11] with the following modifications: (i) elimination of the *P*-518 component from the signals at 554 and 564 nm was achieved by appropriate digital subtractions of the change at 518 nm rather than 531 nm (see Ref. 11 and text); (ii) flash intensity was doubled by illuminating the sample cuvette from opposing directions, at right angles to the measuring beam, with two EG and G FX201 xenon lamps; (iii) flash spectral composition was limited to greater than 650 nm by Kodak Wratten 70 filters and had a 4 μs pulse width at half peak height; (iv) flash frequency was 1.5 Hz. A single flash from both lamps resulted in at least 90% saturation of the 518 nm change. Resolution of the relaxation kinetics was achieved by non-linear least-squares regression analysis on a CDC

7600 computer. Calculated half-times fell within a standard error of ±10% with the exception of those for cytochrome *f* in Fig. 5a and c where the error was ±45%.

Endogenous electron acceptors such as HCO_3^- were omitted from the reaction medium to prevent reoxidation of NADPH and promote cyclic electron flow. Calculations, based on a photosynthetic unit size of 1/500 chlorophylls and a total $NADP^+$ plus NADPH pool of 10 nmol/mg chlorophyll in the stroma [15], indicated that 20 flashes should completely reduce the $NADP^+$ pool; consequently, 20–30 preilluminating flashes were given prior to acquisition of data.

The photophosphorylation of endogenous stromal ADP was measured as previously described [6].

Results

Fig. 1 displays a light minus dark difference spectrum for intact chloroplasts subjected to weak continuous red illumination in the presence of monensin and NaCl. The absorbance changes associated with the α-bands of cytochrome *f* (λ_{max} = 554 nm) and cytochrome b_6 (λ_{max} = 564 nm) are superimposed on a large positive signal with a peak at about 518 nm (P518). If 545 and 575 nm

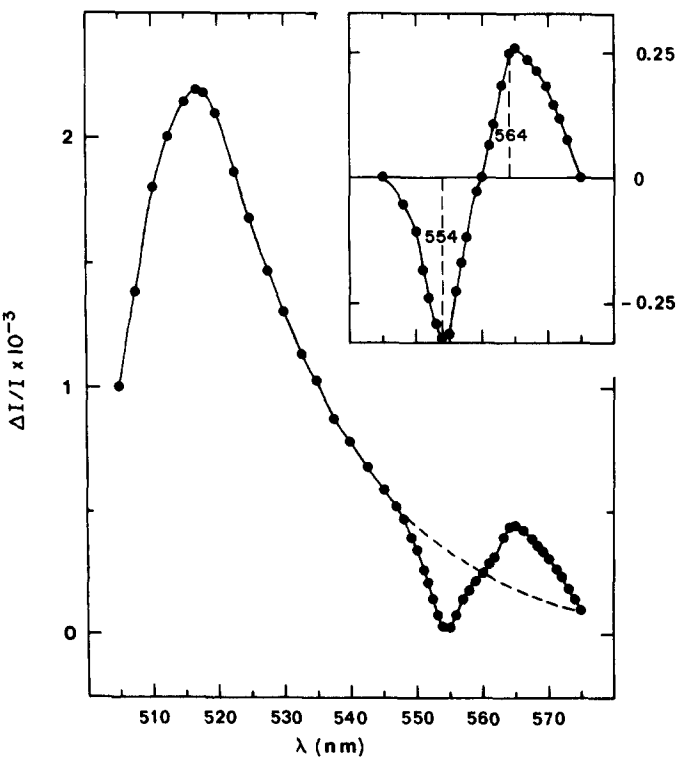

Fig. 1. Light minus dark steady-state difference spectrum of intact chloroplasts. Illumination was with 50 W/m² of red (Corning 2-58) light. Samples were prepared in 'reaction buffer' supplemented with 2 μM monensin and 20 mM NaCl. Inset: curve obtained by subtracting solid curve from dashed line.

are assumed to be isosbests for the cytochrome responses [5,17], and the tail of the *P*-518 response is extrapolated within this region (dashed line) then the α-band difference spectrum shown in the inset can be obtained by subtraction. In this corrected spectrum cytochromes *f* and b_6 are clearly responsible for the distinct peaks at 554 nm and 564 nm. The broad absorbances of plastocyanin [35], *P*-700$^+$ [23] and *P*-518 [11] in the α-band region may contribute to the net displacement observed in the spectrum.

The above conclusion is reinforced by the flash excitation studies presented in Fig. 2. The absorbance spectrum 700 μs after an excitation flash is predominantly that of the electrochromic effect (*P*-518) with peaks at 480 and 518 nm, as was also obtained by Vredenberg and Schapendonk [17] with intact chloroplasts. This spectrum also agrees well with that observed 320 μs after flash illumination of *Chlorella* [18]. The cytochrome changes, at 700 μs, are superimposed upon the tail of the *P*-518 signal as in the steady-state spectrum of Fig. 1. After 44 ms, the cytochrome changes have completely relaxed and the spectrum between 500 and 575 nm is essentially that of *P*-518 [11]. In order to remove the *P*-518 absorbance contribution from the α-band region, the 44 ms spectrum was normalized to match the 518 nm peak in the 700 μs spectrum and then subtracted. The result, displayed in the inset of Fig. 2, shows that 700 μs after the flash there is a substantial oxidation of cytochrome *f* together with a smaller apparent reduction of cytochrome b_6. This finding agrees with earlier reports showing that cytochrome *f* is fully oxidized 700 μs after a flash [11,18] whereas cytochrome b_6 is only partially reduced. The half-time for cytochrome b_6 reduction has been estimated at 1.3 ms [11].

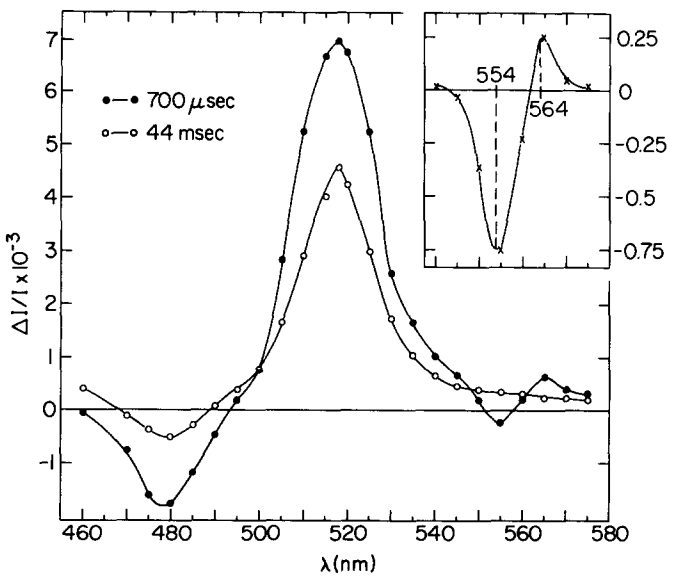

Fig. 2. Wavelength dependence of flash-induced absorption changes 700 μs and 44 ms after the flash. Samples contained 2 μM monensin and 20 mM NaCl and were otherwise as in Materials and Methods. The data at 700 μs (•———•) and 44 ms (o———o) for each wavelength represent the average of between 128 and 1024 flashes given at 3 Hz. Inset: spectrum obtained by normalizing the 44 ms curve at 518 nm and subtracting the normalized curve from the 700 μs curve.

From the above spectral observations was determined the number of repetitive absorbance transients, measured at 518 nm, required to strip the *P*-518 contribution from the absorbance changes in the α-band region. Values of 7.2% and 5.4% were used for the relative *P*-518 absorbance contribution at 554 and 564 nm, respectively, in subsequent curve-stripping procedures (Figs. 4 and 5).

Fig. 3 documents the effect of flash frequency on the electrochromic change plotted as the relative amplitude at selected time intervals after the flash. The signal amplitude is relatively constant for repetitive flash frequencies between 0.5 and 2 Hz but at higher frequencies there is a linear decline in amplitude indicative of incomplete relaxation between flashes. This is consistent with the 500 ms dark recovery time for the electrochromic effect recently observed with intact chloroplasts [17]. The secondary rise in the 518 nm change (see Fig. 4b for example) shows a similar behavior with the minor exception that its peak height occurs at progressively shorter time intervals (as noted in brackets) after the flash. The dark relaxation of cytochromes *f* and b_6 is essentially complete within 50 ms (cf. Fig. 2) therefore a flash frequency of 1.5 Hz was chosen to maximize both the cytochrome and electrochromic response in the following study.

The oxidation kinetics of cytochrome b_6 and the reduction kinetics of cytochrome *f* should both depend on the coupling state of the chloroplast if the cyclic pathway involves cytochrome b_6 and utilizes the coupled step between plastoquinone and cytochrome *f* [1,5]. These expectations are in agreement with the flash kinetics for intact chloroplasts depicted in Fig. 4. When chloroplasts are examined without additions (Fig. 4a) the amplitudes of the cytochrome b_6 and *f* responses to a flash are approximately equal and their dark

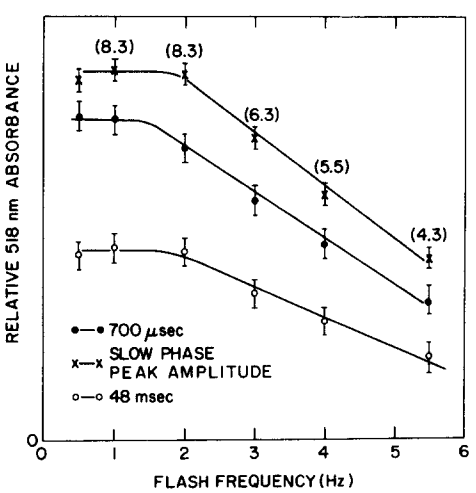

Fig. 3. The response of the 518 nm change at selected time intervals after the flash as a function of flash frequency. Samples contained 2 μM monensin and 20 mM NaCl as in Fig. 2. Data points indicate the relative signal amplitude 700 μs (•), 48 ms (○) or at the number of mseconds after the flash given in brackets (X). Each point represents the average of 128 sweeps. See Fig. 4b for example of actual signal trace.

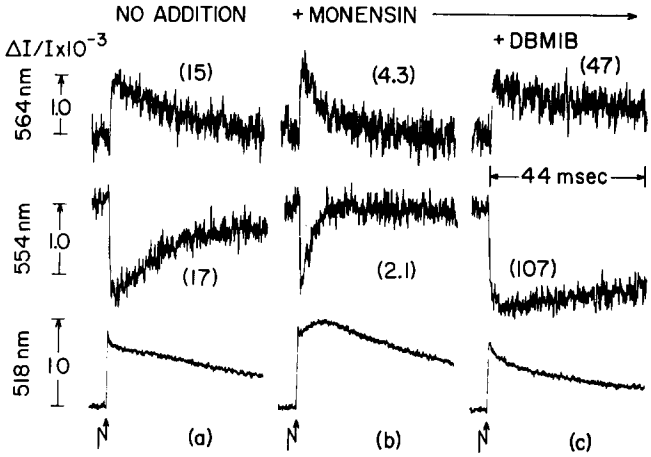

Fig. 4. Kinetic traces for the flash-induced absorption changes attributable to cytochrome b_6, cytochrome *f* and *P*-518 in intact chloroplasts. The 518-nm traces represent the accumulated average of 128 sweeps. The cytochrome b_6 response was obtained by appropriate digital subtractions to strip 5.4% of the averaged 518 nm absorbance from that measured after 512 sweeps at 564 nm. The cytochrome *f* response was obtained by subtraction of 7.2% of the averaged 518 nm absorbance from that measured after 512 sweeps at 554 nm. Otherwise the absorption changes were recorded as described in Materials and Methods and Ref. 11. Chloroplast samples were prepared with 'reaction buffer' supplemented with 20 mM NaCl. Other additions were as follows: trace a, none; trace b, 2 μM monensin; trace c, 2 μM monensin plus 2 μM DBMIB. Numbers in brackets give the computed first-order half-times for cytochrome b_6 oxidation and cytochrome *f* reduction in mseconds.

relaxations nearly complete within 44 ms. Calculation gives a ratio of approximately 1.0 for cytochrome b_6 to *f* heme turnover, based upon extinction coefficients of 17 and 22 $mM^{-1} \cdot cm^{-1}$, respectively [16]. This suggests that, when the acceptor $NADP^+$ is not regenerated due to lack of added electron acceptors, light-driven electron transfer from cytochrome *f* to b_6 is not in competition with pseudocyclic electron flow [19]. It is also noteworthy that the 554 nm signal represents about half of the total chloroplast cytochrome *f* content of one heme/500 chlorophylls as determined from chemical redox titrations.

When monensin is added (Fig. 4b), proton uptake is diminished [20] and the $t_{0.5}$ value for cytochrome b_6 oxidation is decreased to 4.3 ms while that for cytochrome *f* reduction is lowered to 2.1 ms. These accelerated turnovers indicate the existence of a coupling site between cytochromes b_6 and *f*. Subsequent addition of DBMIB, shown in Fig. 4c, decreases the oxidation rate of cytochrome b_6 ten-fold and also drastically slows the reduction rate of cytochrome *f*. Presumably a quinone component functions in the coupled electron transfer step between cytochromes b_6 and *f*.

The discrepancy, seen in Fig. 4b, between the rates of cytochrome b_6 oxidation and cytochrome *f* reduction could be the result of a direct competition between electrons from Photosystem II and reduced cytochrome b_6 for a common pool of oxidized plastoquinone. If this assumption is correct, reduction of the plastoquinone pool by the cyclic route should be enhanced when electron flow from Photosystem II is at least partially blocked by 3-(3,4-dichlorophyl)-1,1-dimethylurea (DCMU). Table I summarizes this apparent poising effect of DCMU [4,6] on the cyclic phosphorylation activity of intact

TABLE I

DCMU POISING OF CYCLIC PHOSPHORYLATION

Values indicate the amount (nmol/mg chlorophyll) of endogenous stromal ADP phosphorylated after 15 s of continuous red (Corning 2-58, 100 W/m^2) illumination. Initial dark distributions of ATP, ADP and AMP in nmol/mg chlorophyll were 3.6, 15.4 and 4.5, respectively. Additions as indicated were 1.0 μM antimycin, 0.1 μM DCMU and 3.3 mM NH_4Cl. Measurements as in Ref. 6.

ATP		No addition	+DCMU	+DCMU + NH_4Cl
Total	— antimycin	7.2	9.8	2.1
	+ antimycin	5.7	6.2	—
Cyclic	(difference)	1.5	3.6	

chloroplasts illuminated with red light. Phosphorylation of the stromal ADP pool is greater when the partially inhibitory concentration of 0.1 μM DCMU [6] is present. This finding agrees with earlier observations that low concentrations of DCMU also increase energization of the thylakoid membrane [6]. The differences between the DCMU-poisoned and unpoisoned samples clearly show that DCMU more than doubles the antimycin-sensitive (cyclic) contribution to the overall phosphorylation process. Repetitive flash studies require that antimycin be used at less than fully inhibiting levels and for comparative purposes, these conditions were retained in Table I. Accordingly, the low DCMU concentration used enhanced phosphorylation, even when antimycin was present.

Kinetic traces for the cytochrome responses under the 0.1 μM DCMU-poisoned conditions are shown in Fig. 5. The oxidation of cytochrome b_6, presented in Fig. 5a, is slower than in the unpoisoned control shown earlier (Fig. 4a). There is also a considerable loss of cytochrome *f* signal amplitude and the reduction rate becomes biphasic with development of 8.6-ms and 80-ms components as seen earlier [11] with coupled chloroplasts. This decreased activity is apparently the result of a more tightly coupled state since the addition of NH_4Cl (Fig. 5b) greatly accelerates the rates of cytochrome b_6 oxidation and cytochrome *f* reduction while restoring the signals to their original amplitudes (Fig. 4a). With the exception of some residual slow oxidation of cytochrome b_6, the $t_{0.5}$ value of 1.9 ms, seen with DCMU and NH_4Cl present, more nearly approximates the corresponding 2.3 ms half-time for cytochrome *f* reduction. Similar results were obtained using monensin (as in Fig. 4) however, to illustrate the generality of uncoupler effects on these kinetics, data for NH_4Cl are presented.

Rapid cytochrome turnover, and presumably cyclic electron flow, is inhibited by antimycin as shown in Fig. 5c. The half-time for cytochrome b_6 oxidation is increased to 48 ms while the cytochrome *f* reduction half-time is also greatly extended by antimycin.

The phosphorylation and kinetic data, presented above, support the view that a low concentration of DCMU increases coupled electron flow through a pathway that is antimycin sensitive.

A slow rise in the 518 nm absorption change becomes prominent in the presence of uncouplers (Figs. 4 and 5b). Similar traces (not shown) were

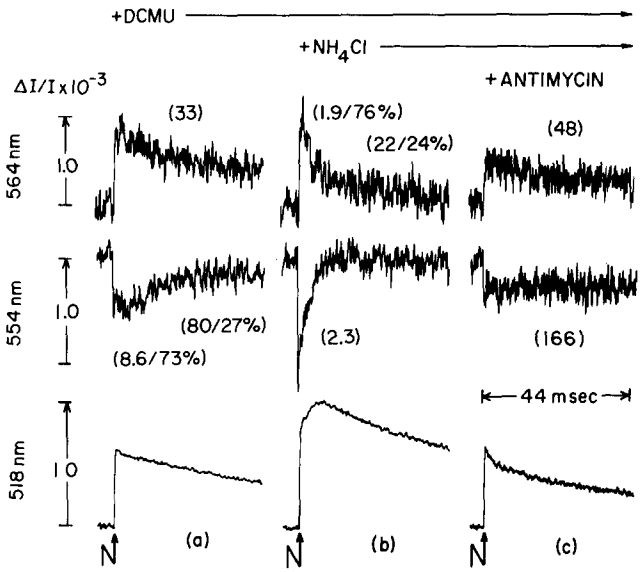

Fig. 5. Kinetic traces for the flash-induced absorption changes in intact chloroplasts partially inhibited with DCMU. Measurements as described in Fig. 4 and Materials and Methods. Chloroplast samples were prepared with 'reaction buffer' containing 0.1 μM DCMU. Further additions were as follows: trace a, none; trace b, 3.3 mM NH_4Cl; trace c, 3.3 mM NH_4Cl plus 2 μM antimycin. Numbers in brackets give the computed half-times in mseconds for oxidation or reduction and the computed percentage of the component relaxing with the given half-time (when more than one half-time is evident).

obtained with additions of 1.0 μM nigericin or 0.5 μM carbonylcyanide-*m*-chlorophenylhydrazone (CCCP). This rise is evidently associated with rapid electron transfer through cytochrome b_6, plastoquinone and cytochrome *f* since both DBMIB (Fig. 4c) and antimycin (Fig. 5c) abolish the rise while inhibiting electron flow in this region of the electron transport chain. Antimycin and DBMIB similarly block the slow rise when cyclic electron flow around Photosystem I has been induced by addition of dithionite to chloroplasts poisoned with 20 μM DCMU [36]. In order to estimate the formation rate of the secondary rise, the inhibited traces (Figs. 4c and 5c) were subtracted from the traces where only uncoupler was present (Figs. 4b and 5b). Regardless of uncoupler or inhibitor used, the results in Fig. 6a and b give a 2 ms half-time for development, which is comparable to the uncoupled electron transfer rate between the cytochromes. The amplitude of this rise (Fig. 6a) is approximately one-half the maximum signal observed 50—100 μs (1—2 signal averager address times) after the flash in unpoisoned chloroplasts (Fig. 4a). Subtractions of traces obtained under coupled conditions with DBMIB or antimycin (data not shown) also reveal the occurrence of a slow rise; however, the formation rate is considerably decreased as would be expected if a transthylakoid pH gradient, produced by repetitive flashes, slowed coupled electron transfer. The rise is indicative of an electrogenic rather than a light-scattering change since its spectrum (Fig. 6c) is similar to the electrochromic shift depicted in Fig. 2. The generation of such a shift milliseconds after a 4 μs flash and well beyond residual flash 'tails' is evidence for formation of a transthylakoid electrochemi-

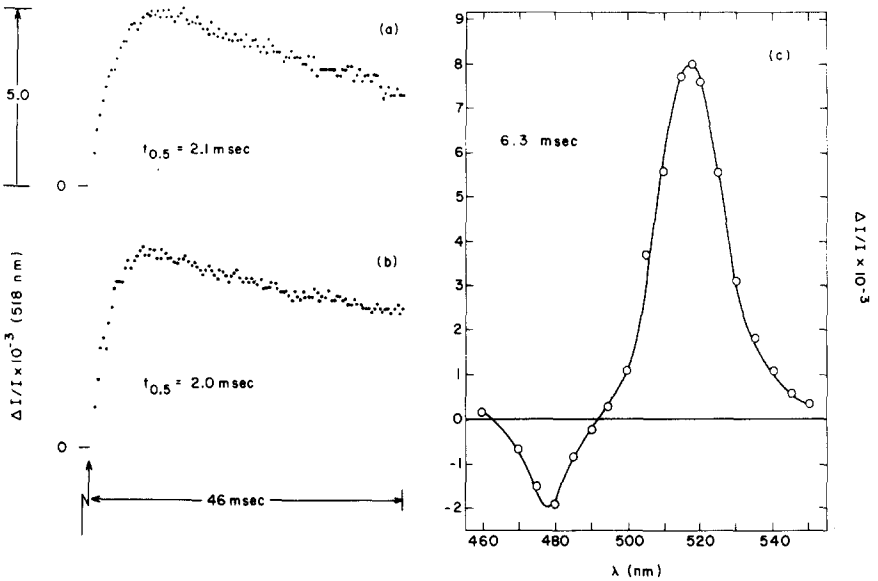

Fig. 6. Dark formation rate of the secondary 518 nm signal. Curve a was obtained by subtracting the trace (Fig. 4c) with DBMIB plus monensin from the trace (Fig. 4b) with monensin alone. Curve b was obtained by subtracting the trace (Fig. 5c) with NH_4Cl plus antimycin from the trace (Fig. 5b) with NH_4Cl alone. Curve c is the spectrum for the electrochromic shift obtained 6.3 ms after the flash as in Fig. 2.

cal potential dependent on postillumination transfer of electrons through a portion of the cyclic route containing a coupling site.

Conclusions

Under conditions favoring cyclic electron flow, relaxations of flash-induced changes in the cytochrome α-band absorbances are superimposed on the decay of a broad band electrochromic shift having a positive peak at 518 nm. After correction for this change, cytochromes b_6 and *f* are found to be responsible for essentially all of the absorbance changes between 540 and 570 nm. The absence of significant absorbance changes at 550 nm and 559 nm suggests that C550 [21] and cytochrome *b*-559 [5,16] are not involved in the cyclic pathway. This is not surprising since there is currently no evidence linking these components wit Photosystem I-mediated cyclic electron transport. The cyclic pathway does include *P*-700 as the primary photoreceptor [22] and the broad absorption peak for *P*-700$^+$ could contribute to the absorbance shift in the α-band region [23]. However, the rapid reduction rate of *P*-700$^+$, with estimated half-times of 10 and 200 μs [24,25], would seem to preclude a significant contribution at times greater than 700 μs after a flash.

The major conclusions to be drawn are:

(i) Cytochromes b_6 and *f* function on the electron donor and acceptor sides of the cyclic coupling site, respectively. Agents which facilitate a collapse in the pH gradient across the thylakoid membrane accelerate the rates of cytochrome

b_6 oxidation and cytochrome *f* reduction by seven-fold under repetitive flashes. Such accelerations are typical of those observed when linear, but not pseudocyclic electron flow is uncoupled [7].

(ii) The coupling site between the cytochromes involves a quinone since electron transfer is blocked by DBMIB. Experimental evidence from several laboratories suggests this quinone may be the same plastoquinone which functions in linear electron flow. Trebst and coworkers [1,3] have shown that the plastoquinone antagonist DBMIB inhibits linear electron flow and phosphorylation systems which require the participation of plastoquinone. CO_2 fixation with pyruvate as a substrate is dependent only on cyclic ATP production, and was shown to be DBMIB sensitive in intact chloroplasts [2]. Spectroscopic measurements by Böhme and Cramer [5] indicated that DBMIB inhibited reoxidation of cytochrome b_6 in the dark and increased the extent of cytochrome *f* photooxidation. Furthermore, work of Arnon and Chain [4] along with others [6,26] has shown that partial suppression of electron flow from Photosystem II is an essential prerequisite for optimizing cyclic activity in red light. This 'poising effect' presumably arises from the need to maintain a partially oxidized plastoquinone pool so that electrons can enter this intermediate by the cyclic route. Recent fluorescence measurements support the view that cyclic electron flow reduces the plastoquinone pool [27].

(iii) The antimycin inhibition site lies between the chloroplast cytochromes b_6 and *f*. Location of this site previously rested on the assumed analogy between chloroplasts and other energy-transducing membrane systems which involve a cytochrome *b* to cytochrome *c* electron transition [8—10]. This study provides the first kinetic evidence for such a site in chloroplasts. The lack of inhibitory effects on linear electron flow [7] indicates that the site of antimycin action lies between cytochrome b_6 and the point at which cyclic electron flow enters the electron carrier sequence between the photosystems.

(iv) Electron transfer through the cytochrome b_6 and *f* region of the cyclic pathway results in an electrochromic shift indicative of a charge separation process across the chloroplast thylakoid membrane. In its 2 ms rise time and its sensitivity to antimycin inhibition, this process is quite similar to the slow charge separation seen in chromatophores of *Rhodopseudomonas sphaeroides* [9,10] and *Rhodopseudomonas capsulata* [9], and invites comparison between the chloroplast and chromatophore electron transfer mechanisms. Past inability to observe or characterize the slow 518 nm rise could be the result of poor endogenous cyclic activity following the loss of soluble cofactors [4] in the preparation of chloroplast thylakoid membranes.

(v) The rate of cyclic phosphorylation is potentially significant as a supplement to coupled ($P/2\,e = 1.3$) linear electron flow in chloroplasts capable of sustaining CO_2 fixation rates of 100—200 μmol/mg chlorophyll per h [7]. Under the coupled condition of Fig. 4a, one can estimate a steady-state phosphorylation rate of 40 μmol ATP/mg chlorophyll per h even when ADP regeneration is slowed by the absence of either 3-phosphoglycerate or HCO_3^- [28]. This estimate is based on the turnover of half the total cytochrome *f* present in 40 ms and on the assumptions that (a) 1 H^+/e^- is transported during the oxidation of plastoquinone by cytochrome *f* and (b) 3 H^+ are required for each ATP formed [29]. During rapid ADP regeneration in the presence of

3-phosphoglycerate or HCO_3^- [28] a larger ADP pool would presumably permit cyclic electron flow at a rate 3—4-fold faster and comparable to that seen in the presence of uncouplers (Figs. 4b and 5b). Under coupled conditions, such a rate would be sufficient to sustain phosphorylation of 120 μmol ADP/mg chlorophyll per h (assuming only half the total cytochrome *f* participates). Though conservative in nature, these estimates are consonant with reported cyclic phosphorylation rates [1,3,4,26] and suggest that cyclic phosphorylation is capable of supplying a portion of the ATP needed for CO_2 fixation [30].

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