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Thylakoid membrane protein phosphorylation modifies the equilibrium between Photosystem II quinone electron acceptors

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Chloroplast thylakoid membrane protein phosphorylation inhibits Photosystem-II-mediated electron transfer at saturating light intensities. Measurements of room temperature chlorophyll fluorescence have been used to monitor electron transfer reactions between the primary (Q_A) and secondary (Q_B) quinone acceptors of Photosystem II. The initial transfer from Q_A^- to Q_B is not affected by the phosphorylation, although a second slow reoxidation phase is even slower after phosphorylation. After equilibrium is achieved between $Q_A^-Q_B$ and $Q_AQ_B^-$ there appears to be a higher concentration of Q_A^- in the phosphorylated membranes. This is explained by a phosphorylation-induced destabilisation of the anionic semiquinone, Q_B^-, as seen from the DCMU-stimulated fluorescence increase.

Introduction

In chloroplasts, several protein kinases have been reported which have been associated with the outer envelope [1,2], the soluble stromal fraction [3] and the thylakoid membranes [4]. At least one protein kinase, which is thylakoid membrane-bound, catalyses the ATP-dependent phosphorylation of several polypeptides in the molecular weight range of 5000–70000 [5,6] when the plastoquinone (PQ) pool is totally or partially reduced [7,8]. It is now well established that the major thylakoid phosphoproteins are the 25–29 kDa polypeptides of the light-harvesting chlorophyll *a/b* complex of photosystem II (PS II) (LHC II) (see, e.g., Ref. 9). After phosphorylation a certain subpopulation of phospho-LHC II moves from the granal regions of the thylakoid membranes to the stromal areas [10,11], where it transfers excitation energy to photosystem I (PS I) [12,13]. This is believed to be the mechanism of the so-called State 1–State 2 transitions (see, e.g., Ref. 14). Recent studies have shown that a number of the other phosphoproteins are linked with PS II [6,15]. A 43 kDa phosphoprotein has been associated with CPa-2 (the chlorophyll-containing polypeptide close to the PS II reaction centre) [15] while the 32 and 34 kDa phosphoproteins have been assumed to be the D1 (or herbicide-binding protein) and D2 proteins, respectively [15]; thought to make up the PS II reaction centre [16]. However, the exact

Abbreviations: Chl, chlorophyll; D1, herbicide-binding protein (*psbA* gene product); D2, protein associated with PS II (*psbD* gene product); DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea; DPC, diphenylcarbazide; DPIP, 2,6-dichlorophenolindophenol; F_0, initial Chl fluorescence; F_M, maximal Chl fluorescence; Hepes, *N*-2-hydroxyethylpiperazine-*N'*-2-ethanesulphonic acid; LHC, light-harvesting Chl *a/b* protein complex; pBQ, phenyl benzoquinone; PQ, plastoquinone; PS, Photosystem; Q_A, primary quinone electron acceptor of PS II; Q_B, secondary quinone electron acceptor of PS II.

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origins of these phosphorylated proteins is still not clear, as Millner et al. [6] have suggested that the 32 kDa phosphoprotein is not D1. Another phosphoprotein of approx. 8 kDa is also closely associated with PS II but its role is not yet known, although it is believed not to be cytochrome b_{559} nor a hydrophobic subunit of the ATPase [17], but it shows a certain amino-acid sequence homology with the N-terminal region of LHC II [18].

Several other effects, other than the well-documented redistribution of excitation energy, have been reported in the literature, which could be a result of the phosphorylation of the PS II-associated polypeptides. These effects include: an enhanced stability of Q_B^-, the anionic semiquinone form of the secondary electron acceptor of PS II [19], an increased ability of PS II herbicides like DCMU, atrazine and dinoseb to inhibit the Hill reaction [20], a stimulation of a hydroxylamine-sensitive cyclic flow of electrons around PS II [21], protection against photoinhibition [22], an increased negative surface charge density near to Q_A [23] and an inhibition of light-saturated PS II electron transfer [24,25]. Such observations suggest that thylakoid protein phosphorylation has a direct affect on photosynthetic electron transfer. The inability of DPC to restore the PS II inhibition and the effect of phosphorylation on the form of fluorescence induction curves in the absence of DCMU [25] strongly suggest that the site of inhibition is on the acceptor side of PS II.

In this work we investigate the electron transfer steps on the acceptor side of PS II as a function of the phosphorylation state of the thylakoid membranes. This was carried out by measuring the reoxidation kinetics of Q_A^- and the relative concentration of Q_B^- after one or several saturating preflashes as well as the stability of Q_B^- after a single flash.

Materials and Methods

Chloroplasts of either pea or lettuce were isolated as described in Ref. 25 and resuspended as a concentrated stock in 0.33 M sorbitol, 5 mM $MgCl_2$ and 20 mM Hepes (pH 7.5).

Phosphorylation was carried out as follows: Chloroplasts were osmotically shocked in 10 mM $MgCl_2$ for 20 s and double-strength medium was added to give a final concentration of 133–200 μg Chl/ml, 0.33 M sorbitol, 10 mM Hepes (pH 7.8, NaOH), 5 mM $MgCl_2$, 10 mM KCl and 10 mM NaF. To generate phosphorylated samples 500 μM ATP was included in the medium and the thylakoids were incubated for either 30 min in the dark in the presence of 5 μM *Spirulina maxima* ferredoxin (Fd) and 500 μM NADPH or 20 min in the light (intensity = 10 $W \cdot m^{-2}$) at room temperature (18–22°C). Nonphosphorylated samples underwent the same treatments but in the absence of ATP. Before subsequent measurements the dark/Fd/NADPH-treated thylakoids underwent a single centrifugation for 1 min to remove the reductants, before being diluted 20–100 times in the same media as above. The single centrifugation step and the dilution were enough to allow the total oxidation of the PQ pool in the dark, as measured by chlorophyll fluorescence techniques.

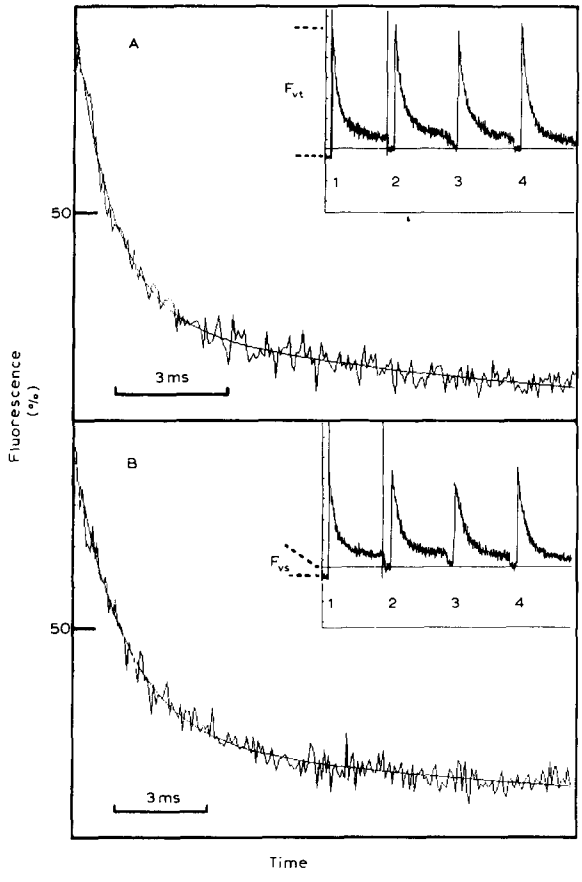
The reoxidation kinetics of Q_A^- were measured at 10 μg Chl/ml in the phosphorylation media by the chlorophyll fluorescence decay (24 accumulations) measured, after a series of saturating flashes, by a train of non-actinic detection flashes as previously described [26]. The luminescence was subtracted from the overall signal.

The stability and concentration of Q_B^- was measured using the DCMU-induced fluorescence technique [27]. The experimental apparatus and the protocol used have been described elsewhere [26]. The thylakoids were resuspended at 10 μg Chl/ml in the same media as above.

Room temperature chlorophyll fluorescence induction was measured at 2 μg Chl/ml in the presence or absence of 10 μM DCMU using the apparatus described in Ref. 28. Oxygen evolution rates were measured, using a Clarke-type O_2 electrode, at 15 μg Chl/ml in the presence of 300 μM pBQ and 2 μM gramicidin at 19°C using white light (intensity = 2000 $W \cdot m^{-1}$).

Results

Fig. 1 shows typical fluorescence decay curves, measured after one saturating flash, exhibited by nonphosphorylated (A) and phosphorylated (B) thylakoid membranes. The best fit of a two-exponential analysis is shown by the line drawn through the original signal. Such curves and



analyses were used to calculate the half-times ($t_{1/2}$) for the different Q_A^- reoxidation phases, as shown in Table I. The two component analysis gave rise to a rapid phase and a slow phase but did not take into account a very slow reoxidation component due to the fact that we chose the asymptotic fluorescence level at 500 ms after the flash (see insets of Fig. 1). Table I gives the $t_{1/2}$ values of the two components and the average $t_{1/2}$ for nonphosphorylated and phosphorylated membranes produced either in the light or in the dark with added reductants. It is interesting to note that the different protocols for phosphorylating the proteins did not give rise to significantly different results, as judged by statistical analyses using the *t*-distribution (*t*-test). The 'apparent'

Fig. 1. The reoxidation of Q_A^-, as measured by the chlorophyll fluorescence decay after a saturating flash, exhibited by (A) nonphosphorylated and (B) phosphorylated thylakoid membranes. The best fit of a two-exponential analysis is shown by the solid line through the original signal. The insets show the original fluorescence decay signals after 1, 2, 3 and 4 saturating flashes. The horizontal line indicates the asymptotic fluorescence level used in the kinetic analyses. The difference between this level and the real F_0 level gives the F_{VS} level (see Table I).

TABLE I

KINETICS OF Q_A REOXIDATION AFTER A SATURATING FLASH IN PHOSPHORYLATED (Phos) AND NONPHOSPHORYLATED (Nphos) THYLAKOIDS AS MEASURED BY CHLOROPHYLL FLUORESCENCE

System	Half-times (ms)			Amplitudes (%)		Fluorescence	
	t_1	t_2	t_{mean}	A_1	A_2	F_M/F_0	F_{VS}/F_{VT}
Dark/Fd/NADPH							
Nphos	0.301	4.640	2.123	58	42	3.17	0.19
Phos	0.356	5.370	2.462	58	42	3.08	0.23
Nphos/Phos [a]	0.846	0.864	0.862	1.0	1.0	0.06	0.82
Light							
Nphos	0.281	4.260	1.475	70	30	2.69	0.15
Phos	0.248	4.880	1.869	65	35	2.74	0.17
Nphos/Phos	1.133	0.873	0.780	1.08	0.86	0.98	0.88
Light							
Nphos [b]	0.342	6.846	2.553	66	34	2.69	0.15
Phos [b]	0.318	7.564	3.506	56	44	2.74	0.17
Nphos/Phos	1.075	0.905	0.728	1.18	0.77	0.98	0.88

[a] The ratio of Nphos/Phos parameter values.

[b] Two-exponential decay analyses carried out on transformed fluorescence data by using the calculated relationship fluorescence/Q_A^- from induction curves.

variations between the two phosphorylation treatments arise because they were never carried out on the same plant material on the same day. It can be seen that phosphorylation mainly increased the $t_{1/2}$ of the slow (ms) component (a significant difference at $\alpha < 5\%$ using the *t*-test) with no significant change in either the rapid (μs) decay (standard deviation of ± 50 μs (Nphos) and ± 86 μs (Phos)) or the amplitudes of the two phases. This led to an increase in the average $t_{1/2}$ by approx. 20% after phosphorylation, which was also observed after 2, 3 and 4 flashes (not shown). However, it is well known that fluorescence is not proportional to the concentration of Q_A^- (see Ref. 29), and therefore the $t_{1/2}$ values calculated from the fluorescence are not 'real' kinetic values for the reoxidation of Q_A^- but only comparable values if the relationship between fluorescence and concentration of Q_A^- is the same. The differences observed in Table I could therefore represent a change in the relationship between fluorescence and Q_A^- after phosphorylation due to the proposed alteration in PS II connectivity [30]. Therefore, we have carried out a transformation of the fluorescence data, obtained for light-activated phosphorylation, into a Q_A decay by using the calculated fluorescence/Q_A^- relationship measured from a fluorescence induction curve in the presence of DCMU. Such a manipulation, in fact, enhanced the differences between the phosphorylated and nonphosphorylated thylakoids (an increase in the average $t_{1/2}$ of 37% after phosphorylation instead of only 20%, see Table I) by increasing the slight change in the amplitude of the slow component (as well as making the slow phase even slower). Table I also gives various fluorescence parameters measured from the fluorescence decays similar to those shown in Fig. 1. It can be seen that the F_M/F_0 ratio was unchanged after phosphorylation (as already reported from steady-state fluorescence induction curves (e.g. Ref. 30)), although there was a quenching of fluorescence due to the phosphorylation of LHC II. However, the proportion of very slowly reoxidised Q_A^- (F_{VS}) (see inset of Fig. 1B) was increased after phosphorylation with respect to the total variable fluorescence (F_{VT}), as seen from the F_{VS}/F_{VT} ratio in Table I.

Table II gives certain room temperature chlorophyll fluorescence induction curve parameters measured in the presence and absence of DCMU, as well as the rates of oxygen evolution at saturating light intensities, exhibited by the non- and phosphorylated thylakoids used in the fluorescence experiments (Figs. 1–3). A 20% decrease in both F_M and F_0 was observed in the presence of DCMU after phosphorylation so that the F_M/F_0 ratio stayed the same (as in Table I). The $t_{1/2}$ for the increase in variable fluorescence increased after phosphorylation. These observations show that the conditions used to carry out the phosphorylation brought about LHC II phosphorylation, which led to a decrease in PS II absorption cross section. Furthermore, autoradiographs of ^{32}P-labelled thylakoids after gel electrophoresis showed that under both of our phosphorylation conditions numerous polypeptides were phosphorylated, including those associated with PS II (data not shown). In the absence of DCMU the phosphorylated membranes showed a lower F_M/F_0 ratio but

TABLE II

CHLOROPHYLL FLUORESCENCE INDUCTION CURVE PARAMETERS FROM PHOSPHORYLATED (Phos) AND NON-PHOSPHORYLATED (Nphos) THYLAKOIDS IN THE PRESENCE AND ABSENCE OF DCMU AND LIGHT-SATURATED PS II ELECTRON-TRANSFER RATES

System	+DCMU		−DCMU		O_2 evolution (μmol O_2/mg chl per h)
	F_M/F_0	$t_{1/2}$ F_V (ms)	F_M/F_0	F_I/F_{VT}	
Nphos	4.58	46	5.71	0.12	180
Phos	4.54	54	5.12	0.18	150
Nphos/Phos [a]	1.01	0.85	1.12	0.67	1.20

[a] The ratio of Nphos/Phos parameter vlaues.

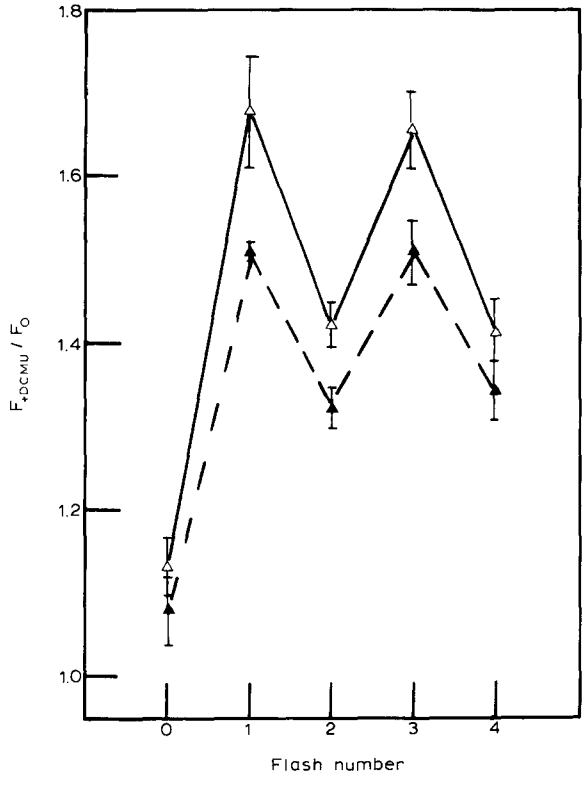


Fig. 2. The DCMU-induced increase in chlorophyll fluorescence (F_{+DCMU}), normalised to the F_0 level after no preflash and no DCMU addition, exhibited by nonphosphorylated (△) and phosphorylated (▲) thylakoids (light activated) as a function of saturating flash number. The F_{+DCMU}/F_0 ratio gives a value representative of the concentration of Q_B^- after the final flash. 50 mM hydroxylamine was added at the same time as 100 μM DCMU.

an increased proportion of the fast 'o-i' rise (F_I/F_{VT}). This increase in the rapid rise has been shown previously for the dark/reductant method of phosphorylation [25] but not for light-activated phosphorylation. However, this increase in the proportion of the fast rise could be due to the decrease in the total variable fluorescence seen after phosphorylation, which is probably due to the inability of the phosphorylated membranes to reduced fully the PQ pool at the light intensity used to carry out the measurement. A calculation carried out for a similar F_M/F_0 ratio (as for +DCMU) shows that there is still a greater proportion of the 'o-i' rise in the phosphorylated thylakoids, of which the values are very similar to the F_{VS}/F_{VT} ratio shown in Table I from the Q_A^- reoxidation experiments.

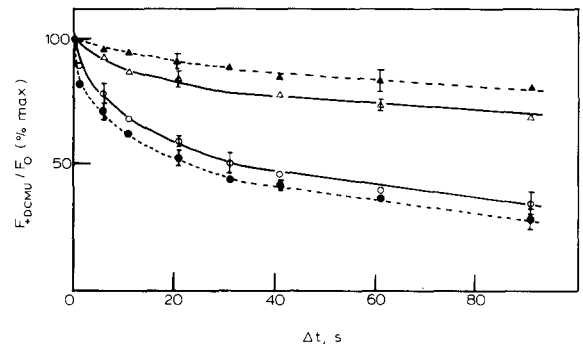


Fig. 3. The DCMU-induced increase in chlorophyll fluorescence after one flash as a function of time between the flash and the addition of DCMU (final concentration, 100 μM) in nonphosphorylated (open symbols) and phosphorylated (closed symbols) thylakoids (light-activated) in the absence (circles) and presence (triangles) of hydroxylamine. The 100% level is the F_{+DCMU}/F_0 ratio exhibited by the thylakoids in the presence of 50 mM hydroxylamine and 100 μM DCMU measured 1 s after the flash.

When DCMU is added to preilluminated thylakoids it causes a deplacement to the right of the equilibrium between $Q_B^-Q_A \rightleftharpoons Q_A^-Q_B$ + DCMU → Q_A^-DCMU which leads to an increase in chlorophyll fluorescence if the reoxidation of Q_A^- by the S-states is slower than its reduction. The DCMU-induced increase in fluorescence is therefore related to the initial concentration of Q_B^- after a given number of flashes. Fig. 2 shows the relative increase in fluorescence by the addition of DCMU, normalised to F_0 (F_{+DCMU}/F_0), to non- and phosphorylated thylakoids (generated by light) after 1–4 flashes. It can be seen that after phosphorylation there is always less Q_B^- present approx. 500 ms after the last flash. The binary oscillation of the DCMU-induced fluorescence rise indicates the occurrence of the two-electron gate mechanism of electron transfer [31] and the stability of Q_B^-. The extent of the oscillations are not affected by the phosphorylation as there is always approx. 22% less DCMU-stimulated fluorescence after 1, 2, 3 or 4 flashes in the phosphorylated thylakoids.

The lifetime of the reduced secondary acceptor can be studied by varying the time between the saturating preflash and the addition of DCMU. Fig. 3 shows the DCMU-induced fluorescence increase after one flash as a function of dark time between the flash and DCMU addition (Δt) for

TABLE III

STABILITY OF Q_B^- IN PHOSPHORYLATED (Phos) AND NONPHOSPHORYLATED (Nphos) THYLAKOIDS

System	F_{+DCMU}/F_0 ($+NH_2OH$)	$t_{1/2}$ (s)	% rapid phase ($t_{1/2} < 5$ s)
Nphos	2.34	32	35
Phos	2.07	25	45
Nphos/Phos [a]	1.13	1.28	0.78

[a] The ratio of Nphos/Phos parameter values.

non- and phosphorylated membranes (light-activated) carried out in the absence and presence of hydroxylamine (to inhibit the reoxidation by the electron-accumulating states of the oxygen-evolving complex, S_2 and S_3). In the absence of hydroxylamine the stimulation of fluorescence by DCMU addition decreased with an average $t_{1/2}$ of 25 s in the phosphorylated material, whereas in the nonphosphorylated samples the average $t_{1/2}$ was 35 s (see Table III). It must be noted that 100% was taken to be the F_{+DCMU}/F_0 ratio measured 1 s after the flash in the presence of hydroxylamine (Table III). A two-exponential component analysis of the log of the decays shown in Fig. 3 showed that phosphorylation did not significantly alter the $t_{1/2}$ values of the two phases but increased the proportion of a rapid ($t_{1/2} \leqslant 5$ s) decay (Table III). In the presence of hydroxylamine the phosphorylated membranes appeared to give rise to a more stable DCMU-induced fluorescence signal.

Discussion

It has been seen that PS II electron transfer, under saturating light conditions, to DPIP [24,25], diaminodurene [24] and pBQ (this work, Table II) is inhibited by protein phosphorylation. This inhibition is not due to the phosphorylation of the LHC II [32]. Such an effect could be caused by a number of different phosphorylation-induced changes, such as the inability to carry out a stable charge separation, a change in the equilibrium between Q_A and Q_B electron transfer or an altered equilbrium between D1 and PQ at the Q_B site. The changes in equilbria could be caused by a slower electron transfer between Q_A and Q_B (as suggested in Ref. 25), an altered binding of the PQ to the Q_B site or protonation of the reduced semiquinone, Q_B^-, or a cycling of electrons around PS II (as proposed in Ref. 21).

The reoxidation kinetics of Q_A^- has been measured from the decay of chlorophyll fluorescence after a flash. It has been previously reported that the $t_{1/2}$ for the reoxidation of Q_A^- when the Q_B site is occupied by a PQ is between 100 and 300 μs (see, e.g., Ref. 33) whereas a slower phase reflects the apparent affinity constant of PQ binding to the Q_B site. After less than 1 s the equilibrium $Q_A^-Q_B \underset{k_{-1}}{\overset{k_1}{\rightleftarrows}} Q_AQ_B^-$ is achieved [34] and the level of fluorescence reflects the concentration of Q_A^-. Protein phosphorylation does not appear to slow down the initial reoxidation of Q_A^- by Q_B (Table I) but it might alter the equilibrium between the PQ and the Q_B site, as seen from the increase in the slow $t_{1/2}$. The increase in the fluorescence level after equilibrium has been set up (F_{VS}/F_{VT}, Table I) shows that there is a higher concentration of Q_A^- after phosphorylation. The changes in the two kinetic components and the higher fluorescence level after phosphorylation are very similar to those seen between herbicide-sensitive and -resistant plants [35], in which an altered equilibrium has been proposed. These observations suggest that the stability of Q_B^- could be affected by protein phosphorylation, which alters the equilibrium constant between $Q_A^-Q_B$ and $Q_AQ_B^-$ by a decrease in k_{-1} ($Q_B^-Q_A \rightarrow Q_A^-Q_B$).

The instability of Q_B^- due to phosphorylation can be seen from Fig. 2, in which the DCMU-induced fluorescence increase after a given number of saturating flashes is always lower in the phosphorylated thylakoids. This could reflect an increase in the 'non-B' type of PS II centres (see Ref. 36) or a lower binding affinity for the DCMU (contrary to that reported in Ref. 20). The difference in relative concentration of Q_B^- between phosphorylated and non-hosphorylated thylakoids is always approx. 22%, regardless of flash number. Such a change can be explained, like the altered Q_A^- reoxidation kinetics, by an increased instability of Q_B^- after phosphorylation. This is a conclusion which is the opposite to that of Jursinic and Kyle [19], who interpreted their results by an increased stability of Q_B^- after phosphorylation

due to observing a higher DCMU-induced fluorescence increase in phosphorylated membranes along with a complete loss of binary oscillations. Such observations are clearly not seen in Fig. 2 and might be explained by the poor control used in Ref. 19, in which the non-phosphorylated thylakoids were not given a light treatment.

The increase in the F_I/F_{VT} ratio and the lower F_M/F_0 ratio (Table II) after phosphorylation can also be explained by an altered electron transfer equilibria on the acceptor side of PS II. Again, such changes, although larger, have been reported for herbicide-resistant plants [37].

In the absence of hydroxylamine, Q_B^- (and Q_A^-) are less stable in the phosphorylated thylakoids than in the nonphosphorylated membranes, as seen from the DCMU-induced fluorescence rise data in Fig. 3. This change can also be easily explained by a phosphorylation-induced alteration in $Q_A^-Q_B \rightleftharpoons Q_AQ_B^-$ equilbrium. As both Q_A^- and Q_B^- recombine with the S_2 state of the oxygen-evolving complex ($Q_A^-S_2 \rightarrow Q_AS_1$; $t_{1/2} = 1$–5 s [34,38], $Q_B^-S_2 \rightarrow Q_bS_1$; $t_{1/2} = 25$–40 s [34,39]), and the fluorescence level depends on both the concentration of Q_A^- and Q_B^-, the faster decrease in the DCMU-induced fluorescence after phosphorylation reflects the increased proportion of the rapid reoxidation of Q_A^- by S_2. This can be seen from Table III, in which the fast phase ($t_{1/2} \leqslant 5$ s) calculated from Fig. 3 increases in the phosphorylated membranes. In the presence of hydroxylamine the phosphorylated thylakoids give rise to a more stable DCMU-induced fluorescence rise, which can be interpreted as being due to the preferential reoxidation of Q_B^- by oxygen with respect to Q_A^-.

However, we cannot say if the altered equilibrium arises from the instability of Q_B^-, due to an affect of the increased negative surface charge density (see Ref. 23), and/or from an altered PQ affinity for the Q_B site, leading to less bound PQ at any given time. The significance of such an inhibition is also not known. It could be that it protects the thylakoid from accumulating Q_B^- when there is an imbalance in excitation energy distribution between the two photosystems, which could lead to photoinhibition (a phosphorylation-induced protection from photoinhibition has already been reported [22]). It has been proposed recently that protein phosphorylation might be involved in signal transmission and/or an identification process which occur prior to the adjustment of protein stoichiometry in the thylakoid membrane [40] and could therefore mark the proteins for degradation [41].

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