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Stimulation of the chlororespiratory electron flow by Photosystem II activity in *Chlamydomonas reinhardtii*

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When dark-adapted cells of the *Chlamydomonas reinhardtii* mutant strain F15$^+$ deficient in Photosystem I are illuminated by a single saturating flash (2 μs duration), a transitory O_2 uptake process is observed. This O_2 uptake transient was recorded by using either an amperometric or a mass spectrometric technique. The light-induced signal was inhibited by DCMU, but insensitive to DBMIB, which is known to block the electron transport between the plastoquinone pool and the cytochrome (cyt) b_6f complex. A similar O_2 uptake signal was observed in a mutant strain (FuD6) deficient in the cytochrome b_6f complex. We conclude from these data that when the photosynthetic electron transport is impaired after the plastoquinones, flash illumination induces an electron transport from Photosystem II to O_2; this transfer involves the quinones of the plastoquinone pool, but not the cytochrome b_6f complex. The Photosystem II-dependent O_2 uptake transient is shown to be inhibited by both antimycin A and myxothiazol which were recently reported to inhibit chlororespiration (Ravenel and Peltier (1991) Photosynth. Res. 28, 141–148). We therefore suggest the involvement of chlororespiratory electron carriers in this process and propose that flash-induced Photosystem II activity transitorily increases the chlororespiratory electron flow. By studying the effect of DCMU on the flash-induced O_2 signal recorded in wild-type *Chlamydomonas* cells, we show that such a stimulation of chlororespiration by Photosystem II also occurs when both photosystems are active but develops slower than its inhibition by Photosystem I.

Introduction

In confirmation of the early suggestions of Goeddher [1], it has been recently demonstrated that chloroplasts of the green alga *Chlamydomonas* contain a respiratory activity [2–5]. This activity, called chlororespiration [2], in order to distinguish it from mitochondrial respiration (mitorespiration) is thought to be supported by an electron transport chain interacting with the photosynthetic electron transport chain within thylakoid membranes [2–5]. During photosynthetic reactions, Photosystem II (PS II) normally achieves the reduction of the plastoquinone pool (PQ) whereas the cyt b_6f complex and PS I are involved in its oxidation. Chlororespiration would carry out a dark reduction of the PQ pool probably catalyzed by chloroplastic activities such as the thylakoid-bound NADH-PQ oxidoreductase [6–8] or the succinate dehydrogenase [9] discovered in *Chlamydomonas* cells. Other electron carriers, such as cytochromes have been suggested to be involved in chlororespiration [5,6,10,11]. The inhibition of the dark reoxidation of the PQ pool by two cytochrome complex inhibitors, myxothiazol and antimycin A, was taken as an evidence for the participation of cytochromes to chlororespiration [5]. Following nitrogen starvation, two thylakoid-bound cytochromes have been shown to accumulate in the *Chlamydomonas* thylakoids [6]. This was accompanied by a strong increase in the activity of chlororespiration, suggesting again the involvement of cytochromes in the chlororespiratory electron chain [6]. According to these studies, the PQ pool would function as a central electron carrier, being reduced or oxidized by both photosynthetic and chlororespiratory chains [2–5]. Close interactions between photosynthetic and respiratory electron transport chains which share the same redox carriers have also been described in photosynthetic bacteria, cyanobacteria and chloroplasts of higher plants. This subject has been recently reviewed [12,13].

The effect of light on the chlororespiratory activity has been taken as evidence for an interconnection between photosynthetic and chlororespiratory electron transport chains [4,5]. For instance, using flash illumi-

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Abbreviations: cyt, cytochrome; DBMIB, 2,5-dibromomethylisopropyl-*p*-benzoquinone; DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea); PS, Photosystem; PQ, plastoquinone.

nation and a combination of amperometric and mass spectrometric techniques. Peltier et al. [4] concluded that flash-induced PS I activity transitorily inhibited the chlororespiratory activity. This was explained by a diversion towards PS I of the electron flow that would otherwise participate in chlororespiration. Because of the central location of the PQ pool, one should expect PS II activity to stimulate chlororespiratory activity. To investigate such a possibility, we have studied in this paper the O_2 transients in mutant strains of *Chlamydomonas* deficient either in PS I or in cyt b_6f. We observed that flash excitation induces a transitory O_2 uptake process in these mutants. Based on the properties of this signal such as the apparent affinity towards O_2 or the sensitivity towards different electron transport inhibitors, we conclude that the O_2 transient is due to a stimulation of chlororespiration by PS II. Moreover, we show that both a stimulation and an inhibition of chlororespiration can occur successively when wild-type algae are illuminated by a single flash. The importance and the physiological signification of chlororespiration in photosynthetic organisms is discussed.

Materials and Methods

The mutant strain F15$^+$ of *Chlamydomonas reinhardtii* deficient in PS I and the mutant strain FuD6 deficient in the cyt b_6f complex were isolated as described by Lemaire et al. [10] and were generously provided by Dr. Girard-Bascou. Both mutant strains were grown in the dark in a Tris-acetate-phosphate medium [2]. The *Chlamydomonas* wild-type strain was grown as previously described [14]. During exponential growth, cells were harvested by low speed centrifugation ($1500 \times g$) and resuspended in a 50 mM Tris buffer (pH 7.2) containing 1 mM sodium acetate and 0.1 M KCl to provide a sufficient conductivity for the amperometric measurements.

Flash-induced oxygen exchange measurements were performed using a bare platinum electrode system similar to that described by Schmid and Thibault [15]. The cells were allowed to settle on the electrode for about 20 to 30 min before measurements were made. Oxygen was flushed at the surface of the sample to maintain a sufficient oxygen concentration at the algal level. The electrode system was covered by a conic reflector in which apertures for xenon flash and optic fibers were adapted. A xenon flash (EG and G, FX 201, 2 μs duration) was used to provide flash illumination.

Unidirectional oxygen exchange were measured using $^{18}O_2$ and a magnetic sector mass-spectrometer (type 14-80, VG Instruments, UK). The algal suspension (1 ml) was introduced in a closed chamber (total volume of about 4 ml) onto a polyethylene gas permeable membrane. Gases dissolved in the algal sample

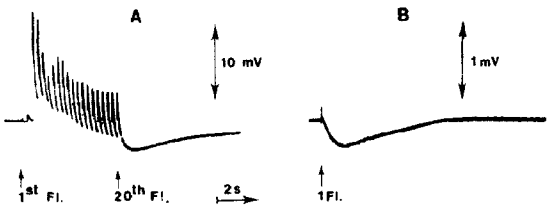

Fig. 1. Amperometric signal recorded following the flash (Fl.) illumination of *Chlamydomonas* mutant F15$^+$ lacking PS I activity. Cells were deposited on a bare O_2 platinum electrode and illuminated by flashes. (A) 20 flashes spaced by 0.5 s were fired. (B) A single flash was fired. Algae were adapted in the dark for 3 min before measurements. Note differences in intensity scales.

were allowed to diffuse from the chamber to the mass spectrometer through a vacuum line. After bubbling the suspension with N_2, $^{18}O_2$ (98.1% ^{18}O from C.E.A., Saclay, France) was injected to obtain an initial O_2 concentration of about 25% O_2. The concentrations of the isotopic species $^{16}O_2$ ($m/e = 32$) and $^{18}O_2$ ($m/e = 36$) were simultaneously and continuously recorded. As for the amperometric measurements, the cells were allowed to settle on the membrane before measurements were made. Under these conditions, the $^{18}O_2$ signal decreased in the dark at a constant rate, due to the O_2 consumption by the mass spectrometer and by the algal respiration and due also to unavoidable gas leaks. The $^{16}O_2$ signal remained at a steady low level corresponding to an equilibrium between O_2 consumption processes and leaks.

Results

When the *Chlamydomonas* F15$^+$ mutant, which is deficient in PS I, is deposited on a bare platinum electrode, adapted to the dark for 15 min and illuminated by a series of flashes, the pattern shown on Fig. 1 is observed. As is the case in wild-type algae [4], oxygen is produced by PS II after the third and the following flashes with a periodicity of 4 in the amplitude of the O_2 signals. This typical pattern was first observed by Joliot et al. [16]. It was later interpreted by Kok et al. [17] in terms of the accumulation of four positive charges necessary to photolyse two molecules of H_2O on the donor side of PS II.

Following a single flash, wild-type algae exhibit a positive oxygen signal which has been previously shown to be due to a transitory inhibition of a respiratory process [4]. This signal was characterized by a half-rise time much slower ($t_{1/2} = 350$ ms) than that for the oxygen evolution signal ($t_{1/2} = 2$ ms). Additional studies using inhibitors suggested that the flash-induced respiratory signal was the consequence of the inhibition of chlororespiration by PS I activity [4,5]. In the F15$^+$ mutant, this positive respiratory signal is clearly

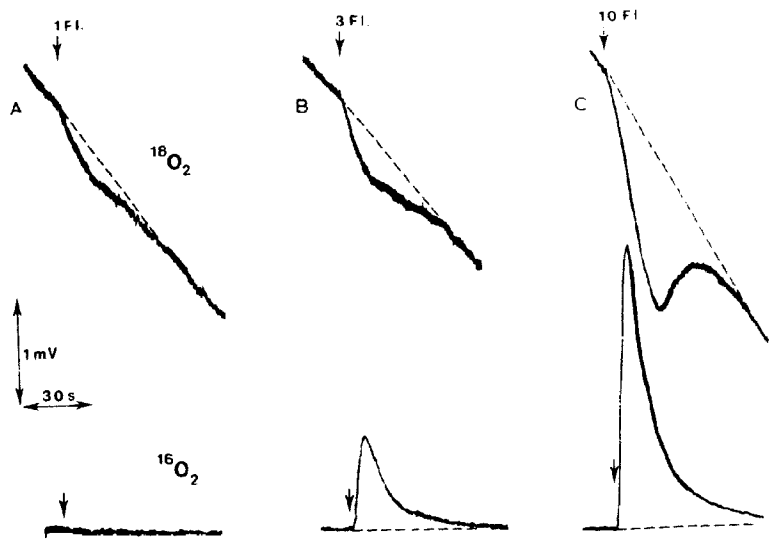

Fig. 2. Mass spectrometric measurements of $^{18}O_2$ ($m/e = 36$) and $^{16}O_2$ ($m/e = 32$) concentrations following the flash (Fl.) illumination of the *Chlamydomonas* mutant F15$^-$ after dark adaptation. (A) 1 flash. (B) 3 flashes. (C) 10 flashes. Flashes were spaced by 30 ms. Background signal values before flashes were fired: mass 32 = 330 mV; mass 36 = 5130 mV.

absent (Fig. 1A). However, by carefully studying the single flash-induced transient, we observed a negative signal indicating the existence of an oxygen uptake process (Fig. 1B). This signal was characterized by a half-rise time of 1050 ± 200 ms.

In order to confirm the existence of an oxygen uptake signal and to get rid of any possible artefactual signal of the platinum electrode, a mass spectrometry experiment was carried out. Flash-induced O_2 exchanges were monitored by mass spectrometry in experiments in which $^{16}O_2$ was replaced by $^{18}O_2$ (Fig. 2). The use of isotopes makes it possible to distinguish between O_2 uptake (by following the $^{18}O_2$ signal) and photosynthetic O_2 production originating from the photolysis of water ($H_2^{16}O$) which is specifically given by the $^{16}O_2$ signal variations. In the experiment shown in Fig. 2 the $^{18}O_2$ concentration regularly decreased in the dark mainly due to algal respiration. A single flash illumination induced a transitory acceleration of this consumption thus corroborating the previous amperometric measurements. Moreover, this experiment establishes that O_2 uptake takes place not only after the first flash, but also during the following flashes, which could not be observed in the amperometric experiment due to the photosynthetic O_2 production. The O_2 evolution signal observed with the O_2 electrode after 3 or more flashes is therefore the resultant between O_2 uptake and O_2 evolution processes. The areas of the flash-induced variations of the mass spectrometric O_2 signals have been plotted on Fig. 3 as a function of the number of flashes. Oxygen uptake progressively increased from 1 to 12 exciting flashes, whereas O_2 evolution required a minimum of three consecutive flashes to be recorded at a significant level. The parallel evolution of both $^{18}O_2$ and $^{16}O_2$ curves observed in the *Chlamydomonas* mutant F15$^-$ (Fig. 3) indicates that in the absence of PS I, all of the electrons provided by flash-induced PS II activity are used for the reduction of O_2.

In order to determine what are the electron carriers involved in the flash-induced O_2 uptake process, we studied the effect of some inhibitors of the photosynthetic electron transport (Fig. 4). The transient is

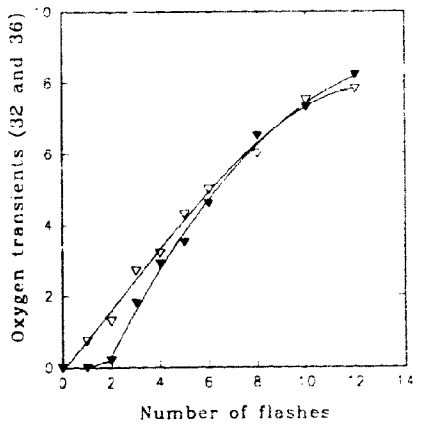

Fig. 3. Light-induced variations of the mass spectrometric signals (▼), 32 ($^{16}O_2$); (▽), 36 ($^{18}O_2$) following the flash illumination of dark-adapted cells of the *Chlamydomonas* F15$^-$ mutant. Conditions were the same as in Fig. 2.

inhibited by DCMU (Fig. 4B) showing that PS II activity and the Q_A to Q_B transfer are both necessary for the interaction between O_2 and the electron transport to occur. On the other hand, the transient was insensitive to DBMIB (Fig. 4C), an inhibitor of the PQ oxidation by the cyt b_6f complex [18], thus indicating that the interaction occurred at the PQ pool level. This interpretation was confirmed by the existence of a similar flash-induced O_2 uptake transient in a mutant strain of *Chlamydomonas* (FuD6) deficient in the cyt b_6f complex (Fig. 4D). The O_2 uptake transient of the cyt b_6f-deficient mutant was shown to be sensitive to the same chemicals as the transient of the PS I-deficient mutant (data not shown). One should however notice a difference between the two signals, the half-response time of the signal observed in the FuD6 mutant ($t_{1/2} = 500 \pm 100$ ms) being significantly shorter than the half-response time observed in the $F15^+$ mutant ($t_{1/2} = 1050 \pm 200$ ms).

Accordingly, the flash-induced O_2 interaction could take place at two different levels: either directly at the PQ pool level or through a transfer towards specific electron carriers, not involved in the photosynthetic electron transport, and able to mediate electron transfer from the plastoquinones to O_2. Chlororespiration has been shown to be able to oxidize the PQ pool [2,4] and to involve some specific electron carriers able to carry out this oxidation [5]. This part of the chlororespiratory chain was shown to be inhibited by antimycin A as well as by myxothiazol [5]. Fig. 4 shows that the O_2 uptake transient is inhibited by 5 μM antimycin A and by 5 μM myxothiazol, thus arguing in favor of a probable O_2 interaction through the chlororespiratory chain (Fig. 4E and F).

In another experiment, we compared the apparent oxygen affinity of the flash-induced O_2 uptake stimulation observed in the $F15^+$ mutant to the apparent affinity of the flash-induced O_2 uptake inhibition observed in wild-type cells and reported to be related to chlororespiration (see Ref. 4). In this experiment, the relative O_2 concentration is given by the background value of the amperometric signal. Both signals exhibited a quite close apparent affinity for O_2 (0.5 and 0.4 mV, respectively; see Fig. 5). This again argues in favor of the involvement of the chlororespiratory electron transport chain in the flash-induced O_2 uptake process.

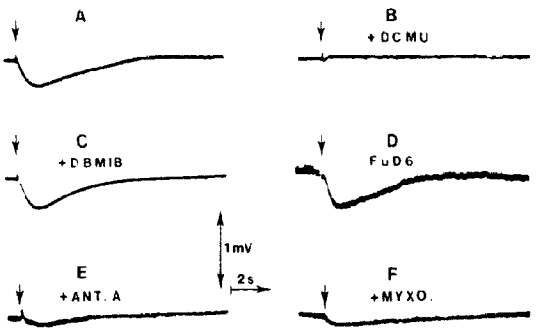

Fig. 4. Effect of different inhibitors and mutations on the amperometric signal induced by a single flash illumination in dark-adapted *Chlamydomonas* cells. (A) $F15^+$ mutant deficient in PS I; (B) $F15^+$ mutant treated by 10 μM DCMU; (C) $F15^+$ mutant treated by 10 μM DBMIB; (D) FuD6 mutant deficient in the cyt b_6f complex; (E) $F15^+$ mutant treated by 5 μM antimycin A (ANT.A); (F) $F15^+$ mutant treated by 5μM myxothiazol (MYXO.).

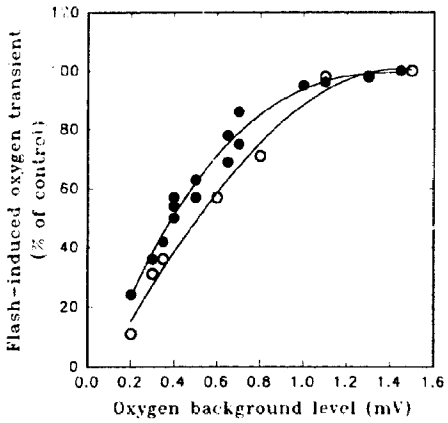

Fig. 5. Effect of the oxygen background level on the intensity of the amperometric signals induced by a single flash in dark-adapted *Chlamydomonas* cells. (●), *Chlamydomonas* wild-type. (○), $F15^+$ mutant deficient in PS I.

Flash-induced PS I activity has been previously reported to inhibit chlororespiration [4]. On the other hand, we show in the present paper that PS II activity would stimulate this electron pathway. Because in wild-type algae both PS I and PS II are active, the two effects (inhibition and stimulation) should be combined. However, flash excitation was reported to inhibit chlororespiration in wild-type algae [4]. In order to figure out whether the flash-induced transient was composed of two different components (components of inhibition and of stimulation), we studied the effect of DCMU on the transient kinetics. Fig. 6 shows that the flash-induced inhibition signals recorded before and after the addition of DCMU are different. The difference between the two curves, which reveals the contribution of PS II to the whole transient has been plotted on Fig. 6. It shows that PS II induces a stimulation of O_2 uptake which seems similar ($t_{1/2}$ of approx. 1.5 s) to that observed in the PS I-deficient mutant.

Discussion

Amperometric and mass spectrometric data presented in this paper demonstrate that in a *Chlamydomonas* strain deficient in PS I, the flash-induced

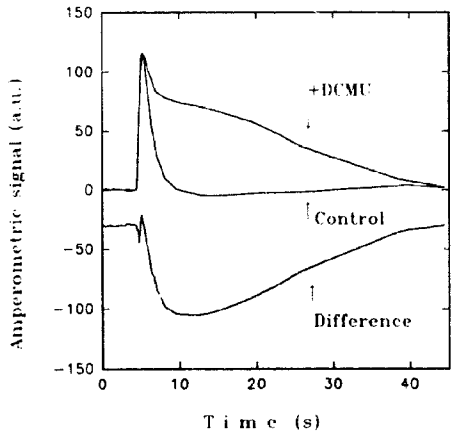

Fig. 6. Effect of DCMU (10 μM) on the flash-induced O_2 transient kinetics in *Chlamydomonas* wild-type algae.

activity of PS II provokes a transitory oxygen consumption. The inhibition of this signal by DCMU and its insensitivity to DBMIB indicate that the plastoquinones of the PQ pool, but not the cyt b_6f are involved in this electron pathway. This is confirmed by the existence of a similar flash-induced O_2 uptake transient in a mutant of *Chlamydomonas* deficient in the cyt b_6f complex. The flash-induced O_2 uptake was inhibited by the two inhibitors of cytochrome complexes antimycin A and myxothiazol, which were recently shown to inhibit chlororespiration [5]. From the above experiments, we suggest that the flash-induced O_2 uptake transient is due to a stimulation of chlororespiration by PS II activity.

A functional sheme of the interactions between the chlororespiratory chain, PS II and PS I activities is proposed on Fig. 7. As suggested in previous studies [5,6], a cytochrome complex sensitive to myxothiazol

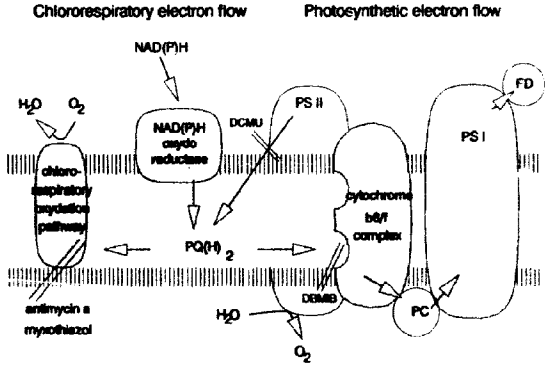

Fig. 7. Hypothetical scheme showing the interactions between photosynthetic and chlororespiratory electron flow in *Chlamydomonas* cells.

and antimycin A would oxidize the PQ pool in the dark. Flash-induced PS I activity, by oxidizing the PQ pool, can inhibit chlororespiration [4]. On the other hand, PS II activity can reduce the PQ pool and therefore stimulate the chlororespiratory electron pathway. Because in wild-type algae both PS I and PS II are active, the two effects (inhibition and stimulation) should be combined. However, only a flash-induced inhibition of chlororespiration has been previously reported in wild-type algae [4]. This can be explained by two different reasons. First, the difference in the half-response times of the two processes: the $t_{1/2}$ of the inhibition is of 350 ms (see Ref. 4) whereas the $t_{1/2}$ of the stimulation is 1050 ms. Therefore, when both photosystems are active, the inhibition is observed before the stimulation. However, the flash-induced inhibition could be observed when acetate was added to the culture medium [4]. Under such conditions respiratory metabolism is presumably enhanced and the PQ pool probably more reduced in the dark. Indeed, we observed that addition of acetate to algae increased the initial fluorescence level (F_0) recorded during a transient, thus indicating a higher redox state of both Q_A and the PQ pool (data not shown). Under these conditions, chlororespiration is presumably operating at a higher rate [4], favoring its inhibition by PS I rather than its stimulation by PS II. In agreement with such an interpretation, we observed that both the shape and the kinetics properties of the O_2 transient were modified depending on the acetate supply of algae [13].

The shape of the flash-induced transient observed in wild-type algae treated with DCMU appears to result from the superposition of two different processes (Fig. 6). However, according to our interpretation, this transient would result only from the inhibition of chlororespiration by PS I. Such kinetics of inhibition could be explained in two different ways. First, one can imagine the existence of two inhibitory processes characterized by different time constants. For example, in the case of photosynthetic bacteria, two processes have been suggested to explain the light-induced inhibition of respiration: a light-induced diversion of electron from the respiratory chain to the photooxidized reaction center and a control of respiration by the photoinduced proton electrochemical gradient [19]. Another explanation would be to consider that the chlororespiratory electron transport can occur at different rates depending on the state of association between the electron carriers within the thylakoids membrane. According to this view, it has been recently shown that in *Chlamydomonas* a change in the lateral distribution of the cyt b_6f complex along the thylakoid membranes occurs during state transitions [20]. Experiments are presently carried out to explain the kinetics of the flash-induced inhibition of chlororespiration observed in DCMU-treated algae.

The response time of the flash-induced stimulation of chlororespiration observed in the F15$^+$ mutant ($t_{1/2}$ = 1050 ± 200 ms) was significantly longer than the one observed with the FuD6 mutant ($t_{1/2}$ = 500 ± 100 ms). Besides this difference, the two signals exhibited the same properties towards inhibitors (DCMU, antimycin A, myxothiazol; data not shown). This faster inhibition recorded in the cyt b_6f deficient mutant cannot however be considered as the consequence of a non-functional electron transport between the PQ pool and PS I since DBMIB had no effect on the transient kinetics in the F15$^+$ mutant. We therefore assume that in the absence of cyt b_6f complexes, the thylakoid membrane might be organized in a different manner which would cause a difference in the kinetics properties of the electron transport.

By using $^{18}O_2$ and mass spectrometry, Peltier and Thibault [21] reported that *Chlamydomonas* mutants impaired in the photosynthetic electron transport after the PQ pool, when illuminated by continuous light, exhibit a photo-dependent O_2 uptake associated with an O_2 evolution by PS II at a rate ranging from about 150 to 230 nmol O_2/min per mg Chl. It was concluded that a part of this O_2 uptake was due to a light-induced stimulation of mitochondrial respiration. The authors suggested that, as previously observed in photosynthetic bacteria [22,23], light may provide energy for a reverse electron flow which could reduce NAD or NADP [21]. NAD(P)H could in turn be transported towards the mitochondria, via metabolic shuttles, and be used as respiratory substrate [21]. The results presented above suggest that another part of this continuous electron transport from PS II to O_2 should be represented by the electron transfer occurring via the chlororespiratory pathway. It should therefore be possible, by re-examining the properties of this continuous electron flow in different light conditions and in the presence or absence of different chlororespiratory inhibitors, to estimate the maximal rate of electron which can transit through the chlororespiratory pathway. This point is presently under investigation in our laboratory.

Besides *Chlamydomonas*, chlororespiratory activity has also been detected in another unicellular alga, *Mantoniella squama*, by using pulse-modulated fluorescence measurements [24]. Although the occurrence of chlororespiration seems well-established in green algae [2–6,24], very few studies have been published concerning higher plants. By using flash-induced absorption change measurements Garab et al. [25] concluded that the same type of interaction between photosynthetic and respiratory electron transport chains that those reported in photosynthetic bacteria, cyanobacteria or green algae were occurring in higher plant thylakoids (see also Refs. 12 and 13 for a review). However, attempts to record a flash-induced inhibition of chlororespiration using either amperometric or mass spectrometric techniques in higher plant protoplasts (unpublished data) or photoautrophic cell cultures [26] have been unsuccessful. It seems therefore possible that chlororespiration may not occur in the same way in all oxygenic photosynthetic organisms.

An important question to elucidate concerning chlororespiration is to establish the physiological significance, if any, of the phenomenon. In the dark, chlororespiration has been suggested to establish a proton gradient [2] the effect of which could be to maintain chloroplastic ATPases in an active form [4]. In illuminated cells, the quantitative importance of chlororespiration is difficult to establish. Flash illumination apparently provokes a transitory inhibition, followed by a transitory stimulation of chlororespiration. In continuous low light, the activity of chlororespiration has been suggested to be inhibited by PS I activity [27]. Under conditions of continuous, intense illumination the activity of chlororespiration is difficult to estimate, but probably depends on the steady state redox level of the PQ pool. Chlororespiration might play a role in the dissipation of energy in conditions where the reducing power is generated in excess by photosystems compared to its utilisation rate. This can occur in different stress conditions, such as exposure to high light intensities or nitrogen deficiency [6]. By recycling the reducing power through an electron transport chain, chlororespiration could help to the dissipation of energy and therefore avoid possible damages to the photosynthetic apparatus. However, further experiments have to be carried out to conclude to a role of chlororespiration in the protection of photosynthetic organisms to stress conditions.

In conclusion, we have identified an O_2 uptake site between the two photosystems. According to its properties (sensitivity towards inhibitors, apparent affinity for O_2), this site is likely related to chlororespiration. The activity of chlororespiration in *Chlamydomonas* cells is regulated by light, inhibited by PS I and stimulated by PS II.

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