Biochimica et Biophysica Acta, 591 (1980) 381—390

BBA 47873

THE RELATIONSHIP OF THE CYCLIC AND NON-CYCLIC ELECTRON TRANSPORT PATHWAYS IN CHLOROPLASTS

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(Received August 20th, 1979)

Key words: Plastocyanin; P-700; Ferredoxin; Rieske iron-sulfur center; Cyclic electron transport; Non-cyclic electron transport

Summary

Light-induced redox changes of plastocyanin, the Rieske iron-sulfur center, and *P*-700 have been studied in situ in spinach chloroplasts. Plastocyanin and the Rieske center behaved in an analogous manner in that their steady states were fully oxidized in the light in the presence or absence of 3-(3,4-dichlorophenyl)-1,1-dimethylurea when an electron acceptor is present. After illumination under conditions of non-cyclic electron transfer from water to an electron acceptor, followed by a short dark period, the steady state of both shifted to a more reduced level. A 3-(3,4-dichlorophenyl)-1,1-dimethylurea-sensitive photoreduction of the Rieske center was observed in ferricyanide-washed chloroplast fragments. With reduced ferredoxin as electron donor, it was possible to demonstrate a reduction in the dark of these electron carriers and of *P*-700; this reduction was insensitive to 3-(3,4-dichlorophenyl)-1,1-dimethylurea but was inhibited by antimycin A. These findings are discussed in relation to a function for these electron carriers in the cyclic electron transport pathway in chloroplasts and to their function in the non-cyclic electron transport pathway.

Introduction

Chloroplasts are known to contain two electron transport pathways: (i) a non-cyclic pathway that involves two photoreactions, designated Photosystem II and Photosystem I, in which electrons released from water are transferred to NADP with a concomitant synthesis of ATP, and (ii) a cyclic route, which involves only Photosystem I, in which ATP is made but no stoichiom-

Abbreviations: DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea; Tricine, *N*-tris(hydroxymethyl)methylglycine.

etric amounts of oxidant or reductant are produced [1—3].

A widely accepted hypothesis is that, in the dark, electrons are transferred in the non-cyclic pathway from the primary electron acceptor of Photosystem II to the reaction center chlorophyll of Photosystem I (*P*-700) and that the electron carriers in this dark chain are plastoquinone, cytochrome *f*, and plastocyanin [2,3]. Other electron carriers in this dark sequence have been proposed, but their roles have not been fully characterized [4—6]. Electron carriers of the cyclic pathway have been less well defined, although it is likely that cytochrome b_6 (*b*-563) is a component [6—10]. The mechanism of cyclic electron transfer and the relationship of cyclic electron carriers to those in the non-cyclic pathway remain unresolved problems. It is now known that ATP produced via cyclic electron flow is required for CO_2 fixation in chloroplasts [11,12], and, thus, interest in this portion of the chloroplast electron chain has a new importance in studies of chloroplast bioenergetics.

In this communication we report results of studies of light-induced oxidation-reduction changes of several electron carriers that were previously assigned functional roles as electron carriers in the non-cyclic electron transport chain. We have studied these carriers under conditions of both cyclic and non-cyclic electron flow: that is, when the source of reducing equivalents was either reduced ferredoxin, as would be the case in cyclic flow, or water, as in non-cyclic flow. Evidence will be presented for a role in cyclic and non-cyclic transport for a newly identified electron carrier [4,13], designated the Rieske iron-sulfur center, as well as for plastocyanin and *P*-700. The interaction of the cyclic electron pathway with the non-cyclic pathway will be considered in relation to our findings.

Materials and Methods

Chloroplasts were isolated as previously described [14] from greenhouse-grown spinach leaves freshly picked for each series of experiments. The final resuspension was in a solution containing 0.02 M Tricine-KOH buffer (pH 8.35) and 0.01 M NaCl. Ferricyanide-washed chloroplast fragments, in which the bound electron carriers were in the oxidized state, were prepared by adding 2 mM $K_3Fe(CN)_6$ to the hypotonic breaking solution, followed by centrifugation to isolate the membranes and then washing in a standard hypotonic solution in the absence of ferricyanide.

Redox reactions of plastocyanin, the Rieske iron-sulfur center, and *P*-700 were monitored by their characteristic electron paramagnetic resonance (EPR) signals. We were interested in following the redox reactions of these carriers at physiological temperatures and used a two-step procedure because the EPR signals of oxidized plastocyanin and the reduced Rieske center are detected only at cryogenic temperatures. Chloroplast samples were mixed with appropriate reagents (see figure legends) in 13 × 100-mm test tubes at 4°C, transferred to standard 3-mm quartz EPR tubes, and illuminated in white light (intensity, 10^6 ergs $\cdot$ $cm^{-2} \cdot s^{-1}$) at room temperature. After a measured dark period, the EPR tubes were immersed in isopentane maintained at about 120 K in liquid nitrogen to rapidly freeze the samples. The samples were then stored

at 77 K prior to examination at 15 K in the EPR instrument.

Because the free-radical of $P\text{-}700^+$ is readily observed at physiological temperatures, kinetics of this electron carrier could be followed directly both during and after illumination. In these experiments, 0.05 ml samples were placed in capillary tubes (1.0 mM (inner diameter) × 100 mm) which were inserted into standard 3-mm diameter quartz EPR tubes. Samples for study of *P*-700 were illuminated directly in the EPR cavity with either red light (Corning 2-58 filter, intensity $2 \cdot 10^5$ ergs $\cdot$ cm$^{-2} \cdot$ s^{-1}) or far-red light (Baird-Atomic 740 nm interference filter, 10 nm half-band width, intensity $1 \cdot 10^4$ ergs $\cdot$ cm$^{-2} \cdot$ s^{-1}).

EPR spectra at 15 K were recorded in a modified JEOL X-band spectrometer operated at a frequency of 9.20 GHz with a modulation frequency of 100 KHz. Spectra at 25°C were recorded in a Bruker Model ER 200TT X-band spectrometer operated at a frequency of 9.18 GHz with a modulation frequency of 100 KHz. EPR conditions are given in the respective figure legends.

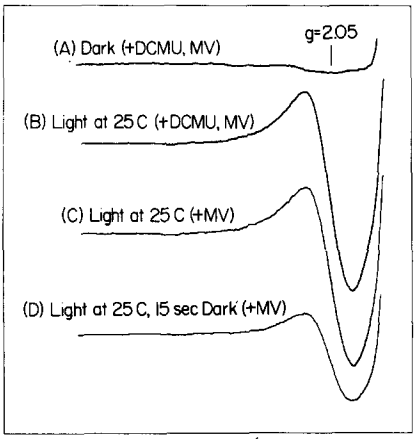
Results

Studies of electron carriers under conditions of non-cyclic electron transfer

We compared the redox response of plastocyanin and the Rieske iron-sulfur center under conditions of non-cyclic electron transfer. The redox state of these carriers is monitored most conveniently by characteristic EPR signals at cryogenic temperatures: oxidized plastocyanin shows a signal at $g = 2.05$ [15], and the reduced Rieske center shows a signal at $g = 1.89$ [13]. Both signals can be detected in the same sample in the temperature range from 15 to 30 K.

As shown in Fig. 1A, plastocyanin is fully reduced in dark-adapted chloroplasts, and illumination at 25°C in the presence of an electron acceptor (methyl viologen) and 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU) (Fig. 1B) photooxidized plastocyanin. We previously demonstrated that this photooxidation can be activated by Photosystem I illumination [15]; however, white light was used in the present experiments. When DCMU is omitted (Fig. 1C), non-cyclic electron transfer from water to methyl viologen occurs; under these conditions, the steady-state level of plastocyanin in the light is almost fully oxidized (cf. Fig. 1B and C). When illumination under these conditions is followed by a 15 s dark period prior to freezing, as shown in Fig. 1D, a reduction of oxidized plastocyanin occurs, presumably because of dark electron flow from the reduced plastoquinone pool. Under comparable conditions, a similar pattern has been observed for cytochrome *f* [16].

When we follow the redox changes of the Rieske iron-sulfur center in the same sample as that used for Fig. 1, the results shown in Fig. 2 are obtained. The Rieske center is reduced in dark-adapted chloroplasts (Fig. 2A), and it can be photooxidized in white light in the presence of DCMU and methyl viologen (Fig. 2B). Similar results are obtained in far-red light (715 nm). Under conditions of non-cyclic electron flow (Fig. 2C), the steady state is oxidized, although to a lesser extent (approx. 20%) than that of plastocyanin. After illumination, a dark reduction of this carrier can be observed (Fig. 2D) and 50% of the carrier is found in the reduced state 15 s after illumination. This finding is comparable to the extent of dark reduction of oxidized plastocyanin (cf. Fig.



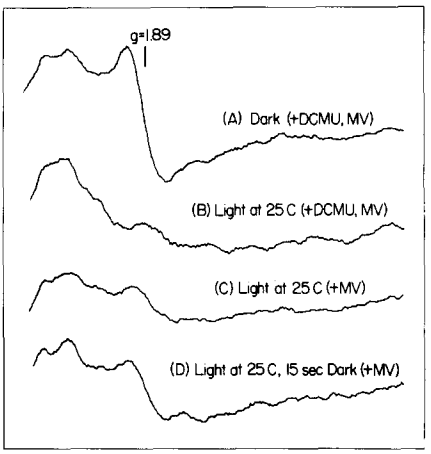


Fig. 1. Photoreactions of plastocyanin in spinach chloroplasts. The reaction mixture contained 100 mM Tricine/KOH buffer (pH 8.35), 5 mM $MgCl_2$, 1 mM ADP, 1 mM potassium phosphate, chloroplasts at a chlorophyll concentration of 3.0 mg/ml, and, where indicated, 100 μM methyl viologen (MV) and 25 μM DCMU. In samples illuminated at 25°C, the illumination time was 30 s and the sample was frozen in the light unless otherwise indicated. EPR conditions: field setting, 3200 ± 250 G; modulation amplitude 10 G; microwave power, 5 mW; temperature, 15 K.

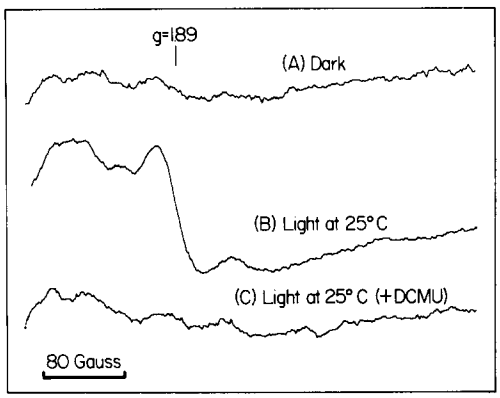
Fig. 2. Photoreactions of the Rieske iron-sulfur center in spinach chloroplasts. The same EPR sample from Fig. 1 was used to study the photoreactions of the Rieske iron-sulfur center. EPR conditions: field setting, 3500 ± 250 G; modulation amplitude, 10 G; microwave power, 10 mW; temperature, 15 K.

1C and D). In this experiment, we found an almost complete analogy under conditions of non-cyclic electron flow between the redox reactions of plastocyanin and the reactions of the Rieske iron-sulfur center.

Because the Rieske iron-sulfur center is in the reduced state in isolated chloroplast membranes, a direct study of its photoreduction was not possible. We used chloroplast fragments washed with $K_3Fe(CN)_6$, a treatment that results in the bound electron carriers being in the oxidized state in the dark, for a study of the photoreduction of this carrier. As shown in Fig. 3A, no $g = 1.89$ signal is found in these fragments in the dark because the iron-sulfur center has been oxidized. Fig. 3B shows that illumination at room temperature results in the photoreduction of the Rieske center, as evidenced by the appearance of the $g = 1.89$ signal; the addition of DCMU (Fig. 3C) totally inhibits the photoreduction of this component.

Studies of electron carriers in the cyclic electron transfer pathway

Conditions for the study of cyclic electron transfer include reduced ferredoxin as an electron donor. Under physiological conditions, reduced ferredoxin would be formed in catalytic amounts and would be reoxidized by bound components of the cyclic pathway. Ferredoxin is considered to be a cofactor for cyclic electron transfer in chloroplasts [17], and there have been several recent reports of ferredoxin-catalyzed cyclic electron flow [12,14,16,18–20]. One important feature of the ferredoxin-catalyzed cyclic pathway, used experimentally because of its discrimination relative to other pathways, is its sensitivity to the inhibitor antimycin A [12,14,21]. We used



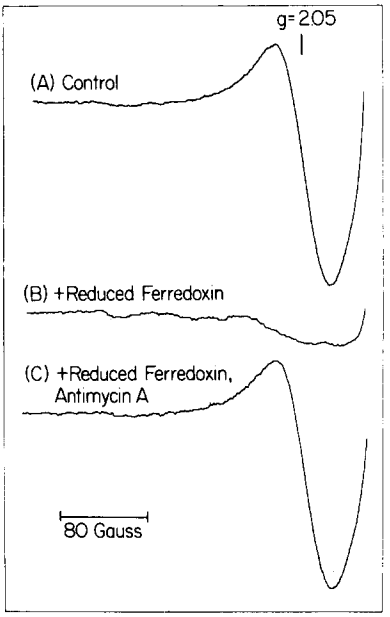


Fig. 3. Photoreduction of the Rieske iron-sulfur center in ferricyanide-washed chloroplasts. The reaction mixture contained 100 mM Tricine/KOH buffer (pH 8.35), 5 mM $MgCl_2$, ferricyanide-washed chloroplasts at a chlorophyll concentration of 2.8 mg/ml, and, where indicated, 25 μM DCMU. The sample was illuminated for 15 s at 25°C, and a 5 s dark period followed before it was frozen. EPR conditions as in Fig. 2.

Fig. 4. Dark reduction of chloroplast plastocyanin by reduced ferredoxin and the effect of antimycin A. The reaction mixture contained 100 mM Tricine/KOH buffer (pH 8.35), 5 mM $MgCl_2$, 1 mM ADP, 1 mM potassium phosphate, 25 μM DCMU, chloroplasts at a chlorophyll concentration of 3 mg/ml, and, where indicated, 25 μM spinach ferredoxin, 10 mM NADPH, and 54 μM antimycin A. The reaction sequence is given in Materials and Methods and includes a 15 s illumination period followed by a 5 s dark period. EPR conditions as in Fig. 1.

antimycin A in our examination of the redox changes of several electron carriers (plastocyanin, the Rieske iron-sulfur center, and *P*-700), with reduced ferredoxin as the electron donor. This could be done most conveniently by adding ferredoxin, in catalytic amounts, and a substrate amount of NADPH because the bound chloroplast ferredoxin-NADP reductase will catalyze the transfer of electrons from reduced pyridine nucleotide to the low-potential iron-sulfur protein.

The effects of reduced chloroplast ferredoxin and antimycin A were studied during reactions at physiological temperatures, followed by rapid freezing of the samples. The following experimental protocol was appropriate: because electron carriers of interest were in the reduced state, chloroplast samples were illuminated in the presence of DCMU and an autooxidizable electron acceptor (methyl viologen) to photooxidize the carriers. A short dark period preceded freezing of the samples, during which time electron donation by reduced ferredoxin to rereduce the carriers occurred. In control experiments, little or no reduction of the photooxidized carriers occurred if the dark period was sufficiently short (less than 10 s). Some variability in the rate of reduction of photooxidized plastocyanin and the Rieske iron-sulfur center was observed, and

it was necessary in each experiment to determine the exact conditions for optimum inhibition by antimycin A. Altered were the concentration of NADPH and of ferredoxin and the length of the dark period after illumination, to regulate electron donation into the membrane system. These studies were in the presence of DCMU to prevent electron donation by water, which is insensitive to inhibition by antimycin A [17].

In dark-adapted chloroplasts, plastocyanin is in the reduced state and no EPR signal at $g = 2.05$ is observed. After illumination of chloroplasts under the conditions described above, followed by a 5 s dark period before freezing, a large signal at $g = 2.05$ is observed (Fig. 4A), an indication that plastocyanin has undergone photooxidation in the light in a DCMU-insensitive reaction, with little rereduction in the ensuing dark period. With NADPH and ferredoxin in the reaction mixture and a light-dark regime identical to that of Fig. 4A, plastocyanin is in the reduced state after the dark period (Fig. 4B) because of electron donation from reduced ferredoxin to photooxidized plastocyanin. The possibility exists that under the reducing conditions of Fig. 4B plastocyanin was not photooxidized but remained in the reduced state. Evidence against this possibility is seen in another report (Ref. 16), in which cytochrome *f* was predominantly in the oxidized form under comparable conditions. That electron donation from reduced ferredoxin to oxidized plastocyanin does not occur by a nonspecific pathway is suggested by the results of Fig. 4C, in which is shown the effect of the inhibitor, antimycin A. In that experiment, antimycin A was added in the presence of NADPH and ferredoxin and the reduction of photooxidized plastocyanin by reduced ferredoxin was completely inhibited, i.e., the magnitude of the $g = 2.05$ signal was comparable to that observed in the absence of reduced ferredoxin (cf. Fig. 4A and C). These findings indicate that photooxidized plastocyanin can be reduced by reduced ferredoxin in a reaction that is sensitive to antimycin A.

It was possible to follow the redox state of the Rieske iron-sulfur center in the samples that were used for detection of the state of plastocyanin. The spectra in Fig. 5A indicate that this component was photooxidized by white light in the presence of DCMU and methyl viologen and that little reduction occurred in the 5 s dark period before the samples were frozen (little or no $g = 1.89$ signal). The addition of NADPH and ferredoxin (Fig. 5B) led to the appearance of a $g = 1.89$ signal from the reduced iron-sulfur center, and antimycin A inhibited this reduction by reduced ferredoxin (Fig. 5C). Thus, these results are analogous to those obtained in the experiments with plastocyanin.

Because the EPR signals from oxidized plastocyanin and the Rieske iron-sulfur center are observed only at cryogenic temperatures, it was not possible to study in detail the kinetics of electron donation by reduced ferredoxin. The reactions of *P*-700 could be monitored at physiological temperatures through observation of the $g = 2.0026$ EPR signal that originates from the oxidized component.

Electron donation by the NADPH-ferredoxin system to *P*-700 could be studied using far-red illumination to photooxidize this carrier. Light of wavelength 740 nm was used to activate the system; normally, 740 nm light would activate only Photosystem I, but, because of the high concentration of chlorophyll in our EPR experiments, we observed some activation of Photosystem II

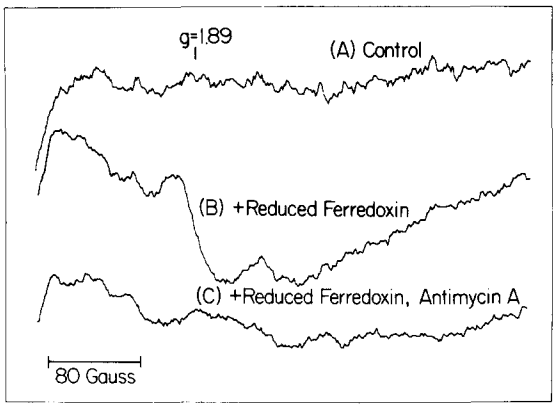


Fig. 5. Dark reduction of the chloroplast Rieske iron-sulfur center by reduced ferredoxin and effect of antimycin A. The same EPR sample that was used in Fig. 4 was used for study of the Rieske iron-sulfur center. EPR conditions as in Fig. 2.

as well. As shown in Fig. 6, under conditions of primarily cyclic electron flow, *P*-700 is predominantly oxidized in the steady state in far-red light; the addition of DCMU (Fig. 6B) leads to an enhanced oxidation because inhibition of the residual electron flow from water to $P\text{-}700^+$ occurs in the 740 nm light. A

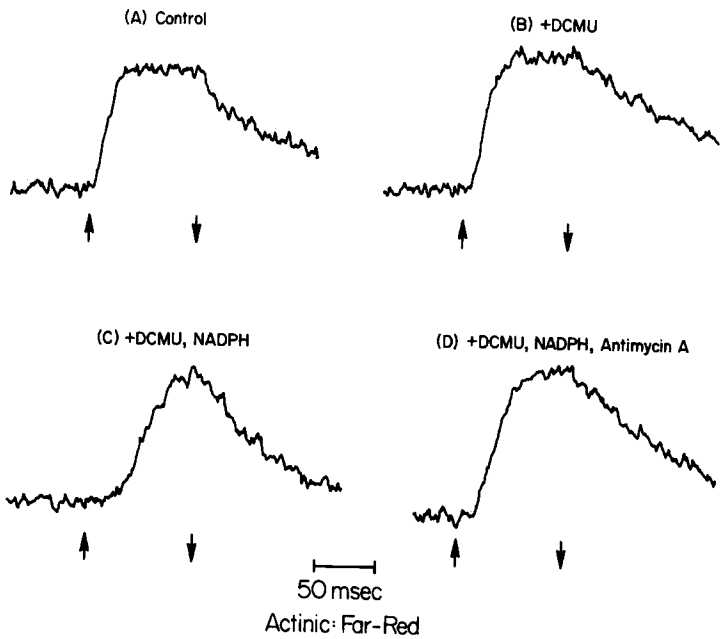


Fig. 6. Reactions of *P*-700 during illumination with far-red light under conditions of electron donation by reduced ferredoxin and effect of antimycin A. The reaction mixture contained 100 mM Tricine/KOH buffer (pH 8.35), 5 mM $MgCl_2$, 1 mM ADP, 1 mM potassium phosphate, chloroplasts at a chlorophyll concentration of 2.5 mg/ml, and, where indicated, 25 μM DCMU, 50 μM spinach ferredoxin, 10 mM NADPH, and 45 μM antimycin A. *P*-700 was monitored at the high-field peak of the first derivative EPR signal centered at $g = 2.0026$ with the following EPR instrument conditions: microwave power, 25 mW; modulation amplitude, 5 G; time-response, 10 ms; sweep time, 500 ms; temperature, 25°C. The sample was illuminated in the EPR cavity with far-red light (Baird-Atomic 740 nm interference filter).

slow reduction of $P\text{-}700^+$ occurs after the light is turned off. A markedly different pattern is observed when NADPH is added in the presence of DCMU (Fig. 6C): a lag in the photooxidation of *P*-700 was observed and the rate of dark reduction after illumination was increased over that observed in the absence of NADPH. Antimycin A also produced two effects (Fig. 6D): the lag in photooxidation is decreased, as is the rate of reduction. The most reasonable explanation for the effect of NADPH and antimycin A at this light intensity is that reduced ferredoxin, generated from NADPH, keeps *P*-700 reduced in the light and prevents the accumulation of $P\text{-}700^+$, thereby producing a detectable lag and accelerating the rereduction in the ensuing dark period. Antimycin A inhibits this electron donation and thereby decreases the lag in photooxidation and the accelerated dark decay. In general, the same conclusions could be drawn from experiments in which red light was used for activation.

Discussion

An important characteristic of electron carriers in the non-cyclic electron transport chain that connects the two light reactions in chloroplast photosynthesis is their ability to undergo both photooxidation and photoreduction. We previously demonstrated such effects on plastocyanin [16] and this antagonistic effect was observed earlier by other workers who studied cytochrome *f* [22,23].

In our present work we have shown that the Rieske iron-sulfur center can be photooxidized in a DCMU-insensitive reaction after illumination with white light; Whitmarsh and Cramer [24] have also shown that this photooxidation occurs in far-red light, which activates primarily Photosystem I. We have also been able to demonstrate a photoreduction of the oxidized center in a reaction that is sensitive to DCMU. The photoreactions of the Rieske center under steady-state illumination were found to be analogous to those of plastocyanin, an electron carrier that has been well documented as functioning between the two chloroplast light reactions. Such results would strongly support a role for the Rieske iron-sulfur center in the dark electron transport chain connecting Photosystems I and II, as has been concluded recently by Whitmarsh and Cramer [24].

It is interesting to note that, in the presence of an electron acceptor, the steady-state level of the Rieske center was more reduced than was that of plastocyanin: studies of cytochrome *f* have shown that the cytochrome is almost fully oxidized in red light in the presence of a non-cyclic electron acceptor [16,23]. These results are consistent with the site of function on the reducing side of cytochrome *f* previously proposed for the Rieske center on the basis of studies with a photosynthetic mutant [4].

We have also shown that reduced ferredoxin, generated from NADPH in the presence of ferredoxin-NADP reductase, can reduce several of the electron carriers that have been proposed to function in the dark electron transport chain connecting Photosystems I and II. The carriers characterized in our work are *P*-700, plastocyanin and the Rieske iron-sulfur center. A forthcoming paper will contain a report of similar effects on cytochrome *f* [16]. In all cases, the dark reduction of the carriers was inhibited by antimycin A, a known inhibitor

of ferredoxin-catalyzed cyclic photophosphorylation.

The electron carriers have been examined under conditions duplicating as closely as possible those used in our studies of ferredoxin-catalyzed phosphorylation. We propose that the electron carriers demonstrated in this work to undergo ferredoxin-dependent reduction are also involved in the ferredoxin-dependent phosphorylation pathway. The previously reported antimycin A sensitivity of these reactions would be consistent with this conclusion. Thus, high-potential electron carriers of the non-cyclic electron transport chain would appear to be involved in the ferredoxin-catalyzed cyclic electron transport pathway.

On the basis of studies of *C*-550 in the presence of reduced ferredoxin, it was concluded that electrons from the cyclic chain enter the non-cyclic chain in the region of plastoquinone [18]. This conclusion was based on the finding that reduction of *C*-550 with electrons from reduced ferredoxin was sensitive to DCMU and to antimycin A but not to dibromothymoquinone. The latter inhibitor was reported to act as a plastoquinone antagonist [21,25] but our recent work suggests a site of action for dibromothymoquinone nearer the Rieske iron-sulfur center [26]. In either case, dibromothymoquinone acts after the plastoquinone pool, and its effect in the above-mentioned experiments would be consistent with electron donation by reduced ferredoxin to plastoquinone. The demonstrated ability of dibromothymoquinone to reduce high-potential electron carriers in the dark precluded our study of its effect in these experiments [26]. However, earlier reports [14,18], taken in conjunction with our new findings, indicate that the ferredoxin cyclic pathway enters the non-cyclic chain on the reducing side of the Rieske iron-sulfur center which is on the reducing side of the site of inhibition by dibromothymoquinone; it then uses the high-potential electron carriers (Rieske iron-sulfur center, cytochrome *f*, plastocyanin, and *P*-700) of the non-cyclic chain.

Acknowledgements

This work was supported in part by a grant from the National Institutes of Health. We thank Dr. A.J. Bearden of Donner Laboratory for the continued use of his EPR facilities and Dr. W.A. Cramer for sending a copy of his manuscript on the Rieske iron-sulfur center.

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